Acta Physiol Scand 1997, **161**, 459–464

Effects of endurance training on oxidative capacity and structural composition of human arm and leg muscles

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ABSTRACT

Six healthy subjects performed endurance training of the same duration with legs and arms consecutively. Performance and muscle structure were measured before and after training in lower and upper limbs. Training induced similar increases in maximal oxygen consumption (6 ± 1 vs. 7 ± 2 mL min^{-1} kg^{-1}: legs vs. arms, $P > 0.05$) and mitochondrial volume in leg and arm muscles (42 ± 12 vs. 31 ± 11%: legs vs. arms, $P > 0.05$). The gain in mitochondrial volume after training was achieved solely by increasing the fraction of mitochondria (+40 ± 11%, $P < 0.05$) in the same muscle volume (+2 ± 2%, $P > 0.05$) in the legs. In contrast, increased muscle volume (+14 ± 3%, $P < 0.05$), in addition to a tendency for an increase in mitochondrial fraction (+16 ± 11%, $P > 0.05$), occurred in the arms after training. Thus, similar improvements in muscle oxidative capacity in upper and lower limbs were brought about by different mechanisms. It is suggested that due to infrequent use and a lack of load-bearing function, arm muscle volume is underdeveloped in untrained, sedentary or detrained/injured subjects and that the mode of endurance training used in this study is sufficient to enlarge arm muscle volume as well as aerobic capacity.

Keywords maximal oxygen consumption, mitochondria, muscle oxidative capacity.

Received 14 April 1997, accepted 25 July 1997

In a previous study, 6–8 weeks of leg endurance training led to a significant increase in maximal oxygen consumption, accompanied by increases in mitochondrial and lipid volume densities, but not in muscle cross-sectional area of trained leg muscles (Hoppeler *et al.* 1985). A companion report indicated that non-trained arm musculature (deltoid muscle) underwent a decrease in ultrastructural indices of muscle oxidative capacity such as mitochondrial volume density during leg training (Rosler *et al.* 1985). These findings suggest that the muscle ultrastructure of arms and legs may adapt to (de)training independently. In this study, we therefore tested the hypothesis that limb specific endurance training, designed to elicit a similar improvement in maximal oxygen consumption, would lead to similar changes in muscle oxidative capacity and structure in arms and legs.

Endurance training (high volume, low intensity) is generally considered to increase muscle oxidative capacity with little or no change in muscle volume (Hoppeler *et al.* 1985). On the other hand, strength training (low volume, high intensity) often increases muscle volume at a constant or reduced muscle oxidative capacity (MacDougall *et al.* 1979, Saltin & Gollnick 1983, Luthi *et al.* 1986). The increases in arm oxidative capacity *and* muscle volume after arm training observed in this study collectively suggest that certain training programmes may increase both oxidative capacity and strength in specific muscle groups (Dudley & Fleck 1987).

MATERIALS AND METHODS

Subjects

Six healthy male subjects (23 ± 1 years old, 179 ± 1 cm in height and weighing 75 ± 2 kg) participated in the study, which had been approved by the University of Bern ethical committee (Ferretti *et al.* 1992). None of the subjects had taken part in systematic training programmes for cycling or specific arm exercise, although they were recreationally active.

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Performance tests

Discontinuous, incremental performance tests were conducted for leg cycling and arm cranking exercise before and after training of legs and arms, respectively. Subjects sat quietly on or next to the ergometer whilst resting measurements of cardiorespiratory and metabolic variables were made using standard techniques. The initial low power output was then performed, followed by a recovery period of 10 min or, during higher power outputs, until blood lactate concentration ($[La]_b$, mmol L^{-1}) returned to below 2 mmol L^{-1}. For the leg cycling test, the incremental power output step was 50 W from an initial 50 W; for the arm cranking test the incremental power output step was 18 W from an initial 54 W and each step was performed for 4–6 min. Subjects pedalled at the same rate as during leg and arm training (90–95 rpm). Maximal oxygen consumption ($\dot{V}_{O_2\,max}$, mL min^{-1} kg^{-1} STPD) was estimated from the plateau of $\dot{V}_{O_2}$ with increasing power output. When a plateau in the $\dot{V}_{O_2}$ vs. power output relationship was not observed, $\dot{V}_{O_2\,max}$ was established on the following basis: (1) attainment of maximal heart rate ($f_{card\,max}$; increment of less than 3 beats min^{-1} with an increase in power output); (2) $[La]_b$ higher than 7 mmol L^{-1}; (3) subject's inability to maintain a higher power output for longer than 3 min.

