The Effect of Endurance Training on Muscle Strength and Power

by

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ABSTRACT

In order to investigate possible negative effects of endurance training on muscle strength and power, 10 healthy young men underwent 10 weeks of endurance training. Subjects trained unilaterally on a cycle ergometer so that their opposite leg served as a control. Training consisted of 30 min per day for 3 days per week and progressed to 60 min per day for 5 days per week by the seventh week of training. This volume of endurance training exceeds the upper limits of that normally performed by athletes who compete in power sports. The exercise intensity was initially ~75% of pre-training maximum power output and was increased over time to maintain a training heart rate of 140-160 beats per minute. Endurance, strength, and power variables were assessed in each leg before and after the training period. Measurements included electrically-stimulated twitch characteristics of the quadriceps, single-leg $\dot{V}O_{2peak}$ and lactate threshold (T_{lac}), single-leg take-off vertical jump power indices from a force platform, and maximal leg press strength at a low- (60°/s) and high-velocity (300°/s). Needle biopsies were taken from the quadriceps femoris before and after the training period, and analyzed for fibre-type proportions, fibre area, oxidative enzyme activity and capillary density. Post-training, subjects increased leg $\dot{V}O_{2peak}$ (7%) and T_{lac} in the trained leg. Leg press strength was unaffected by training. Vertical jump power was not impaired nor were evoked twitches. Following training, there was a decrease in % type IIb and an increase in % type IIa fibres ($p<0.05$). There were no significant changes in fibre area. Percent fibre area increased for type I and IIa and decreased for type IIb fibres in the trained leg ($p<0.05$). These data indicate that a 10-week endurance-training program increases aerobic power but does not impair muscle strength or power.

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CHAPTER I

REVIEW OF LITERATURE

1.1 INTRODUCTION

Depending upon the event, athletic performance is often determined by the aerobic capacity of skeletal muscle or by its strength and power characteristics. Some sports require a high degree of both aerobic fitness and muscle power; however the physiological adaptations that enhance aerobic capacity may have a negative effect on power production. The physiological responses that occur in skeletal muscle as a result of strength training and endurance training are divergent in nature (Bell et al., 2000). For example, the aerobic capacity of muscle would benefit from muscle fibres with small diameters and a large proportion of slow-twitch (type I) fibres due to a reduction in diffusion distance for oxygen and increased resistance to fatigue. In contrast, power is dependent on muscle force and velocity of shortening. Since small muscle fibre diameters are correlated with low force outputs and type I fibres are known to have a slow velocity of shortening (Schluter and Fitts, 1994), one would expect that a large proportion of type I fibres would not result in exceptional strength and power performance. Costill (1967) observed low levels of muscle power in well-trained endurance athletes and thus it seems that achieving maximal levels of both aerobic power and muscle power may be a physiological contradiction. Fast-twitch (type II) muscle fibres have larger cross-sectional areas and faster speeds of contraction than type I fibres (Larsson and Moss, 1993). As a result, type II fibres contribute most to the fast, forceful muscle contractions involved in short-term speed and power activities. A large

proportion of type II muscle fibres would therefore be desirable for events characterized by high power outputs.

The dynamic nature of muscle is reflected by its ability to adapt to altered functional demands by making qualitative changes in fibre type composition (Pette, 1998). Specific physiological adaptations occur in response to the demands imposed by training and affect contractile characteristics accordingly. Aerobic or endurance training increases the body's work capacity through changes in the cardio-vascular and skeletal muscle systems (Saltin et al., 1976). Prolonged sub-maximal endurance training is known to improve aerobic power (Andersen and Henriksson, 1977a) but would not normally be expected to increase muscle fibre area, strength, or power (Kraemer et al., 1995). The adaptations to contrasting exercise modes have important implications for training program design in sports that require high levels of both aerobic and muscular power, such as ice hockey. It is possible that the adaptations to endurance training may cause impairment in explosive strength events such as jumping, short sprinting, or throwing, due to divergent adaptations in muscle fibre type and fibre cross-sectional area. This review will document the effect of endurance training on aerobic capacity, strength and power characteristics and fibre type composition of skeletal muscle.

1.2 MUSCLE FIBRE TYPES

Human skeletal muscle fibres are generally categorized as slow-twitch (type I) or fast-twitch (type II, Brooke and Kaiser, 1970). Additionally, using histochemical classification, the fast-twitch group contains two main subtypes, the type IIa and type IIb fibres (Schluter and Fitts, 1994). Human skeletal muscle contains varying proportions of

both fibre types and is functionally dependent on its fibre composition. The 3-fibre type delineation has been based on muscle contractile properties (Larsson and Moss, 1993), metabolic enzyme activities (Essen and Borges, 1986), myosin ATPase activity (Howald, 1982), and myosin heavy chain (MHC) isoform composition (Schluter and Fitts, 1994). Each fibre type has a characteristic profile and is best suited for a particular type of activity. The fibre-type composition (as determined by MHC isoforms) is the major determinant of the diversity in contractile properties among human muscles (Harridge et al., 1996). For example, the smaller, slow-contracting, fatigue-resistant type I fibres are beneficial to endurance activities and the fast-contracting, larger type II fibres produce high levels of force for optimal performance in powerful movements.

1.2.1 Contractile Properties. The primary muscle contractile elements are actin and myosin filaments. After removal of the regulating-protein tropomyosin from binding sites on actin, myosin cross-bridge heads attach to the actin filaments. The cross-bridge attachment then pulls the actin closer to the midline of the sarcomere, shortening the muscle fibre and causing movement to occur. Myosin ATPase catalyzes the hydrolysis of ATP into ADP and a phosphate ion to allow for the cross-bridge cycle and muscle contraction (Howald, 1982). The speed at which the cross-bridge cycling occurs determines the shortening velocity of the fibre. The wide diversity in the contractile properties of different muscle fibre types is due to their myosin composition. Each fibre type contains a specific myosin isoform that contains the site of ATP hydrolysis (Wagner and Giniger, 1981). The type II fibres have faster a faster myosin ATPase than type I

fibres (Burke, 1981) and therefore will have faster velocities of shortening (Schluter and Fitts, 1994).

Power is a function of force and velocity. In humans, high power outputs may be enhanced by a large proportion of type II fibres due to their higher speeds of contraction and greater force generation (Perrine, 1986). Type II fibres can produce 5-10 times more power than type I fibres (Schluter and Fitts, 1994) and peak power is positively correlated with the percentage of type II fibres (Fitts et al., 1991). Type IIb fibres have the fastest velocity of shortening (contraction speed) and type I fibres have the slowest (Schluter and Fitts, 1994). Type IIa fibres are considered intermediate fibres because they rank between type IIb and type I fibres with respect to their velocity of shortening and oxidative capacity (Jansson and Kaijser, 1977; Peter et al., 1972), making them well-suited for prolonged high-intensity contractions. Muscle power is dependent on this velocity of shortening and also on the force production capacity of the muscle fibres. Muscle force, or strength, is defined as the maximal force generated by a muscle at a specified velocity (Knuttgen and Kraemer, 1987) and the strength of an individual muscle fibre is proportional to its cross-sectional area (Widrick et al., 1996a). Type II fibres typically have a larger cross-sectional area and can produce more absolute force than type I fibres (McDonagh et al., 1980) and consequently produce more power.

Fibre contractile properties may change if an altered functional demand is imposed on the muscle. Elite distance runners (with 20 years of training) have been shown to have striking differences in their muscle fibre morphology and contractile properties compared to age-matched sedentary controls. In the trained runners, the type I and type IIa fibres had significantly smaller cross-sectional areas and produced less force

and peak power than those of age-matched sedentary individuals (Widrick et al., 1996a; 1996b). Fitts et al. (1998) have also shown a decreased force production in type I fibres after endurance training. This impairment in force production was attributed to a reduced fibre cross-sectional area that may contribute to the compromised power characteristics observed in endurance athletes. For example, vertical jump height, considered an index of lower body power, has been shown to be significantly lower in cross-country runners than in control subjects (Costill, 1967). These results suggest that strength and power athletes should avoid a training program comprised exclusively of endurance training, because it may compromise performance.

The development of in-vitro, single-fibre preparations for studying the contractile characteristics of individual muscle fibres has advanced our knowledge of muscle force-velocity properties (Lynch and Williams, 1994). Maximum shortening velocity is commonly expressed as V_{max} (Fitts and Holloszy, 1977; Larsson and Moss, 1993; Widrick et al., 1997) or V_o (Edstrom and Grimby, 1986; Widrick et al, 1996b). V_{max} and V_o are thought to reflect the maximal rate of cross-bridge cycling and contraction time (Larsson and Moss, 1993) and likely depend on the fibre's myosin heavy chain composition. Researchers distinguish between V_{max} and V_o by the method by which each property is determined. V_{max} is extrapolated from the force-velocity curve and may vary with the accuracy of the curve (Widrick et al, 1997). V_o is determined from a single fibre "slack-test" and is considered to be a more sensitive indicator of maximal shortening velocity (Widrick et al, 1996b). Studies have found similar values for V_{max} and V_o in type I fibres (Widrick et al., 1997) but the values observed for V_{max} and V_o have not always been equal in all fibre types (Widrick et al., 1996b). The results of single-fibre

contractile studies should be interpreted with caution because within-species differences in measures of contractile speed may exist. The V_{max} of both monkeys and rats were greater than their corresponding V_o values (Widrick et al., 1997) suggesting that these two properties may not always be identical in mammals. In addition, inter-species differences in power production have been observed. The soleus in rats has been shown to produce more peak power than monkeys or humans when normalized to fibre diameter (Widrick et al., 1997). Small animals may also have an increased normalized peak power output because of increased maximal cross-bridge cycling rate, but peak fibre tension is quite similar across mammalian species (Widrick et al., 1997).

It is believed that V_{max} is determined by the rate of disassociation between the myofilaments (Fitts et al., 1991) and it has been shown to correlate with myosin ATPase activity in single muscle fibres (Edman et al., 1988). The V_{max} of type IIb and type IIa fibres is approximately 10 and 3 times faster, respectively, than that of type I fibres (Larsson and Moss, 1993). Therefore, it would be expected that a fibre expressing a higher percentage of fast myosin or a muscle with a higher percentage of type II fibres would be capable of a faster speed of contraction. A fibre's V_{max} is highly dependent on its MHC composition because different MHC isoforms have different shortening velocities (Fitts et al, 1991; Larsson and Moss, 1993) and the myosin molecule contains the site for ATP hydrolysis (Wagner and Giniger, 1981). The different myosin ATPases found in each fibre type exhibit a cause-effect relationship between MHC composition and the fibre's velocity of shortening. For example, the V_{max} of the mammalian type IIx fibre was greater than the V_{max} of the type IIa fibres (Widrick et al., 1996b), likely due to the type IIa fibre myosin contributing to a longer time for cross-bridge detachment. The

changes in a fibre's contractile characteristics reflect subtle modifications in the composition of the fibre's contractile proteins. Hybrid fibres that contain small amounts of slow myosin and large amounts of fast myosin have been shown to have a slower V_o in comparison to fibres composed of fast myosin only (Fitts et al., 1998). This observation supports the hypothesis that MHC composition is the main determinant of the velocity of shortening, although unloaded shortening velocity may be proportional to the myosin light chain composition the IIa, IIx and IIb fibres in the rat (Bottenelli et al., 1994).

The V_{max} of type I fibres increased following endurance training (Fitts and Holloszy, 1977; Fitts et al., 1991). Training may affect V_o in a manner similar to V_{max}. Elite distance runners had a greater type I fibre V_o in comparison to controls (Widrick et al., 1996a) and, after 8-12 weeks of endurance training, the V_o of rat soleus type I fibres was elevated by 23% (Schluter and Fitts, 1994). In humans, swimming increased the V_o of the type I fibres in the deltoid but decreased the V_o of the type II fibres by 45% in comparison to pre-training values (Fitts et al., 1989). The V_o of a fibre is correlated with the amount of the fast MHC isoform (Edstrom and Grimby, 1986). As noted earlier, hybrid fibres have an intermediate V_o value suggesting that even a small amount of MHC-I will decrease the V_o in comparison to a pure type II fibre (Fitts et al., 1998). The changes in the contractile properties of a fibre after training are likely due to the exercise-induced expression of fast myosin in slow fibres and slow myosin in fast fibres (Fitts et al., 1989). In summary, a fibre's velocity of shortening is primarily determined by the composition of its MHC proteins but there may also be some contribution from the myosin light chain proteins (Pette et al., 1976).

The maximum tension of a fibre (P_o) is measured as a peak tetanic isometric contraction (Widrick et al., 1996a; 1997) and is directly dependent on fibre diameter (Fitts et al., 1991). Fitts and Holloszy (1977) found that P_o decreased in rats following endurance training, indicating that training can alter contractile properties. Fast-twitch fibre cross-sectional area (CSA) is normally greater than slow-twitch CSA (Lexell et al., 1988) as is peak force (McDonagh et al., 1980). The maximum tension of a fibre is very important in determining overall muscle strength and power. Peak twitch force is influenced by the quantity of contractile protein and adaptations in the excitation-contraction coupling process (Keen et al., 1994). Fitts et al. (1998) believe that differences in peak force between muscle fibres are due entirely to differences in cross-sectional area. Therefore a larger proportion of type II fibres would be beneficial in generating a greater power production. Endurance training and long-term muscle stimulation have been suggested to impair both force (P_o) and the rate of force development. Maximum muscle tension was found to decrease in endurance swim-trained rat soleus muscle (Lynch and Williams, 1994) but not in endurance run-trained rat soleus muscle (Schluter and Fitts, 1994). Stimulation of the tibialis anterior muscle reduced maximum tension and increased time-to-peak twitch (Salmons and Sreter, 1976). Endurance training has also been shown to increase the time-to-peak tension in type I fibres (Peter et al., 1972). These results suggest that endurance training should be avoided if power production is the main training objective.

Specific tension is defined as maximum tension normalized to fibre cross-sectional area. In order to compare force-generating capabilities of the different motor unit types, it is necessary to have, in addition to absolute force output, estimates of

specific tension (McDonagh et al., 1980). This enables researchers to determine if there is a difference in the quality of force production of the contractile proteins between fibres, rather than just observing a greater force production due to an increased quantity of contractile protein (greater CSA). There is some debate as to whether type II fibres have a greater specific tension than type I fibres. Type II fibres belong to fast-fatiguing (FF) motor units and type I fibres belong to slow-fatiguing (S) motor units (McDonagh et al., 1980). Studies of motor units in cats suggest that FF motor units have a greater specific tension than S motor units (McDonagh et al., 1980) and may be due to the greater fibre number per motor unit and size of each fibre (Bodine et al., 1987). The difference between the FF motor unit (4.9kg/cm2) and the S motor unit (1.5kg/cm2) (McDonagh et al., 1980) was very large but interpretation is complicated by the fact that the force was measured at the tendon and not directly from the fibres. In contrast to the motor unit findings, direct measures from single fibre experiments have found similar specific tensions across fibre types in humans (Fitts et al., 1991). In comparison, Widrick et al. (1996a) found peak tension (calculated as the peak isometric force divided by the cross-sectional area of the muscle fibre) to differ between fibre types in sedentary but not in endurance-trained subjects. Therefore, there may not be a difference in the force-generating capacities of type I and type II fibres when expressed as specific tension and this also may be determined by the level of contractile activity. The researchers had no definite explanation for these equivocal results. Additional studies of specific tension have shown no fibre differences in humans (Donaldson et al., 1978) or in rhesus monkeys (Fitts et al., 1998). However, more recent work in humans has suggested that the specific tension depends on MHC composition because specific tension was observed to be

significantly lower in type I compared to both type IIa and IIb fibres (Bottenelli et al., 1996). Experimental techniques may also affect the outcomes of measures of specific tension, since specific tensions have been found to be lower in freeze-dried fibres, than in chemically skinned fibres (Larsson and Moss, 1993). These authors observed that, in freeze-dried fibres, the type IIb fibres had a significantly greater specific tension than type I fibres. Future studies need to determine the most accurate method for determining specific tension and methods should also attempt to sub-group the type II fibres in order to determine possible differences between the type IIb and type IIa fibres.

In addition to the myosin heavy chain isoform, there are other components that may influence contraction speed. The sarcoplasmic reticulum (SR) controls the release and re-uptake of calcium to participate in the cross-bridge cycle at the contractile apparatus. Calcium is required to bind to the regulatory protein troponin, in order for the tropomyosin to be removed and allows myosin to bind with actin. Sarcoplasmic reticulum surface area is a contributing factor to twitch contraction time in addition to cross-bridge cycling rate, external load, and fatigue. The SR of the type II fibres is much more developed in comparison to the type I fibre (Burke, 1981) suggesting a greater capacity for calcium release in muscle contractions. Alway et al. (1988) demonstrated that, in humans, the volume density of the sarcoplasmic reticulum-transverse tubule network was more than 2 times greater in type II fibres than in type I fibres. Prior to this study, all work had been done in rodents. Long-term electrical stimulation of rodent muscle decreased the calcium re-uptake ability of the SR (Mabuchi et al., 1982) and this may contribute to the development of a slower muscle fibre.

Based on the contractile properties of each specific fibre type, one would expect that type IIb fibres would contract with a higher velocity (higher V_{max}), especially when mechanically loaded (if there is a fibre difference in specific tension). Thus, the attributes of a type IIb fibre would promote maximal muscle power output and performance in strength and power events. However, a transition to a slower myosin heavy chain isoform (Andersen and Schiaffino, 1997) and a reduction in fibre cross-sectional area may occur with endurance training (Andersen and Henriksson, 1977b; Klausen et al., 1981; Saltin et al., 1976). These changes would negatively affect contractile properties such as velocity of shortening and peak tension and could impair muscle power.

1.2.2 Biochemical Properties. Each fibre type has a specific enzyme profile dictating the extent to which the different metabolic pathways are utilized during exercise at a given intensity. Essen and Borges (1986) found greater concentrations of glycolytic enzymes in type II fibres when compared to type I fibres, allowing the type II fibres to generate ATP for muscle contraction at a faster rate. Type II muscle fibres also contain more glycogen and phosphocreatine (PCr) than type I fibres to fuel anaerobic energy production, but there is no difference in the concentration of ATP (Casey et al., 1996; Esbjörnsson et al., 1999). Therefore the type II fibres have a greater glycolytic capacity and can produce energy at faster rates to optimally support performance in high power activities. In contrast, the type I fibres have a greater oxidative capacity profile. The volume of mitochondria, oxidative enzymes, and the concentration of myoglobin are rich in type I fibres and provide for an increased oxidative capacity and resistance to fatigue

(Thompson, 1994; Wang et al., 1993). The oxidative enzyme activity of type IIa fibres has been shown to be higher than that of IIb fibres (Staron et al., 1983). Oxidative enzyme activity of type IIb fibres is low and, even following training, has been shown to reach only 60% that of type I fibres (Stone et al., 1996). Therefore, the type IIb fibre is poorly suited for endurance activities, but the type IIa has an intermediate enzyme profile (Jansson and Kaijser, 1977) and would be beneficial in activities requiring high force outputs for an extended duration. Endurance training results in physiologic adaptations that are specific to the imposed demands and therefore would be expected to enhance the oxidative capacity of all fibres.

