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Review

Effects of strength training on muscle fiber types and size; consequences for athletes training for high-intensity sport

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Training toward improving performance in sports involving high intense exercise can and is done in many different ways based on a mixture of tradition in the specific sport, coaches' experience and scientific recommendations. Strength training is a form of training that now-a-days have found its way into almost all sports in which high intense work is conducted. In this review we will focus on a few selected aspects and consequences of strength training; namely what effects do strength training have of muscle fiber type composition, and how may these effects change the contractile properties of the muscle and finally how will this affect the performance of the athlete. In addition, the review will deal with muscle hypertrophy and how it develops with strength training. Overall, it is not the purpose of this review to give a comprehensive up-date of the area, but to pin-point a few issues from which functional training advises can be made. Thus, more than a review in the traditional context this review should be viewed upon as an attempt to bring sports-physiologists and coaches or others working directly with the athletes together for a mutual discussion on how recently acquired physiological knowledge are put into practise.

When watching athletes in action, it is obvious even for the untrained eye that some athletes are ''faster'' or more ''explosive'' than others. Likewise, it is evident that some athletes manage to perform certain movements quicker than others. No doubt much of this can be attributed to superior technical skills achieved through many hours of practice, but any coach will tell you that ''fast'' and ''explosive'' are qualities the athlete had already before he or she was molded through endless training sessions; he/she had ''talent.'' Thus, both coaches and scientists know that it is not possible to turn a donkey into a racehorse by means of exercise and training. Hard work will, at the most, turn the donkey into a fast and explosive donkey! With this in mind, a number of fundamental questions can be asked. What and how much can we improve through training, and what are the factors that matter? These questions are unfortunately extremely complex and difficult to answer. Nevertheless, a number of crucial physical parameters can be identified.

We know that the ability of a muscle to conduct a fast and forceful contraction contribute positively to performance in certain athletic advents. Within muscle physiology it has been know for many years that the maximum speed at which a muscle can contract is to a high extent explained by the its composition of fast and slow muscle fibers (Harridge et al., 1996; Bottinelli & Reggiani, 2000). Likewise, the maximum force and power produced by the single muscle fiber is strongly positively related to its content of fast myosin (Bottinelli et al., 1999), which can also be observed during *in vivo* muscle contraction in the intact human (Aagaard & Andersen, 1998). The purpose of this review is to look at what happens with human skeletal fiber type composition and fiber size when exposed to strength training, and how these changes might affect athletic performance. It should be emphasize that the aim of this paper is not to give an extensive review of the literature within the area, but to pin-point a few selected aspects and issues that are of relevance in the exercise planning for elite athletes.

Defining the terms ''strength training'' or ''resistance training'' may be a little more difficult than it seems at first glimpse. A number of variables such as; type of exercise, order of exercises, load or intensity, total volume of exercises and rest are obvious parameters that can be regulated in a training regimen (Fleck & Kraemer, 2004). On top of this we can add other variables such as; speed of contraction, the choice between exercising in machines or with free weights and overall periodization principals (Fry, 2004). Thus, there is no doubt that the end-result will be influenced by how these variables are combined (Fry, 2004). For the purpose of this review

we will define strength training as; "Training that in a *efficient* manner induces a measurable increase in muscle strength or/and hypertrophy." Thus, this review will focus on training that typically engage relatively heavy loads (e.g. 70–100% of 1 RM), performed in series of relative few repetitions (e.g. ≤ 12), as this loading modality appears to be highly efficient of producing muscle hypertrophy (Fry, 2004).