Muscle structure

The muscle structure of the legs and arms was also studied before and after the respective training programmes. The cross-sectional areas of gross anatomical compartments at the mid-thigh level of both legs and at 60% of total upper arm length of both arms (measured from the bottom of the humerus) were calculated from images collected using computed tomography (125 kV peak, 5 s exposure, 230 Ma s^{-1}; Somatom SF, Siemens, Germany) and an image analysis system (MOP AMO 3: Kontron, Switzerland). Total, muscle, subcutaneous fat and bone cross-sectional areas (cm^2) were measured on whole images and the mean of five separate measurements on both left and right limbs was used in calculating individual values included in group means. In addition, the cross-sectional area of the extensor and flexor muscle groups in the upper arm were measured separately.

Muscle biopsies were taken, using local lidocaine anaesthesia, from the left vastus lateralis and deltoideus muscles and processed for electron microscopy by fixation for 1 h in a 6.25% solution of glutaraldehyde. Micrographs for morphometry were taken on 35 mm films with a Philips EM 300 electron microscope. Morphometry was performed according to stereological methods appropriate for human muscle (Hoppeler *et al.* 1985). The absolute volume of mitochondria (V(mt), mL) and myofibrils (V(fi), mL) in a 1 cm deep representative tissue slice was calculated by multiplying volume density of mitochondria (Vv(mt,f)) or myofibrils (Vv(fi,f)) by muscle cross-sectional area and correcting for muscle density (1.06 g mL^{-1}; Mendez & Keys 1960). Total capillary length (J(c), km) in a 1 cm representative tissue slice was calculated by multiplying capillary density (Na(c,f), mm^{-2}) by muscle cross-sectional area, accounting for both muscle density and capillary tortuosity (×1.24; Conley *et al.* 1987). Capillary to fibre perimeter ratio (B_B(O)) was calculated according to Mathieu-Costello *et al.* (1991), assuming a capillary diameter of 6 μm.

Endurance training

Six week endurance training programmes were performed on mechanically braked cycle ergometers which were calibrated with a purpose-built motor-driven device. Training bouts were undertaken at constant work rate for 30 min on five continuous days each week at a pedalling rate of between 90 and 95 rpm. For arm training, the crank axis was adjusted for each individual to mid-sternum level with the upper arms at 90° from the body, with slight elbow flexion in the extended position. The subjects were not restrained during arm training, aside from sitting on a high-backed chair with the feet kept in contact with the floor. The initial training power output for each individual was estimated from f_{card} and $[La]_b$ vs. power output relationships based on preliminary incremental performance tests for both leg and arm exercise cycloergometry. End-bout f_{card} during leg and arm training bouts was maintained at ≈98 ± 1% of $f_{card\,max}$ and training bouts were typically accompanied by a $[La]_b$ above 6 mmol L^{-1} for all but the first 5 min. Each subject was nearly exhausted at the end of each training bout and it took between 1 and 2 min for f_{card} to return to 130 beats min^{-1}. An increase of >15 W in power output midway through a training bout was unsustainable with a rapid increase in f_{card} and $[La]_b$ to maximal values and exhaustion.

Statistics

Values are reported as mean ± SE ($n = 6$). Paired Student's t-tests were used to identify significant differences between means before and after training or for changes due to training ($P < 0.05$).

RESULTS

Performance

$\dot{V}_{O_2\,max}$ significantly increased by a similar amount after muscle-specific training (6 ± 1 vs. 7 ± 2 mL

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min^{-1} kg^{-1}: legs vs. arms, $P > 0.05$). This was accompanied by similar increases in maximal power output for both legs and arms (58 ± 12 vs. 59 ± 7 W: legs vs. arms, $P > 0.05$). $f_{card\ max}$ was not affected by training, but $[La]_{b,max}$ increased following arm training (Table 1).