1.2.3. Motor Unit Recruitment. The motor unit (MU) is the functional connection between the nervous system and muscle, and consists of a motor neuron and all the muscle fibres that it innervates. Based on earlier histochemical studies of glycogen-depletion patterns, three MU types were identified: fast glycolytic (FG), fast oxidative glycolytic (FOG), and slow oxidative (SO) (Edstrom and Kugelberg, 1968). Another classification scheme based on evoked motor unit contractile characteristics has identified fast-fatigable (FF), fast fatigue-resistant (FR), and slow (S) motor units (Burke, 1981). The S, FR, and FF motor units are presumed to be comprised of type I, type IIa, and type IIb muscle fibres, respectively (McDonagh et al., 1980). Typically the MU classification accurately describes the motor unit's contractile characteristics, but each category is subject to considerable variation in force production and conduction velocity. The FF motor units and FR fibres produce greater amounts of force (Burke et al., 1971) due to greater fibre numbers and size per motor unit (Bodine et al., 1987) and have a fast

velocity of shortening (Burke, 1981). Slow motor units produce the smallest amounts of force, have the slowest velocity of shortening (Burke, 1981), and have axons with a slow conduction velocity (Borg et al., 1978).

The strength of a muscle contraction depends on the number of motor units recruited and their firing rate. Motor units are thought to be recruited in accordance with the "size principle of recruitment", meaning that there is a sequential recruitment of motor units based on their size (Henneman et al., 1965). As contraction force is increased, the small slow motor units are recruited first followed by larger fast motor units (Milner-Brown et al., 1973). It is generally accepted that the S motor unit is recruited first and the FF motor unit is recruited last and only at high workloads. A maximal contraction will demand complete recruitment of all motor units and a maximum firing rate for each motor neuron. Endurance activities, characterized by relatively slow speeds of contraction and low force output, do not require recruitment of all available motor units or maximum firing rate for each motor neuron. Burke (1981) suggests that a fibre's oxidative capacity is correlated to its fatigability. During low intensity exercise, slow motor units are recruited first and as those units fatigue during prolonged exercise, additional motor units are recruited. Fatigue is defined as a decrease in the force output from a motor unit below maximal or a required level. Therefore, during progressive exercise the larger motor units would be recruited to contribute to force production to maintain activity.

The work by Henneman et al. (1965) on motor unit recruitment provides the basis for the theory of selective recruitment in endurance activity. At sub-maximal intensities, slow motor units are preferentially recruited and contribute to the majority of tension

development. Gollnick et al. (1973c) have shown preferential glycogen depletion of type I fibres during sub-maximal cycling (31-84% $\dot{V}O_{2max}$) as has Costill et al. (1973), with sub-maximal distance running. Furthermore, Gollnick et al. (1973a) observed selective hypertrophy of slow twitch fibres after a 20 week endurance training program, suggesting that the slow twitch fibres are selectively recruited during moderate-intensity endurance activities. Slow twitch fibres are the first to be depleted of glycogen in sub-maximal exercise bouts (Costill et al., 1973) and fast twitch fibres are progressively recruited because slow twitch fibres become glycogen depleted (Gollnick et al., 1973c). After 60 min of exercise at 61% of $\dot{V}O_{2max}$, glycogen breakdown was observed in all type I fibres and in about 65% of type IIa fibres (Vollestad and Blom, 1985). In repeated bouts of exercise at 150% of $\dot{V}O_{2max}$, slow twitch and fast twitch fibres exhibit equal glycogen depletion (Gollnick et al., 1973b). The results indicate a primary reliance on slow twitch fibres in sub-maximal endurance exercise and an increase in fast twitch recruitment in later stages of exercise and at supra-maximal workloads.

1.2.4. Normal Muscle Fibre Distribution. The quadriceps is a large muscle group composed of the vastus lateralis, vastus medialis, vastus intermedius, and rectus femoris and controls knee extension. The quadriceps is highly active in endurance activities such as cycling (Gollnick et al., 1972) and running (Widrick et al., 1996a) and power activities such as jumping (Häkkinen and Komi, 1986). The vastus lateralis in untrained individuals is characterized by a large proportion of type I fibres (45-59%), a moderate type IIa proportion (30-39%), and a small type IIb proportion (11-15%) (Esbjörnsson et al., 1999; Simoneau and Bouchard, 1989). However, there is a wide

variation between individuals and type I, type IIa, and type IIb proportions have been observed to range between 15-79%, 13-77%, and 0-44%, respectively (Simoneau and Bouchard, 1989). The vastus medialis and the vastus lateralis contain similar percentages of type I fibres, 52.1% (Gollnick et al, 1972) and 51.4% (Kuzon et al., 1990) respectively. However, as many as 79% of the gastrocnemius (Costill et al., 1976b) and 88% of the soleus fibres (Johnson et al, 1972) may be type I. The relatively high proportion of type I fibres in the quadriceps, gastrocnemius and soleus is probably due to the fact that these are postural and locomotive muscles that are active for a long duration, but at low contractile forces. In contrast, muscles of the upper body, such as the triceps and biceps, are used for brief forceful contractions and composed of high proportions (60-70%) of type II fibres (Johnson et al, 1972; MacDougall et al, 1982; 1984).

1.2.5 Muscle Fibre Type and Athletic Performance. The physical activity background of individuals may influence and account for their muscle fibre composition or alternatively, the inherent fibre type composition of an individual may influence his or her selection of events. Endurance trained athletes have a greater composition of type I fibres (Costill et al., 1976a; 1976b; Jansson and Kaijser, 1977; Larsson and Ansved, 1985; Tesch and Karlsson, 1985) and a lower composition of type IIb fibres (Friden et al., 1984; Ingjer, 1978b; Tesch et al., 1984) compared to control subjects. In a cross-sectional study designed to examine the contrast in muscle fibre composition between elite athletes, sprinters had a small proportion (24%) of slow twitch fibres in comparison to the large slow twitch fibre (69%) composition in distance runners (Costill et al., 1976a). Whether the difference is due to training, or selection of the sport as influenced

by genetics, is unknown. Tesch and Karlsson (1985) hypothesize that long-term endurance training may influence muscle fibre composition. Not only was their hypothesis supported by a large proportion of type I fibres in the vastus lateralis of runners, but also by a large proportion of type I fibres in the deltoid muscle of kayakers and wrestlers (Tesch and Karlsson, 1985). Differences in fibre type have been shown to be associated with significantly greater maximal oxygen consumption in endurance-trained athletes (Ingjer, 1978b). Classical dancers also have a significantly greater proportion of type I fibres and a lower proportion of type IIb fibres in the vastus lateralis, when compared to sedentary individuals (Dahlströhm et al., 1997). This greater proportion of type I fibres would benefit these performers, because their activities are of low intensity and long duration. Larsson and Ansved (1985) conclude that fibre type make-up is not determined exclusively by heredity and can adapt to physical demands.

Weightlifters and untrained individuals were shown to have similar type I percentages, but the weightlifters had a lower average percentage area of the type I fibres (Gollnick et al, 1972). Percent fibre area is calculated as a product of the percent fibre type and the fibre area, as a proportion of the entire muscle area. Percent fibre area may be more important than fibre proportion in determining function. A greater type II percent area would result in a larger proportion of fast myosin heavy chain isoforms in the muscle and therefore a faster velocity of shortening. Tesch & Karlsson (1985) found the type II fibre area of weightlifters to be significantly greater than in wrestlers or kayakers and that runners had the smallest fibre areas for the vastus lateralis. The small fibre areas of the runners would contribute to a lower force production and a possible impairment in power production.

1.3 DETERMINATION OF FIBRE TYPE

1.3.1 Histochemical Classification. Histochemistry is the most common method for fibre identification and uses myofibrillar (myosin) ATPase reactions to indicate the myosin heavy chain (MHC) isoform of the muscle fibre (Gauthier, 1979; Jostarndt et al., 1998; Staron and Pette, 1986). Histochemical analysis of myosin ATPase is the most popular method used in the literature because it is quick, simple, and effective. The technique is based on the difference in the sensitivity of a muscle fibre's myosin ATPase activity, after exposure to different pH incubations (Brooke and Kaiser, 1970). Prior to histochemical identification of myosin ATPase, methods included staining for mitochondrial enzyme activity to identify type I fibres, or for glycolytic enzyme activity to identify type II fibres (Gollnick et al., 1972). Recently, the use of gel electrophoresis to separate myosin heavy chain isoforms has become more common and may allow for a more accurate quantification of the myosin composition of muscle samples (Betto et al., 1985).

Originally, only two muscle fibre types were identified with histochemical methods (Andersen and Henriksson, 1977a), but advanced techniques have enabled the sub-groups of the type II fibres to also be distinguished (Brooke and Kaiser, 1970). The delineation of the three or more fibre types is accomplished by staining three serial sections of a muscle biopsy following specific pH incubations (Staron, 1997). The type I fibres stain positive at pH 4.3 and 4.6, the type IIa fibres stain positive at pH 10.0, and the type IIb fibres stain positive at pH 4.6 and 10.0 (Andersen and Schiaffino, 1997). Incubation at a pH of 4.6 allows for the distinction between type II muscle fibre

subgroups (Smerdu et al., 1994). Small proportions of additional fibre types (Ic, IIc, IIac, and IIab) have also been observed in humans through ATPase histochemistry and coexpress different amounts of myosin heavy chains (Staron and Hikida, 1992). The delineation of type I, type IIa, and type IIb fibres is essential for a true understanding of the contractile and oxidative properties of muscle. Unfortunately, histochemical analysis of needle biopsy samples may have a significant sampling and method error that may leave changes in fibre type going undetected (Gollnick et al., 1973a; Baumann et al., 1987). The variation in inter-biopsy fibre types from the same muscle of one individual has been reported to be as high as 6% (Staron, 1997).

1.3.2 Classification According to Myosin Heavy Chain (MHC) Isoforms.

Myosin heavy chain measurement establishes the proportion of a specific fibre type in a muscle biopsy. Myosin is the major protein of the thick filament, consists of two heavy chains and is a suitable marker of fibre type diversity (Pette, 1998). There is a difference in the MHC composition of type I and type II fibres; therefore, these two fibre types are presumably products of different genes (Howald, 1982; Alway et al, 1989). The MHC contains the site of ATP hydrolysis and is a major determinant of contraction speed (Larsson and Moss, 1993; Reiser et al., 1985) and therefore the myosin isoform is classified as to its speed of contraction. There is controversy over the designation of myosin heavy chain types in humans. Three fast (MHC-IIb, MHC-IIx, and MHC-IIa) isoforms and one slow (MHC-I) isoform exist in small mammals, but, in humans there appear to be only three total MHC isoforms, previously labeled as slow (MHC-I) and fast (MHC-IIa and MHC-IIb) (Pette, 1998). It had also been established that small mammals

contain different myosin heavy chain isoforms than humans (Pette, 1998). The human MCH-IIb isoform properties best correlated with the properties of the rodent MHC-IIx isoform, rather than with the rodent MHC-IIb isoform (Smerdu et al., 1994), therefore the human MHC-IIb is a misnomer. Recently, Williamson et al. (2000) identified six fibre types, including three co-expressing up to three different myosin heavy chain isoforms (I, I/IIa, I/IIa/x, IIa, IIa/x, IIx). In addition, this paper labeled the fastest human fibre type as IIx, rather than IIb, as this study showed that the fastest human muscle fibre type corresponded most similarly to the mammalian IIx isoform (Williamson et al., 2000). Therefore, MHC-IIx is the proper designation, yet past studies that have used the designation MHC-IIb will be referenced as so.

Myosin heavy chain analysis determines the amount of each specific MHC isoform in a muscle sample. The myosin heavy chain isoforms are distinguished by gel electrophoresis and are regulated by contraction (O'Neill et al., 1999). Large volumes of muscle contractions have been suggested to alter fibre type and MHC composition. Histochemical analysis of muscle after training has supported this belief (Andersen et al., 1994) but the MHC composition is considered the single best marker of exercise-induced fibre type changes (Pette and Staron, 1997). Each histochemically determined fibre type has a particular MHC profile and associated contractile properties. Perrie and Bumford (1986) associated a specific histochemical ATPase stain with one of the three human MHC isoforms. Post-training fibre type transformations, as measured by histochemistry, have been correlated with equivalent changes in the MHC isoform compositions (Adams et al., 1993; Andersen et al., 1994; Baumann et al., 1987; Staron, 1997; Staron and Pette, 1986). However, histochemistry does not determine the exact proportion of a single

MHC isoform in hybrid muscle fibres. In one study, the MHC composition was different from the histochemically determined fibre type composition, suggesting a proportion of the histochemical type IIb fibre population may have been hybrid fibres (Andersen et al., 1994). This highlights the importance of including MHC analysis when examining fibre composition.

Changes in fibre type and MHC composition occur at the molecular level and therefore reflect changes in genetic expression of proteins. A change in the MHC isoform indicates changes in the molecular structure of specific myosin molecule (Klitgaard et al., 1990). Rodent MHC isoforms show a functional plasticity in response to chronic low-frequency stimulation (Pette and Dusterhoft, 1992) and human MHC isoforms may change with resistance training or endurance training (Adams et al., 1993; Andersen et al., 1994). The magnitude of change in the MHC isoform composition depends on the initial molecular profile of the fibre population before training and the intensity and duration of the training stimulus. The predominate transformation is within the fast twitch subtypes, with very little, if any, measurable change in the slow twitch group (Adams et al., 1993). Many studies have shown variable amounts of two MHC isoforms within a single fibre (Andersen and Schiaffino, 1997; Ciazzo et al., 1996; Harridge et al., 1996; Smerdu et al., 1994). The "hybrid" or "intermediate" fibre has been hypothesized to represent a stable transitional stage in the process of fast to slower fibre type conversion (Andersen and Schiaffino, 1997; Mabuchi et al., 1982). Hybrid fibres may be incorrectly classified in histochemical analysis based on the dominant MHC isoform expressed (Klitgaard et al., 1990; Wang et al., 1993), or the hybrid may be excluded from histochemical analysis (Widrick et al., 1996b). Therefore the separation

of the myosin heavy chains (MHC) provides a better representation of fibre transformation than histochemistry, because the MHC composition analysis can delineate hybrid fibres.

Hybrid fibres may be found after periods of detraining (Fry et al., 1994; Larsson & Moss, 1993) but may be reduced with resistance training (Williamson et al., 2000). Klitgaard et al. (1990) originally hypothesized that extreme contractile activity such as endurance training will permit the co-expression of MHC isoforms in single fibres. However, according to Williamson et al. (2000) this is unlikely because resistance training was actually shown to reduce MHC co-expression in single muscle fibres in older men. Andersen and Schiaffino (1997) documented a time delay between the transcription of MHC mRNA and the expression of the protein. Ciaozzo et al. (1996) also observed a lag time between mRNA transcription and the net accumulation of MHC isoforms. This lag period between protein transcription and expression, following low-frequency stimulation in rodents, may be attributed to the slow turnover of myosin protein isoforms (Pette and Dusterhoft, 1992). The lag time has been suggested as one explanation for the observance of hybrid fibres in MHC analysis (Andersen and Schiaffino, 1997). The expression of slower MHC isoforms could result in a more efficient and fatigue-resistant fibre (Ciaozzo et al., 1996; Pette, 1998). Human type IIb muscle fibres may express slower muscle proteins after endurance training, allowing them to become more efficient for endurance activities, although no research has identified the exact signal for MHC transformations (Pette and Dusterhoft, 1992). Thus, changes in the composition of MHC isoforms may occur with training, depending upon the intensity and duration of the training stimulus.

1.3.3 Species Differences in Fibre Types and MHC Composition.

Analysis of small mammals has identified some skeletal muscles to be composed of exclusively "fast-twitch" or "slow-twitch" muscle fibres (Fitts et al., 1998; Green et al., 1983; Mabuchi et al., 1982; Peter et al., 1972; Pette et al., 1976; Staron and Pette, 1986). In contrast, no human muscles are composed exclusively of one fibre type or myosin heavy chain. In rabbits, histochemical analysis showed that the tibialis anterior is a "fast" muscle and is composed exclusively of type II fibres (Mabuchi et al., 1982; Pette et al., 1976). The soleus of the rabbit contains only type I fibres and is a "slow" muscle with contractile properties antagonistic to those of the tibialis anterior (Staron and Pette, 1986). The gastrocnemius and soleus of the rhesus monkey are composed exclusively of MHC-IIx and MHC-I isoforms respectively (Fitts et al., 1998) and therefore should contribute low-intensity non-fatiguing contractions to activities such as locomotion. Studies have shown small mammals' muscles can contain almost exclusively one fibre type. The rabbit soleus was found to contain 96% type I fibres and the guinea pig vastus lateralis was found to contain 86% type IIb fibres (Peter et al., 1972), but no muscles have been observed that were composed exclusively of type IIa fibres. Even within a single muscle, there may be areas composed of exclusively one fibre type. In the rat, the vastus lateralis can be differentiated into "fast" (superficial) and "slow" (deep) compartments (Green et al., 1983). In comparison, human skeletal muscles are heterogeneous with varying proportions of all fibre types (Klitgaard et al., 1990; Widrick et al., 1996a).

Smaller mammals appear to exhibit more extreme muscle compositions as differences between rats, rabbits, and monkeys have been observed. In the smaller mammals, rats have been found to have a lower proportion of slow MHC isoforms in

comparison to rabbits (Jaschinski et al., 1998). The muscle composition of mammals appears to change with size. Observations of the rhesus monkey have suggested its muscle composition is actually more like that of the human since its contractile properties are closer to those of humans than to rats (Fitts et al., 1998). Unlike the rat, neither the soleus nor the gastrocnemius of the rhesus monkey contained type IIb fibres, but rather consist of considerable amounts of mammalian type IIx fibres (Fitts et al., 1998). The large species differences in MHC composition limits the application of the results of animal studies to humans. As well, training programs between species are difficult to equate because animals may be forced to tolerate heavier workloads than humans (Terrados et al., 1986) and chronic low-frequency stimulation is not possible in human research.

1.4 TRANSFORMATION OF FIBRE TYPES

1.4.1 Chronic Electrical Stimulation. Prolonged electrical stimulation of muscle fibres causes alterations in contractile properties and fibre type. Animal studies have shown that chronic low-frequency electrical stimulation can change the contractile properties of entire muscles, such that a fast muscle becomes slower (Salmons and Vrbova, 1969). Changes in muscle fibre type or MHC isoform composition may be related to the intensity and duration of the stimulus. In some studies, low-frequency long-term electrical stimulation may convert type II fibres to type I fibres in small animals (Edstrom and Grimby, 1986; Jaschinski et al., 1998). However, in one investigation, stimulation caused conversion only within the type II subgroups (from type

IIb to type IIa fibres) and no change in the overall proportions of the type II or type I fibres (Mabuchi et al., 1982).

Studies that involve de-nervating motor units and re-innervating the fibres with a new motorneuron are called cross-innervation studies. Cross-innervation experiments that supply type II motor units with type I motorneurons can convert type II motor units to type I, reduce the maximum tension of a fibre, and increase the time-to-peak twitch of a fibre (Salmons and Sreter, 1976). Cross-innervation studies can therefore cause adaptations similar to those observed in long-term electrical stimulation models.