Skeletal muscle fibers contain a large number of different proteins facilitating contraction; some are purely structural, with the sole purpose of maintaining the physical structure of the fiber as force is produced, whereas others have their main function in the actual contractile process (Schiaffino & Reggiani, 1996). Although several contractile proteins play important roles when a muscle fiber contracts, the two main players are myosin (the thick filament) and actin (the thin filament). When a contraction is initiated the two proteins couple, change conformation, one slides past the other as they move in opposite directions, uncouple, reload while preparing for coupling with the next actin/myosin that passes by, constantly repeating the cycle. In the human skeletal muscle actin exists in a singular form only (Schiaffino & Reggiani, 1994). Myosin (or to be more exact the heavy chain of the myosin molecule; MyHC), on the other hand, exists in three different forms (know as isoforms; essentially different versions of the same protein taking care of the same task) in human skeletal muscle (Schiaffino & Reggiani, 1994). Each of these MyHC isoforms do, when present in a muscle fiber endow the fiber with specific functional characteristics, the most important being the velocity of contraction. A number of other proteins contribute to or modulate the outcome but the absolute governing element in the equation is the MyHC isoform present. Thus, muscle fibers can be readily separated into different fiber types with specific contraction characteristics via identification of the MyHC isoform(s) present in the individual fibers. Obviously, other criteria for fiber type differentiation can be set up, e.g. metabolic characteristics (Essén et al., 1975), however these are beyond the scope of this review. The three different MyHC isoforms should in principle leave us with three different major muscle fiber types. In human skeletal muscle, however, one often find that two MHC isoforms are present alongside each other in the same fiber, which depending on the degree of details could expand the number of different fiber types from three to five or even into a continuum of slow-to-fast fiber types. The three MyHC isoforms present are; MyHC I, MyHC IIA and MyHC IIX [in older literature often refereed to as "IIB," (Smerdu et al., 1994)] (Schiaffino & Reggiani, 1996). Fibers containing only MyHC I, MyHC IIA and MyHC IIX constitute the "pure" fiber types, but also "hybrid fibers" co-expressing MyHC I and MyHC IIA as well as MyHC IIA and MyHC IIX are commonly found (Andersen et al., 1994).

It is possible to determine the maximum contraction velocity of single human skeletal muscle fibers through relative simple but time-consuming experiments. When doing that a clear pattern emerges; fibers containing MyHC I are the slowest and fibers containing MyHC IIX are the fastest, and a relative solid rule of thumb says that the order of contraction velocity for the different fiber types is, MyHC I < MyHC I/IIA hybrids < MyHC IIA < MyHC IIA/IIX hybrid < MyHC IIX (Harridge et al., 1996; Bottinelli, 2001). The difference in maximum shortening speed, when determined in single fibers between fibers containing only one of the three MyHC isoforms (MyHC I:MyHC IIA:MyHC IIX) is in the order of magnitude of 1:3:8 or 1:4:10, where co-expression hybrid fibers are placed nicely in-between fibers containing only one MyHC isoform (Fitts & Widrick, 1996; Harridge, 2007). These data are results of experiments conducted at relatively low temperature (15–18 °C). While this is substantially below the temperature in the intact muscle, recent data conducted at 35 °C indicate that the fiber type difference at more physiological relevant temperature is much less and in the magnitude of 1:2 between MyHC I and MyHC II fibers (Lionikas et al., 2006).

The next question that arrives is if this difference in shortening velocity between "slow" and "fast" fibers can be observed in the intact muscle. The question asked could be; is there a correlation between fiber type composition of a muscle and the velocity properties of the intact muscle? A number of studies have exploited this question, and strong relationship have been demonstrated both in different muscles with different fiber type composition in the same individual (Harridge, 1996; Harridge et al., 1996) and in the same muscle between different individuals with different fiber type composition (Tihanyi et al., 1982; Yates & Kamon, 1983; Aagaard & Andersen, 1998). The relationship between fiber type composition and muscle contractile velocity does not emerge at slow contraction velocities, because slow fibers in this case have ample time to build up force to more or less to the same level as the fast fibers (Aagaard & Andersen, 1998). Consequently, the close relationship between maximal concentric muscle strength and the percentage of MyHC II in intact human skeletal muscle first becomes readily apparent at high contraction velocities (Aagaard & Andersen, 1998). Translated to functional terms this mean that a person with a relative large proportion of fast fibers will be able to achieve higher muscle force and power output during fast movements including the early acceleration phase than a person with a low relative

proportion of fast fibers. Likewise, muscles characterized by a large proportion of fast muscle fibers (high relative MyHC II content) are substantially more "explosive" [i.e. demonstrating a greater rate of force development (RFD)] than muscles with fewer fast fibers (low relative MyHC II content), as reflected by an elevated contractile RFD (Harridge et al., 1996), hence demonstrating an enhanced capacity for rapid force production.

Thus, as it is established that that a person with high relative amount of fast fibers, all other things equal, will be more suited for sports in which fast, explosive-type movements performed over shorter periods of time is crucial, another question raises; "Can we change the fiber type composition of our muscles through training?" The short (disappointing) answer is; "Not really" (Andersen et al., 2000). The long answer has some uplifting nuances. Animal studies have shown that exposing a muscle with predominantly fast muscles fibers to huge amounts of low-frequency electrical stimulation, similar to what is received by slow muscles fibers, over time will gradually change the MyHC composition from fast to slow. Likewise a complete removal of the nerve impulse to a slow muscle, e.g. by cutting the motor nerve, will over time induce a switch from slow to fast MyHC (Pette & Staron, 2000). Similar findings were demonstrated some 50 years ago in animal studies in which fast and slow motor nerves were switched between a fast and slow muscle leading to a switch in contraction velocity characteristics between the two muscles (Buller et al., 1960). Later it has been shown that these shifts were the consequence of a change in MyHC isoforms expression from fast to slow and vice versa in the muscles (Pette, 2001).