Gross muscle composition

Total cross-sectional area of the mid-thigh remained unchanged after leg training (4 ± 2%, $P > 0.05$), whereas upper arm total cross-sectional area increased significantly after arm training (+11 ± 3%, $P < 0.05$). The increase in total cross-sectional area in the upper arm was due to a significant increase in muscle cross-sectional area (+14 ± 3%, $P < 0.05$), whilst fat cross-sectional did not change (−5 ± 10%, $P > 0.05$). The increase of arm muscle cross-sectional area was due to a significant increase in flexors (25 ± 3 vs. 31 ± 4 cm^2, $P < 0.05$) rather than extensors (28 ± 4 vs. 31 ± 5 cm^2, $P > 0.05$: before vs. after arm training).

Muscle ultrastructure

Leg training led to a significant increase in total mitochondrial volume density (Vv(mt,f), +40 ± 11%, $P < 0.05$) in the legs, mainly due to a significant proliferation of intermyofibrillar volume density (4.1 ± 0.2 vs. 5.6 ± 0.2: before vs. after training, $P < 0.05$) rather than subsarcolemmal volume densities (0.9 ± 0.3 vs. 1.2 ± 0.2: before vs. after training, $P > 0.05$). Vv(mt,f) in arm muscle tended to increase after arm training (+16 ± 11%, $P > 0.05$). The increase in Vv(mt,f) in the legs after leg training was accompanied by a small, significant decrease in myofibrillar volume density (Vv(fi,f), −5 ± 1%) and a small, significant increase in fibre area (a(f), 14 ± 7%). Capillary density (Na(c,f), 15 ± 7%), capillary-to-fibre ratio (Nn(c,f), 31 ± 8%) and B_B(O) (23 ± 9%) all significantly increased after leg training in the legs, but only showed a tendency to increase in the upper arm after arm training (Table 1).

Combination of gross and ultrastructural data

V(mt) of the leg tissue slice significantly increased, by +42 ± 12%, following leg training, mostly due to the increase in Vv(mt,f) and not muscle volume (Mm; 2 ± 2%, $P > 0.05$; Fig. 1). On the other hand, V(mt) in the arm tissue slice significantly increased, by +31 ± 11%, following arm training due to a significant increase in Mm (14 ± 3%, $P < 0.05$) and a tendency for Vv(mt,f) to increase. J(c) of the leg tissue slice increased significantly following leg training (+19 ± 9%, $P < 0.05$), but only tended to increase in the arm tissue slice following arm training (+13 ± 10%,

Table 1 Changes induced by endurance training of either legs or arms in humans

		Legs	Arms
Performance			
$\dot{V}_{O_2\ max}$ (mL min^{-1} kg^{-1})	Before	49 ± 2	34 ± 1
	After	55 ± 2*	41 ± 2*
P_{max} (W)	Before	267 ± 14	130 ± 3
	After	325 ± 17*	190 ± 7*
$f_{card\ max}$ (min^{-1})	Before	186 ± 2	175 ± 2
	After	183 ± 3	177 ± 4
$[La]_{b,max}$ (mmol L^{-1})	Before	9.6 ± 0.3	8.0 ± 0.5
	After	9.4 ± 0.6	10.6 ± 0.9*
Muscle composition			
Total (cm^2)	Before	235 ± 3	72 ± 2
	After	244 ± 6	80 ± 3*
Muscle (cm^2)	Before	193 ± 6	56 ± 2
	After	196 ± 5	64 ± 3*
Fat (cm^2)	Before	35 ± 4	12 ± 1
	After	41 ± 5	12 ± 2
Bone (cm^2)	Before	7 ± 0	4 ± 0
	After	7 ± 0	4 ± 0
Muscle ultrastructure			
Vv(mt,f)	Before	5.0 ± 0.5	3.9 ± 0.2
	After	6.8 ± 0.2*	4.5 ± 0.4
Vv(li,f)	Before	0.5 ± 0.1	0.3 ± 0.1
	After	1.0 ± 0.3	0.5 ± 0.1
Vv(fi,f)	Before	81.0 ± 1.1	81.0 ± 0.7
	After	76.8 ± 0.9*	80.0 ± 1.0
Vv(re,f)	Before	13.9 ± 0.8	14.7 ± 0.7
	After	15.4 ± 0.8*	15.1 ± 0.8
Na(c,f) (mm^{-2})	Before	473 ± 14	460 ± 35
	After	540 ± 19*	451 ± 25 (4)
Nn(c,f)	Before	1.71 ± 0.12	1.60 ± 0.18
	After	2.21 ± 0.14*	1.77 ± 0.21 (4)
a(f) (um^2)	Before	3637 ± 299	3462 ± 225
	After	4083 ± 209*	3894 ± 348 (4)
B_B(O)	Before	0.12 ± 0.01	0.12 ± 0.01
	After	0.15 ± 0.1*	0.13 ± 0.01 (4)
V(mt) (mL)	Before	9.6 ± 0.7	2.2 ± 0.2
	After	13.3 ± 0.4*	2.9 ± 0.3*
V(fi) (mL)	Before	155.3 ± 6.1	45.1 ± 1.5
	After	150.8 ± 4.7	50.7 ± 2.6*
J(c) (km)	Before	11.2 ± 0.5	3.2 ± 0.3
	After	13.2 ± 0.7*	3.6 ± 0.5 (4)