The changes in the muscle fibre composition and contractile properties are determined by the genetic response to the stimulus. Chronic (24 hour) stimulation can cause complete fibre transition in rabbits in approximately 21 days (Pette et al., 1976), whereas six days of 24 hour chronic stimulation was required to induce a significant change in the expression of MHC proteins (Jaschinski et al., 1998). Contractile velocity is altered during fibre type transitions, resulting in an increase in the time-to-peak twitch tension (Pette et al., 1976; Salmons and Sreter, 1976). Fibre transformations occur concomitant with sequential shifts in the MHC isoform composition (Jaschinski et al., 1998). The structural and functional responses to electrical stimulation suggest an increase in the synthesis of slow myofibrillar proteins and, therefore, a transition from fast to slow myosin isoforms.

Electrical stimulation models support the hypothesis that fibre transitions may also occur in response to voluntary muscle activation in an exercise model, if the exercise stimulus is of a sufficient intensity and duration. The magnitude of the required stimulus is unclear, although the majority of endurance training studies have shown a shift in fibre

composition in rodents (see Pette and Staron, 1997). Chronic low-frequency stimulation animal experiments are excellent models for eliciting a change in fibre composition because they allow the administration of a standard contractile activity, uniform activation, and a guaranteed intensity and duration (Pette, 1998). However, the findings of these studies are not applicable to the human model, due to the duration of the stimulus. It is not physically or ethically possible to have subjects exercise for 24 hr per day for several weeks. Therefore, the ability to use electrical stimulation protocols makes animal studies very attractive but not applicable and only human training studies allow for the extrapolation of results to strength and power sport training programs.

1.4.2. Training. In animals, the proportion of type I fibres has been consistently shown to increase with chronic low-frequency stimulation (Pette and Dusterhoft, 1992). Type I fibres (from S motor units) are recruited during all activities (Henneman et al., 1965) and therefore undergo a large volume of contractile activity regardless of intensity, so it may be hypothesized that endurance training would induce similar fibre transformations. Green et al. (1984) found an increase in type I and IIa fibres in the deep portion of the vastus lateralis of rats after 15 weeks of endurance training. Regular endurance exercise may alter the function and structure of muscle fibres due to the expression of different contractile proteins (Fitts et al., 1991), but the magnitude of change would likely be less than the response to low frequency electric stimulation observed in animals (Pette, 1998). Pette and Staron (1997) have concluded that an increased neuromuscular loading (contractile activity) generally shifts rodent muscle MHC composition to slower isoforms in a graded and sequential manner. Muscle fibres

that are recruited frequently and for a sufficient duration may switch off the transcription of the MHC-IIb gene in favour of the MHC-IIa gene (Jaschinski et al., 1998). In rats, even a small stimulus (10 contractions, 40 seconds of muscle tension) increased the composition of MHC-IIx and MHC-IIa isoforms and decreased the expression of the MHC-IIb isoform (Ciazzo et al., 1996). The result of resistance training in rats was a motor unit that was more fatigue-resistant, but still capable of high mechanical power. At the protein level, these changes are primarily due to a transition from the MHC-IIb to the MHC-IIa isoform (Jaschinski et al., 1998) and there may not be an increase in the MHC-I isoform. Fibre transformations toward a slower myosin isoform are generally accepted as occurring in response to training, but it is important to examine changes within the type II subgroups as well as changes between type I and type II fibres.

Human skeletal muscle fibres are not fixed units but exist in a dynamic continuum. The nature of muscle allows transformation in response to altered functional requirements (Baumann et al., 1987; Trappe et al., 1995) by adapting the molecular composition of individual muscle fibres (Baumann et al., 1987). The intensity and duration of the contractile stimulus are likely major determinants of a muscle's plasticity and the adaptations that the muscle makes in response to training. In further support of the concept of muscle plasticity, training adaptations have been shown to regress during periods of detraining (Staron et al., 1991) and sedentary individuals were found to have a large composition of type IIb fibres (Klitgaard et al., 1990). It appears that muscle fibre composition and its dependent contractile properties are in part determined by the functional demand imposed on the motor unit. The relationship between the structural and functional properties of a muscle fibre, in response to the demand of a stimulus, have

been summarized according to Pette and Staron (1997), in the following statement, "functional demand affects structural adaptation dictating functional expression".

Type I fibres are the dominate fibre type for force production in aerobic activities such as distance running because they can sustain low force contractions for a long duration. As a result of their selective recruitment in activity (Costill et al., 1973), the composition of type I fibres has been shown to increase with endurance training (Howald et al., 1985), resistance training (Staron et al., 1991), and concurrent (strength and endurance) training (Sale et al., 1990). The proportion of type I fibres also increases with age (Trappe et al., 1995). Endurance training can result in a shift to a slower myosin heavy chain isoform, such as the replacement of MHC-IIx by MHC-IIa and MHC-IIa by MHC-I (Andersen and Schiaffino, 1997). The induced changes in MHC isoform expression result primarily from altered pre-transcriptional events. A change in the MHC isoform may be responsible for the change in fibre composition (as determined histochemically). A slower myosin isoform enables the muscle to sustain work at a lower energy cost (Howald et al., 1985), because these fibres are more efficient (require less ATP per unit of force produced) and may enhance endurance (Fitts et al., 1991; Schluter and Fitts, 1994).

The type IIb fibre appears to be the most adaptable type in response to stimulation and contractile activity, since the majority of studies demonstrate that an increase in contractile activity will alter the proportion of type IIb muscle fibres and MHC-IIb composition. One month of endurance training (cross-country skiing) has been shown to decrease the number of type IIb fibres in the triceps brachii by 13% (Schantz and Dhoot, 1987). Myosin heavy chain analysis showed that the MHC-IIb composition was almost

completely absent following endurance training (Baumann et al., 1987; Klitgaard et al., 1990). Endurance training, performed for at least 6 weeks, results in sufficient contractile activity to reduce the proportion of type IIb fibres (Howald et al., 1985) and increase the number of type IIa fibres (Andersen and Hendriksson, 1977a). Short-term, high-intensity aerobic training is clearly an adequate stimulus for inducing changes in protein expression of the contractile apparatus (Baumann et al., 1987). O'Neill et al. (1999) found a suppression of MHC-IIx mRNA within seven days after initiating daily endurance training. The suppression was only observed immediately after the exercise bout, on day 7 of the training regimen. Higher pre-training MHC-IIx isoform proportions resulted in a greater post-exercise depression of MHC-IIx synthesis (O'Neill et al., 1999). The pre-training muscle composition is an important contributing factor in the degree of transition that will occur with training. The activity of myosin ATPase decreased in type II fibres after 12 weeks of endurance training (Bell et al., 2000). This adaptation suggests that the training caused a shift towards slower contractile properties.

Additional forms of training, such as resistance training, have resulted in similar patterns of change in fibre composition (Hather et al., 1991; Simoneau et al., 1985). High-volume resistance training caused a significant type IIb to type IIa transition in previously untrained males (Hather et al., 1991; Kraemer et al., 1995) and females (Staron et al., 1989) and the changes can occur as rapidly as within 14 days (Staron et al., 1994). Even short-term (6 weeks) sprint training can induce a reduction in type IIb fibres (Esbjörnsson et al., 1993; Jacobs et al., 1987) although not all sprint training studies support this (Thorstensson et al., 1975). Fibre transitions can also occur in a graded manner in response to the high-volume contractile activity of team sports. Green et al.

(1979) observed an increase in the type I and IIa populations and a corresponding decrease in type IIb fibre proportions of elite hockey players over the course of a season. The hockey players began the season with a significantly lower type IIb population in comparison to controls and were still able to further decrease the type IIb proportion over the season (Green et al., 1979). Therefore, repeated performances of brief explosive activity can induce a type II subtype inter-conversion similar to that commonly found in endurance training studies.

Very few papers documenting training studies have reported a decrease in type I fibres, however, a study by Andersen et al. (1994) reported that sprint training resulted in a shift from type I to IIa fibres. In that study the training caused a decrease in type I and type IIb fibres and MHC-I and MHC-IIb isoform proportions and increased type IIa fibre and MHC-IIa isoform composition. This unique bi-directional shift toward the intermediate type IIa fibre suggested that mechanical loading up-regulates the expression of the MHC-IIa isoform.

Within the subgroups, the transition of type IIb fibre to type IIa fibres is common, but there is little evidence for a shift of type II to type I fibres (Gollnick et al., 1973a). However, one endurance training study resulted in a decrease in the proportion of type IIb fibres, without a corresponding change in type IIa or type I fibre proportions (Baumann et al., 1987). A hybrid fibre may also exist and probably represents a transitional stage between the type IIb and type IIa fibres. Hybrid fibres containing both the MHC-IIb and MHC-IIa isoforms have been observed following endurance training and are referred to as the MHC-IIab isoform (Andersen et al., 1994; Schantz and Dhoot, 1987). Only MHC analysis will enable accurate identification of the hybrid fibres.

Some long-term training studies have failed to show fibre transformation. Klausen et al. (1981) did not find any change in fibre proportions, despite a significant increase (15%) in aerobic performance. Studies in which no changes in fibre type proportions were reported (Saltin et al., 1976) may be the result of the exclusive methods that were used for identifying fibre types and by the fact that type II subgroups were not examined (Andersen and Henriksson, 1977a). For example, Gollnick et al. (1973a) did not distinguish between the two type II subgroups and found no significant fibre transitions despite a 20-week program of endurance training. Therefore advanced analysis techniques, such as single fibre type subgrouping using SDS-PAGE and myosin heavy chain analysis are necessary to accurately identify possible transitions in fibre types after a period of training.

It is believed that fibre type transformations occur in response to significant changes in the amount and pattern of muscle activity. High training volumes, regardless of intensity, appear to be most effective in causing a transformation of type IIb to type IIa fibres. Fibre type and MHC isoform conversions require changes to occur at the molecular level. The molecular changes may occur if the gene for a fast contractile protein can be shut off and the gene for a slower muscle protein turned on (Edstrom and Grimby, 1986) and would theoretically occur in response to a large volume of slow velocity contractions. Contractile force does not appear to be a factor, since both endurance (Howald et al., 1985) and resistance training (Staron et al., 1989) can cause the type IIb to type IIa shifts. The loss of type IIb fibres that occurs with increased contractile activity may impair power (due to the high shortening velocity of type IIb fibres), but it does not appear to impair strength. Resistance training that causes a loss of

type IIb fibres may still result in improvements in strength because the remaining type IIa fibres may have an increase in cross-sectional area (Esbjörnsson et al., 1999; Simoneau and Bouchard, 1989; Staron et al., 1989). Therefore, provided there is no difference in specific tension between the type II fibre subgroups, decreases in strength will not necessarily result from the transition of type IIb to type IIa fibres. These findings also have implications for endurance training. Although the effect of endurance training on fibre area is debatable, if no decrease in fibre cross-sectional area occurs and there is no difference in specific tension between fibres, there should be no decrease in muscle strength even if there is a change in fibre type.

1.5 ADDITIONAL ADAPTATIONS TO ENDURANCE TRAINING

Endurance training is characterized by sub-maximal exercise, performed with a large muscle mass, for an extended duration. The muscular demands of endurance exercise are in contrast to those of strength and power exercise and would be expected to cause different training adaptations. Adaptations to endurance training would normally not be expected to improve performance in high-intensity power activities. Constable and colleagues (1980) support this hypothesis and showed no changes in strength or power measures after 8 weeks of endurance exercise. In fact, adaptations to endurance training are commonly considered antagonistic to the development of strength and power (Kraemer et al., 1995) such as a type IIb to type IIa fibre transition (Howald et al., 1985). Endurance training is also known to increase the oxidative capacity of muscle fibres and to increase the capacity of the cardiovascular system.

1.5.1 Maximal Aerobic Power. Maximal aerobic power ($\dot{V}O_{2max}$) is the maximal rate that ATP can be synthesized aerobically in muscle. It is indirectly quantified by measuring the rate that oxygen is taken up by the body while performing maximal exercise, with the assumption that the rate that oxygen is consumed equals the rate of aerobic ATP production. It has thus become common to use the term maximal aerobic power synonymously with the term maximal oxygen consumption or $\dot{V}O_{2max}$. Maximal oxygen consumption is determined by the maximal rate that oxygen can be delivered to the muscles (maximal cardiac output) and the maximal rate that it can be taken up or extracted by the muscle. Both of these limiting factors can be improved with training. At the muscle level, $\dot{V}O_{2max}$ could be expected to increase with endurance training due to changes including an increase in blood flow and capillary density (Saltin and Rowell, 1980), oxidative enzymes (Trappe et al., 1995), and a decrease in fibre area (Kraemer et al., 1995).

Howald et al. (1985) concluded that the aerobic capacity of all muscle fibre types could be improved following a period of endurance training. The magnitude of change in maximal oxygen consumption after training depends on the intensity and duration of the exercise stimulus and the initial training status of the individual. Longitudinal studies (4-8 weeks) have shown increases of 8% to 25% in $\dot{V}O_{2max}$ (Andersen and Henriksson, 1977a; Dudley and Djamil, 1985; Gollnick et al., 1973a; Hickson, 1980; Howald et al., 1985; Ingjer, 1978b; Kraemer et al., 1995). The $\dot{V}O_{2max}$ observed in unilateral cycle ergometer exercise is approximately 70-80% of two-leg exercise (Saltin et al., 1976). Four weeks of unilateral cycle ergometer exercise training at an intensity of 75% $\dot{V}O_{2max}$ has been shown to increase $\dot{V}O_{2max}$ 20% and 6% in the trained and untrained legs

respectively (Saltin et al., 1976). Therefore single-leg endurance training is a sufficient stimulus for increasing aerobic power in the trained leg and a relatively minor portion of this adaptation is probably due to improvements in oxygen delivery (maximal cardiac output). In addition, changes in capillary density and enzyme activity can influence aerobic power.

1.5.2 Capillary Supply. Capillaries transport blood to the muscle fibre, delivering oxygen and energy substrates during prolonged bouts of exercise. Endurance training is usually a powerful stimulus for increasing capillary density (number of capillaries per unit area) causing an increase of as much as 20% in 8 weeks (Andersen and Henriksson, 1977b; Ingjer, 1978a). Resistance training has also been shown to cause angiogenesis (Hather et al., 1991) but the normal result of muscle hypertrophy is a reduction in capillary density (MacDougall et al., 1986). Bell et al. (2000) found an increase in capillary density only after a program of strength and endurance training, but not after endurance training alone. An increased vascularization of skeletal muscle would facilitate the oxidative capacity of the muscle fibres and would be an expected adaptation following endurance training. Additionally, comparative studies show that endurance-trained individuals have a greater capillary density in comparison to age-matched sedentary controls (Klausen et al., 1981; Terrados et al., 1986; Tesch et al., 1984: Trappe et al., 1995). The increase in capillary density parallels the increase in maximal aerobic power (Andersen and Henriksson, 1977b). If capillary density is low, the diffusion distance for oxygen into the muscle fibres would be increased and this would represent a peripheral limitation to aerobic performance. Therefore it would be

expected that a long-term program of endurance training would elicit an increase in capillary density.

Capillary density can also be influenced by muscle fibre cross-sectional area. Small muscle fibres would be expected to have a greater capillary density, because it would enable more capillaries to be within a given area. In comparison to control subjects, orienteers did not have a greater capillary supply (number of capillaries per muscle fibre), but they did have a smaller fibre area (Jansson and Kaijser, 1977). Therefore, it can be hypothesized that the endurance-trained orienteers had a greater capillary density. In contrast, fibre hypertrophy could decrease capillary density because there would be more fibre area and a smaller number of capillaries in a given area. Researchers commonly choose to express the capillary characteristics as capillary-to-fibre ratio (supply) instead of capillary density because the determination of capillary density also depends on the orientation the muscle biopsy sample (Andersen and Henriksson, 1977b). For example, a biopsy viewed obliquely, as opposed to a true cross-sectional view, would overestimate fibre size and underestimate capillary density. The measure of muscle capillarization must be distinguished when comparing studies.

1.5.3 Mitochondrial Volume Density and Enzyme Activity. Endurance training is known to increase the number and size of mitochondria in trained muscles and results in an increased oxidative capacity of the muscle (Howald et al., 1985). Citrate synthase is a mitochondrial enzyme and a marker of the muscle fibre's oxidative capacity. As endurance training increases mitochondrial density, the activity of the mitochondrial enzymes should increase as well. The reaction velocity, at a given

substrate concentration, is a function of total enzyme concentration (Gollnick and Saltin, 1982), therefore the greater the amount of citrate synthase, the greater the rate of oxidative reaction for a given concentration of substrate. Because citrate synthase is a marker for other TCA enzymes, an increase in citrate synthase activity indicates an increase in other enzymes as well (Hickson et al., 1988; Schluter and Fitts, 1994). Additionally, comparative studies show that aerobic fitness is correlated with enzyme concentration (Holloszy and Coyle, 1984; Trappe et al., 1995), although the activity of enzymes such as citrate synthase increase to a much greater extent than the increase in maximal oxygen consumption (MacDougall et al., 1998). Even chronic low-frequency stimulation has been shown to increase citrate synthase activity in rabbits (Sutherland et al., 1998). In untrained humans, citrate synthase values are approximately 10.8, 8.5, and 6.5 umol/g/min in type I, type IIa, and type IIb fibres respectively (Essen et al., 1975), although all fibres have the ability to increase oxidative enzyme capacity (Gollnick et al., 1973a). The potential for mitochondrial enzyme concentration to increase after endurance training is reduced if accompanied by hypertrophy of the muscle fibre (Stone et al., 1996). Therefore, when fibre hypertrophy accompanies endurance training, there may not be an increase in mitochondrial enzyme activity.

1.5.4 Lactate Threshold. During incremental exercise the "Lactate threshold" (T_{lac}) is the workload at which the rate of lactate production in skeletal muscle exceeds its rate of removal, leading to an exponential increase in blood lactate concentration (Brooks, 1985). During exercise, blood lactate concentrations do not increase significantly until the T_{lac} inflection point is reached (~60-80% $\dot{V}O_{2max}$), at which point

lactate increases non-linearly and continues to increase with further increases in exercise intensity. Endurance training shifts the T_{lac} in untrained individuals so that it occurs at a higher intensity post-training and it also occurs at a higher intensity in endurance trained individuals compared to untrained (Hurley et al., 1984; Gaesser & Poole, 1988).

1.5.5 Fibre Area. In fibres that have been identified by their oxidative and glycolytic enzyme concentrations, fast-glycolytic (type IIb) fibres normally have the greatest cross-sectional area (McDonagh et al., 1980). However, type IIa fibre cross-sectional area has also been shown to be greater than the type IIb fibre area (Esbjörnsson et al., 1999; Simoneau and Bouchard, 1989) and type I fibre area is consistently the smallest of all fibres (Costill et al., 1976a). Hypertrophy, an increase in the size of the muscle fibre, is the typical response to the mechanical overload applied in resistance training. Endurance exercise may also mechanically overload the muscle in addition to overloading the oxidative system. However, the metabolic requirements of endurance training are relatively greater than the magnitude of the imposed overload on the contractile stimulus and therefore hypertrophy is not commonly observed after endurance training programs. Bell et al. (2000) did not observe any increase in fibre size after 12 weeks of endurance training and suggest that a lack of hypertrophy would limit increases in strength. Endurance exercise might even be expected to reduce the cross-sectional area of the fibre to maximize the surface area-to-volume ratio and to decrease the diffusion distance for gas and metabolite exchange. A decrease in the number of myofibrils and amount of contractile protein per fibre would decrease fibre cross-sectional area, thereby reducing force output and decreasing power potential. The final

outcome of a long-term program of endurance training that reduces fibre area could impair strength and power.

