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ORIGINAL ARTICLE

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Effects of strength, endurance and combined training on myosin heavy chain content and fibre-type distribution in humans

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Abstract This study investigated the effect of strength training, endurance training, and combined strength plus endurance training on fibre-type transitions, fibre cross-sectional area (CSA) and MHC isoform content of the vastus lateralis muscle. Forty volunteers (24 males and 16 females) were randomly assigned to one of four groups: control (C), endurance training (E), strength training (S), or concurrent strength and endurance training (SE). The S and E groups each trained three times a week for 12 weeks; the SE group performed the same S and E training on alternate days. The development of knee extensor muscle strength was S > SE > E ($P < 0.05$) and has been reported elsewhere. The reduction in knee extensor strength development in SE as compared to S corresponded to a 6% increase in MHCIIa content ($P < 0.05$) in SE at the expense of the faster MHCIId(x) isoform ($P < 0.05$), as determined by electrophoretic analyses; reductions in MHCIId/x content after S or E training were attenuated by comparison. Both S and SE induced three- to fourfold reductions ($P < 0.05$) in the proportion of type IIA/IID(X) hybrid fibres. S also induced fourfold increases in the proportion of type I/IIA hybrid fibres within both genders, and in a population of fibres expressing a type I/IID(X) hybrid phenotype within the male subjects. Type I/IIA hybrid fibres were not detected after SE. Both S and SE training paradigms induced similar increases (16–19%, $P < 0.05$) in the CSA of type IIA fibres. In contrast, the increase in CSA of type I fibres was 2.9-fold greater ($P < 0.05$) in S as compared to SE after 12 weeks. We conclude that the interference of knee extensor strength development in SE versus S was related to greater fast-to-slow fibre-type transitions and attenuated hypertrophy of type I fibres. Data are given as mean (SEM) unless otherwise stated.

Keywords Endurance training · Fibre types · Myosin heavy chain · Strength training

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Introduction

Skeletal muscle is a heterogeneous tissue composed of different fibre types that are differentiated by their MHC isoform contents and metabolic properties (Pette and Staron 1997). The MHC composition of muscle fibres is a major determinant of the contractile characteristics, influencing energetic economy of contraction, maximum muscle shortening velocity and maximum power output (Bottinelli et al. 1996, 1999; Galler et al. 1997; Stienen et al. 1996). When exposed to different functional demands, muscle fibres display considerable plasticity that extends to all aspects of their biochemical and morphological properties (for reviews see Abernethy et al. 1994; Pette 2002). Changes in the patterns of MHC isoform expression and fibre cross-sectional area (CSA) in response to various exercise training regimes, for example, represent especially important adaptations that underlie changes in skeletal muscle strength and power output (Bottinelli et al. 1999; Shoepe et al. 2003; Widrick et al. 2002).

Previous studies have reported that strength training in humans resulted in a lower proportion of histochemically identified type IIB fibres, a concomitant increase in the proportion of hybrid fibres expressing MHCIIa, and greater CSA of all fibre types (e.g. Haddad et al. 1998; Hostler et al. 2001; Kraemer et al. 1995; Staron et al. 1989, 1991, 1994). In contrast, endurance training of a similar duration may not induce the same

degree of fast-to-slow fibre type transitions, if the intensity of repeated contractions is low (Fitzsimons et al. 1990). High-intensity endurance training, however, appears to induce similar (Fitzsimons et al. 1990; Simoneau et al. 1985) and perhaps even greater transformation, also encompassing an increase in the proportion of type I fibres (Fitzsimons et al. 1990; Simoneau et al. 1985). In comparison to strength training, where the CSA of type I and type II fibres were increased, after endurance training the CSA of fibres remain unchanged (Carter et al. 2001; McCarthy et al. 1995; Sipila et al. 1997) or decrease (Kraemer et al. 1995). Thus, it appears that strength- and endurance-training regimes may yield differences with regard to the degree of MHC-based fast-to-slow fibre type transitions and changes in fibre CSA. These differences may provide the basis for the interference of strength development when these two training regimes are applied concurrently. Indeed, recent studies have shown that concurrent strength- and endurance-training paradigms are antagonistic and limit the development of muscle strength (e.g. Bell et al. 2000; Kraemer et al. 1995; Leveritt et al. 1999). However, the question remains as to whether the interference with knee extensor strength development, when strength- and endurance-training paradigms are concurrently applied, is related to qualitative and quantitative changes in the patterns of MHC protein isoform expression.