Likewise, in humans a number of critical conditions can introduce large changes in MyHC compositions in skeletal muscle, e.g. after a spinal cord injury leading to paralysis. This condition will after a while leads to an almost complete abolishment of the slow MyHC isoforms in the affected muscles, leaving the muscle to exclusively express the two fast MyHC isoforms (Andersen et al., 1996). Thus, these experiment and observations tells us that a more or less complete switch between expression of fast and slow MyHC isoforms is possible in most skeletal muscles. Nevertheless, the above described scenario of a complete change in expression from slow to fast MyHC after a spinal cord injury and other similar situations are highly un-physiological, and not within the frame of physical training.

What are the limits of fiber type changes that we can introduce with physical training, and in our case strength training? Numerous studies have shown that heavy resistance exercise training will decrease the expression of MyHC IIX in human skeletal muscle and simultaneously increase the expression of MyHC IIA, whereas the expression of MHC I is much more unaffected by the resistance exercise (Hather et al., 1991; Adams et al., 1993; Andersen & Aagaard, 2000). This is a highly solid observation and a general consensus on this point exists among people working in the field (Fry, 2004; Folland & Williams, 2007). Likewise, cessation of resistance training will induce, or re-induce MyHC IIX at the expense of MyHC IIA (Andersen & Aagaard, 2000; Andersen et al., 2005). Whether or not the number of fibers expressing MyHC I is increased or decreased after strength training is debateable, but most likely, there is no or only very subtle changes in the number of fibers expressing MyHC I (Andersen & Aagaard, 2000; Fry, 2004). Thus, the general rule of MyHC isoform plasticity in human skeletal muscle appears to be: introduction of or increase in the amount of resistance training lead to decrease in MyHC IIX and increase in MyHC IIA, while a withdrawal or decrease in resistance training lead to increase in MyHC IIX and decrease in MyHC IIA, leaving MyHC I relatively unaffected (Andersen & Aagaard, 2000; Fry, 2004).

From a functional point of view the disappearance of MyHC IIX with strength training may seem somewhat unfavorable since this MyHC isoform has the fastest contraction velocity and highest power production, and removal from the muscle should lead to a slowing and reduced power output of the muscle. Theoretically that is the case when looking at the individual fiber, but when looking at the capacity of the whole and intact muscle this apparent slowing is, in most athletic settings, more than out-weighted by the increase in contractile strength, power and RFD of the trained muscle (Aagaard, 2004). In consequence, maximal unloaded limb movement speed is observed to increase (Schmidtbleicher & Haralambie, 1981; Aagaard et al., 2003) or remain unaltered (Andersen et al., 2005) following 3–4 months of heavy-resistance strength training. The enhancement in muscle force, power and RFD observed following heavy-resistance strength training to a large extent is caused by the fast fibers demonstrating a twofold greater hypertrophy than the slow fibers in response to heavy-resistance strength training (Aagaard et al., 2001; Kosek et al., 2006). Moreover, a differentiated hypertrophy of the fast and slow fibers with heavy resistance training, in favor of the fast fibers will eventually give rise to not only a bigger muscle but also a muscle in which a relatively lager proportion of the cross-sectional area is being occupied by fast fibers (Andersen & Aagaard, 2000; Aagaard, 2004).

Data from our lab indicate that heavy resistance training followed by detraining can evoke a boosting in proportions of the MyHC IIX isoform. In a

strength training study involving a group of young healthy male subjects is was observed that the MyHC IIX percentage in the vastus lateralis muscle of the subjects decreased from 9% to only 2% in a 3 months training period, but somewhat more remarkable the MyHC IIX percentage subsequently increased to 17% after a additional period of 3 months of detraining (Andersen & Aagaard, 2000). The MyHC IIX level at the end of the study were significantly higher than both the level after training, but also the level before the resistance training period (Andersen & Aagaard, 2000). In a similar study, we found that the MyHC IIX boosting after detraining were accompanied by a parallel increase in RFD in the trained muscles of the subjects (Andersen et al., 2005), however detraining also resulted in a loss in muscle mass that returned to levels comparable to that observed before the training period. This apparent boosting of the MyHC IIX isoform with detraining (and potentially also by tapering) is highly interesting if the goal of a long-term training program is to increase the relative amount of MyHC IIX in the muscle of a specific athlete, typically an athlete competing in an athletic event in which no endurance type of work is necessary, and contractile speed, power and/or explosiveness (RFD) is dominantly favored (e.g. a high- or long jumper). At this point in time we do not know how the muscle will react beyond the experimental period of 3 months, but it can be expected that the level of MyHC IIX will eventually return to the original pre-training value. A least one study with a somewhat different design seems to indicate that this is a likely scenario (Staron et al., 1991).