The response of muscle fibre cross-sectional area to endurance training appears to be variable. Several studies in humans support an increase in fibre area after endurance training (Andersen and Henriksson, 1977b; Klausen et al., 1981; Saltin et al., 1976). The intensity of training may influence the response. In horses, muscle fibre area did not increase after 7 weeks of training at 60% $\dot{V}O_{2max}$, however continued training at higher intensities (intervals of 100% $\dot{V}O_{2max}$) increased fibre area by 20% in 25 weeks (Tyler et al., 1998). In contrast, alternative studies suggest that fibre area can decrease as a result of endurance training (Jansson and Kaijser, 1977; Terrados et al., 1986; Widrick et al., 1996a) and the decreases may occur in type I (Kraemer et al., 1995) and type II fibre groups (Gollnick et al., 1973a). The majority of studies show no change in fibre area (Bell et al., 2000; Constable et al., 1980; Fitts et al., 1989; Schluter and Fitts, 1994). A comparative study of runners, cyclists, and sedentary controls showed no difference in the mean fibre area of the vastus lateralis (Tesch et al., 1984). However, in the same study, cyclists had a greater FT/ST area ratio than the runners, suggesting that the mode of training may have some bearing on muscle overload and selective hypertrophy (Tesch et al., 1984). Concurrent training for both strength and endurance did not increase fibre area (Sale et al., 1990). Therefore, endurance training may attenuate the normally observed increases in fibre area associated with resistance training. Further examination of the adaptive response of concurrent strength and endurance training is warranted.

It is important that the type II subgroups are identified in analysis because there may be differences in size and in response to training interventions. Some studies

express an average fibre area for the type IIa and type IIb fibres (Constable et al., 1980; Saltin et al., 1976) but others distinguish only between type II and type I fibres (Gollnick et al., 1973a). The delineation of the type II fibre to its subgroups is an essential component of proper analysis, because of the significant differences between the two subtypes in oxidative and contractile function.

1.6 INTERFERENCE EFFECTS OF CONCURRENT STRENGTH AND ENDURANCE TRAINING

The compatibility of concurrent strength and endurance training has been examined in many studies. Some studies have shown impaired increases in strength from combined training when compared to only strength training (Hickson, 1980; Hunter et al., 1987; Dudley and Djamil, 1985). One study showed impaired strength in leg extension but not leg press after 12 weeks of concurrent training (Bell et al., 2000). Other studies have found no impairment, or only a trend towards impaired performance (Kraemer et al, 1995). Concurrent training programs rarely impair the development of aerobic power and Nelson et al. (1984) were the only group that found the greatest strength gain to occur in the concurrent training group while they also observed an impaired improvement in aerobic capacity. Strength and endurance training, in isolation, result in antagonistic physiological responses and demonstrate the principle of "specificity in training". For example, resistance training results in an increase in strength, muscle mass (Kraemer et al., 1988; 1990) and fibre size (MacDougall et al., 1977) but can decrease muscle fibre capillary density (Schantz, 1983) and mitochondrial volume density (MacDougall et al., 1979). A muscle fibre is required to repeatedly produce low force contractions during

endurance exercise and these fibres may adapt by decreasing muscle fibre cross-sectional area (Kraemer et al., 1995). A smaller fibre area would enhance gas diffusion, transport of wastes and energy substrates and should enhance oxidative capacity. However, fibre adaptations that increase aerobic power might do so at the expense of strength and power. Costill et al. (1967) observed an impaired vertical jump in distance runners, compared to controls and Hunter et al. (1987) found a compromised increase in vertical jump in the concurrent training group when compared to the strength-training group.

The effect of concurrent training on fibre area is unclear. Sale et al. (1990) found no inhibitory effect while McCarthy et al. (1995) observed a lesser increase in fibre size with concurrent training in comparison to strength training alone. The outcomes have been highly variable because of study design and the multitude of factors that influence the interaction of the two modes of exercise. For example, different endurance training modes may influence results. Bell et al. (2000) observed an increase in leg press strength following endurance training only, and attributed this to the mode of training, since cycling may increase the strength of the hip and knee extensors. Distance running does not appear to increase leg strength (Kraemer et al., 1995). Concurrent strength and endurance protocols may also have impacted results by causing subjects to become overtrained. A state of overtraining would be expected to be detrimental to both endurance and strength performance. Finally, the study design may have affected the results, based on the order of the training since the initial training mode may interfere with the intensity of the second training component. Subjects in the study by Nelson et al. (1984) may not have trained optimally for endurance, due to fatigue from the prior strength training and this may account for the unexpected results. Concurrent strength

and endurance training appears to impair strength performance in some, but not all, situations while endurance does not appear to be negatively affected.

1.7 SUMMARY

Endurance training and strength training result in markedly different adaptations. Endurance training increases the oxidative capacity of the muscle through increases in capillary density, oxidative enzyme activity, possible reductions in fibre size, and the transition to a slower muscle fibre type or MHC isoform. Muscle fibre transitions are a well-accepted phenomenon in response to an increase in contractile activity and muscle plasticity is especially pertinent under human physiological conditions, for example, well-trained athletes. It is generally accepted that fibre conversions (type II to type I) do not occur in human training studies, but these may occur in animal chronic stimulation models. Fibre transitions can be measured by histochemical methods or by MHC composition analysis because both reflect changes in the MHC isoforms. These adaptations may be antagonistic to gains in strength (Kraemer et al., 1995). Power is a measure closely related to strength and speed of contraction. Therefore, it can be hypothesized that adaptations to endurance training may impair power production. It is extremely important for athletes to train properly for improvements in specific sport-related variables. Research has proven that adaptations are specific to the mode of exercise. Since the exercise stimulus causes changes at the molecular level to support the functional demand, improvements would not normally be expected in variables unrelated or antagonistic to the training mode. It should be remembered that elite athletes train at extremely high levels for many years and this may limit the applicability of short-term

training studies on untrained individuals. In summary, muscle fibres are dynamic functional units in accordance to their structure and they respond to training by altering their structure in accordance to functional demands.

1.8 PURPOSE

Endurance training may cause changes in the fibre's contractile proteins that could impair strength and power production and would be counterproductive to athletes engaged in sports requiring rapid, explosive movements (e.g. basketball or ice hockey). The purpose of the present study was to determine whether a prolonged period of intensive endurance training would have an effect on muscle strength and power and, if so, to examine the possible mechanisms behind this effect. We hypothesized that the endurance training program would impair strength and power production, possibly due to changes in fibre type or fibre area.

A unilateral training model was used to test this hypothesis, by having subjects perform 10 weeks of high volume cycle ergometry training with one leg so that the contralateral leg could serve as a control. A series of measurements of endurance and strength and power performance were made on each leg before and after the training period. Changes in muscle fibre area, fibre type, capillarization and oxidative enzyme activity were also assessed from needle biopsy samples of vastus lateralis, before and after the training period.

CHAPTER II

MEASUREMENT TECHNIQUES

2.1 EVOKED MUSCLE TWITCH

Force-time characteristics of the evoked maximal twitch were measured in the quadriceps of both legs before and after the 10 week training period. The subject sat on the dynamometer with his back and thighs supported, so that the thighs were in the horizontal plane, with a trunk-thigh (hip) angle of 100°. Velcro straps across the middle and proximal thigh prevented extraneous movement. The lower leg was strapped to an aluminum plate, which was attached to a steel shaft whose axis was coincident with the axis of the subject's knee joint. The shaft-plate combination was adjusted to set the knee angle to 90°. The steel shaft was instrumented with a strain gauge that sensed the torque developed by isometric actions of the knee extensors. The signal from the strain gauge (sample rate of 3 kHz) was amplified and filtered, AD converted (Model DI420, Dataq Instruments, Inc.) and analyzed with both customized and ACODAS (Dataq Instruments Inc.) software on an IBM-compatible personal computer (Hamada et al., 2000).

2.1.1 Stimulation and Electromyogram Recording. Twitch contractions of the knee extensors (quadriceps femoris) were evoked by indirect percutaneous nerve stimulation. Two carbon-impregnated rubber electrodes were used for stimulation; the cathode (4x4.7 cm) placed on the skin over the femoral nerve in the inguinal crease, the anode (4.5 x 10 cm) placed over the mid-portion of the thigh. Before attaching the electrodes to the skin, electrode gel was applied to the contact surface. The underlying

skin was prepared by shaving, sanding and rubbing with isopropyl alcohol. The stimuli were rectangular voltage pulses of 200 μs duration, delivered from a stimulator (Devices 3072, Medical Systems). Ag/AgCl electromyographic (EMG) disposable recording electrodes, 3.8 mm diameter, were applied to the skin over the belly of vastus medialis (stigmatic), about 20 mm distal and medial to the patella (reference), and on the postero-lateral aspect of the thigh (ground). EMG signals (sample rate 3 kHz) were amplified (1000x) and filtered (10 Hz to 2 kHz). AD conversion and analysis was the same as for twitch torque (Hamada et al., 2000).

2.1.2 Protocol. Subjects were instructed to abstain from both alcohol and caffeine for 24 h before testing and to refrain from participating in any activities that may cause muscle damage (prolonged endurance training, sprinting, resistance training of the lower body, or sports involving high-intensity activities) for 48 h before testing. Subjects sat resting for about 20 min before their leg was placed in the testing apparatus. A maximum pre-MVC (maximum voluntary contraction) twitch response was elicited by delivering a series of single stimuli of increasing intensity until a plateau of twitch torque and muscle compound action potential (M-wave) amplitude was obtained (Hamada et al., 2000).

2.1.3 Twitch measurements. Twitches were analyzed on a computer with a custom-made computer software program. Twitch measurements included peak torque, time-to-peak torque, rise time (10% to 90% peak torque), and half-relaxation time. These

data were collected in collaboration with Tim Rogers as part of a larger study on the effects of endurance training on post-activation potentiation.

2.2 SINGLE-LEG $\dot{V}O_{2peak}$

Maximal oxygen consumption (maximal aerobic power) is determined by the maximal rate that oxygen can be delivered to the muscles (maximal cardiac output) and the maximal rate that it can be taken up or extracted by the muscle. Single-leg $\dot{V}O_{2peak}$ was determined by an incremental exercise test to exhaustion on a cycle ergometer. Subjects pedaled at a cadence of 60 revolutions per minute (RPM) on an electronically braked cycle ergometer (Erich Jaeger, Hoechberg, Germany) for 2 min intervals. To allow for proper movement, the exercising foot was taped to the pedal. Power output was increased by 20-45 watts at the end of each interval until volitional exhaustion, defined as the point at which subjects could no longer maintain 60 RPM. Expired gases were collected with one-way air-flow valves (Hans Rudolph #2700, Hans Rudolph Inc., Kansas City, MO) and analyzed on-line every 30 s by an IBM PS1 computer (International Business Machines, Armonk, NY). Analysis was performed with TurboFit software (Vacumetrics, Ventura, CA) coupled with an AMETEK S3A/1 oxygen analyzer (Applied Electrochemistry, Pittsburgh, PA) and a Hewlett Packard 78356A carbon dioxide analyzer (Hewlett Packard, Mississauga, Ont.). Both analyzers were calibrated prior to and following each test using gases of known O_2 (12.1%) and CO_2 (5.1%) content. The highest 1 min average score was deemed to be the single-leg $\dot{V}O_{2peak}$. Three electrodes (leads) were also attached to the subject's chest to measure heart rate (HR) throughout the testing period and a finger-tip blood sample was collected at the end of

each leg $\dot{V}O_{2peak}$ test for analysis of whole blood lactate concentration (YSI 2300 glucose-lactate analyzer, Yellow Springs, OH).

2.3 LACTATE THRESHOLD

The lactate threshold (T_{lac}) was approximated from the ventilatory record of the leg $\dot{V}O_{2peak}$ test for each leg, with the assumption that the T_{lac} and ventilatory threshold (VT) would occur at similar power outputs (Wasserman et al., 1973). Ventilatory threshold was inferred to be that workload where a major rise in ventilation was observed, along with a drop in the fraction of expired carbon dioxide (F_ECO_2) and an increase in RER. This exponential increase in ventilation is thought to be due to the generation of additional CO_2 by the bicarbonate buffering of H^+ (Wasserman et al., 1973), although the association between lactate accumulation and VT is not universally accepted. While some physiologists maintain that T_{lac} causes VT (Wasserman et al., 1986; Davis, 1986), others have reported a coincidental rather than causal relationship (Neary et al., 1985). Nonetheless, the VT can be used as an approximate indicator of the workload that elicits the T_{lac}. The T_{lac} can then be more accurately identified with a follow-up test, using smaller increments in workload with blood sampling after each.

Subjects returned to the lab to determine their lactate threshold a minimum of 24 hours after their leg $\dot{V}O_{2peak}$ test. The initial workload was set at 20 W below the estimated VT, and for each successive stage the workload, was increased in increments of 10 W until the investigators were satisfied that the T_{lac} had been attained. The criterion for reaching T_{lac} was a marked exponential increase in blood lactate from the previous workload. The same workloads were used in the post-training testing, so that lactate

accumulation could be measured at the same absolute intensity. Subjects performed exercise in 3-minute bouts at each workload, followed by a 2-minute resting period. A fingertip sample of blood (0.25 ml) was collected immediately after each workload and analyzed for whole blood lactate concentration (YSI 2300 glucose-lactate analyzer, Yellow Springs, OH). A blood sample was also taken at rest, before the start of each test. The subject's HR and $\dot{V}O_{2max}$ were monitored throughout the entire test with the same electrode and gas collection system used in the leg $\dot{V}O_{2peak}$ test. These data were collected in collaboration with Manik Bhan and are part of a larger study on central vs. peripheral adaptations to endurance training.

2.4 VERTICAL JUMP

The vertical jump is a common test of muscle "explosive" power. Subjects performed a vertical jump with a 1-leg takeoff. The measurements of force and impulse at the time of take-off were measured on a multi-component force plate (AMTI model OR6-5) and the vertical force component was digitally converted (Data Translation model DT2801A-12-bit resolution) at 100 Hz and stored on computer disk (Dowling and Vamos, 1993). A 2-step approach was allowed and subjects contacted the platform with the take-off foot. They were instructed to jump as high as possible and to limit the horizontal component of the jump. Subjects landed on a second platform placed so that it transmitted a force back to the force platform to indicate the time of landing. A custom-made computer program analyzed the jump performance, to determine power output and impulse. Subjects performed 3 jumps and had their best jump used in final analysis.

Measurements included take-off velocity, maximum positive power, peak force, and impulse.

2.5 STRENGTH

Muscle force, or strength, is defined as the maximal force generated by a muscle at a specified velocity (Knuttgen and Kraemer, 1987) and is proportional to the cross-sectional area of the muscle fibre (Widrick et al., 1996a). Unilateral voluntary strength was measured with a Cybex II isokinetic dynamometer (Lumex, Ronkonkoma, NY) coupled to a leg press unit by a gear-and-chain system to accommodate the high forces generated with this movement (Vandervoort et al., 1984). Subjects sat in the testing apparatus with the foot attached to the leg-press lever arm via footplates and began the movements with their knee angle standardized to 90°. Torque output was recorded and analyzed on a computer. Each subject performed 3 trials at a slow speed (60°/s) and then 3 trials at a fast speed (300°/s) with a 30 s rest between attempts. All trials for one leg were completed prior to repeating the tests for the remaining leg. The highest torque recorded for each leg at each velocity of movement was used in analysis.

2.6 NEEDLE BIOPSY

Before and after training, 2 muscle biopsy samples (~80-100 mg) were obtained from the vastus lateralis under local anesthesia, using the Bergström needle biopsy technique (Bergström, 1962) and manual suction. The first sample was used for histochemical analysis and the second for biochemical analysis. All visible fat and connective tissue was dissected free from the biopsy sample. The sample for

histochemistry was mounted in cross-section on cork blocks with OCT (optimum cutting temperature) embedding medium with the aid of a microscope and immediately frozen in isopentane cooled in liquid nitrogen (-159° C). Samples were stored at -70° C until analysis. Before training, only one leg (the leg that would be trained) was sampled but, following the training period, samples were taken from both legs.

2.7 HISTOCHEMISTRY

Histochemistry is the most common method for fibre identification and uses myofibrillar (myosin) ATPase reactions to indicate the myosin heavy chain (MHC) isoform of the muscle fibre (Gauthier, 1979; Jostarndt et al., 1998; Staron and Pette, 1986). Histochemical techniques can be used to determine fibre type and fibre area. Myosin ATPase histochemical analysis is quick and effective and the technique is simply based on the difference in the sensitivity of a fibre's myosin ATPase activity, following exposure to different pH incubations (Brooke and Kaiser, 1970). Tissue samples were brought to -20° C and sectioned (7 μm) in series in a cryostat (Microtome HM 500 OM, Microm International) and mounted on glass slides. The sample was stained for myofibrillar adenosinetriphosphatase (mATPase) activity following separate pre-incubations of pH 4.3, 4.6, and 10.0 (Brooke and Kaiser, 1970). The pH 4.6 preparation enabled all three fibres types to be distinguished since, in this preparation, the type I fibres stain darkly, type IIa fibres stain lightly, and type IIb fibres stain intermediately (see figure 1). The stained slides were photographed (10x magnification, Spot camera, Diagnostics Instruments, Inc.) under a light microscope (Olympus BX60, Carsen Group,

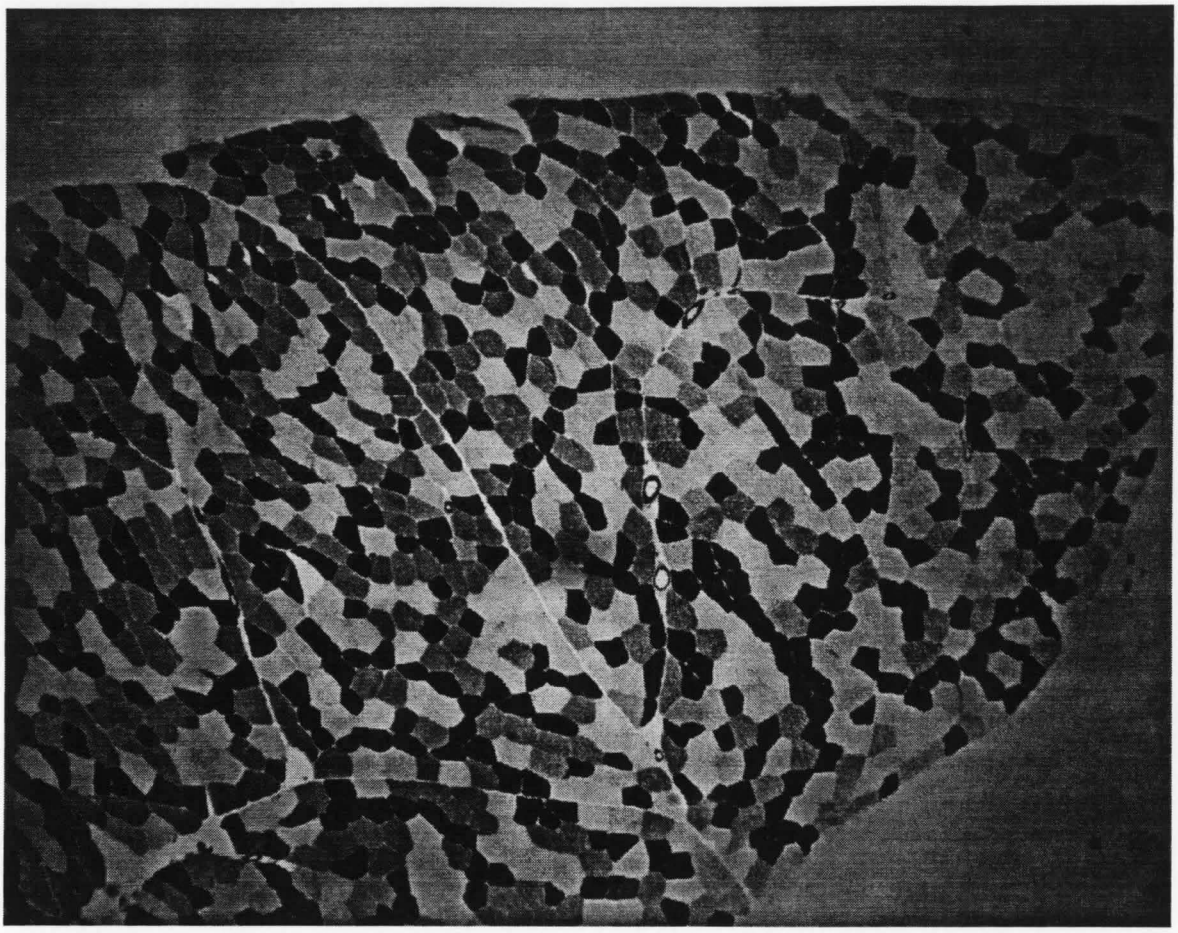


Figure 1. Needle biopsy of the vastus lateralis stained after pre-incubation at pH 4.6. The dark fibres represent type I, the light fibres represent IIa, and the Intermediate fibres are type IIb.