The purpose of the present investigation was to examine the patterns of MHC protein isoform expression, fibre type distribution and fibre CSA in a group of previously studied subjects who displayed lower knee extensor strength development when progressive resistance and endurance training paradigms were completed concurrently (Bell et al. 2000). In contrast to previous studies, we used sensitive immunohistochemical and electrophoretic analytical methods to study changes in the patterns of MHC protein isoform expression. Subjects were studied before and after 6 and 12 weeks of strength- (S), endurance- (E) or concurrent strength and endurance-training (SE). We hypothesized that training-induced increases in CSA would be lower and fast-to-slow fibre type transitions would greater when strength and endurance training were completed concurrently (i.e. SE), as compared with S training alone.

Methods

Subjects

Forty volunteers (24 males and 16 females) participated in this study after providing written informed consent. The study was approved by the University of Alberta Ethics Research Board. The age, height and weight of the males were [23 (0.6) years, 181.2 (1.0) cm and 79.1 (1.9) kg, respectively (mean (SEM)]; the corresponding values for the females were 22 (0.9) years, 166.5 (1.3) cm and 64.5 (1.9) kg. Males and females were randomly assigned separately to one of four groups: control (C), endurance training (E), strength training (S) or combined strength and endurance training (SE). Physiological testing, as well as muscle biopsy procedures were performed before training and after 6 and 12 weeks. The C group was studied at the beginning of the study and after 12 weeks. Details of the physiological testing methods are reported elsewhere (Bell et al. 2000).

Training

The training protocols used in this study have been previously described (Bell et al. 2000). Briefly, E training was performed on Monark cycle ergometers three times a week, on non-consecutive days. Continuous endurance training was conducted twice a week; the duration began at 30 min and was progressively increased to 42 min per session. Initial intensity was equivalent to that power output which elicited ventilation threshold. The third endurance training session was comprised of cycling intervals at 90% $\dot{V}O_{2max}$ (i.e. 3 min work/3 min rest). Each subject began at four sets and progressively increased until seven sets were completed each session. The S training program was comprised of a combination of free-weight and machine-loading systems for four lower body exercises (bilateral inclined leg press, unilateral knee extension, unilateral knee flexion and bilateral calf raises) and four upper body exercises (seated row, bench press, upright row, lat pull downs) and was performed 3 days per week on non-consecutive days. Upper body exercises were included to provide a balanced whole body program. The S program was based on a pyramidal system in which the number of sets was varied between 2 and 6 and the repetitions with each set ranged from 4 to 10. The mean relative intensity was progressively overloaded from approximately 70–85% of 1 RM. Rest intervals between sets were limited to 1–2 min, and between exercises to 2–3 min. The SE group performed the same strength and endurance training on alternate days (6 days a week).

Muscle sample collection

Skeletal muscle biopsies were taken from the lateral aspect of the right vastus lateralis muscle (Bergstrom 1975) at rest before training was initiated, and after 6 and 12 weeks of training. To eliminate variations in skeletal muscle sampling, biopsies were taken from the same region and depth. Samples were rapidly mounted on cork in O.C.T. embedding compound, frozen in melting isopentane and stored at −80°C.

Primary immunohistochemical analyses

Fibre type determination was completed immunohistochemically on 10-μm-thick, frozen sections according to

(Putman et al. 1999, 2000, 2003) using monoclonal antibodies directed against various MHC isoforms: anti-MHCIβ(clone BA-D5); anti-MHCIα (clone F88 12F8,1); anti-MHCIIa (clone SC-71), anti-MHCembryonic (clone BF-45) and anti-MHCIIb (clone BF-F3). Controls were run in parallel in which the primary antibody was omitted or a non-specific mouse-lgG antibody (Santa Cruz, Calif., USA) was substituted. Type I, IIA and IIB fibres were identified by their direct immunohistochemical reactions with the corresponding antibody. Type IID(X) fibres were identified as those fibres that remained unstained using this panel of antibodies. Analyses were completed with a Leitz Diaplan microscope (Leitz Wetzlar, Germany) fitted with a Pro Series High Performance CCD camera (Media Cybernetics, USA) and an analytical imaging software program (Image Pro Plus 4.0). At least three separate regions and a total of 623 (125) fibres were analysed per muscle sample. Fibres that stained for both MHCI and MHCIIa, were classified as type I/IIA hybrids. Fibres that stained lightly for MHC-IIa but no other MHC-isoforms were identified as IIA/D(X) hybrid fibres. Neither of MHCIIb, MHCembryonic nor MHCIα was observed in extrafusal fibres. All fibres were accounted for using this classification system. The camera was calibrated with a known measurement scale. Muscle fibres from video images of the stained cross sections were outlined using the Image Pro program. The cross-sectional area of each fibre type was determined by the mean of at least 50 muscle fibres per muscle sample; CSA was not determined for those fibre types that did not reach this threshold for all muscle samples (i.e. type IIA/IID(X) and type I/IID(X) hybrid fibres).