The question remains, however, if a high relative amount of MyHC IIX in the major skeletal muscles is interesting to other than athlete's participation in very specialised compositions? The fact is that muscle fibers containing predominantly MyHC IIX are also fibers that relay on a metabolism that enables them to produce very high amounts of energy in short time (i.e. exerting very high power), but only over a very limited period of time (seconds) (Harridge, 1996; Harridge et al., 1996). Consequently, the IIX fibers need to rest to avoid exhaustion. Sufficient rest they will not get in any of the major ball sports, or other sports in which continues work over longer periods are need. Thus, fibers containing MyHC IIA might be preferable to athletes that compete in events in which a relative fast but also somewhat enduring muscle is desirable; i.e. in 400–1500 m runners, rowers, kayakers, cycling events like sprint and team pursuit etc. Training to meet these conditions is much "easier" to plan than training to provoke fibers to express exclusively MyHC IIX. However, if the intention is to produce a very fast 100 or 200 m sprinter (i.e. targeting the latter training regime) the scheme would roughly be: avoid training involving hours of continues work at a moderate aerobic level, as this type of exercise may lead to an increased number of fibers expressing MyHC I (Schaub et al., 1989) and/or fibers co-expressing MyHC I and MyHC IIA. Further, aerobic exercise may fully or partially blunt the hypertrophic muscle response from concurrent resistance training (Glowacki et al., 2004; Baar, 2006; Nader, 2006; Coffey et al., 2009). Training exercises should comprise high-intensity intermittent work along with substantial amounts of resistance exercise (strength training), the former giving rise to an improved short-term endurance of the type IIA fibers, and the latter giving rise to a preferential hypertrophy in the type II muscle fibers. The end-result will be a muscle with is optimized toward the highest possible relative amount of MyHC IIA at the expense of both MyHC I and MyHC IIX. Needless to say, this scenario favors athletes that have a relatively high amount of type II fibers to begin with. Whether or not these type II fibers contain MyHC IIA or MyHC IIX to begin with is of less importance, since the transformation MyHC IIX $\rightarrow$ MyHC IIA inherently will be introduced through training.

In many ways it seems trivial to repeat that the training-induced increase in muscle strength and muscle hypertrophy go hand in hand. This have been observed in many long-term studies conducted on human subjects, especially involving subjects with no or limited prior history of heavy load resistance exercise training (Staron et al., 1991; Adams et al., 1993; Andersen & Aagaard, 2000; Aagaard et al., 2001). An interesting aspect of muscle adaptation to strength training, that is sometimes overlooked or toned down, is the background of the individual who is exposed to the training. When planning strength training for a given athlete it is important to know and take into account the training background of the athlete: A certain amount/volume of training might introduce significant muscle hypertrophy in one athlete with no prior strength training experience, whereas another athlete having conducted large amounts of resistance training may experience regular atrophy of his/her muscles if conducting the same amount and type of resistance training that is prescribed for a more inexperienced athlete, simply because the stimulus to his/her muscles and nervous system are less intense than the muscle-CNS signaling that they normally receive. The point here is that we should bear in mind that a very hypertrophied muscle is not in "equilibrium," and will strive toward a less hypertrophied status if the stimulus to the muscle is lowered or removed.

For muscular hypertrophy to occur a number of things have to happen. After the initial stimuli, being the resistance training, several cellular and hormonal

signal pathways will be activated (Bickel et al., 2005; Bamman et al., 2007; Coffey & Hawley, 2007), descriptions of which are beyond the scope of this review. Essentially these signal-pathways govern the processes leading to hypertrophy. Two of the major processes evidentially leading to hypertrophy are (i) increase in muscle protein synthesis (Kumar et al., 2009) and (ii) myogenic satellite cell proliferation (Kadi et al., 2005). Even though hypertrophy only is manifested, or more rightly so; measurable, after 4–6 weeks of intensive resistance training from the untrained state (Seynnes et al., 2007), the processes leading to hypertrophy commence already within the first exercise session (Atherton et al., 2005). Although the two processes will be initiated directly after the training session, one will contribute much more to the increase in muscle mass than the other. The increase in protein synthesis is the immediate response of the muscle fibers to the training stimulus received, whereas the activation (proliferation) of satellite cells are trailing somewhat behind, as if the muscle fibers are "waiting" to see if this stimulus are withheld over a longer period, before the costly affair of incorporating new nuclei into the fibers are implemented (Kadi et al., 2005; Kosek et al., 2006; Seynnes et al., 2007).