Inc., Markham, Ont.) for examination of fibre type. The stained slides were also photographed under a light microscope (40x magnification) and analyzed using computer software (Image Pro® Plus, Version 4.0 for Windows, Media Cybernetics) to determine mean single fibre cross-sectional area. Percent fibre type and fibre areas were determined from an average of 300 and 100 fibres per biopsy, respectively. Percent fibre area was calculated from % fibre type and the average area for that fibre type.

2.8 MYOSIN HEAVY CHAIN ISOFORM ANALYSIS

Myosin heavy chain measurement establishes the proportion of the specific MHC isoform in a skeletal muscle fibre and is a suitable marker of fibre type diversity (Pette, 1998). In humans there are three MHC isoforms, designated as slow (MHC-I) and fast (MHC-IIa and MHC-IIb) (Pette, 1998). (Recently it has become more common to use the term IIx, rather than IIb, to designate the fastest MHC isoform (Williamson et al., 2000). Consequently the classifications I, IIa and IIx are used in the present study).

Myosin heavy chain composition of the vastus lateralis was studied after electrophoretic separation of slow MHC-I, fast oxidative MHC-IIa, and fast glycolytic MHC-IIx isoforms from needle biopsies obtained at pre- and post-training in accordance with the methods of Carraro (1983). These analyses were conducted at the University of Waterloo in collaboration with Dr. H. J. Green.

2.9 CITRATE SYNTHASE ACTIVITY

Citrate synthase (CS) is a mitochondrial enzyme and a marker of the muscle fibre's oxidative capacity. A portion of each biopsy sample was used to determine CS

activity. First, the muscle was homogenized. Approximately 15-25 mg of muscle was weighed and the exact mass was recorded. The majority of 300 μL of homogenizing buffer was added to the muscle sample and the sample was pulverized on ice by grinding 50 times and the solution was then transferred to an Eppendorf tube. The remainder of the buffer was used to rinse the homogenizer to ensure all tissue and solution was transferred into the Eppendorf tube. The tube was then sonicated for 5 s and from this solution the protein concentration was determined using the Bradford assay for micro-samples. The Bio-rad protein assay is based on the method of Bradford (1976) and is a simple and accurate procedure for determining concentration of solubilized protein. The assay involves the addition of acidic dye to a protein solution, and subsequent measurement at 595 nm with a microplate reader (Microplate Manager® 4.0 Bio-Rad Laboratories, Inc.). Comparison to a standard curve provides a relative measurement of protein concentration.

Citrate synthase activity detection is based on this reaction:

Acetyl CoA + oxaloacetate + H_20 $\leftarrow\rightarrow$ Citrate + CoA-SH + H^+.

In a blank well, the following solutions are added in this order:

1 mL of Tris buffer (heated to 37°C in a water bath).

10 μL DTNB.

2 μL acetyl CoA.

10 μL muscle homogenate and mix (mix with cuvette mixing rods).

At this point, the UV spectrophotometer (Hewlett Packard 8453, Mississauga, Ont.) was zeroed and 10 μL of oxaloacetate were added to initiate the reaction. The absorbance of the reaction was read (RF-Mini 150, Recording Fluorometer, Mandel

Scientific Co. Ltd., Shimadzu Corp., Columbia, MD) and recorded in 30 s intervals over a 3 min period at 412 nm. The first minute was necessary for the reaction to initiate and the change in absorbance from min 1 to min 3 was used to calculate enzyme activity. The samples and other reagents were kept on ice until they were ready for use. Only 2-4 samples were thawed at a time to ensure enzyme activity was not lost. All samples were homogenized and measured on the same day. The enzyme activity was calculated and reported in micromoles per milligram of muscle (wet weight) per minute. The assay was performed in duplicate and the coefficient of variation was ± 1.44%.

Activity = (OD/0.0136) (dilution) (300µl/wet weight muscle) (mg/ml PRO) (60/100).

OD = (absorbance at 3 min-absorbance at 1 min) / 2 min

Dilution = dilution factor (vol. of Tris buffer/homogenate added) = (1 mL/10 µL)

300µl = volume of homogenate medium

wet weight muscle = wet weight of muscle sample

mg/ml PRO = protein concentration

Units: mol·kg Protein^{-1}·h^{-1}

2.10 CAPILLARY DENSITY

Since most of the capillaries that supply skeletal muscle run parallel to the muscle fibres, capillarization can be estimated from histochemically stained tissue mounted in cross-section. Samples were brought to -20° C and serially sectioned (7 µm) in series in a cryostat (Microtome HM 500 OM, Microm International). Slides were stained with hemotoxylin and eosin, photographed (20x magnification, Spot camera, Diagnostics Instruments, Inc.) and examined under the light microscope (Olympus BX60, Carsen

Group, Inc., Markham, Ont.). Capillaries were counted and expressed per square millimeter of tissue. Tissue area was calculated using computer software (Image Pro® Plus, Version 4.0 for Windows, Media Cybernetics). Repeated estimates of capillary density on the same sample were found to vary less than 2.2%.

CHAPTER III

THE EFFECT OF ENDURANCE TRAINING ON MUSCLE STRENGTH AND POWER

(A paper to be submitted for publication)

INTRODUCTION

In many team sports, athletes require high levels of both aerobic fitness and "explosive" muscle power. Endurance training can enhance aerobic fitness, but it is possible that some of the adaptations to this mode of training may have a negative effect on the muscle's ability to generate power. Some investigators believe that endurance training reduces strength and power (Kraemer et al., 1995), or that it impairs power development when endurance training and strength training are performed concurrently (Hickson, 1980). The objective of athletes and coaches is to maximize the development of each attribute without compromising performance in the other.

Endurance training is known to increase maximal aerobic power (Andersen and Henriksson, 1977a; Dudley and Djamil, 1985; Gollnick et al., 1973a; Hickson, 1980; Howald et al., 1985; Kraemer et al., 1995) and may also alter fibre type composition (Andersen and Hendriksson, 1977a; Howald et al., 1985). A transformation of type IIb motor units to slower contracting units (type IIa or I) would enhance their oxidative capacity (Jansson and Kaijser, 1977), but may compromise maximal contraction velocity. Endurance training may increase (Saltin et al., 1973) or decrease (Gollnick et al., 1973a) fibre cross-sectional area. Although a smaller fibre area could also promote a greater oxidative capacity because of its decreased diffusion distance for oxygen, energy substrates and metabolites, it would not be capable of contracting as forcefully.

Muscle fibre types and their contractile properties are determined by the fibre's myosin heavy chain (MHC) isoform. The MHC isoform correlates with fibre shortening velocity (Edman et al., 1988) and therefore determines the expression of contractile properties. Myosin heavy chain composition has been shown to change in response to low-frequency electrical stimulation (Pette et al., 1976), resistance training (Staron et al., 1994) and endurance training (Andersen and Schiaffino, 1997). Such changes could be important to the athlete because, ultimately, they might alter the expression of force at the muscle level. For example, an athlete concerned with explosive activities (such as a jumper or sprinter) could find the loss of type IIb fibres detrimental to performance. The type IIb fibres have a greater cross-sectional area (Esbjörnsson et al., 1999; Simoneau and Bouchard, 1989) and a 10-fold faster rate of shortening velocity than type I fibres and a 3-fold faster rate of shortening velocity than type IIa fibres (Schluter and Fitts, 1994). Theoretically these attributes would permit type IIb fibres to have a greater power output.

Anecdotal reports from athletes and cross-sectional comparisons of cross-country runners with sedentary controls (Costill, 1967) suggest that excessive endurance training might have a negative effect on the ability to perform rapid, powerful movements. Although many investigators have examined the potential inhibitory effect of concurrent strength and endurance training, no study, to date, has isolated the effects of endurance training alone on the ability of the muscle to perform explosive forceful contractions. Consequently, the purpose of this study was to determine whether or not several months of endurance training might reduce strength and power in the trained muscle group and, if so, to examine the possible mechanisms. It was hypothesized in the current study that mechanisms by which such training might impair muscle power might include a

reduction in fibre area and/or a transformation in fibre type towards slower twitch units. We chose a unilateral training model, whereby our subjects trained only one leg, so that the opposite leg could act as a control for any changes in physical activity pattern (other than endurance training). The duration of the training program (10 weeks) and volume of training (progressing to 60 min per day, 5 days per week) was selected in order to exceed the upper limit of that normally performed by athletes who compete in sports that have both a high endurance and power component. The rationale for this was that, if it were found that the intervention had no inhibitory effect on strength and/or power, the issue could be considered a lesser concern for such athletes.

METHODS

Subjects. Ten healthy males were recruited (age 21.4 ± 0.3 yr, body mass 76.8 ± 2.2 kg) to perform a 10 week period of high-volume endurance training. Each subject had one leg randomly assigned to the training condition, such that 5 subjects trained their dominant leg and 5 subjects trained their non-dominant leg. The dominant leg was defined as the leg that was favoured in a vertical jump with a 1-foot take-off. All subjects were physically active, but untrained and had not been involved in a resistance-training or endurance-training program in the previous 6 months. Subjects continued their regular activity patterns throughout the duration of the study and were instructed not to perform any additional structured exercise outside of the training sessions. They were provided remuneration for completing the study and any missed training sessions were made up in following weeks or at the end of the training period, prior to testing, to ensure 100% compliance. The purpose of the experiment and possible risks were explained to each

subject prior to obtaining written consent and the study was performed in accordance with the Human Ethics Committee of McMaster University (see Appendix C).

Pre-testing. Each subject was tested for electrically-stimulated twitch characteristics of the quadriceps, leg $\dot{V}O_{2peak}$ and lactate threshold (T_{lac}) using progressive exercise tests, vertical jump height with a 1-foot take-off, and maximal leg press strength at slow- and high-velocities. Each test was performed on each leg, in random order, with at least 6 hours of rest prior to testing for leg $\dot{V}O_{2peak}$ or T_{lac} of the opposite leg. A standardized rest period (24 hours) was provided between tests on the same leg to control for any residual fatigue. All testing equipment was calibrated to ensure valid and reliable measures. After all performance tests, and on a separate day, a needle biopsy sample of the vastus lateralis was obtained from the designated training leg for measurements of fibre-type proportions, fibre area, oxidative enzyme activity, and capillary density.

Testing order. Testing order was randomized according to leg-dominance.

Test 1 – Muscle twitch characteristics (both legs)

Test 2 – Single-leg $\dot{V}O_{2peak}$ (minimum of 24 hours after Test 1)

Test 3 – Single-leg $\dot{V}O_{2peak}$ (minimum of 6 hours after Test 2)

Test 4 – T_{lac} (minimum of 24 hours after Test 3)

Test 5 – T_{lac} (minimum of 6 hours after Test 4)

Test 6 – Strength and power (minimum of 24 hours after Test 5)

Test 7 – Muscle biopsy (minimum of 24 hours after Test 6)

Training. Each subject performed unilateral cycle ergometry for 10 weeks such that one leg trained and the other served as a control. The cycle ergometers (Monark) were modified so that the subject could pedal in a recumbent position. Subjects cycled at 60 RPM (in cadence with a metronome) at a power output that initially corresponded to 75% of the pre-training trained maximum power output. Exercise intensity was adjusted to either meet this intensity or to keep the subject's heart rate between 140 and 160 beats per minutes for the duration of the training session. All training sessions were performed under supervision of an investigator. Each training session was initiated by a 2 min warm-up (this time was included in the recorded exercise duration). Training in week 1 consisted of 3 sessions of 30 min and the following weeks progressed as follows:

Week 2 = 4 d * 30 min

Week 3 = 4 d * 35 min

Week 4 = 4 d * 40 min

Week 5 = 5 d * 40 min

Week 6 = 5 d * 45 min

Week 7 = 5 d * 50 min

Week 8 = 5 d * 55 min

Weeks 9 and 10 = 5 d * 60 min

Post-testing. All pre-training tests were repeated in the same order as the pre-training schedule. Post-testing was delayed for 48 hours after the last training session to permit a standard recovery period. After all performance tests were complete, resting biopsies were obtained from the trained and control legs.

PERFORMANCE MEASUREMENTS

Evoked twitch. Twitch contractions of the knee extensors (quadriceps femoris) were evoked by indirect percutaneous nerve stimulation as detailed in chapter II (p. 42-44) and described by Hamada et al. (2000). Twitch measurements included peak torque, time-to-peak torque, rise time (10% to 90% peak torque), and half-relaxation time.

Single-leg $\dot{V}O_{2peak}$. Single-leg $\dot{V}O_{2peak}$ was determined for each leg by an incremental cycle ergometry test to exhaustion. Power outputs were increased every 2 minutes and $\dot{V}O_2$ was continuously recorded by open circuitry spirometry. The highest value (averaged over 60 s) was considered to be the subject's single-leg $\dot{V}O_{2peak}$. Further details are provided in chapter II (p. 44).

Lactate threshold (T_{lac}). Subjects returned to the lab to have their lactate threshold determined after a minimum of 24 hours following their same-leg $\dot{V}O_{2peak}$ test. The workload at which T_{lac} was expected to occur was estimated from the ventilatory threshold of the $\dot{V}O_{2peak}$ test (Chapter II, p. 44-46). The initial workload was set 20 watts below this and subjects performed 3 min bouts separated by 2 min of recovery, during which a fingertip blood sample was taken for immediate analysis of blood lactate. The power output of each successive stage was increased by 10 watts until the investigators were satisfied that the T_{lac} had been attained.

Vertical jump. Subjects performed a vertical jump from an in-floor mounted force platform with a 1-foot takeoff to determine muscle power. They performed 3 jumps with each leg and had their best jump used in final analysis (see Chapter II, p. 46). Measurements included take-off velocity, maximum power, peak force and impulse.

Strength. Unilateral voluntary isokinetic strength was measured with a Cybex dynamometer (Lumex, Ronkonkoma, NY) modified to measure low and high velocity leg press strength (Vandervoort et al., 1984). Each subject performed 3 trials at a slow speed (60°/s) and then 3 trials at a fast speed (300°/s) with a 30 s rest between attempts. All trials for one leg were completed prior to repeating the tests for the remaining leg. The highest torque recorded for each leg at each velocity of movement was used in analysis (see Chapter II, p. 47).

TISSUE MEASUREMENTS

Needle biopsy. Before and after training, 2 muscle biopsy samples (~80-100 mg) were obtained from the vastus lateralis under local anesthesia using the Bergström needle biopsy technique (Bergström, 1962) and manual suction. Before training, only one leg (the leg that would be trained) was sampled but, following the training period, samples were taken from both legs (see Chapter II, p. 47).

Histochemistry. The muscle sample was stained for myofibrillar adenosinetriphosphatase (mATPase) activity following separate pre-incubations of pH 4.3, 4.6, and 10.0 (Brooke and Kaiser, 1970). The stained slides were photographed (10x

magnification, Spot camera, Diagnostics Instruments, Inc.) under a light microscope (Olympus BX60, Carsen Group, Inc., Markham, Ont.) for fibre type determination and to measure mean single fibre cross-sectional area with computer software (Image Pro® Plus, Version 4.0 for Windows, Media Cybernetics, see Chapter II, p. 48-50). Fibre type and fibre areas were determined from an average of 300 and 100 fibres per biopsy, respectively. Percent fibre area was calculated from % fibre type and the average area for that fibre type.

Myosin heavy chains. Myosin heavy chain composition of the vastus lateralis was studied after electrophoretic separation of slow MHC-I, fast oxidative MHC-IIa, and fast glycolytic MHC-IIb isoforms from needle biopsies obtained at pre- and post-training (see Chapter II, p. 50).

Citrate synthase activity. A portion of each biopsy sample was used to determine citrate synthase activity. First, the muscle sample was homogenized and protein concentration was determined by Bradford technique. Citrate synthase activity was then determined using a UV spectrophotometer (Hewlett Packard 8453, Mississauga, Ont.) and recording fluorometer (RF-Mini 150, Recording Fluorometer, Mandel Scientific Co. Ltd., Shimadzu Corp., Columbia, MD). The enzyme activity was calculated and reported in micromoles per milligram of muscle (wet weight) per minute (see Chapter II, p. 50-52).

Capillary density. Slides were stained with hemotoxylin and eosin, photographed (20x magnification, Spot camera, Diagnostics Instruments, Inc.) and examined under the light microscope (Olympus BX60, Carsen Group, Inc., Markham, Ont.). Capillaries were counted and expressed per square millimeter of tissue (see Chapter II, p. 52). Tissue area was calculated using computer software (Image Pro® Plus, Version 4.0 for Windows, Media Cybernetics).

