

Muscular characteristics of detraining in humans

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ABSTRACT

MUJICA, I., and S. PADILLA. Muscular characteristics of detraining in humans. *Med. Sci. Sports Exerc.*, Vol. 33, No. 8, 2001, pp. 1297–1303. Skeletal muscle is characterized by its ability to dynamically adapt to variable levels of functional demands. During periods of insufficient training stimulus, muscular detraining occurs. This may be characterized by a decreased capillary density, which could take place within 2–3 wk of inactivity. Arterial-venous oxygen difference declines if training stoppage continues beyond 3–8 wk. Rapid and progressive reductions in oxidative enzyme activities bring about a reduced mitochondrial ATP production. The above changes are related to the reduction in $\dot{V}O_{2\max}$ observed during long-term training cessation. These muscular characteristics remain above sedentary values in the detrained athlete but usually return to baseline values in recently trained individuals. Glycolytic enzyme activities show nonsystematic changes during periods of training cessation. Fiber distribution remains unchanged during the initial weeks of inactivity, but oxidative fibers may decrease in endurance athletes and increase in strength-trained athletes within 8 wk of training stoppage. Muscle fiber cross-sectional area declines rapidly in strength and sprint athletes, and in recently endurance-trained subjects, whereas it may increase slightly in endurance athletes. Force production declines slowly and in relation to decreased EMG activity. Strength performance in general is readily maintained for up to 4 wk of inactivity, but highly trained athletes' eccentric force and sport-specific power, and recently acquired isokinetic strength, may decline significantly. **Key Words:** TRAINING CESSATION, MUSCLE, ENZYMATIC ACTIVITY, FIBERS, STRENGTH

One of the most important characteristics of skeletal muscle is its dynamic nature. Skeletal muscle tissue has an extraordinary plasticity and is therefore able to adapt to variable states of functional demands, neuromuscular activity, and hormonal signals by reversibly changing its functional characteristics and structural composition (17,25,34,44). Physical training is a process consisting of a series of physiological stresses that bring about or preserve specific adaptations to enhance a subject's ability to tolerate the stressing factors arising from training (8,16,30,52). Therefore, training-induced skeletal muscle adaptations are such that the trained muscle increases its tolerance to exercise (30). On the other hand, inherent to the concept of training adaptation is the principle of training reversibility or detraining, according to which the stoppage or marked reduction of training leads to a partial or complete reversal of training-induced adaptations, thus compromising athletic performance (8,23,41). Skeletal muscle tissue is no exception to this rule and also readjusts to the reduced physiological stressors during periods of reduced training stimuli or complete training cessation (6,8,9,14,22,30,48,52,54).

It was the aim of this review to compile and briefly synthesize the data reported in the exercise science literature concerning the muscular characteristics of detraining, in both highly trained athletes, on the one hand, and moderately or recently trained individuals, on the other hand.

MUSCLE CAPILLARIZATION

The effects of training stoppage on capillary density in athletes have not been clearly established, as contradictory results have been reported in the literature. Indeed, Houston et al. (31) reported a 6.3% reduction in capillary density after only 15 d without training in well-trained endurance runners. As well, semiprofessional soccer players' number of capillaries around ST fibers decreased significantly from 6.0 to 5.8 after 3 wk of training cessation (3). In contrast with these results, Coyle et al. (11) reported that seven endurance-trained subjects' trained-state muscle capillarization was unchanged after 84 d without training and that capillarization in this population was about 50% higher than in sedentary controls. The authors discussed that the retention of the increased capillary density contributed to the observed partial maintenance of the ability to attain a high percentage of $\dot{V}O_{2\max}$ without large increases in blood lactate concentration.

The only available report on the effects of training cessation on recently trained individuals' muscle capillarization indicated that capillaries per mm^2 and capillaries per fiber represented, respectively, 108.7 and 106.3% of the pretraining baseline values after 4 wk of training stoppage, but these were significantly lower than the 121.7 and 120.3% values registered immediately after the 8-wk training program. In addition, posttraining capillaries around ST, FTa, and FTb fibers were 123.4, 120.8, and 129.7% of pretraining, but they declined to 108.6, 108.6, and 115.0% in 4 wk, and to 103.7, 108.6, and 112.2% in 8 wk of training cessation. In view of these results, the authors discussed that there was a favorable long-term effect on the average diffusion distance

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between the capillaries and the muscle fibers, due to a decreased fiber area during training cessation (35).