Values are means ± SE; $n = 6$ except where noted.
*, significantly different from before training ($P < 0.05$).
$\dot{V}_{O_2\ max}$, maximal oxygen consumption; P_{max}, maximal power output; $f_{card\ max}$, maximal heart rate; $[La]_{b,max}$, maximal blood lactate concentration; total, total cross-sectional area from CT scans; muscle, cross-sectional area of skeletal muscle; fat, cross-sectional area of fat; bone, cross-sectional area of bone; Vv(mt,f), total mitochondrial volume density (unitless); Vv(li,f), lipid volume density (unitless); Vv(fi,f), myofibrillar volume density (unitless); Vv(re,f), remaining volume density (unitless); Na(c,f), capillary density; Nn(c,f), capillary to fibre ratio (unitless); a(f), mean fibre cross-sectional area; B_B(O), capillary to fibre perimeter ratio (unitless); V(mt), total volume of mitochondria in a 1 cm representative tissue slice; V(fi), total volume of myofibrils in a 1 cm tissue slice; J(c), total capillary length in a 1 cm tissue slice.

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$P > 0.05$). The training-induced increases in V(mt) or J(c) of legs and arms were not significantly different. V(fi) did not increase in the legs after leg training (−3 ± 3%, $P > 0.05$), but significantly increased in arms after arm training (+13 ± 4%, $P < 0.05$). The training-induced increase in V(fi) of arms was significantly greater than in the legs.

DISCUSSION

The new main finding of this study was primarily that leg training predominantly altered leg muscle ultrastructure and not muscle volume, whereas arm training led to increases in V(mt) and V(fi) mainly by gains in muscle volume. Thus, muscle-specific endurance training increases muscle-specific oxidative capacity by different mechanisms in arms than in legs.

Similarity of training programmes and differences in muscle specific adaptation

The results of leg training are quantitatively the same as in previous studies (Hoppeler *et al.* 1985, Rosler *et al.* 1985). Endurance training of the upper body has also been shown to increase arm-specific $\dot{V}_{O_2\,max}$ by an equal amount or to a greater degree (Magel *et al.* 1978, Bhambhani *et al.* 1991; Table 1). Importantly for this study, the increase in $\dot{V}_{O_2\,max}$ was similar after leg and arm training (i.e. +6–7 mL min^{-1} kg^{-1}). With this point of reference, the main hypothesis of this study was to test if training-induced changes in leg and arm muscle architecture were also similar in nature.

The increase in V(mt) following training was similar in legs and arms (+42 ± 12 vs. 31 ± 11%: legs vs. arms, $P > 0.05$); however, the increases were achieved in different ways. Changes in muscle ultrastructural composition, and not muscle volume, were primarily responsible for the training-induced effects in legs, whilst a significant increase in muscle cross-sectional area had a major role in adaptation of arm muscle to training. The design of the study may have been improved by (1) including a control 'non-exercise' group for comparison of training-induced changes, and (2) measurements of muscle force production (see below).