STATISTICAL ANALYSIS

A two factor (leg x time) repeated measures within-subject analysis of variance (ANOVA) was used to determine significant differences between the trained and untrained legs for single-leg $\dot{V}O_{2peak}$, lactate threshold, vertical jump, and strength before and after training. An additional one factor repeated measures within-subject ANOVA was used to determine significant differences within legs over time for single-leg $\dot{V}O_{2peak}$. A one factor repeated measures within-subject ANOVA was used to determine significant differences between the pre-training trained-leg, post-training control-leg, and post-training trained-leg for histochemical fibre composition, myosin heavy chain isoform composition, capillary density, and citrate synthase activity. When main-effects were found, a Tukey's HSD post-hoc test was applied to determine pair-wise differences. The level of significance was set at $p<0.05$. All statistical analysis was performed with Statistica© V. 5.0 software (StatSoft Inc., Tulsa, OK) for Windows. Values are expressed as means ± SE.

RESULTS

Subject data. Subject descriptive data is presented in Table 1. There was no change in body mass from pre- to post-training.

PERFORMANCE MEASUREMENTS

Evoked muscle twitch characteristics (figure 1). There was a main effect for time ($p<0.05$) since peak twitch torque was significantly greater post-training in both legs combined, but there was no interaction between time and leg. There was no change in time-to-peak torque, rise time, or half-relaxation time, before and after training or between legs.

Single-leg $\dot{V}O_{2peak}$ (figure 2). There was a significant ($p<0.05$) main effect for time for leg $\dot{V}O_{2peak}$, but no interaction between legs, following training. The changes in $\dot{V}O_{2peak}$ were 7% in the trained leg and 3% in the untrained leg. The lack of interaction may have been due to the relatively large differences between the dominant and non-dominant legs of several individuals (see raw data in Appendix A). When data were re-examined with a separate 1-way ANOVA for each leg, the increase in the trained leg was significant ($p<0.05$), but that in the untrained leg was not ($p>0.05$). The maximal power output achieved also increased (16%) significantly in the trained leg (165 ± 8.2 W vs. 192.5 ± 9.44 W, $p<0.01$), but not in the untrained leg (165 ± 8.2 W vs. 175.5 ± 9.98 W, $p>0.05$).

Table 1: Subject Descriptive Data

Subject	**Age (y)**	**Height (cm)**	**Pre-training Mass (kg)**	**Post-training Mass (kg)**	**Dominant Leg**	**Training Leg**
1	22	187.5	81.5	82.5	R	R
2	21	187.5	72.0	69.8	L	R
3	22	182.5	75.4	76.2	L	L
4	21	180.0	78.0	76.0	L	R
5	21	175.0	75.0	74.5	R	R
6	20	160.0	67.3	67.0	L	R
7	22	172.5	84.2	85.5	L	L
8	22	180.0	85.0	85.5	R	L
9	20	170.0	67.9	69.8	R	R
10	23	177.5	81.8	84.0	R	L
AVE.	**21.4 ± 0.3**	**177.3 ± 2.7**	**76.8 ± 2.0**	**77.1 ± 2.2**		

Results are mean ± SEM.

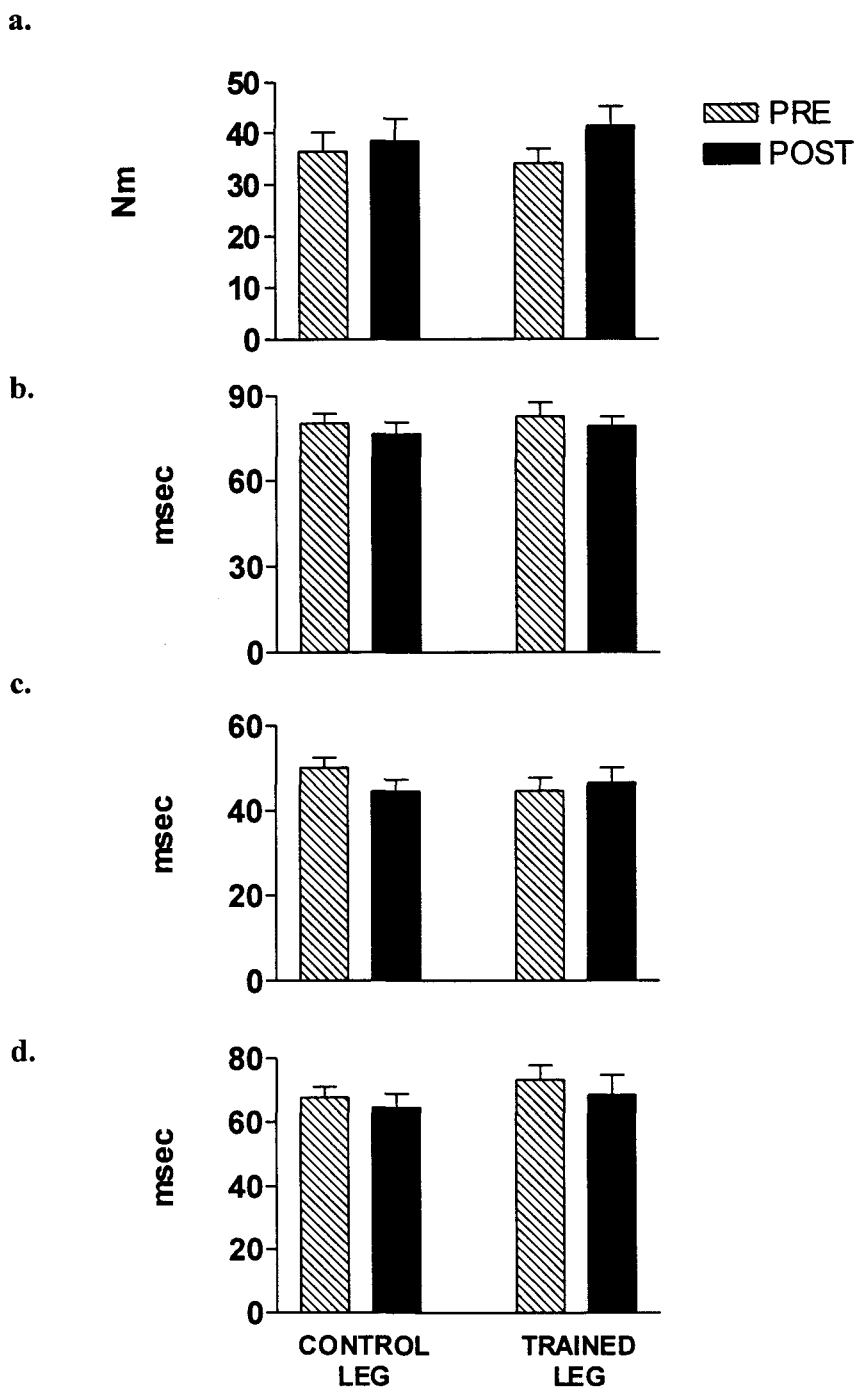
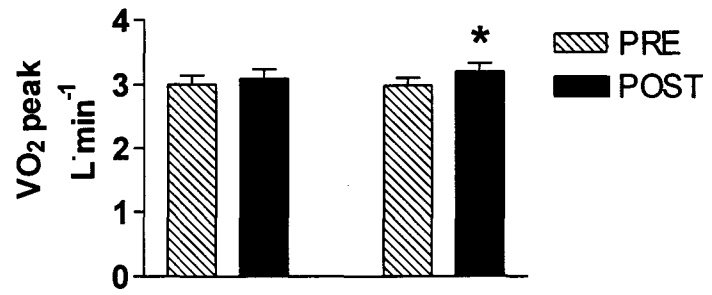
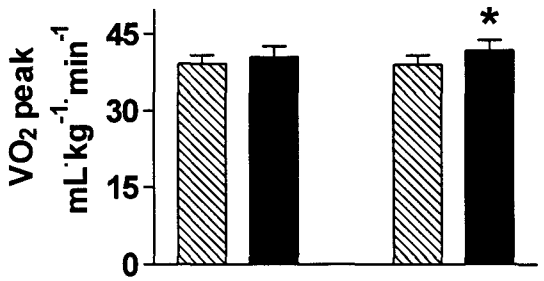


Figure 1. Evoked twitch results for the control and trained leg. (a) Peak torque (b) Time-to-peak torque (c) Rise time (d) Half-relaxation time (mean $\pm$ SE)

a.



b.



c.

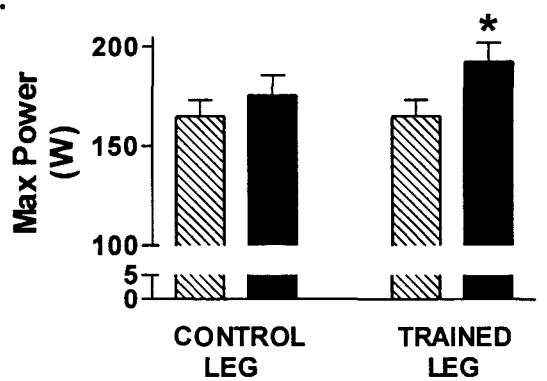


Figure 2. Maximal aerobic power of the control and trained leg.

(a) Absolute $\dot{V}O_2$ peak (b) Relative $\dot{V}O_2$ peak (c) Maximal power output

(mean $\pm$ SE) *significantly greater than pre-training value ($p<0.05$)

Lactate threshold (figure 3). Following training, there was a shift in blood lactate concentration ([lactate]) for the trained leg, such that at a given sub-maximal workload, [lactate] was lower compared to pre-training, or to the untrained leg. Following training, the blood lactate in the trained leg was significantly lower (3.09 ± 0.57mM vs. 5.14 ± 0.88mM, $p<0.05$) on the completion of exercise at the highest workload completed in the pre-training test. For example, if a subject's final workload in the pre-training test was 100 watts, the corresponding lactate accumulation at that workload was compared to the lactate response at 100 watts after training. A significant decrease was also observed in blood lactate accumulation for the untrained leg following the training period (4.45 ± 1.03mM vs. 5.46 ± 0.66mM, $p<0.05$) but this decrease was significantly less than that for the trained leg.

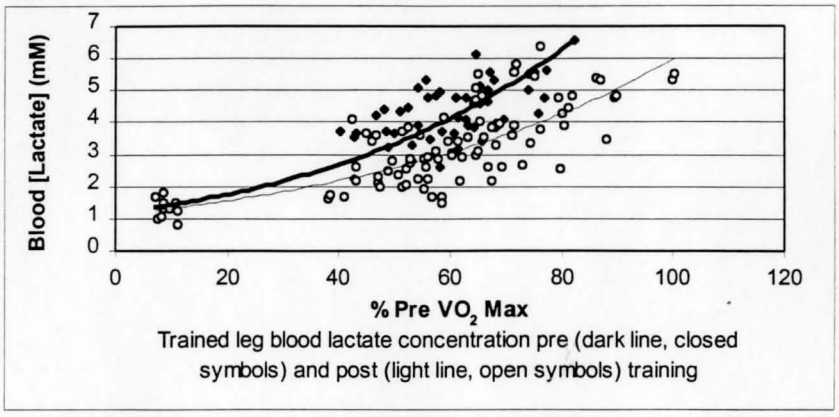
Strength (figure 4). Low- and high-speed strength did not change in either leg after training.

Vertical jump power (figure 5). There was a trend toward a decrease in take-off velocity (8%) in the trained leg but this was not significant ($p>0.05$). Maximum positive power also tended to decrease by 8% in the trained leg but this was also not significant ($p>0.05$). There were no changes in peak force or impulse over time or between legs.

TISSUE MEASUREMENTS

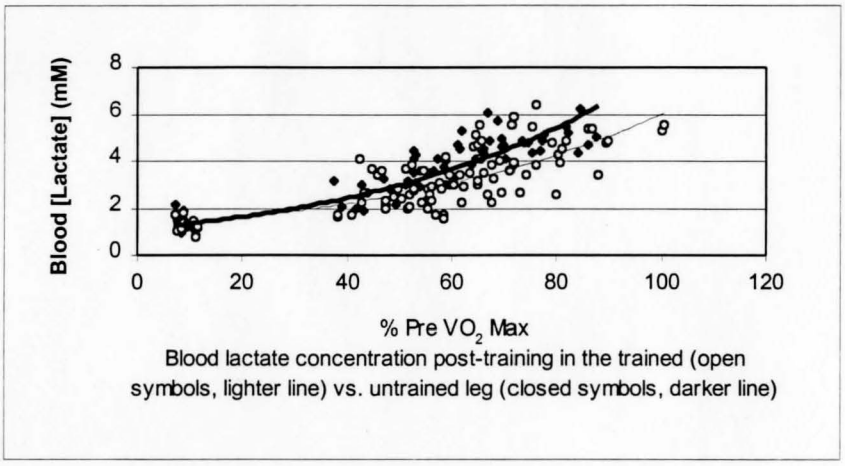
Fibre type (figure 6a). In the trained leg, the percentage of type I fibres was identical following training (47.9 ± 1.3 %) to that before training (47.5 ± 1.9 %). There

a.



Trained leg blood lactate concentration pre (dark line, closed symbols) and post (light line, open symbols) training

b.



Blood lactate concentration post-training in the trained (open symbols, lighter line) vs. untrained leg (closed symbols, darker line)

c.

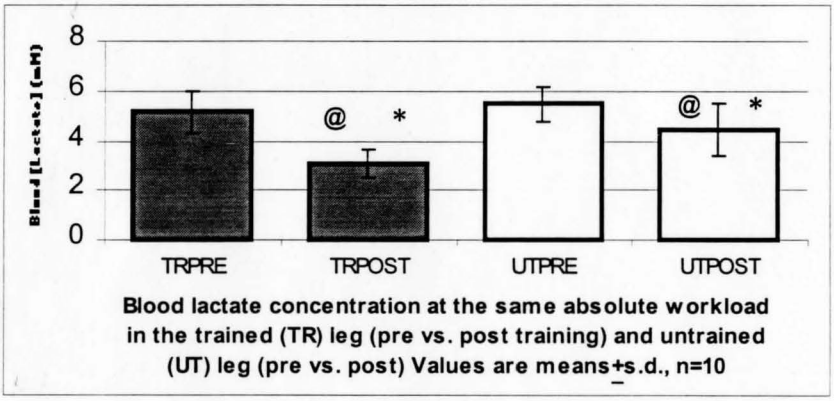
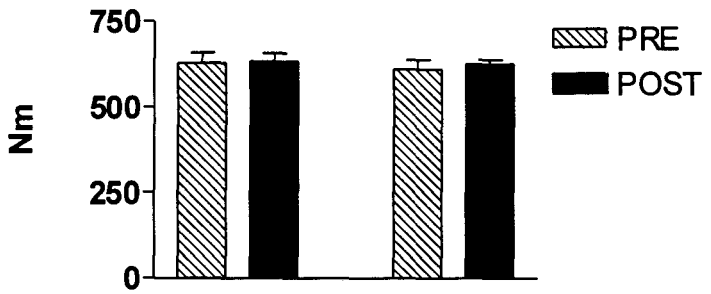


Figure 3. *significantly lower than pre-training value ($p<0.05$)

@decrease in lactate significantly greater for trained leg than for untrained leg ($p<0.05$)

a.



b.

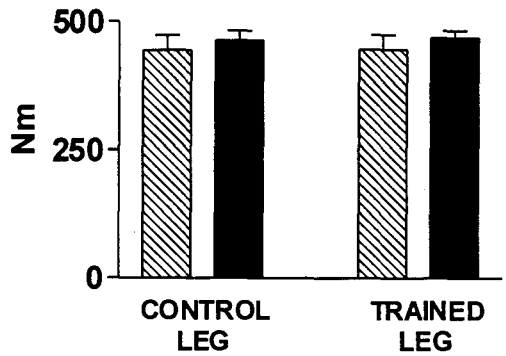


Figure 4. Low and high speed strength of the control and trained legs.

(a) 60° per second (b) 300° per second

(mean ± SE)

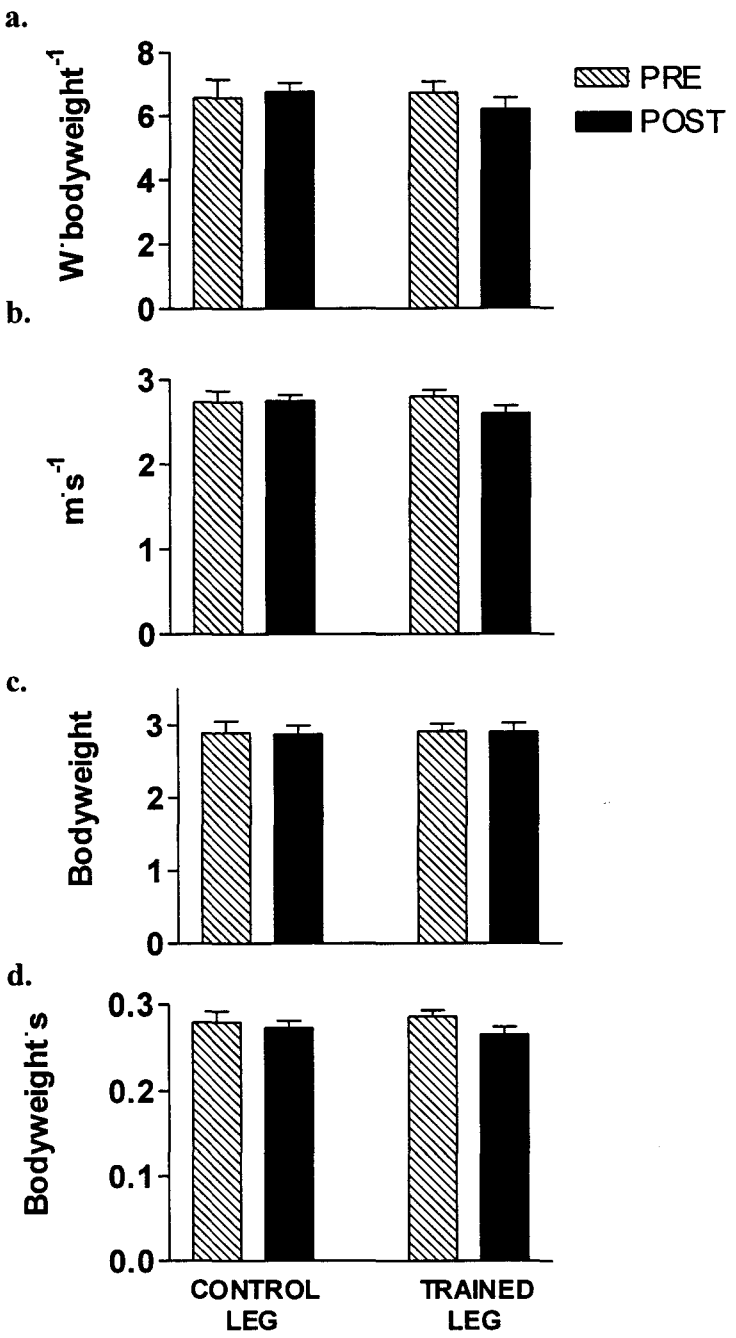


Figure 5. Vertical jump measurements of the control and trained legs.

(a) Maximum positive power (b) Take-off velocity

(c) Peak force (d) Impulse (mean ± SE)

was, however, a significant ($p<0.05$) reduction in the percentage of type IIb fibres (from 20.9 to 15.9 %) and a significant increase in the percentage of IIa fibres (from 31.6 to 36.2%) as a result of the training.