ARTERIAL-VEIN OXYGEN DIFFERENCE

To the best of the authors' knowledge, only one study has reported data on arterial-venous oxygen difference during training cessation in highly trained individuals (11). According to this study, short-term inactivity (21 d) did not bring about any change in the maximal arterial-venous oxygen difference of seven endurance-trained athletes (15.1, 15.1, and 15.4 mL·100 mL⁻¹ at days 0, 12, and 21 of training cessation, respectively). After 56 and 84 d without training, on the other hand, values dropped to 14.5 (4% reduction) and 14.1 mL·100 mL⁻¹ (7% reduction), respectively. These observations led the authors to suggest that the initial $\dot{V}O_{2\max}$ loss observed in athletes refraining from training is due to a decreased stroke volume, whereas the decreased arterial-mixed venous oxygen difference would be responsible for the reduction in $\dot{V}O_{2\max}$ observed between the 3rd and 12th wk of training cessation in endurance athletes (11).

MYOGLOBIN CONCENTRATION

In a 12-wk training cessation study, Coyle et al. (11) reported that myoglobin concentration in the gastrocnemius muscle of seven endurance-trained runners and cyclists (43.3 mg·g protein⁻¹) did not change significantly after 3 wk (41.0 mg·g protein⁻¹) and 12 wk (40.7 mg·g protein⁻¹) of training cessation. In addition, these muscle myoglobin concentrations were not different from that of eight sedentary controls (38.5 mg·g protein⁻¹).

ENZYMATIC ACTIVITIES

One of the main characteristics of muscular detraining is a marked decrease in skeletal muscle oxidative capacity, as shown by markedly reduced mitochondrial enzyme activities. Coyle et al. (10,11) observed that seven endurance-trained subjects' citrate synthase activity declined from 10.0 to 7.7 mol·kg protein⁻¹·h⁻¹ during the first 3 wk of training cessation, then continued declining to 6.0 mol·kg protein⁻¹·h⁻¹ by the 56th d, and stabilized thereafter. Succinate dehydrogenase, β -hydroxyacyl-CoA dehydrogenase, and malate dehydrogenase declined roughly in parallel with citrate synthase, i.e., about 20% in 3 wk and 40% in 56 d of training stoppage, also stabilizing thereafter at that level, which was 50% higher than that of sedentary counterparts. Quite similar results were reported by Chi et al. (4), with citrate synthase, succinate dehydrogenase, β -hydroxyacyl-CoA dehydrogenase, malate dehydrogenase, and β -hydroxybutyrate dehydrogenase declining an average of 36% after 42–84 d of training stoppage in endurance athletes. As in the previous study, detrained state oxidative enzyme values were nevertheless 40% higher than control. Interestingly, these authors also showed that mitochondrial enzyme levels decreased almost to untrained levels in ST fibers, but they remained 50–80% higher in FT fibers. During a 7-wk training/3-wk detraining paradigm, Moore et al. (40) ob-

served a 45% decline in citrate synthase activity in previously trained athletes, even though pretraining citrate synthase activity did not change in response to training. As well, Houmard et al. (27,28) reported a 25.3% decline in citrate synthase activity in a group of 12 distance runners who stopped training for 14 d. Similar results (27% decline in citrate synthase and β -hydroxyacyl-CoA dehydrogenase) have been reported in soccer players not training for 3 wk (3), triathletes (28.6% decline in citrate synthase activity) not training for 10 d (38), and adolescent soccer players (37.5% decline in citrate synthase activity) not training for 4–8 wk (2). Madsen et al. (37) reported a 12% lower β -hydroxyacyl-CoA dehydrogenase activity after 4 wk of insufficient training in nine endurance athletes, and Houston et al. (31) measured a 24% lower succinate dehydrogenase in six distance runners who did not train for 15 d. A similar 25% lower succinate dehydrogenase has been observed in six rugby league players' lateral head of the gastrocnemius muscle 6 wk after the end of their competitive season (1). Moreover, 16 male and female runners' skeletal muscle lipoprotein lipase activity was reduced by 45–75% during 2 wk of training cessation, whereas this enzyme's activity increased by 86% at the adipose tissue level. The adipose tissue lipoprotein lipase/muscle lipoprotein lipase ratio increased from 0.51 to 4.45, indicating a tendency for the storage of circulating lipids in the adipose tissue (47). It has been suggested that all of the above changes in mitochondrial enzymatic activities are primarily regulated by altered protein synthesis rates (30) and that the observed reductions are associated with the concomitant long-term reductions in $\dot{V}O_{2\max}$ and arterial-venous oxygen difference (1,11).