Secondary immunohistochemical analyses

In order to further examine the co-expression of fast and slow MHC isoforms, especially with regard to type I/IID(X) polymorphism, secondary analyses were completed using a dual labelling immunofluorescent method. Frozen sections (6 μm thick) were mounted on gelatin-chromagen (0.1% gelatin, 0.01% chromium potassium sulfate)-coated glass coverslips. Sections were incubated for 30 min in a blocking solution (BS) containing 2% BSA and 2% rabbit serum in PBS and then for 1 h with a mouse mc anti-MHCI (IgA, clone S58) diluted 1:10 in BS. Sections were washed and incubated for 1 h with rhodamine-conjugated goat anti-mouse IgA (diluted 1:50 in BS) (Sera-Lab, Cedarlane Laboratories, Hornby, Ontario). Sections were washed as before and incubated for 1 h with mouse mc anti-MHC-fast (IgG, MY-32; Sigma, St. Louis, Mo.), diluted 1:1,000 in BS. Sections were washed again and incubated for 1 h with FITC-conjugated goat anti-mouse IgG (diluted 1:50) (Zymed Laboratories, San Francisco, Calif., USA). Sections were covered with an aqueous mounting medium and the coverslips applied. Fluorescence was visualized on a Leitz Diaplan microscope equipped with N2.1 (excitation 515–560 nm; emission >580 nm) and K2 (excitation 470–490 nm; emission >515 nm) filters. Sections were photographed on the same day and printed at a final magnification of 320×. The number of fibres expressing fast, slow or combined fast and slow MHC was determined from photographs in 78 (24) randomly selected fibres for each biopsy sample. This analytical approach was found to be representative of the composition of the whole biopsy ($r^2 = 0.92$), as determined by counting all fibres in three biopsies. The correlation between observations on different days ($r_{xy} \geq 0.99$) and between investigators ($r_{xy} \geq 0.99$) was very high.

MHC isoform content

MHC extracts were prepared, and the relative contents of the adult MHC protein isoforms expressed were determined electrophoretically according to (Putman et al. 2003). One microgram of total protein was loaded per lane and gels were run at 275 V for 24 h.

Statistical analyses

Data were analysed using a three-way ANOVA (group and gender) with repeated measures on one factor (training). One-way ANOVA was used to compare the mean decrease in the proportion of MHCIId(x) between groups after a significant main effect and interaction were observed. Differences between group means were assessed using Fisher's pairwise post-hoc test for planned comparisons. Because it was specifically hypothesized a priori that the CSAs of type I and IIA fibres would increase as a result of strength training, these data were analysed using a one-tailed paired dependent-samples Student's *t*-test. Data are summarized as mean (SEM). Differences were considered significant at $P < 0.05$.

Results

MHC content

The electrophoretic method used to quantify the various MHC isoforms is shown in Fig. 1; changes in MHC content are summarized in Fig. 2. No significant gender by time by group interaction was found for MHC content. Therefore, the data were examined independent of gender. Only the SE training paradigm resulted in an increase in MHCIIa after 12 weeks of training. In contrast, MHCIId(x) content was reduced to the same level after 12 weeks of S or E training. In the SE group the decrease in MHCIId(x) was first detected after 6 weeks and was further reduced after 12 weeks of training; the decrease in MHCIId(x) was greatest in the SE group ($P < 0.05$).

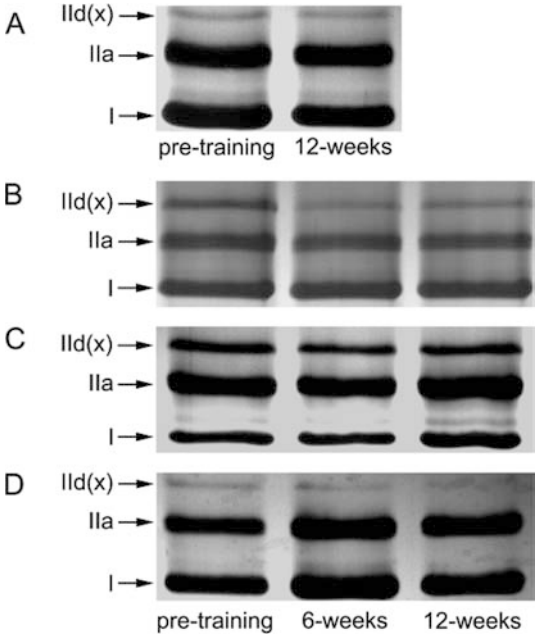

Fig. 1A–D Representative MHC Gels. Sample shown are from: **A** control group; **B** strength training group; **C** endurance training group; **D** combined strength and endurance training group. MHC isoforms are denoted as follows: MHCIId(x) = IId(x); MHCIIa = IIa; and MHCI = I

