UNIT 1---- CONTINUED

Long-Term Adaptations to Exercise Training

Adaptations of Skeletal Muscle and Bone

Skeletal muscle adapts to endurance training chiefly through a small increase in the cross-sectional area of slow-twitch fibers, because low- to moderate-intensity aerobic activity primarily recruits these fibers (Abernethy, Thayer, Taylor 1990). Prolonged endurance training (i.e., months to years) can lead to a transition of FTb fibers to FTa fibers, which have a higher oxidative capacity (Abernethy, Thayer, Taylor 1990). No substantive evidence indicates that fast-twitch fibers will convert to slow-twitch fibers under normal training conditions (Jolesz and Sreter 1981). Endurance training also increases the number of capillaries in trained skeletal muscle, thereby allowing a greater capacity for blood flow in the active muscle (Terjung 1995).

Resistance-trained skeletal muscle exerts considerably more force because of both increased muscle size (hypertrophy) and increased muscle fiber recruitment. Fiber hypertrophy is the result of increases in both the size and number of myofibrils in both fast-twitch and slow-twitch muscle fibers (Kannus et al. 1992). Hyperplasia, or increased fiber number, has been reported in animal studies, where the number of individual muscle fibers can be counted (Gonyea et al. 1986), and has been indirectly demonstrated during autopsies on humans by using direct fiber counts to compare dominant and nondominant paired muscles (Sjöström et al. 1991).

During both aerobic and resistance exercise, active muscles can undergo changes that lead to muscle soreness. Some soreness is felt immediately after exercise, and some can even occur during exercise. This muscle soreness is generally not physically limiting and dissipates rapidly. A more limiting soreness, however, may occur 24 to 48 hours following exercise. This delayed-onset muscle soreness is primarily associated with eccentric-type muscle action, during which the muscle exerts force while lengthening, as can happen when a person runs down a steep hill or lowers a weight from a fully flexed to a fully extended position (e.g., the two-arm curl). Delayed-onset muscle soreness is the result of structural damage to the muscle; in its most severe form, this damage may include rupture of the cell membrane and disruption of the contractile elements of individual muscle fibers (Armstrong, Warren, Warren 1991). Such damage appears to result in an inflammatory response (MacIntyre, Reid, McKenzie 1995).

Total inactivity results in muscle atrophy and loss of bone mineral and mass. Persons who are sedentary generally have less bone mass than those who exercise, but the increases in bone mineral and mass that result from either endurance or resistance training are relatively small (Chesnut 1993).

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The role of resistance training in increasing or maintaining bone mass is not well characterized. Endurance training has little demonstrated positive effect on bone mineral and mass. Nonetheless, even small increases in bone mass gained from endurance or resistance training can help prevent or delay the process of osteoporosis (Drinkwater 1994).

The musculoskeletal system cannot function without connective tissue linking bones to bones (ligaments) and muscles to bones (tendons). Extensive animal studies indicate that ligaments and tendons become stronger with prolonged and high-intensity exercise. This effect is the result of an increase in the strength of insertion sites between ligaments, tendons, and bones, as well as an increase in the crosssectional areas of ligaments and tendons. These structures also become weaker and smaller with several weeks of immobilization (Tipton and Vailas 1990), which can have important implications for musculoskeletal performance and risk of injury

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