A biopsy sample was not taken from the control leg before the training period, but the post-training control sample surprisingly showed a significantly lower percentage of type I fibres than that in the trained leg before the training period (41.2 ± 1.4 % vs. 47.5 ± 1.9 %). In addition, the percentage of IIa fibres in the control leg was significantly greater than in the pre-training sample for the trained leg (36.8 ± 1.0 % vs. 31.6 ± 1.5 %). The percentage of type IIb fibres in the control leg was also significantly higher than in the trained leg following training (22.0 ± 1.1% vs. 15.9 ± 1.3 %).

Mean fibre area (figure 6b). There were no significant differences between legs or measurement times for mean fibre area, although there was a trend for the post-training type I fibre area to be greater than the control value ($p=0.08$). As well, there was a trend for the post-training type IIa fibres to be greater than the pre-training and control values ($p=0.05$). There were no significant changes in type IIb fibre area.

Percent fibre area (figure 6c). In the trained leg, type I percent fibre area was identical following training (41.9 ± 1.2 %) to that before training (41.5 ± 2.4 %). There was, however, a significant ($p<0.05$) increase in type IIa percent area (from 36.9 % to 42.6 %) and a significant ($p<0.05$) decrease in type IIb percent area (from 21.6 % to 15.5 %) as a result of training.

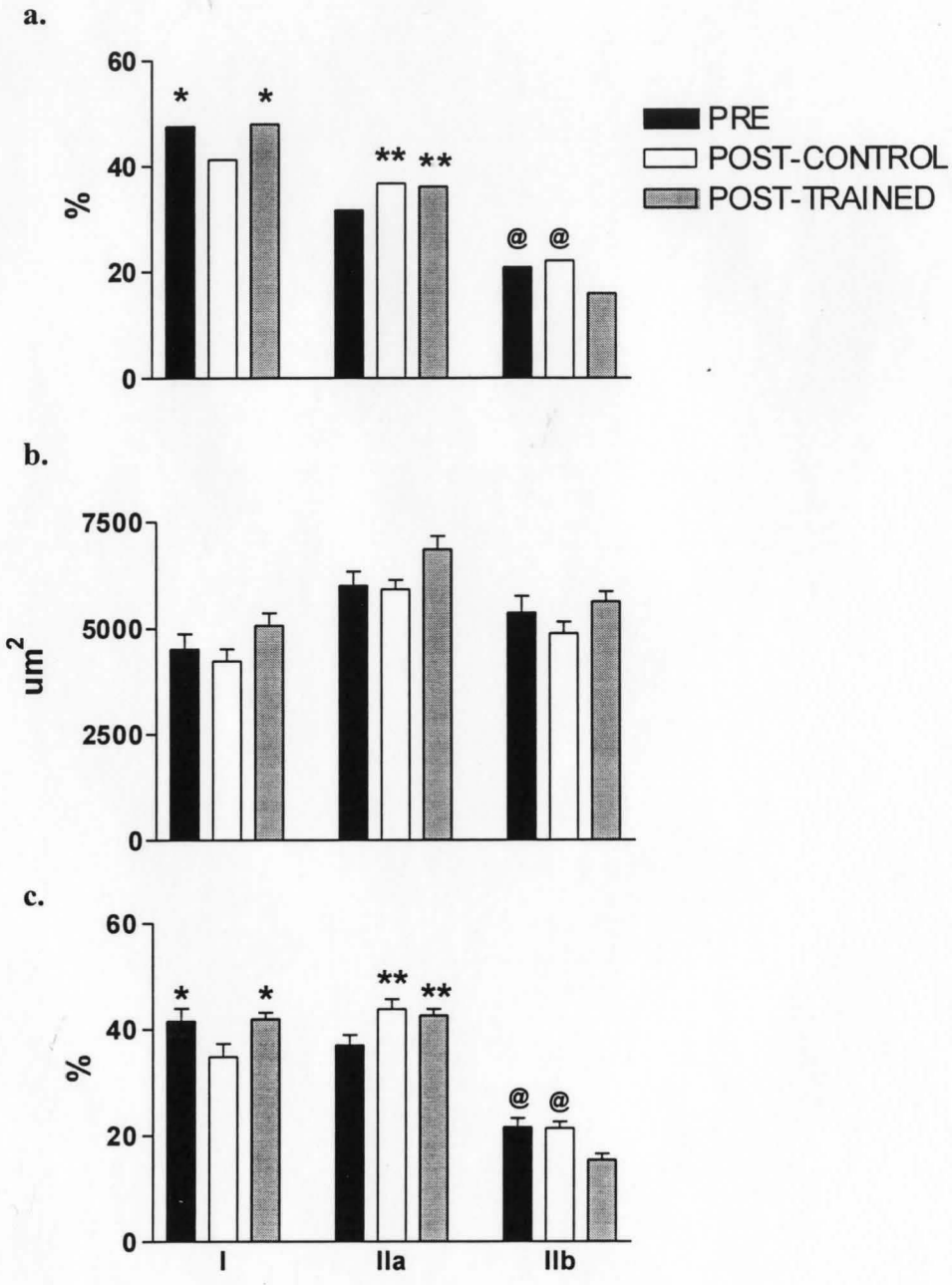


Figure 6. Tissue measurements of the control and trained leg.

(a) Percent fibre type (b) Fibre area (c) Percent fibre area (mean $\pm$ SE)

*significantly greater than POST-CONTROL, $p<0.05$.

**significantly greater than PRE, $p<0.05$.

@significantly greater than POST-TRAINED, $p<0.05$.

The post-training sample of the control leg surprisingly showed a significantly lower type I percent area than that in the trained leg before the training period (34.9 ± 2.4 % vs. 41.5 ± 2.4 %). In addition, type IIa percent area of the control sample was significantly greater than in the pre-training sample for the trained leg (43.8 ± 1.9 % vs. 36.9 ± 1.9 %). The type IIb percent area in the control leg was significantly higher than in the trained leg following training (21.4 ± 1.2 % vs. 15.5 ± 1.1 %).

Citrate synthase activity (figure 7). There was no difference in the trained leg over time. The control leg had a significantly lower citrate synthase activity in comparison to trained leg at pre-training (3.88 ± 0.41 $mol \cdot kg^{-1} \cdot h^{-1}$ vs. 4.78 ± 0.37 $mol \cdot kg^{-1} \cdot h^{-1}$, $p<0.05$) and post-training (3.88 ± 0.41 $mol \cdot kg^{-1} \cdot h^{-1}$ vs. 5.11 ± 0.53 $mol \cdot kg^{-1} \cdot h^{-1}$, $p<0.05$).

Capillary density (figure 8). There were no differences between legs or changes over time for capillary density.

DISCUSSION

As indicated by the increase in single-leg $\dot{V}O_{2peak}$, lactate threshold, and % type IIa fibres in the trained leg, it is apparent that the 10 week training program had a significant aerobic training effect. In spite of this, and contrary to the original hypothesis, there was no decline in strength or power as result of the training.

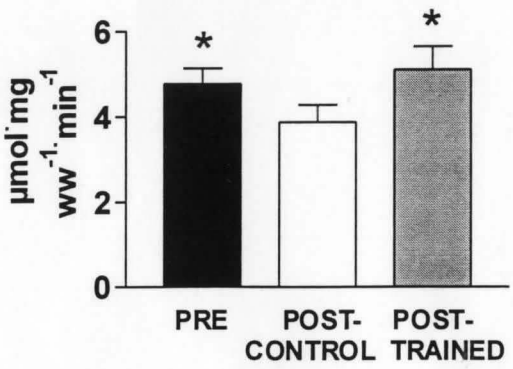


Figure 7. Citrate synthase activity of the control and trained leg (mean ± SE)

*significantly greater than post control, $p<0.05$.

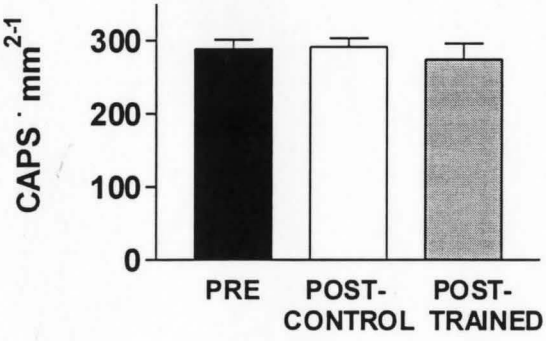


Figure 8. Capillary density of the control and trained leg (mean ± SE)

Single-leg $\dot{V}O_{2peak}$. The observed increases in leg $\dot{V}O_{2peak}$ are within the ranges reported in previous literature (Andersen and Henriksson, 1977a; Dudley and Djamil, 1985; Gollnick et al., 1973a; Hickson, 1980; Howald et al., 1985; Ingjer, 1978b; Kraemer et al., 1995). However, the improvements are less than those observed following a shorter unilateral endurance training program (Saltin et al., 1976). In that study, the investigators observed an increase in $\dot{V}O_{2peak}$ of 20% and 6% in the trained and untrained legs respectively compared to the 7% and 3% in the present study. The slight increase in the $\dot{V}O_{2peak}$ of the untrained leg was probably due to cardiovascular adaptations (increased muscle blood flow) whereas the greater increase in the trained leg would have been due to a combination of cardiovascular adaptations and adaptations within the muscle.

Lactate threshold. In the trained leg, there was a shift in the blood lactate accumulation such that at a given sub-maximal $\dot{V}O_2$, it was lower post-training in comparison to pre-training, as well as being lower than that for the untrained leg. This shift was likely the result of peripheral adaptations, such as a decrease in lactate production, or an increase in lactate uptake by other fibres within the muscle (Donovan and Brooks, 1983). Although the blood lactate concentration was reduced to a greater extent in the trained leg after training compared to the untrained leg, a minor shift in the blood lactate response was also observed at the same absolute submaximal intensity in the untrained leg. Since no training stimulus was applied to the control leg, this effect may have been due to an enhanced lactate removal rate (in liver or perhaps other inactive muscle fibres) but any differences between the trained and untrained legs can be attributed to adaptations within the muscle itself.

Citrate synthase. In the trained leg, there was no change in citrate synthase activity over time. This was somewhat surprising, since single leg training studies using lower training volumes have resulted in major increases in activity of this enzyme (Melissa et al., 1997; Sale et al., 1990). An increase in the mitochondrial density of a trained muscle will increase its oxidative capacity (Howald et al., 1985), and should increase citrate synthase activity as well. However, the potential for the mitochondrial enzymes to increase after endurance training might also be impaired if accompanied by hypertrophy of the muscle fibre (Stone et al., 1996). The slight hypertrophy that occurred in the subjects of this study may explain the lack of change in enzyme activity within the trained leg. The differences between the control and pre-training values were unexpected.

Capillary density. Capillary density did not increase after the 10 weeks of endurance training. Endurance training is typically considered a powerful stimulus for increasing capillary density (Andersen and Henriksson, 1977b; Ingjer, 1978a) although Bell et al. (2000) found an increase in capillary density only after a program of strength and endurance training, and not after endurance training alone. Thus, increased capillarization is not a universal adaptation to endurance training. Fibre hypertrophy could result in a reduction in capillary density (MacDougall et al., 1986) because there would be a greater fibre area and a smaller number of capillaries in a given area. Therefore the lack of capillarization in the present study may also have been due to the slight increases observed in cross sectional area of the type I and IIa fibres.

Muscle twitch characteristics. The slight increase in peak twitch torque observed in the trained leg would contribute to greater force production by the muscle and may be associated with the slight increases in fibre size. Elite distance runners have been shown to produce less peak force and to have significantly smaller fibre areas in comparison to age-matched sedentary controls (Widrick et al., 1996a; 1996b). Muscle force is proportional to muscle cross-sectional area (Widrick et al., 1996a) and peak twitch force is influenced by the amount of contractile protein in the fibre (Keen et al., 1994). Hypertrophy could be an important compensatory mechanism to maintain muscle power output, if in fact endurance training affects the velocity of shortening. However, there were no changes in the force/time characteristics of the twitch. The inability of training to affect the % of type I fibres likely had the greatest impact on the maintenance of the muscle's force/time characteristics. Although the type IIb fibres have the fastest velocity of shortening (Schluter and Fitts, 1994), it is likely that a significant increase in type I fibre number is necessary for the muscle to display slower contractile properties. As well, Widrick et al. (1996a) have shown that long-term endurance training is associated with an increase in the velocity of shortening of type I fibres.

Vertical jump power. Power is dependent on muscle force and velocity of shortening. Muscle force did not decrease as reflected in the maintenance of strength over the training period. This may have been due to the slight increase in cross sectional area of the type I and IIa fibres. A decrease in type IIb fibre number could impair take-off velocity and vertical jump performance, since the type IIb fibres have been shown to have a 10- and 3-fold greater rate of shortening velocity than the type I fibres and type IIa

fibres, respectively (Schluter and Fitts, 1994). Also, Fitts et al. (1991) positively correlated peak power with the percentage of type II fibres, showing the importance of the type II contractile properties for power production. However, there was no decrease in the overall proportion of type II fibres, suggesting that the type IIa fibre contractile properties are sufficient for the maintenance of power. The slight increase in fibre size, and therefore a possible increase in force production capacity, may negate the slight decrease in type IIb contractile properties since fibre diameter has been indicated by Widrick et al. (1996a) to be an important factor in determining power production.

Fibre type and fibre area. Short-term, high-intensity endurance training has been shown to be an adequate stimulus for inducing changes in protein expression of the contractile apparatus in humans (Baumann et al., 1987). Post-training, there was no change in type I fibre number, but there was a significant increase in type IIa and decrease in type IIb fibre numbers. These results agree with the previous literature that suggests type II to type I fibre conversion is minimal and that the type IIb fibre is the most volatile. Within the type II subgroups, the transition of type IIb to type IIa fibres is common, but there is little evidence for a shift of type II to type I fibres (Gollnick et al., 1973a). The type IIb fibre is the most adaptable fibre type in response to contractile activity and sedentary populations have a high composition of type IIb fibres (Klitgaard et al., 1990). It is to be expected then, that a period of increased contractile activity, regardless of intensity, would result in a significant shift of type IIb to IIa fibres. Endurance training, performed for at least 6 weeks, results in sufficient contractile

activity to reduce the proportion of type IIb fibres (Howald et al., 1985) and increase the number of type IIa fibres (Andersen and Hendriksson, 1977a).

Following training, there appeared to be a slight, but non-significant, increase in fibre area of both the type I and IIa fibres. The response of muscle fibre area to endurance training is unclear as shown by the variable responses in longitudinal and cross-sectional studies, as well as between human and animal data. Fibre area may increase (Andersen and Henriksson, 1977b; Klausen et al., 1981), or decrease (Jansson and Kaijser, 1977; Terrados et al., 1986; Widrick et al., 1996a), or not change (Constable et al., 1980; Fitts et al., 1989; Schluter and Fitts, 1994). Theoretically, a smaller fibre area would be of greater benefit to endurance performance because it would result in a shorter diffusion distance for oxygen into the mitochondria and a greater capillary density for greater oxygen delivery. Whether the mode of exercise is a determinant of the response to training is also unclear. In a comparison of runners and cyclists, Tesch et al. (1984) found a greater FT/ST fibre area ratio in cyclists; however, in studies utilizing cycle ergometry as the training mode, Saltin et al. (1976) found an increase in fibre area, but Bell et al. (2000) did not.

Percent fibre area is a reflection of both fibre type and fibre area. A significant change in either variable may cause the percent fibre area to change. A change in percent fibre area could greatly modify the force characteristics of the muscle to a much greater extent than changes in fibre type or fibre proportion alone. The results show that there was no change in percent fibre area of type I fibres, but there was an increase and a decrease of type IIa and IIb percent fibre areas respectively, of the trained leg after training in comparison to the pre-training value. These changes correspond to the slightly

greater increase in type IIa fibre size and the significant decrease in type IIb fibre number. The changes in percent fibre area would indicate that the muscle may have become slightly slower, since the type IIb fibre has the fastest velocity of shortening (Schluter and Fitts, 1994), however the changes did not appear to affect the outcome of the strength or power measurements.

CONCLUSIONS

The results of this study indicate that a 10 week program of endurance training does not impair muscle power in untrained subjects. It is possible that the small reduction in type IIb percent fibre area was offset by the increase in IIa percent fibre area, such that overall strength and power were unaffected. Previously, Bauer (unpublished results) showed that a type IIb to type IIa fibre transformation might not be detrimental to power performance. The versatile nature of skeletal muscle allows it to be modified in response to altered functional requirements (Baumann et al., 1987; Trappe et al., 1995) by adapting the molecular composition of individual muscle fibres (Baumann et al., 1987). However, the magnitude of these changes may need to be greater in order to cause a significant change in performance variables. Therefore, a 10 week program of endurance training can improve aerobic performance variables without compromising strength and power. The possibility that a longer training period or a greater volume of training might have compromised muscle power as suggested by measurements in elite distance runners (Widrick et al., 1996a; 1996b) cannot be dismissed. However, the results indicate that short off-season programs of endurance training can be prescribed with little negative effect for strength and power athletes.

The above finding has important implications for athletes who compete in sports that require high velocity, explosive movements and especially team-sport athletes. In such sports a high level of aerobic fitness is also beneficial during phases at submaximal tempo and for accelerating recovery of muscle phosphagen stores and lactate removal during recovery from high intensity bursts of activity. The present data indicate that such athletes can still "reap the benefits" of aerobic training without compromising their ability to perform explosive maximal intensity efforts.