Primary fibre-type analyses

The immunohistochemical method used to identify the various fibre types is illustrated in Fig. 3. Staining of fibres by clones BF-F3 (i.e. anti-MHCIIb), F88 12F8,1 (i.e. anti-MHCIα) or BF-45 (i.e. anti-MHC-embryonic) was observed only within intrafusal fibres. The fibre type distributions in C and in the various training groups are summarized in Fig. 4. Similar to the MHC data no significant gender by time by group interaction was found for any of the pure fibre types. There was, however, a significant main effect for group indicating that the initial proportion of type I fibres was higher in the S and SE groups as compared to the C and E groups. Nevertheless, no changes were detected in the proportion of pure type I fibres as the result of any of the training conditions. Similarly, the proportion of pure IIA fibres tended to be lower in the SE group; no differences were detected as the result of training. The only changes to manifest from training were within the type I/IIA and IIA/IID(X) hybrid fibre populations. S training alone resulted in an approximate sixfold increase in the proportion of type I/IIA hybrid fibres, while SE resulted in the complete absence of this fibre population. In contrast, the population of fast type IIA/IID(X) hybrid fibres decreased by three- to fourfold after S and by a similar relative amount after SE.

Secondary fibre-type analyses

In order to quantify the proportion of type I/IID(X) hybrid fibres present, secondary immunohistochemical analyses were completed using anti-slow MHC antibody (i.e. clone S58) and anti-MHC-fast antibody (i.e. clone MY32). Because it was determined that neither of the MHCIIb or developmental MHC isoforms were present, it was possible to estimate the proportion of type I/IID(X) fibres as the difference between fibres staining positive for both S58 (slow) and MY32 (fast) (Fig. 5) and those identified as type I/IIA. This analysis revealed that most slow/fast hybrid fibres were type I/IIA. The only exception was observed within the S group of male subjects, which displayed an 8% increase (i.e. fourfold

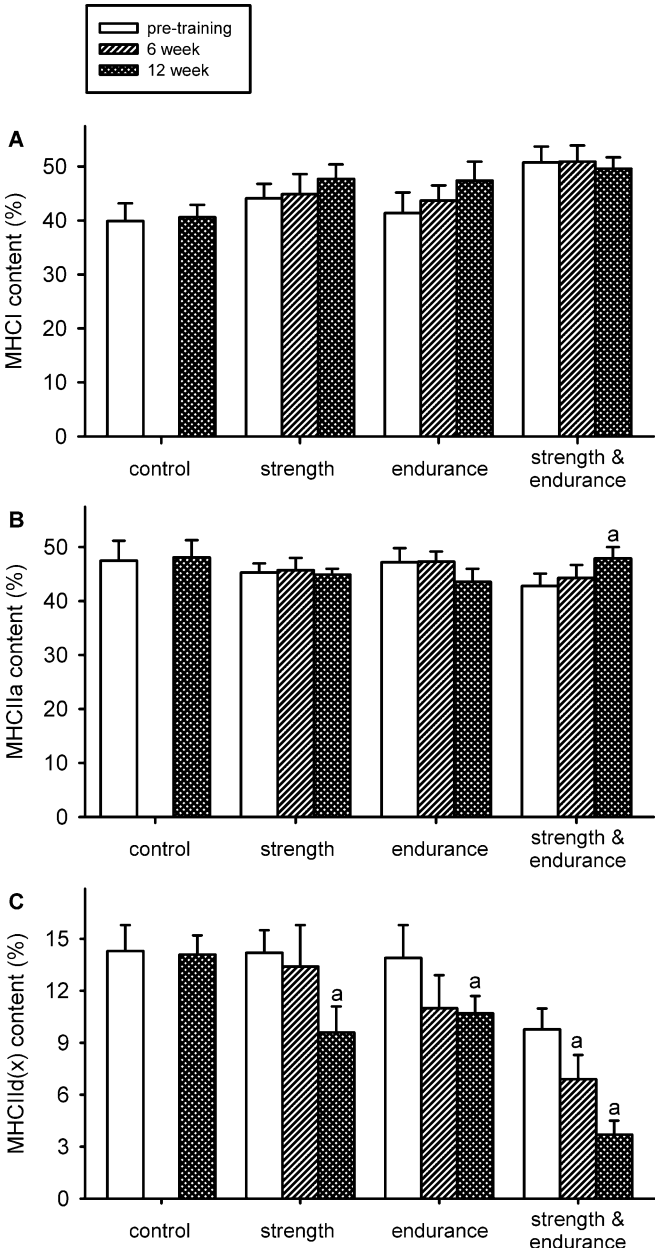

Fig. 2 MHCI (**A**), MHCIIa (**B**) and MHCIId(x) (**C**) contents in the control, strength trained, endurance trained and combined strength and endurance trained groups. *a* indicates different from pre-training control within a group. There was a significant group×time interaction for MHCIId(x) ($P < 0.05$)