Training cessation has also been reported to induce small nonsystematic changes in glycolytic enzyme activities of the highly trained. In endurance athletes not training for 84 d, hexokinase decreased significantly by about 17%, phosphorylase did not change, phosphofructokinase increased nonsignificantly (about 16%), and lactate dehydrogenase increased significantly by about 20% (10). Chi et al. (4) also observed an identical 17% decline in hexokinase, whereas phosphorylase, phosphofructokinase, and lactate dehydrogenase increased by 3.6–21.1% in 42–84 d without training. Houston et al. (31), on the other hand, reported a 13% lower mean lactate dehydrogenase activity after 15 d of training cessation. Competitive swimmers not training for 4 wk showed nonsignificant declines in phosphorylase and phosphofructokinase activities in their posterior deltoid muscle (7). Phosphofructokinase also declined by 16% in rugby players not training for 6 wk (1) and by 54.5% in adolescent soccer players 4–8 wk after their competitive season (2). Glycogen synthase activity has also been shown to decline by as much as 42% after only 5 d without training in seven endurance-trained subjects (39).

Previously sedentary individuals taking part in training/detraining protocols also respond to training cessation with a decline in mitochondrial enzyme activities, which rapidly revert toward pretraining levels. During the inactive phase of a 7-wk training/3-wk detraining paradigm, Moore et al. (40) observed a 25% decline in citrate synthase activity in

previously sedentary subjects, but the final value was nevertheless 19% higher than before training. Using a similar 6-wk training/3-wk detraining protocol, Wibom et al. (53) reported a 4–17% decline in mitochondrial enzyme activities during training cessation, but citrate synthase, glutamate dehydrogenase, and cytochrome-*c* oxidase remained 33, 30, and 50% above pretraining levels, respectively. Klausen et al. (35) reported a progressive decline in succinate dehydrogenase and cytochrome oxidase activities during a training cessation period consecutive to an 8-wk training program. The 30–40% increase obtained in these enzymes through training completely disappeared by the 8th wk of inactivity. After 15 wk of mixed continuous and high-intensity intermittent training, 7 wk of training cessation resulted in a 21.2% decline in β -hydroxyacyl-CoA dehydrogenase and a 27.1% decline in oxoglutarate dehydrogenase activities. Only the former remained above pretraining values (46). Adolescent boys' vastus lateralis succinate dehydrogenase activity decreased to pretraining values in 6 months of training cessation subsequent to 3 months of endurance training, and below pretraining values after a sprint training/detraining paradigm of similar duration (15). Houston et al. (32) did not observe any significant change in the activities of succinate dehydrogenase and β -hydroxyacyl-CoA dehydrogenase, neither after 10 wk of dynamic strength training nor after 12 consecutive wk of training stoppage.

As in the case of highly trained athletes, recently trained individuals' glycolytic enzymes show rather insignificant changes during training/detraining protocols. Wibom et al. (53) reported a significant 17% increase in phosphofructokinase activity of the vastus lateralis muscle after 3 wk of training cessation in recently trained subjects. In contrast, the activities of phosphorylase, phosphofructokinase, and lactate dehydrogenase increased slightly during 8 wk of training but decreased to initial values during 8 subsequent wk of training cessation (35). As well, Fournier et al. (15) reported an increased phosphofructokinase activity after 3 months of sprint training but a return to baseline after 6 months of training cessation. Simoneau et al. (46), on the other hand, noted that hexokinase, phosphofructokinase, and lactate dehydrogenase activities were unchanged after 7 wk of training stoppage in six recently trained but previously sedentary subjects. Finally, Houston et al. (32) did not observe any significant change in the activities of enzymes representative of phosphagen (creatine kinase) and glycolytic (hexokinase, phosphofructokinase, and lactate dehydrogenase) metabolism, neither after 10 wk of dynamic strength training nor after 12 consecutive wk of training stoppage.