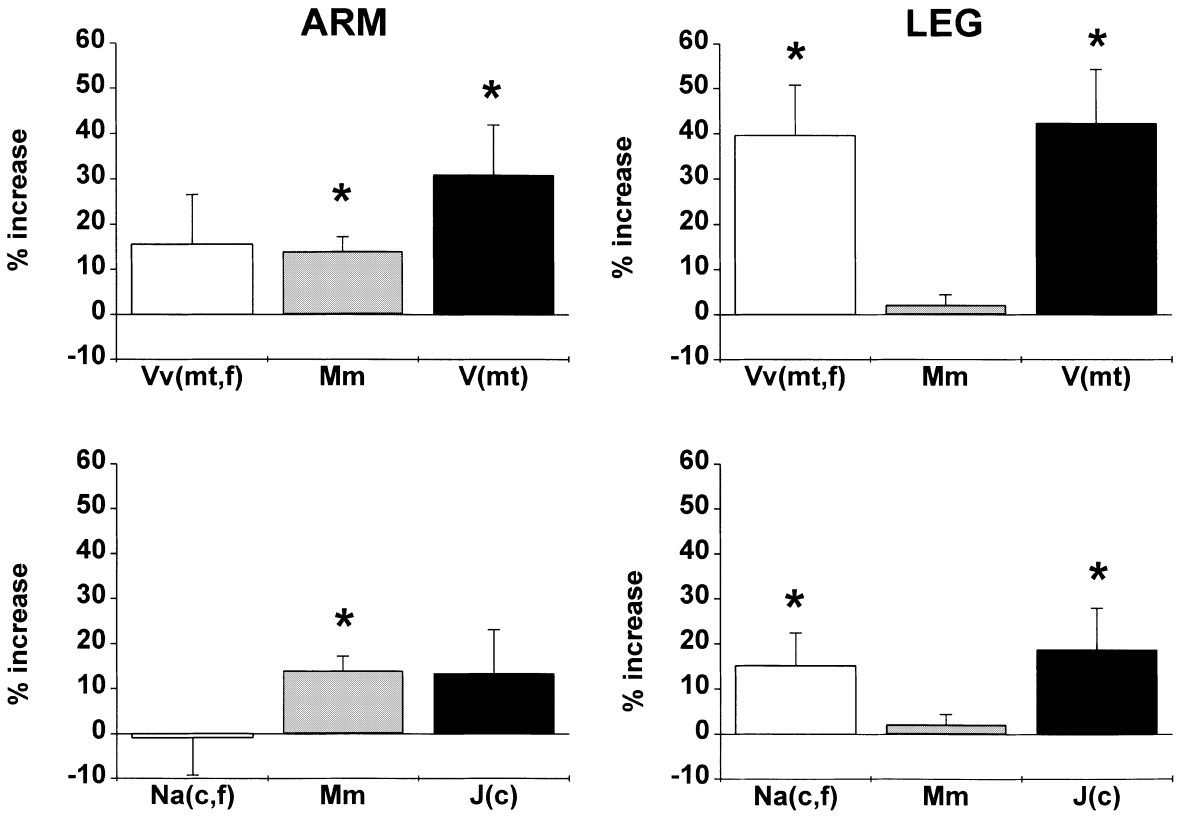

Figure 1 *Upper panel*: The changes (after – before training) of Vv(mt,f), muscle volume (*Mm*) and V(mt) in arms and legs induced by limb-specific endurance training. Values are mean ± SE. *, significant increase after training ($P < 0.05$ vs. zero percentage increase). *Lower panel*: The changes (after – before training) of *Na*(c,f), *Mm* and J(c) in arms and legs induced by limb-specific endurance training. *, significant increase after training ($P < 0.05$ vs. zero percentage increase).