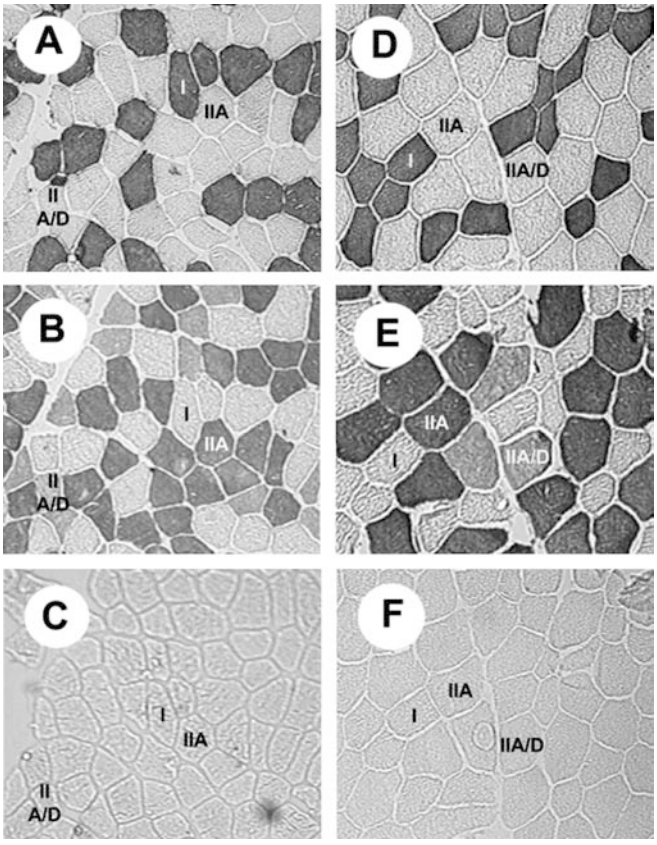

Fig. 3A–F Immunohistochemical staining of human vastus lateralis muscle. Sections were stained using primary anti-MHCIβ (clone BA-D5) (**A** and **D**) or anti-MHCIIa (clone SC-71) (**B** and **E**). Nonspecific mouse-IgG (IgG control, **C** and **F**) was used as a negative control. **A**, **B** and **C** are before training, while **D**, **E** and **F** are after 12 weeks of strength training. Fibres were not stained when primary anti-MHC-embryonic (clone BF-45), anti-MHCIα (clone F88 12F8,1) or anti-MHCIIb (clone BF-F3) were applied. I, IIA and IIA/D indicates type I, type IIA and hybrid type IIA/D(X) fibres, respectively

increase) in the proportion of type I/IID(X) fibres after 6 and 12 weeks of training.

Fibre CSA

Fibre CSAs are summarized in Fig. 6. A significant gender by time interaction was not detected; thus the data were averaged for males and females. It should be noted, however, that there was a main effect for gender indicating that muscle fibre areas of the males were larger than those of the females regardless of fibre type ($P < 0.05$), training group or time point [male versus female (μm^2): type IIA, 4158 versus 3050; type I, 3640 versus 3293]. There was also a significant main effect for fibre types: type IIA muscle fibres were larger than type I fibres ($P < 0.05$). In the S group, type I and -IIA fibre CSAs increased by 17% and 13%, respectively ($P < 0.05$), and were larger than observed in all other conditions; the E group did not display hypertrophy, while the SE group only displayed an 18% increase in the CSA of type IIA fibres ($P < 0.05$).