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APPENDIX A

RAW DATA TABLES

EVOKED TWITCH MEASUREMENTS

PEAK TORQUE (Nm)

	CONTROL LEG		TRAINED LEG	
SUBJECT	**PRE**	**POST**	**PRE**	**POST**
1	21.52	24.51	24.05	26.12
2	25.43	29.58	25.20	32.57
3	34.64	34.64	36.48	42.24
4	43.16	40.39	44.77	37.63
5	39.47	44.08	34.64	46.15
6	44.08	31.19	38.55	42.70
7	55.35	57.20	45.46	65.71
8	44.08	39.24	37.86	38.78
9	18.53	20.14	18.30	26.12
10	38.55	63.87	36.25	55.12
AVERAGE	**36.48**	**38.48**	**34.16**	**41.31**
S.E.	**3.65**	**4.36**	**2.82**	**3.90**

TIME-TO-PEAK TORQUE (ms)

	CONTROL LEG		TRAINED LEG	
SUBJECT	**PRE**	**POST**	**PRE**	**POST**
1	75.28	86.14	71.54	67.42
2	87.64	71.54	108.99	67.42
3	97.54	95.51	92.88	79.78
4	101.12	86.14	95.88	99.25
5	71.16	76.40	75.90	90.64
6	67.91	55.81	50.94	70.57
7	80.90	91.76	82.02	88.76
8	75.28	70.41	84.27	77.15
9	71.16	63.30	89.51	71.16
10	73.03	67.79	74.53	79.78
AVERAGE	**80.10**	**76.48**	**82.65**	**79.19**
S.E.	**3.66**	**4.11**	**5.02**	**3.41**

EVOKED TWITCH MEASUREMENTS

RISE TIME (ms)

	CONTROL LEG		TRAINED LEG	
SUBJECT	**PRE**	**POST**	**PRE**	**POST**
1	44.57	43.07	38.20	40.82
2	60.30	55.06	38.58	41.57
3	49.60	47.57	47.57	35.96
4	62.55	60.67	59.18	56.18
5	49.06	47.94	51.26	55.81
6	40.61	29.96	34.46	46.60
7	48.69	35.21	39.33	67.42
8	52.43	43.82	49.81	51.31
9	52.81	37.45	57.30	39.70
10	40.82	44.19	30.34	31.09
AVERAGE	**50.14**	**44.49**	**44.60**	**46.65**
S.E.	**2.31**	**2.88**	**3.10**	**3.48**

HALF-RELAXATION TIME (ms)

	CONTROL LEG		TRAINED LEG	
SUBJECT	**PRE**	**POST**	**PRE**	**POST**
1	64.04	72.66	75.28	86.52
2	59.55	81.65	76.03	104.87
3	52.60	51.31	66.67	54.68
4	66.67	68.16	64.42	56.93
5	84.64	56.55	69.24	91.39
6	82.56	89.51	107.87	80.23
7	64.04	56.18	85.02	49.44
8	79.78	60.67	56.18	52.43
9	62.92	63.30	70.00	56.18
10	59.55	45.69	61.80	51.69
AVERAGE	**67.64**	**64.57**	**73.25**	**68.44**
S.E.	**3.44**	**4.32**	**4.62**	**6.40**

Leg $\dot{V}O_{2peak}$

$L \cdot min^{-1}$	CONTROL LEG		TRAINED LEG	
SUBJECT	PRE	POST	PRE	POST
1	3.04	3.17	2.69	3.44
2	2.93	3.25	2.91	3.30
3	3.43	3.60	3.42	3.56
4	3.44	3.59	3.21	3.51
5	2.84	2.73	3.00	2.89
6	3.06	3.38	3.28	3.66
7	2.62	2.79	2.66	2.84
8	3.53	3.27	3.47	3.33
9	2.08	2.17	2.19	2.38
10	2.99	2.94	3.01	3.02
AVERAGE	**3.00**	**3.09**	**2.98**	**3.19**
SE	**0.14**	**0.14**	**0.12**	**0.13**

$ml \cdot kg^{-1} \cdot min^{-1}$	CONTROL LEG		TRAINED LEG	
SUBJECT	PRE	POST	PRE	POST
1	37.28	38.37	32.80	41.77
2	40.70	46.59	40.37	47.30
3	45.43	47.30	45.61	46.78
4	44.04	47.20	41.19	45.59
5	37.85	36.70	39.69	38.63
6	45.51	50.51	48.49	54.16
7	31.11	32.67	31.67	33.41
8	41.58	38.20	40.70	38.99
9	30.70	31.13	31.68	34.05
10	36.51	34.95	36.67	36.22
AVERAGE	**39.07**	**40.36**	**38.89**	**41.69**
SE	**1.70**	**2.19**	**1.81**	**2.11**

LEG PRESS

60 degrees per second (Nm)

	CONTROL LEG		TRAINED LEG	
SUBJECT	**PRE**	**POST**	**PRE**	**POST**
1	565.3	540.4	624.0	634.9
2	801.4	648.7	694.4	635.7
3	646.2	675.4	640.2	616.3
4	700.5	720.8	693.7	662.6
5	466.2	588.2	404.5	618.9
6	614.6	630.6	591.6	671.1
7	645.5	623.1	660.2	645.5
8	541.5	514.6	599.9	537.6
9	721.6	755.3	659.8	634.6
10	591.6	641.0	550.2	623.5
AVERAGE	**629.4**	**633.8**	**611.9**	**628.1**
SE	**30.51**	**23.45**	**27.17**	**11.51**

300 degrees per second (Nm)

	CONTROL LEG		TRAINED LEG	
SUBJECT	**PRE**	**POST**	**PRE**	**POST**
1	493.0	464.3	554.7	552.5
2	537.4	445.7	456.6	409.6
3	474.5	536.1	480.2	513.7
4	550.2	499.0	513.7	479.6
5	224.1	399.2	209.0	410.9
6	391.3	433.7	416.5	472.8
7	444.0	484.3	441.4	485.6
8	403.7	348.7	423.7	442.7
9	475.6	546.8	515.6	478.5
10	448.9	482.5	445.1	452.5
AVERAGE	**444.3**	**464.0**	**445.7**	**469.8**
SE	**29.27**	**19.08**	**29.83**	**13.87**

VERTICAL JUMP

MAXIMAL POSITIVE POWER ($W \cdot bodyweight^{-1}$)

	CONTROL LEG		TRAINED LEG	
SUBJECT	**PRE**	**POST**	**PRE**	**POST**
1	3.980	5.780	6.320	3.970
2	10.240	7.740	8.200	7.810
3	7.74	7.57	7.890	7.460
4	7.380	7.390	8.150	6.900
5	5.860	6.320	6.290	6.920
6	8.220	6.280	7.700	5.560
7	5.960	5.450	5.720	5.160
8	5.700	6.970	5.700	5.810
9	4.870	8.000	5.430	6.760
10	5.740	6.100	5.930	5.700
AVERAGE	**6.57**	**6.76**	**6.73**	**6.21**
S.E.	**0.58**	**0.28**	**0.35**	**0.37**

TAKE-OFF VELOCITY ($m \cdot s^{-1}$)

	CONTROL LEG		TRAINED LEG	
SUBJECT	**PRE**	**POST**	**PRE**	**POST**
1	2.242	2.715	2.936	2.130
2	3.594	2.856	3.051	2.904
3	3.059	2.961	3.090	3.066
4	2.982	3.019	3.099	2.798
5	2.515	2.618	2.740	2.735
6	2.880	2.691	2.711	2.514
7	2.519	2.342	2.577	2.285
8	2.613	2.737	2.684	2.441
9	2.324	2.997	2.439	2.672
10	2.682	2.567	2.687	2.451
AVERAGE	**2.74**	**2.75**	**2.80**	**2.60**
S.E.	**0.13**	**0.07**	**0.07**	**0.09**

VERTICAL JUMP

PEAK FORCE (Bodyweight)

	CONTROL LEG		TRAINED LEG	
SUBJECT	PRE	POST	PRE	POST
1	2.227	2.532	2.536	2.190
2	3.544	3.610	3.279	3.727
3	3.207	2.74	3.093	2.871
4	3.051	2.195	3.102	3.070
5	2.705	2.853	2.869	2.966
6	3.720	2.816	3.563	2.708
7	3.024	2.858	2.723	2.703
8	2.567	3.047	2.568	2.871
9	2.277	3.257	2.776	3.162
10	2.573	2.822	2.601	2.737
AVERAGE	**2.89**	**2.87**	**2.91**	**2.90**
S.E.	**0.16**	**0.12**	**0.11**	**0.12**

NET IMPULSE (Bodyweight·s)

	CONTROL LEG		TRAINED LEG	
SUBJECT	PRE	POST	PRE	POST
1	0.229	0.277	0.299	0.217
2	0.366	0.291	0.311	0.296
3	0.312	0.302	0.315	0.313
4	0.304	0.234	0.316	0.285
5	0.256	0.267	0.279	0.279
6	0.294	0.274	0.276	0.256
7	0.257	0.239	0.263	0.233
8	0.266	0.279	0.274	0.249
9	0.237	0.306	0.249	0.272
10	0.273	0.262	0.274	0.25
AVERAGE	**0.28**	**0.27**	**0.29**	**0.27**
S.E.	**0.01**	**0.01**	**0.01**	**0.01**

FIBRE TYPE

TYPE I %

SUBJECT	PRE	POST-CONTROL	POST-TRAINED
1	39.46	35.79	47.23
2	45.94	42.19	48.33
3	43.36	41.76	51.62
4	47.86	39.27	44.68
5	43.66	41.70	44.27
6	58.29	40.36	44.14
7	50.41	51.90	51.33
8	49.84	40.29	42.11
9	41.11	41.57	53.60
10	55.16	37.07	52.14
AVE	**47.51**	**41.19**	**47.95**
S.E.	**1.91**	**1.37**	**1.28**

TYPE IIa %

SUBJECT	PRE	POST-CONTROL	POST-TRAINED
1	30.78	37.63	39.57
2	38.26	39.24	40.83
3	35.73	38.43	31.18
4	37.70	42.92	39.89
5	29.58	33.84	30.43
6	25.50	35.15	36.50
7	28.22	31.55	31.64
8	30.82	36.79	46.86
9	34.19	35.58	32.01
10	25.56	36.66	32.59
AVE	**31.63**	**36.78**	**36.15**
S.E.	**1.47**	**0.98**	**1.73**

TYPE IIb
%

SUBJECT	PRE	POST-CONTROL	POST-TRAINED
1	29.76	26.58	13.19
2	15.80	18.57	10.83
3	20.92	19.80	17.21
4	14.45	17.81	15.43
5	26.76	24.45	25.30
6	16.21	24.49	19.35
7	21.37	16.55	17.04
8	19.34	22.92	11.04
9	24.70	22.85	14.39
10	19.28	26.27	15.27
AVE	**20.86**	**22.03**	**15.90**
S.E.	**1.57**	**1.14**	**1.34**

MEAN FIBRE AREA (um^2)

TYPE I			
SUBJECT	PRE-TRAINING	POST-CONTROL	POST-TRAINING
1	4449.30	3510.90	4374.40
2	3365.70	5620.60	6742.30
3	2955.90	2934.90	4228.10
4	4500.30	4144.20	4537.40
5	6347.00	3747.70	5788.50
6	4097.60	4537.20	5006.50
7	4128.90	4514.90	5480.80
8	6391.80	4697.60	6115.50
9	4971.30	5450.70	4500.30
10	3925.40	3097.90	3908.60
AVE	**4513.32**	**4225.66**	**5068.24**
S.E.	**357.68**	**289.33**	**293.04**

TYPE IIa			
SUBJECT	PRE-TRAINING	POST-CONTROL	POST-TRAINING
1	6846.70	5771.50	6767.70
2	4518.10	5891.70	7666.70
3	5311.90	5673.50	5963.00
4	5611.10	6100.00	6694.00
5	7281.20	6784.60	8104.00
6	5250.60	6717.60	6827.60
7	5242.70	4900.30	8337.30
8	7128.60	4856.10	5975.00
9	7414.20	6670.20	7066.40
10	5444.90	5758.00	5119.60
AVE	**6005.00**	**5912.35**	**6852.13**
S.E.	**331.59**	**217.56**	**317.57**

TYPE IIb			
SUBJECT	PRE-TRAINING	POST-CONTROL	POST-TRAINING
1	6488.30	3914.80	6157.60
2	3608.70	5498.60	6397.20
3	4213.00	4110.90	4855.30
4	5927.10	6138.00	5909.40
5	5840.80	3598.40	5137.00
6	4565.40	5688.40	6112.20
7	4577.00	4387.40	5043.40
8	7544.80	4984.00	5894.00
9	6273.90	5530.80	6428.10
10	4654.30	4952.70	4364.70
AVE	**5369.33**	**4880.40**	**5629.89**
S.E.	**388.14**	**268.05**	**227.85**

PERCENT FIBRE AREA

TYPE I

SUBJECT	PRE-TRAINING	POST-CONTROL	POST-TRAINED
1	30.30	28.13	37.15
2	40.21	41.57	46.01
3	31.56	29.04	44.75
4	42.02	30.48	36.14
5	42.71	32.98	40.49
6	53.46	32.79	37.55
7	45.86	50.76	44.58
8	46.56	39.26	42.74
9	33.35	38.39	43.08
10	48.61	25.18	46.6
AVE	**41.46**	**34.86**	**41.91**
S.E.	**2.43**	**2.43**	**1.22**

TYPE IIa

SUBJECT	PRE-TRAINING	POST-CONTROL	POST-TRAINED
1	36.36	48.57	48.22
2	44.96	40.53	44.2
3	46.74	51.67	38.12
4	41.27	49.04	47.61
5	33.20	48.45	38.97
6	29.97	42.27	42.35
7	32.60	33.50	41.08
8	32.11	37.05	46.47
9	41.36	40.20	40.4
10	31.24	46.29	38.16
AVE	**36.98**	**43.76**	**42.56**
S.E.	**1.93**	**1.88**	**1.22**

TYPE IIb			
SUBJECT	PRE-TRAINING	POST-CONTROL	POST-TRAINED
1	33.34	23.30	14.63
2	14.83	17.90	9.78
3	21.70	19.29	17.13
4	16.71	20.48	16.25
5	24.09	18.57	20.54
6	16.57	24.94	20.1
7	21.55	15.73	13.62
8	21.33	23.70	10.79
9	25.29	21.41	16.52
10	20.15	28.53	15.24
AVE	**21.556**	**21.39**	**15.46**
S.E.	**1.68**	**1.20**	**1.11**

CITRATE SYNTHASE

SUBJECT	PRE	POST-CONTROL	POST-TRAINED
1	3.36	3.77	5.35
2	6.17	5.87	6.31
3	4.68	2.75	6.97
4	5.60	4.77	7.44
5	5.96	5.88	6.30
6	3.59	2.02	2.68
7	5.57	3.61	3.11
8	5.80	3.76	5.43
9	3.55	3.43	3.86
10	3.54	2.91	3.67
AVE	**4.78**	**3.88**	**5.11**
S.E.	**0.37**	**0.40**	**0.53**

CAPILLARY DENSITY (caps/mm^2)

SUB	PRE	POST CONTROL	POST TRAINED
1	308	320	365
2	344	293	248
3	299	272	296
4	346	232	396
5	250	311	205
6	297	368	201
7	259	268	277
8	231	301	259
9	241	260	196
10	304	283	300
AVE	**288**	**291**	**274**
S.E.	**12.95**	**11.86**	**21.45**

APPENDIX B

ANOVA SUMMARY TABLES

STAT. GENERAL MANOVA	Summary of all Effects; design: (rest twitch_peak torque.sta) 1-LEG, 2-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	.6376	18	237.0901	.002689	.959214
2	1*	209.8098*	18*	40.5526*	5.173774*	.035399*
12	1	66.4351	18	40.5526	1.638246	.216817

STAT. GENERAL MANOVA	Summary of all Effects; design: (rest twitch_time 2 pt.sta) 1-LEG, 2-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	69.0901	18	231.2590	.298756	.591375
2	1	125.1391	18	104.1953	1.201005	.287568
12	1	.0714	18	104.1953	.000685	.979404

STAT. GENERAL MANOVA	Summary of all Effects; design: (rest twitch_rise time.sta) 1-LEG, 2-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	28.7133	18	129.0021	.222580	.642749
2	1	32.5261	18	47.8751	.679395	.420584
12	1	147.9556	18	47.8751	3.090449	.095745

STAT. GENERAL MANOVA	Summary of all Effects; design: 1-LEG, 2-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	224.8656	18	306.0375	.734765	.402612
2	1	155.3148	18	157.8610	.983871	.334393
12	1	7.6388	18	157.8610	.048389	.828366

STAT. GENERAL MANOVA	Summary of all Effects; design: (vo2_absolute.sta) 1-LEG, 2-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	.020363	18	.321423	.063352	.804126
2	1*	.234473*	18*	.025971*	9.028182*	.007607*
12	1	.035135	18	.025971	1.352855	.259968

STAT. GENERAL MANOVA	Summary of all Effects; design: (vo2_relative.sta) 1-LEG, 2-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	3.26070	18	71.86036	.045375	.833709
2	1*	41.92973*	18*	5.31265*	7.892436*	.011602*
12	1	5.71649	18	5.31265	1.076016	.313326

STAT. GENERAL MANOVA	Summary of all Effects; design: (watts_vo2.sta) 1-LEG, 2-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	722.500	18	1427.361	.50618	.485917
2	1*	3610.000*	18*	188.472*	19.15402*	.000364*
12	1	722.500	18	188.472	3.83346	.065925

STAT. GENERAL MANOVA	Summary of all Effects; design: (leg press_slow.sta) 1-LEG, 2-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	1360.722	18	8707.522	.156270	.697260
2	1	1059.870	18	3050.484	.347443	.562890
12	1	351.056	18	3050.484	.115082	.738357

STAT. GENERAL MANOVA	Summary of all Effects; design: (leg press_fast.sta) 1-LEG, 2-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	129.240	18	8700.591	.014854	.904346
2	1	4829.006	18	2815.992	1.714851	.206827
12	1	49.062	18	2815.992	.017423	.896452

STAT. GENERAL MANOVA	Summary of all Effects; design: (vj_max positive power.sta) 1-LEG, 2-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	.382203	18	2.362679	.161767	.692271
2	1	.283922	18	1.019085	.278605	.604063
12	1	1.292403	18	1.019085	1.268199	.274896

STAT. GENERAL MANOVA	Summary of all Effects; design: (vj_take-off velocity.sta) 1-LEG, 2-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	.020385	18	.115977	.175769	.679996
2	1	.092641	18	.055166	1.679293	.211390
12	1	.111408	18	.055166	2.019489	.172385

STAT. GENERAL MANOVA	Summary of all Effects; design: (vj_peak force.sta) 1-LEG, 2-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	.006003	18	.211262	.028413	.868022
2	1	.001823	18	.125964	.014468	.905590
12	1	.000090	18	.125964	.000714	.978969

STAT. GENERAL MANOVA	Summary of all Effects; design: (vj_impulse.sta) 1-LEG, 2-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	1	.000009	18	.001124	.008029	.929592
2	1	.001809	18	.000698	2.591675	.124824
12	1	.000511	18	.000698	.732399	.403355

STAT. GENERAL MANOVA	Summary of all Effects; design: (type i percent.sta) 1-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2*	142.8414*	18*	23.23573*	6.147490*	.009228*

STAT. GENERAL MANOVA	Summary of all Effects; design: (type iia percent.sta) 1-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2*	78.80180*	18*	12.21412*	6.451698*	.007716*

STAT. GENERAL MANOVA	Summary of all Effects; design: (type iib percent.sta) 1-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2*	105.7432*	18*	13.08884*	8.078888*	.003134*

STAT. GENERAL MANOVA	Summary of all Effects; design: (type i area.sta) 1-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2	1834376.	18	647787.2	2.831757	.085259

STAT. GENERAL MANOVA	Summary of all Effects; design: (type iib area.sta) 1-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2	1447799.	18	754139.8	1.919802	.175489

STAT. GENERAL MANOVA	Summary of all Effects; design: (type iia area.sta) 1-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2	2682333.	18	767166.6	3.496415	.052133

STAT. GENERAL MANOVA	Summary of all Effects; design: (type i area ratio_calc.sta) 1-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2*	155.9231*	18*	37.70982*	4.134815*	.033294*

STAT. GENERAL MANOVA	Summary of all Effects; design: (type iia area ratio_calc.sta) 1-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2*	130.7578*	18*	25.48929*	5.129913*	.017255*

STAT. GENERAL MANOVA	Summary of all Effects; design: (type iib area ratio_calc.sta) 1-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2*	120.4935*	18*	16.51724*	7.295014*	.004783*

STAT. GENERAL MANOVA	Summary of all Effects; design: (citrate synthase_2.sta) 1-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2*	4.093111*	18*	.794732*	5.150302*	.017033*

STAT. GENERAL MANOVA	Summary of all Effects; design: (capill~1.sta) 1-TIME					
Effect	df Effect	MS Effect	df Error	MS Error	F	p-level
1	2	766.2780	18	2454.890	.312144	.735759