MITOCHONDRIAL ATP PRODUCTION

Even though these authors are unaware of the existence of any study reporting the consequences of training cessation on highly trained athletes' mitochondrial ATP production rate, one study carried out with recently trained individuals participating in a 6-wk endurance training/3-wk training

cessation protocol showed a 12–28% decrease in mitochondrial ATP production rate during the latter, resulting from a 4–17% reduction in individual mitochondrial enzyme activities (53). Considering that, as reported in the previous section, highly trained athletes' mitochondrial enzyme activities are markedly affected by training stoppage, it could be assumed that a marked reduction in mitochondrial ATP production would also take place in athletes undergoing a training cessation period. It is worth noticing, however, that mitochondrial ATP production rate remained 37–70% above pretraining levels in the above mentioned study (53).

MUSCLE FIBER CHARACTERISTICS

The effects of training cessation on muscle fiber distribution appear to be dependent on the duration of the inactivity period. Indeed, short-term training cessation was not enough to induce any change in the fiber distribution of six highly trained distance runners who wore a walking plaster cast for 7 d, then refrained from training for 8 additional days (31). The same was true in the case of four soccer players not training for 3 wk (3), and 12 strength-trained athletes not training for 14 d (26). Longer-term training cessation, on the other hand, has been shown to induce significant changes in the fiber distribution of athletes participating in various sports. Coyle et al. (10) reported a large progressive shift from FTa to FTb fibers in endurance runners and cyclists, the latter increasing from 5% in the trained state to 19% after 56 d of training cessation. Larsson and Ansved (36) reported that the proportion of ST fibers in four elite oarsmen decreased by 14–16% during the 4-yr period after their retirement from competition. An opposite tendency toward a higher oxidative fiber population has been observed in strength athletes. Indeed, a case study on one elite power lifter indicated that oxidative muscle fiber population was 1.4 times greater after 7 months without training (50), and Häkkinen and Alén (18) reported a reduction in %FT muscle fibers (from 66% to 60%) in an elite bodybuilder who underwent training cessation for 13.5 months. However, unchanged muscle fiber distribution has also been observed after 4–8 wk of training cessation in 14- to 15-yr-old soccer players (2). As well, a group of female dancers has been shown not to change their fiber type distribution after long-term (32-wk) training cessation, suggesting that their high percentage of ST fibers may have been the result of natural selection rather than a training-induced adaptation (12).

Mean fiber cross-sectional area has been shown to change during short-term training stoppage. Bangsbo and Mizuno (3), studying samples of the gastrocnemius muscle of male soccer players undergoing 3 wk of training cessation, reported a 7% decline in the mean fiber cross-sectional area. This change, however, was primarily due to a 12.4% decline in FTa fiber area, from 6022 to 5278 μm^2 . Similar results have been observed in 12 weight lifters, whose FT fiber cross-sectional area declined by 6.4% in 14 d. Interestingly, increases were observed in plasma concentrations of growth hormone (58.3%), testosterone (19.2%), and the

testosterone/cortisol ratio (67.6%), whereas cortisol and creatine kinase enzyme levels decreased respectively by 21.5 and 82.3%. According to the investigators, short-term training stoppage in strength athletes specifically affected FT fiber size, and changes in the hormonal milieu accompanying inactivity were propitious for an enhanced anabolic process, but the absence of the overload training stimulus prevented the materialization of such changes at the tissue level (26,28). On the other hand, 14 d of training stoppage did not result in a changed muscle fiber cross-sectional area in a group of endurance runners (27), and it even increased slightly (from 4.05 to 4.52 $\mu\text{m}^2 \cdot 10^{-3}$ in ST fibers, and from 4.20 to 5.22 $\mu\text{m}^2 \cdot 10^{-3}$ in FTa fibers) in a similar group of runners (31).

Longer periods of training stoppage also brought about declines in FT and ST fiber cross-sectional areas, the FT/ST area ratio, and muscle mass in athletes. It has been shown that rugby league players' cross-sectional area of FT fibers decreased to a greater extent than ST fibers, the former being 23% larger at the end of the season but only 9% larger after 6 wk without training. Further, an atrophy of the muscle bulk was suggested by the author in view of the fact that body mass decreased from 79.8 to 76.0 kg, but body fat content remained relatively constant during the inactive period (1). After 7 months of training cessation, an average atrophy of 37.1% was observed in all fiber types of a power lifter, along with a large fat-weight loss (50). As well, an elite bodybuilder's fat-free mass, thigh and arm girth, and average fiber area decreased by 9.3, 0.5, 11.7, and 8.3%, respectively, after 13.5 months without training. In addition, the FT/ST fiber area ratio decreased from 1.32 to 1.04 (18). Häkkinen et al. (21) also reported a reduction in the FT/ST muscle fiber area ratio from 1.11 to 1.04, and a reduced muscle mass after 8 wk of training stoppage in strength-trained athletes, as well as decreased FT and ST fiber areas after 12 wk without training (20). Larsson and Ansved (36) observed a 10% decrease in the relative area of ST fibers in oarsmen after long-term cessation of their athletic activity, and Amigó et al. (2) reported a reduction in the diameter of ST and FT muscle fibers in adolescent soccer players 4–8 wk after the competitive season. Dahlström et al. (12), on the other hand, measured a large increase in fiber areas after 32 wk of training cessation in female dancers, suggesting that smaller fibers were an endurance training-induced adaptation to decrease the oxygen diffusion distance.