Discussion

Previous investigations have shown that heavy progressive resistance training for 8–21 weeks caused fast-to-slow fibre-type transitions within the fast fibre subtypes and considerable fibre hypertrophy (e.g. Adams et al. 1993; Haddad et al. 1998; Hostler et al. 2001; Kadi and Thornell 1999; Staron et al. 1989, 1991, 1994; Kadi et al. 1999). In contrast, endurance training for a similar period typically induces fibre atrophy and, depending on the intensity and volume of repeated contractions, may also enhance the proportion of type I fibres (Fitzsimons et al. 1990). Thus with regard to patterns of MHC

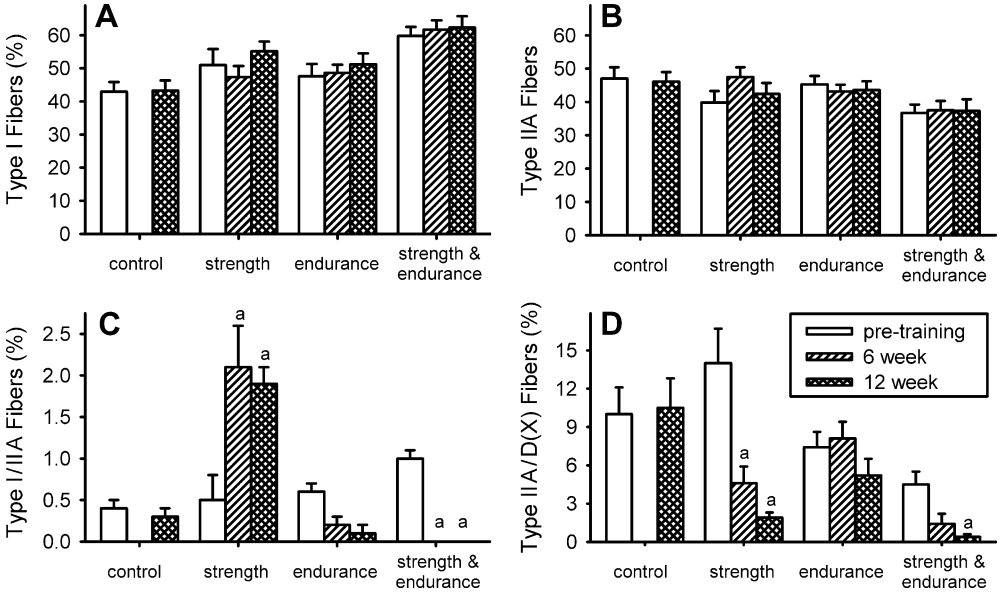

Fig. 4 Proportions of type I (**A**), IIA (**B**), and hybrid type I/IIA (**C**) and IIA/IID(X) (**D**) fibres in the control, strength trained, endurance trained and combined strength and endurance trained groups. *a* indicates different from pre-training control within a group

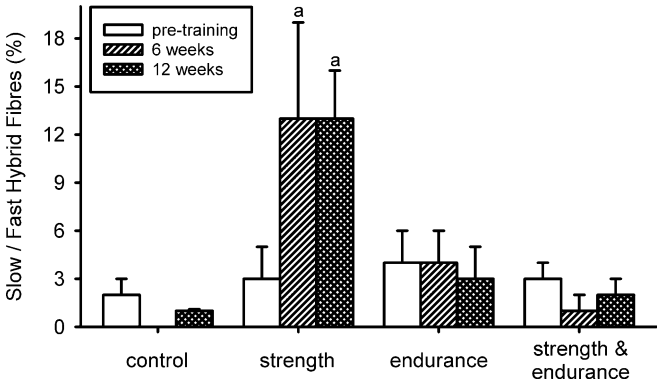

Fig. 5 Proportions of slow/fast hybrid fibres in the male control, strength trained, endurance trained and combined strength and endurance trained groups. *a* indicates different from pre-training control. Clone S58 was used to detect MHCI and clone MY32 was used to detect all fast-MHCs

