www.jcimcr.org

JCIMCR OPEN ACCESS Journal of Clinical Images and Medical Case Reports

ISSN 2766-7820

Short Commentary

Open Access, Volume 3

High intensity exercise performance and muscle damage. A role for free radicals

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Received: Nov 09, 2022

Accepted: Nov 30, 2022

Published: Dec 07, 2022

Archived: www.jcimcr.org

DOI: www.doi.org/10.52768/2766-7820/2189

Abstract

The purpose of this paper is to outline muscle damage and oxidative stress responses to high intensity exercise protocols. The information provided will be important for athletes, coaches, and the general population to provide information related to mechanisms and consequences of high intensity exercise performance. The information will help with recovery strategies from high intensity exercise and consider injury implications for athletic populations. Further to this, the paper outlines the role of antioxidants in the recovery process following exercise and the health benefits for reducing oxidative stress damage.

Introduction

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 myofibrillar disruption, but the exact relationship between these indices has not been fully established. The relative frequency of myofibrillar disturbancesare evident immediately following unaccustomed exercise, and increase in subsequent days due to secondary degradation processes. This process may be initiated by the loss of intracellular calcium homeostasis which could activate several 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 [2]. Most studies implicate aerobic exercise as the fundamental cause of elevated levels of oxygen centered free radicals (e.g.; superoxide radicals $O_2\cdot$, hydroxyl radicals OH·; hydroperoxyl radicals $HO_2\cdot$; and lipid peroxyl radicals LOO·; [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 utilization 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 [5]. Investigated the effects of 30 min running with stepwise increasing intensity (exhaustive, energy demand approx. 50-100% of VO_2 max), 60s supramaximal running (anaerobic, greater than or equal to 125% of VO_2 max) and 40-60 min low intensity running (aerobic, 40-60% of VO_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 fibers 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 fibers of skeletal muscle are known to be responsible for power production during low intensity exercise, whereas fibers of both type I 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. [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 analyzed for hemoglobin, hematocrit, 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 [4] has suggested that due to different metabolic demands of isometric and aerobic exercise, the mass action effect of VO2 can be dismissed as the sole mechanism for exercise induced oxidative stress.

As mentioned earlier, a free radical is a molecule with an unpaired electron in its outer orbital and is produced during normal cellular metabolism. High levels of radicals can damage cells by reacting with cellular components (e.g., proteins and lipids). This form of damage is called oxidation and can result in a lethal injury to cells [3]. Given that radicals are produced during normal metabolism, it is not surprising that cells contain antioxidants (molecules that eliminate radicals) to reduce the risk of radical-mediated injury. Two major classes of antioxidants work together to reduce the potentially harmful effects of radicals: 1) enzymatic antioxidants and 2) non-enzymatic antioxidants. Key antioxidant enzymes include superoxide dismutase, glutathione peroxidase, and catalase [2]. These enzymes are manufactured in the cell and cannot be obtained through dietary supplementation. Important non-enzymatic antioxidants include vitamins E and C, and beta carotene. These antioxidants are included in many foods and can also be obtained through dietary supplements. Vitamin E and beta-carotene are lipid-soluble antioxidants and protect cell membranes from radical damage. Vitamin C is a water-soluble antioxidant and works in conjunction with vitamin E to protect both lipids and proteins in the cell from radical damage.

Radical damage

The adverse effects of excess free-radical formation have been hypothesized to lead to cancer, atherosclerosis, aging, and even exercise-associated oxidative damage [4]. Free radicals are generated from normal oxidative processes in the body and can damage DNA and RNA and inactivate enzymes and other proteins. Free radicals also facilitate oxidation of fatty acids in cell membranes, producing destructive chain reactions that cause cell damage and cell death.

Excess levels of free radicals in plasma and the arterial wall increase Low-Density Lipoprotein (LDL) oxidation leading to cytotoxicity and enhanced plaque formation [2]. Aerobic organisms would not survive without mechanisms that counteract the detrimental effects of free radicals. The system includes the fat-soluble antioxidants such as vitamin E and beta-carotene (a vitamin A precursor); the major water-soluble antioxidant, vitamin C; antioxidant enzymes such as Superoxide Dismutase (SOD), Catalase (CAT), and selenium-dependent Glutathione Peroxidase (GPX); and low-molecular-weight compounds such as glutathione [3]. These components preserve homeostasis during most normal cell function and mild oxidative stress. When free-radical production is excessive, however, or when the antioxidant system is overwhelmed, such as during nutritional deficiencies or exhaustive exercise, such imbalances may favor an “oxidative stress” environment.

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