The muscle mass, or CSA of the individual fibers, is maintained when protein synthesis and muscle protein degradation is in equilibrium. A disturbance in this balance will lead to either muscle hypertrophy or muscle atrophy (Tang et al., 2008; Kumar et al., 2009). Since one of the main the purposes of resistance training frequently is to increase muscle mass obviously it is unfavorable when muscle protein degradation exceeds muscle protein synthesis, as this eventually will result in muscle atrophy. As a matter of fact, muscle protein degradation is increased right after a resistance training session, and the magnitude of degradation may even be bigger than the degree of protein synthesis in the first short period after the training session (a few hours), but provided that the subjects are not in a fasting state the net protein balance (synthesis minus degradation) subsequently becomes positive during the following hours of recovery (Kumar et al., 2009), hence facilitating a hypertrophy response. Furthermore, the increase in synthesis is withheld for a longer period than the increase in protein degradation (Biolo et al., 1995). Thus, the muscle fibers are prepared and will react to resistance training by increasing the net synthesis of contractile (and cytoskeletal) proteins. This is not an inexpensive process, but on the other hand not expensive either in sense that the cellular regulatory machinery is already present and can be set into action right away.

With the onset of fiber hypertrophy the individual muscle fiber increase the myonuclear domain i.e. each nucleus has to serve a lager cytoplasm volume (Kadi et al., 2004; Petrella et al., 2008). It seems that the myonuclei are fully capable of doing this – at least until a certain limit. At some point in the hypertropic process new myonuclei have to be added for cellular hypertrophy to commence, this point in often referred to as the *myonuclear domain ceiling* (Kadi et al., 2004; Petrella et al., 2008). Although it is probably individual for different muscles, fiber types and persons this *myonuclear domain ceiling* has been suggested to arrive around a $\sim$ 25% hypertrophy of CSA of the muscle fibers (Kadi et al., 2004). At this point new myonuclei, from the pool of quiescent satellite cells, will be added to the muscle fiber to ensure that the hypertropic process can continue. Thus, the muscle seems to have two gears; a first reactive gear with an expansion limit, and a second blunt gear with fewer limitations. In the late stage of the hypertropic process the muscle fibers will drive in both gears simultaneously. The interesting part is that the proliferation for the later differentiation of the satellite cells appears to start early in the initiation phase of the resistance-training program, hence preparing the muscle fiber for the situation that may arrive in the future (Petrella et al., 2008).

The plateau in muscle size increase that an athlete often meet typically is around 25% muscle expansion in a intensive hypertropic inducing training program. This plateau or ceiling effect may be related to the individuals ability to activate his/her second "gear-shift," i.e. to activate the pool of myogenic satellite cells. Thus, in a recent study extreme responders, moderate responders and non-responders were identified according to the hypertrophic effect of a 16-week resistance training program, after which extreme responders (cellular hypertrophy of $\sim$ 50%) showed a markedly higher activation (proliferation) of their satellite cells and greater myonuclei addition compared with moderate responders ($\sim$ 25% hypertrophy) and non-responders (0% hypertrophy) (Petrella et al., 2008). Results as these give us strong hints as to why some athletes may react promptly and strongly to resistance training whereas others don't.

In summary, the MyHC composition of human skeletal muscle seems to be modulated when subjected to resistance training and subsequent detraining. Most pronounced is the significant decrease in the expression of the fastest human skeletal muscle MyHC isoform IIX, with a corresponding increase in the MyHC IIA isoform. It is speculated that the increase in the relative amount of MyHC IIA along with a documented twofold greater hypertrophy of the fast fibers, compared with the slow fibers, as well as the training-induced increase in maximal muscle strength are highly beneficial in a wide range of sports. Likewise, the apparent boosting in MyHC IIX isoform content that seems to occur with de-

training following strength training is a phenomenon that should be further examined if the intention is to create a very fast, explosive (albeit non-endurant) type of muscle. In relation to the choice of investing time and efforts in resistance training for a given athlete it is important to closely examine the athletes training background and take into account whether or not the athlete respond with extensive muscle hypertrophy or with almost no hypertrophy. Very recent data indicate that a great deal of difference may exist among different individuals in terms of this particular response, which means that the type and amount of resistance training should be modified accordingly.

Key words: high-intensity sport, fiber types, fiber size, MHC IIX boosting.

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