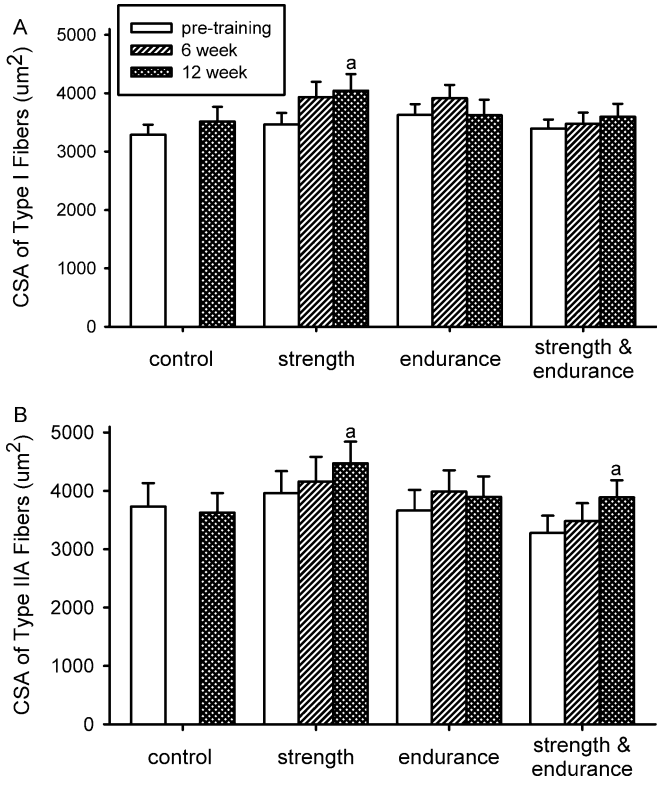

Fig. 6 Cross-sectional areas (CSA) of type I (**A**) and IIA (**B**) fibres in the control, strength trained, endurance trained and combined strength and endurance trained groups. *a* indicates different from pre-training control within a group

protein expression and fibre morphology, resistance- and endurance-training regimes seem to induce competing adaptive responses that may account for attenuated muscle strength development when they are applied concurrently (Bell et al. 2000; Kraemer et al. 1995). The present study was undertaken to investigate the effects of concurrent endurance and heavy resistance training regimes on the patterns of MHC protein isoform expression and fibre CSA. Our results extend the findings of previous studies by demonstrating that attenuated muscle strength development after SE (Bell et al. 2000) was associated with greater fast-to-slow MHC-isoform transitions and preferential hypertrophy of IIA fibres.

Patterns of MHC protein expression

In the present study MHCIIb protein expression was not detected within the vastus lateralis using either immunohistochemical or electrophoretic methods. Thus fibre type transitions within the fast fibre populations were limited to the MHCIIa and MHCIId(x) isoforms. In this regard our findings support suggestions that the IIB fibres reported in previous studies are of the IID(X) phenotype. Furthermore recent studies indicate that most of the MHCIId(x) isoform is expressed in the IIA fibre population forming a population of hybrid IIA/IID(X) fibres (e.g., Gur et al. 2003; Harridge et al. 1998; Widrick et al. 2002). We used a panel of monoclonal antibodies that allowed direct identification of pure type Iβ and IIA fibres, as well as hybrid I/IIA fibres. By virtue of obvious differences in the patterns of staining intensity, not unlike those used in classical myosin-ATPase histochemical stains, it was also possible to clearly discern a population of IIA/IID(X) hybrid fibres. Such fibres and MHCIId(x) were detected in 30 of the 40 subjects, and were evenly distributed between all groups. Both S or E training resulted in reductions in MHCIId(x) content compared to initial pre-training control levels (Figs. 1 and 2); by comparison after SE the reduction in MHCIId(x) was approximately twofold greater than noted in S or E alone. Activity-induced MHCIId(x) $\rightarrow$ MHCIIa transitions have previously been reported for resistance or endurance training (for review see Pette and Staron 1997). This study is, however, the first to note further reductions in MHCIId(x) protein expression after concurrent application of these training paradigms. The functional consequence of such conversion would appear to be lower rate and absolute amount of force development resulting in lower power output (Bottinelli et al. 1996; Stienen et al. 1996; Widrick et al. 2002); thus the more advanced MHCIId(x) $\rightarrow$ MHCIIa conversion associated with SE therefore would seem to account for lower strength development after SE.

Analysis of the proportion of MHCI protein expressed by SDS-PAGE did not reveal any significant changes within any training condition, although an increasing trend was noted after 12 weeks of S training. Subsequent immunohistochemical analyses revealed modest but significant changes in the pattern of MHCI expression, within this group. Increases in a population of hybrid fibres that expressed MHCI and MHCIIa were noted after 6 and 12 weeks of S training (Fig. 4), indicating that some of the IIA fibres began to express

limited amounts of the slow isoform. To further examine the possibility of additional fast/slow polymorphism, secondary immunohistochemical analyses were completed using clones S58 and MY32. Analyses of both female and male subjects revealed the possible existence of a population of type I/IID(X) hybrid fibres after 6 and 12 weeks of S training, but only in the males (Fig. 5), which amounted to ~8% ($P < 0.05$). Such polymorphism has recently been reported in rat skeletal muscles (Caiozzo 2002). The present findings are consistent with the development of similar polymorphism after S.