In recently trained individuals, percentage distribution of fiber types were unaltered during 4 wk of inactivity that followed 8 wk of training, but by the 8th wk of training cessation, the percentage of ST fibers shifted significantly from 44.2 to 38.1%, that of FTa from 36.8 to 39.6%, and that of FTb from 19.1 to 22.2%. In addition, the cross-sectional areas of ST, FTa and FTb fibers, which had increased to 105.2, 104.5 and 104.9% of pretraining values after training, returned to 99.0, 98.9, and 96.5% within 4 wk of training cessation and declined further to 93.3, 98.6, and 94.3% by the 8th wk without training (35). In a 10 wk-training/12-wk detraining protocol, FTb fiber cross-sectional area increased by 18% with dynamic strength training

but decreased by 12% during inactivity (32). Interestingly, four young male subjects exhibited the same $0.10\% \cdot \text{d}^{-1}$ time course of increase during training and decrease during training cessation in their knee extensor muscles' cross-sectional area (42). Finally, the lean body mass of young female subjects training for 7 wk and not training for the subsequent 7 wk increased by 1.1 kg during the active period, but returned to near pretraining levels during the inactive time (49).

STRENGTH PERFORMANCE

According to the data reported in the exercise science literature, athletes can maintain, or suffer a limited decay, in their muscular strength during short periods of training stoppage. Fourteen days of training cessation did not significantly change 12 weight lifters' one-repetition maximum bench press (−1.7%) and squat (−0.9%) performance, isometric (−7%) and isokinetic concentric knee extension force (−2.3%), and vertical jump (1.2%) values. On the other hand, isokinetic eccentric knee extension force and surface EMG activity of the vastus lateralis decreased by 12% and 8.4–12.7%, respectively. The authors concluded that in strength athletes inactive for a short period of time, eccentric strength was specifically affected but that other aspects of neuromuscular performance were unaltered (26). It has also been shown that college swimmers maintained their muscular strength as measured on a swim bench during 4 wk of training cessation, but their swim power, i.e., their ability to apply the force during swimming, declined by 13.6% (43). Longer periods of training cessation are accompanied by more pronounced declines in the strength performance of strength-trained athletes, but this loss is still limited to 7–12% during periods of inactivity ranging from 8 to 12 wk. Häkkinen et al. (21) reported 11.6% and 12.0% decreases in squat-lift and leg extension forces, respectively, after 8 wk of training stoppage. In addition, maximal bilateral and unilateral isometric force decreased by 7.4% and 7.6%, respectively. This was coupled with decreased averaged maximal bilateral (5.6%) and unilateral (12.1%) IEMG. The latter change took place within the first 4 wk of inactivity (19). Results from the same group have shown that both muscle atrophy and a diminished neural activation are responsible for the decline in maximal force that takes place during 12 wk of training cessation, as FT and ST fiber areas, muscle mass, and maximal integrated electrical activity were shown to diminish with training stoppage (19–21).