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A surprising adaptation of arm muscle volume to endurance training

The mechanisms responsible for increasing muscle volume in arms in response to endurance training are not known. However, the marked increase in muscle volume and the consequent increase in $\dot{V}$(fi) of the arms following endurance training suggest that the arms were simultaneously adapting for both endurance and strength, the latter being proportional to muscle cross-sectional area (Dudley & Fleck 1987). Muscle hypertrophy is thought to be caused by high-intensity isometric/isokinetic contractions and is very muscle-specific (Jones *et al.* 1989), and it is striking that the increase in muscle cross-sectional area of the arms in this study (+14 ± 3%) was very similar to the increase in muscle cross-sectional area in legs, which increased isometric force production following strength training (Luthi *et al.* 1986).

The mechanism initiating muscle fibre hypertrophy may involve metabolic signals such as increased [H^+] and [La] or reduced intramuscular [ATP] and [PCr]. Alternatively, muscle fibre hypertrophy may result from physical damage or high intramuscular pressures. Arm muscles have a relative low muscle oxidative capacity (e.g. Vv(mt,f) of untrained arms was 3.9% vs. 5.0% in untrained legs). Additionally, the greater release of lactate and NH^{3+} during arm cycling compared with leg cycling of the same relative intensity has been correlated with a greater breakdown of ATP and a greater metabolic stress (Jensen-Urstad *et al.* 1993). Thus the mode of exercise used to train arm muscle may have accentuated the intensity of the putative metabolic stimuli for muscle hypertrophy. Mechanical stimuli may have been enhanced in this study, as some eccentric, isokinetic or isometric exercise probably occurred due to the posture adopted during training. Strength tests of upper arm before and after arm training were not planned *a priori*, but would be of obvious interest in future investigations.

The relationship between muscle oxidative demand and supply

Increased oxygen demand by a greater $\dot{V}$(mt) has been linked to a greater oxygen supply ($\dot{J}$(c)) at the peripheral level (Hoppeler *et al.* 1991). Capillary density, capillary to fibre ratio and the number of capillaries around all three human muscle fibre types in legs have been shown to increase following endurance training of 6–8 weeks' duration (Andersen & Henriksson 1977, Hoppeler *et al.* 1985, Howald et al. 1995, this study). These increases in indices of capillarity and the increase in B_B(O) after leg training are considered to represent angiogenesis (Hudlicka 1985). B_B(O) is an alternative index of oxygen supply potential that is relatively independent of errors due to muscle fibre hypertrophy and tortuosity (Table 1; Mathieu-Costello *et al.* 1991).

Strength training of legs (and the consequent muscle hypertrophy) normally leads to a reduction or 'dilution' of other muscle components such as capillaries and mitochondria (MacDougall *et al.* 1979, Luthi *et al.* 1986). However, despite a marked increase in muscle cross-sectional area, there was no 'dilution' of capillaries in the arms after arm training in this study; in fact there was a tendency for $\dot{J}$(c) and B_B(O) to increase after arm training. This finding may be interpreted as evidence suggesting that active angiogenesis is in fact occurring in the arms after arm training.

The relationship between oxygen demand (mitochondria) and supply (capillarity) can be expressed as the ratio $\dot{V}$(mt)/$\dot{J}$(c). This ratio was not significantly affected by either muscle specificity (i.e. leg vs. arm) or endurance training (0.9 ± 0.1 vs. 1.1 ± 0.1: before vs. after training of legs; and 0.7 ± 0.1 vs. 0.8 ± 0.1: before vs. after training of arms; repeated measures ANOVA, $P > 0.05$). Thus, in this study, the relationship between muscle oxygen demand and supply is maintained irrespective of muscle location or training history.

In conclusion, this study has shown that an endurance training programme can increase muscle-specific performance and oxidative capacity of similar proportions in legs and arms. Surprisingly, arm training also led to an increase in arm muscle volume, whereas leg muscle volume did not change after leg training. The relationship between muscle oxidative demand (mitochondria) and supply (capillarity) was maintained after endurance training in both leg and arm muscle, despite the different adaptations in muscle volume.

The authors would like to thank Liliane Tuescher and Cristain Moia for their expert technical support. This work was funded by Swiss National Foundation, grant 3.172–0.88 SR.

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