Given the seemingly modest nature of the adaptive changes in the pattern of MHC isoform expression between S, E and SE, it is appropriate to pose the question to what extent could the observed fibre type transitions have caused functionally relevant changes in the contractile properties of whole muscle? We recently observed that a 7% decrease in MHCIIb content ($P < 0.05$) and a corresponding 7% increase in MHCIId(x) content ($P < 0.05$) was associated with a 33% decrease ($P < 0.05$) in the isometric time to peak tension development in rat hindlimb muscles (Gallo et al. 2004). Similarly, it has been reported that rat muscles which differed even less in their relative MHC isoform composition, displayed significant and proportional shifts in their force-velocity curves (Caiozzo 2002; Caiozzo et al. 2003) and in maximum power output (Bottinelli et al. 1999). Recently, Gur et al. (2003) reported that a 10% increase in the proportion of type II fibres in biopsies of human vastus muscles was associated with significant 5% increase in peak isokinetic torque production during knee extension. The lower apparent functional impact in human studies undoubtedly reflects the influence of an intact musculoskeletal relationship. Nevertheless, the adaptive changes noted in the present study were comparable to those studies and highlight the importance of MHC-isoform transitions as a factor contributing to the lower strength development in SE (Bell et al. 2000).

The physiological advantage of relative MHC-isoform transitions probably relates to the greater chemomechanical energy transduction efficiency associated with IIA fibres compared with IID(X) fibres (Stienen et al. 1996; Reggiani et al. 1997). On the other hand, limitations in the degree of fast-to-slow fibre-type transition to pure type I fibres after chronic training appear to be governed by the properties of innervating alpha-motor neurons (Reichmann et al. 1983). Increases in the proportion of type I fibres may be dependent on training regimes that lead to significant fibre damage, followed by fibre regeneration (Staron et al. 1991, 1994) and associated peripheral nerve sprouting (Pette et al. 2002). Thus the absence of detectable muscle fibre damage in our study, as determined by the absence of MHC-embryonic expression within extrafusal fibres, may explain why the proportion of type I fibres remained unchanged by training.

Muscle fibre CSA

Heavy resistance-training activates both low- (i.e. type I fibres) and high-threshold (i.e. type II fibres) motor units inducing hypertrophy of all fibre types (McCall et al. 1996; McCarthy et al. 2002; Staron et al. 1991). In contrast, E training reportedly results in no change or a reduction in fibre CSA in humans (Kraemer et al. 1995). In the present study, the CSA of type I and IIA fibres were not altered after 12 weeks of E training (Fig. 6) and did not result in any appreciable change in knee extensor strength (Bell et al. 2000). In contrast SE training induced an 18% increase ($P < 0.05$) in the CSA of only type IIA fibres, while S training induced increases ($P < 0.05$) in both type I (17%) and IIA (13%) fibres. After 12 weeks of S training this coincided with greater knee extension strength compared with SE (Bell et al. 2000).

The magnitude of the increases in CSA observed in our study are similar to previous reports that also showed corresponding increases in whole muscle or single fibre tension development (Harridge et al. 1998; Kraemer et al. 1995; Widrick et al. 2002). Thus, the differential hypertrophic responses of type I and fast type IIA fibres between the S and SE training appear to also underlie some of the interference effects on knee extensor muscle strength development, as previously suggested by Kraemer et al. (Kraemer et al. 1995). While the underlying cause(s) of lower type I fibre CSA in SE remains uncertain, it is unlikely to have occurred in order to improve O_2 kinetics as previously suggested by this group (Kraemer et al. 1995), as it has been shown that the oxygenation of skeletal muscle fibres increases in humans during maximal exercise (Putman et al. 1995). This should be especially true of the type I fibre population. It seems more plausible that greater recruitment of the type I fibres in SE, in the presence of elevated serum cortisol levels (Kraemer et al. 1995), enhanced the rate of catabolic events within this fibre population.

Mechanisms

An examination of the mechanism(s) underlying the different adaptive responses was beyond the scope of the present investigation and remains the subject of considerable debate. It is interesting to note, however, that is it likely influenced by multiple events (e.g. Goldspink 1998) involving but not limited to differential activation of Ca^{2+}-dependent pathways known to regulate fibre hypertrophy (Dunn et al. 2000) and MHC gene expression (Allen and Leinwand 2002), activation of protease systems involved in regulating the turnover of MHC protein isoforms (e.g. Sultan et al. 2001) and activation of muscle precursor cells (Putman et al. 2000) by autocrine, paracrine or endocrine factors (Düsterhöft et al. 1999; Goldspink 1998, 1999; Mckoy et al. 1999; Yang et al. 1997).

Conclusion

The results of the present study show that concurrent SE training resulted in greater fast-to-slow fibre type transitions and attenuated hypertrophy of the type I fibres compared with S training alone. These findings are consistent with the hypothesis that the interference of knee extensor strength development after SE training resulted from distinctive adaptive processes. However, the regulatory events that caused these phenotypic changes remain to be characterized.

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