Recently acquired strength gains appear to be lost at different rates depending on the type of strength performance measurement. Indeed, Shaver (45) reported that no significant amounts of newly acquired isometric strength gains were lost within 1 wk of training cessation after 6 wk of high-intensity strength training, neither in the trained arm (0.8%) nor in the contralateral arm (0.5%). On the other hand, significant losses were observed in both arms after 4 wk (2.0% and 1.3%), 6 wk (3.1% and 2.0%), and 8 wk (3.2% and 2.1%) of inactivity. However, it is worth noticing

that strength remained elevated above preconditioning levels. A 10-wk one leg dynamic strength training/12-wk detraining study showed that peak torque gains of the trained (39–60%) and untrained (12–37%) legs could be maintained during 4 wk of training cessation and that peak torque output remained above pretraining levels after 12 wk without training, despite 16–21% and 10–15% reductions in the trained and untrained legs, respectively. These force changes were associated with changes in both muscle size and neural factors (32). Similar kinetics have been shown to occur in the knee extensor muscles' cross-sectional area, maximal voluntary contraction and integrated EMG, the time course of increase for the training and that of decrease for training cessation being respectively $0.10\% \cdot d^{-1}$, $0.32\% \cdot d^{-1}$, and $0.7\% \cdot d^{-1}$. Again, these results indicate that hypertrophic and neural contributions to force development seem to exert the same weight during training and training cessation (42). Recently acquired levels of elbow flexors' isokinetic muscular endurance have been shown to decrease at a much higher rate during training stoppage, strength loss amounting 7% in 1 wk, 24% in 3 wk, and 27% in 5 wk (51). An 8-wk strength training/8-wk detraining study carried out with children aged 7–12 yr showed that training-induced strength gains were transient and reversible, as the weekly strength loss during training cessation averaged 3%, and the values of the previously trained children regressed toward untrained control group values within 8 wk of inactivity. These observations led the authors to suggest that children-specific maintenance training programs are necessary to maintain training-induced strength gains (13).

Colliander and Tesch (5) reported that functional strength (3 RM half-squat) was better preserved during 12 wk of inactivity after 12 wk of coupled concentric and eccentric resistance training (18%) than after the same period of concentric-only training (12%). This was basically due to the fact that the former training mode produced greater increases in strength than the latter. The authors concluded that performance of eccentric muscle actions is essential to promote greater and more long-lived neural adaptations to training. In keeping with this conclusion, strength gains achieved through 8 wk of eccentric-only resistance training have been shown to be retained at 100% in the trained limb and at 81% in the contralateral limb during a consecutive 8-wk training stoppage period (29). In addition, a group of college students maintained their physical qualities for speed-strength during a 1.5-month training break, but these decreased by 15.8% after 3 months of training cessation. Interestingly, maintenance of these characteristics was shown to be dependent on the training method used during the previous training period. Indeed, speed-strength was better maintained during training cessation if the previous training method focused on developing explosive strength

(24). Finally, it is worth noticing that in subjects accustomed to weight training without a competitive purpose, a slight decrease in maximal voluntary isometric contraction force and a large (22.5%) increase in the maximal rate of torque development have been reported after 8 wk of inactivity that followed 8 wk of isotonic strength training of the calf muscles, indicating that, during training cessation, the muscle itself can contract faster than during training, most probably due to a fatigue recovery effect (33).

CONCLUSIONS

Skeletal muscle tissue has a plasticity that allows it to adapt to variable levels of functional demands. During periods of marked reduction of physical activity or training cessation, muscular detraining occurs. This is often characterized by a decreased muscle capillary density, which in athletes could take place within 2–3 wk of training cessation. Arterial-venous oxygen difference, unchanged after short-term training stoppage, declines if inactivity continues. Myoglobin concentration, on the other hand, does not seem to be affected by training cessation. Rapid and progressive reductions in oxidative enzyme activities result in a reduced mitochondrial ATP production, and along with the reduced arterial-venous oxygen difference, are directly related with the reduction in $\dot{V}O_{2max}$ observed in individuals undergoing long-term training stoppage. Whereas these muscular characteristics remain above sedentary values in the detrained athlete, recently trained individuals' training-induced muscular adaptations most often return to pretraining values. Glycolytic enzyme activities show nonsystematic changes during periods of training cessation. Muscle fiber distribution remains unchanged during the initial weeks of inactivity, but there may be a decreased proportion of ST fibers and a large shift from FTa to FTb fibers in endurance athletes and an increased oxidative fiber population in strength-trained athletes within 8 wk of training stoppage. A general decline in muscle fiber cross-sectional area is rapidly measurable in inactive strength and sprint-oriented athletes, and in recently endurance-trained subjects, whereas fiber area may increase slightly in endurance athletes. Force production declines slowly and in relation with decreased EMG activity. Strength performance in general is thus readily retained for up to 4 wk of inactivity, but highly trained athletes' eccentric force and sport-specific power may suffer significant declines. The same is true for recently acquired isokinetic strength.

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