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Exercise Induced Muscle Damage and Immunosuppression: A Hindrance to Optimum Performance

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Exercise is beneficial for health and improves one's level of performance. Different exercise protocols are prescribed by medical practitioners not only to prevent but also to cure serious health disorders. Traditional indigenous exercises are also of immense beneficial significance for healthy wellbeing. However, practicing an improper or unscientific training protocol may on the other hand be detrimental to health. Therefore, training guideline should be prescribed by certified personnel and it should be properly adhered to achieve optimum outcome.

There are certain health parameters which react in an adverse manner in response to exercise stress. Even well trained individuals also respond in the same fashion as sedentary people do. For instance, high intensity exercise, training or athletic activity such as competitive running, football etc., impose adverse health effects, e.g., temporary suppression of immune system, exercise induced damage of skeletal and cardiac muscle, hemolysis etc, which not only impair the health of the athletes but also impacts their subsequent athletic performance. Vigorous physical activities lead to damage of cardiac muscle, skeletal muscle and RBC due to oxidative stress resulting from free radical overload that is generated from high intensity exercise and that ultimately impairs athletic performance. Poor nutritional status also makes a person more vulnerable to such adverse effects of exercise.

Exercise Induced Skeletal Muscle Damage

Exercise Induced Muscle Damage (EIMD) results in a temporary decrease in muscle force production due to loss of contractile components of muscle cells, damage to the excitation-contraction (E-C) coupling system, a rise in passive tension, increased soreness and inflammation of the involved muscle group, pain and an increased release of intramuscular proteins. Eccentric exercise leads to a loss of sarcoplasmic reticulum membrane integrity and causes flux of Ca^{2+} into intracellular areas which initiates a cascade of Ca^{2+} dependent proteolytic and phospholipolytic pathways that damage the structural and contractile myofilaments, sarcolemma, cytoskeleton, sarcoplasmic reticulum and mitochondria leading to fibre necrosis [1]. The loss of membrane integrity allows the 'leakage' of intramuscular proteins, e.g., creatine kinase, lactate dehydrogenase, aspartate aminotransferase, alanine aminotransferase, troponin I, myoglobin, myosin heavy chains etc.

Histological examination showed that EIMD involves focal disruption of the myofibrils and cytoskeleton, resulting in Z–disk streaming and damage in mitochondria and the sarcoplasmic reticulum. The factors responsible for Exercise induced muscle damage are of metabolic or mechanical in nature.

EIMD also occurs due to metabolic deficiencies caused due to ischaemia or hypoxia during prolonged exercise. Histological study on marathon runners showed that damage was focal and confined to muscle fibres with complete glycogen depletion.

Exercise Induced Cardiac Muscle Damage

High intensity exercise also imparts considerable oxidative and metabolic stress in cardiac muscle leading to damage of cardiac myocytes and their sarcoplasmic reticulum resulting in increased risk of myocardial infarction during and one hour post exercise. This leads to a series of cardiac dysfunctions collectively known as cardiac fatigue. The myocardium is one of the most aerobic organs having a large mitochondrial density and high oxygen flux. Cardiac fatigue is marked by transitory contractile dysfunction following termination of exercise and there is evidence of cardiac myoctye damage and necrotic episode following high intensity exercise. Free radical production resulted in the disruption of calcium uptake rates of sarcoplasmic reticulum of cardiac muscle and Ca^{2+} ATPase activity. Prolonged endurance exercise results in elevation of specific biomarkers of myocardial injury such as Creatine kinase MB (CK-MB) and Cardiac Troponin T (cTnT) [2,3].

Exercise and Immune Suppression

Exercise stress imposes both positive and negative effects on immune function. Moderate physical activity stimulates immune function resulting a 29% reduction in the incidence of upper respiratory tract infection while vigorous exercise and deliberate heavy training causes temporary suppression of various immune responses, e.g., decreased NK-cell function, impaired lymphocyte proliferation and T- and B-cell functions, decreased in-vitro immunoglobulin production, pro-inflammatory cytokine cascade activation, and altered expression of cytokine receptors and phagocyte activation. Such exercise induced immunosuppression lasts for 3–24 h after exercise depending on the intensity, duration and chronicity of the exercise bouts making the athlete prone to infectious diseases. Exposure to airborne pathogen increases during exercise due to increased rate and depth of breathing that makes an athlete 100–500% more susceptible to infection [2].

C–Reactive Protein (CRP) and plasma Interleukin–6 concentration largely increases during exercise. Contracting muscle fibers release these cytokines and this phenomenon is correlated with muscle injury, inflammation and muscle protein breakdown [4]. Salivary IgA level decreases following intense long-duration exercise although submaximal exercise had no effect on this parameter. Muscle derived IL–6 stimulates hypothalamus to release adrenocorticitropic hormone that in turn increases plasma cortisol level which causes lymphocytopenia, monocytopenia, eosinopenia, and neutrophilia that reach their maximum 4 h after administration. Increased levels of stress hormones during exercise possibly promote entry of less

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mature leukocytes into the circulation from the bone marrow and this may contribute to decreased concentration of immune cells. During exercise, the CD4+ to CD8+ lymphocyte ratio decreases, reflecting the greater increase in CD8+ lymphocytes than CD4+ lymphocytes. The percentage of CD3+ cells (pan T cells) was shown to decline during exercise, whereas CD20+ cells (B cells) did not change in relation to exercise. Moderate exercise boosts neutrophil chemotaxis, phagocytosis and oxidative burst activity whereas a high intensity exercise reduces these functions.

Probable remedial measures to combat the exercise induced muscle damage and immunosuppression

Exercise induced skeletal and cardiac muscle damage and immunosuppression are normal physiological phenomenon which are potentially detrimental towards excelling optimum performance and delays the process of recovery. Therefore it is of significant scientific interest to reduce the quantum of these factors among athletes for the betterment of performance. Since more than last one decade, researchers have tried various nutritional and pharmacological interventions to alleviate these damaging effects of high intensity exercise. However, nothing could be conclusively postulated in this regard.

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