Exercise and Intensity : Implications for Oxidative Stress and Muscle Damage

Editorial

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Exercise induced muscle damage is the temporary, repairable muscle injury which is commonly experienced following performance of physical activity. Eccentric exercise models have been used to study exercise induced muscle damage because it is this component of total muscular work which elicits most myofibrillar disruption and the greatest release of muscle proteins, such as creatine kinase and myoglobin into the blood [1]. Delayed muscle soreness, and loss of strength often accompany the myofibrillar disruption, but the exact relationship between these indices has not been fully established. The relative frequency of myofibrillar disturbances evident immediately following unaccustomed exercise increases in subsequent days due to some secondary degradation process.

This process may be initiated by the loss of intracellular calcium homeostasis which could activate a number of proteolytic and lipolytic systems [2]. Infiltration of mononuclear cells also occurs in the days after unaccustomed exercise. Release of secondary degradation products may increase osmotic pressures in the vicinity of the damage, and this mechanism may then account for the elevated intramuscular fluid pressures and muscle swelling that may occur the days after exercise completion.

A growing amount of evidence indicates that free radicals play an important role as mediators of skeletal muscle damage and inflammation [3]. Free radicals are molecules with an unpaired electron in their outer orbit. When aerobic animals respire, oxygen becomes reduced to form water, during this process, ATP is also formed. However, certain physical restrictions dictate that oxygen can only receive one electron at a time, and four electrons are required to produce water. This univalent pathway of oxygen reduction transiently leads to the production of free radicals [4]. Most studies implicate aerobic exercise as the fundamental cause of elevated levels of oxygen centred free radicals (e.g; superoxide radicals O_2•, hydroxyl radicals OH•; hydroperoxyl radicals HO_2; and lipid peroxyl radicals LOO; Alessio, et al. [4]. During exercise, two of the potentially harmful free radical generating sources are semiquinone in the mitochondria and xanthine oxidase in the capillary endothelial cells. During high intensity exercise the flow of oxygen through the skeletal muscle cell is greatly increased at the same time as the rate of ATP utilisation exceeds the rate of ATP generation. The metabolic stress in the cells causes several biochemical changes to occur, resulting in a markedly enhanced rate of production of oxygen free radicals from semiquinone and xanthine oxidase.

Consequently, a substantial attack of free radicals on the cell membranes may lead to a loss of cell viability and to cell necrosis, and could initiate the skeletal muscle damage and inflammation caused by exhaustive exercise in addition to any beneficial training effect. Takala, et al. [5] investigated the effects of 30 min running with stepwise increasing intensity (exhaustive, energy demand approx 50-100% of $\dot{V}\ O_2$ max), 60s supramaximal running (anaerobic, greater than or equal to 125% of $\dot{V}\ O_2$ max) and 40-60 min low intensity running (aerobic, 40-60% of $\dot{V}\ O_2$ max) on serum concentrations of muscle derived proteins.

Carbonic anhydrase III (CAIII) was used as a marker of protein leakage from type I (slow oxidative) muscle fibres and myoglobin (Mb) as a non-selective (type I and II) muscle marker. The fractional increase in S-CA III was 0.37, 0.1 and 0.46 1 hour after exhaustive, anaerobic and aerobic exercise respectively. The corresponding values for delta Mb were 1.45, 0.39 and 0.67. The value for the delta CAIII / Mb ratio was 0.68 after the aerobic exercise, but only 0.25 - 0.26 after the high intensity exercise. Since type I fibres of skeletal muscle are known to be responsible for power production during low intensity exercise, whereas fibres of both typeI and type II are active at higher intensities, the delta CAIII / delta Mb ratio may depend on the recruitment profile of type I vs type I + II. Increased serum concentrations of intracellular proteins are generally accepted as good indicators of muscle damage. Goodman, et al. [1] investigated protein concentrations following running performance. Twenty male runners completed a 21 km run in as fast as possible. Blood samples were obtained from each subject pre, immediate post and 24 hr after the run. Samples were analysed for haemoglobin, haematocrit, creatine kinase (CK), myoglobin (Mb) and malondialdehyde (MDA) concentrations and corrected for changes in plasma volume (PV).

Percutaneous muscle biopsies were taken from the lateral gastrocnemius muscle of the six subjects 24 h before and 24 h after the run and examined by electron microscopy. Mb levels in the serum increased significantly ($P < 0.05$) immediately post - exercise, while CK levels increased ($P < 0.05$) at 24 hours post - exercise. The PV corrected serum MDA levels were not significant immediately post - exercise ($P > 0.05$). Ultrastructural

examination of pre - exercise samples revealed evidence of muscle changes consistent with exercise. No further damage was evident at 24 hours post - exercise. It was therefore suggested that the increased levels of CK and Mb may be the result of free radical induced cell membrane damage and increased permeability, as evidenced by elevated serum MDA levels, and not due to mechanical muscle damage.

Recent research by Alessio, et al. [4] has suggested that due to different metabolic demands of isometric and aerobic exercise, the mass action effect of $\dot{V}O_2$ can be dismissed as the sole mechanism for exercise induced oxidative stress.

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