The Asian Journal of Animal Science (June, 2011), Vol. 6 Issue 1 : 8-13 | RESEARCH ARTICLE

Exercise prevents ethanol-induced oxidative stress : regulation of selected dehydrogenase activities in the skeletal muscle fibers of male albino rat with reference to aging

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Received : December, 2010; Accepted : January, 2011

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ABSTRACT

Alcohol consumption is associated with several injuries of organs, including liver, lung, heart as well as skeletal muscle; as a result of numerous physiological, morphological and functional changes occur in the tissues. Wistar strain male albino rats of two age groups 3 months (young) and 18 months (old) were divided into four groups, Group I sedentary control(SC); Group II, exercise trained (ExT) (30 min, at a speed of 23 m/min/day/5 days/week for a period of 8 weeks); Group III, ethanol treated (Et) (20% ethanol, 2 gm/kg body weight); Group IV, exercise trained + ethanol treated (ExT + Et) as described in group II and group III. The animals were sacrificed after 24 hours of the last treatment by cervical dislocation and the skeletal muscle fibres such as gastrocnemius (GN) and soleus (SOL) were isolated from the hind limbs of rats and selected enzymes such as succinate dehydrogenase (SDH), lactate dehydrogenase (LDH), malate dehydrogenase (MDH) and isocitrate dehydrogenase activites were assayed. The activities of SDH, LDH and MDH were increased in both skeletal muscle fibers of ExT group. However, a significant decrease was observed in the same parameters with ethanol intoxication in both skeletal muscle fibers when compared to their sedentary control group. From the results obtained in this study, we conclude that 8 weeks exercise training is beneficial to the animal in preventing the ethanol induced toxicity.

Key words : Exercise, Alcohol, Skeletal muscle fibers, Dehydrogenases, Albino rat

Exercise physiology is one of the active fields of research in modern days, since it presents the material essential for understanding relevant changes in various mechanisms of the body that occur during the onset of exercise. As the body is subjected to repeated bouts of exercise, long term adaptation in the bodily function occur (Wilmore, 1982). Exercise can be accomplished only through a series of complex interactions within the body involving all body systems. Exercise increase the oxygen consumption rate by 10 to 15 folds and may result in oxidative stress due to increased production of reactive oxygen species. It also enhances the antioxidant enzyme activity (AOE). Exercise generates adenosine as a break down product of ATP and provides cytoprotection against oxidative stress. Arther Simman (1988) reported increased blood cell composition in exercising animal. It is generally accepted that the increase in mitochondrial oxidative capacity that occurs with training leads to the altered metabolic response to exercise. Spina *et al* . (1996) have shown an increase in the activity of some mitochondrial enzymes during short-term training and have associated, with this smaller increase in blood lactate and lower respiratory exchange ratio values during sub maximal exercise after training. However, Phillips *et al*. (1996) have shown that the metabolic adaptations will occur before increase in enzyme activities during the same type of training.

Alcohol abuse is associated with severe damage of several biological functions and activities within the cell (Harper and Krill, 1990; Lieber, 1991), including electrolyte homeostasis (Gandhi and Ross, 1988).Chronic intake of ethanol produces alterations in several tissues, including skeletal muscle. In chronic alcohol patients, progressive wasting and weakness of proximal muscle groups seems to be associated with lesions of skeletal muscle (Urbano-Marquez *et al*., 1989). Ethanol has been described as producing atrophy of human type II fibres (Martin and Peter, 1985) and sometimes necrosis (Urbano-Marquez *et al*., 1985) in patients affected by alcohol. While some authors consider that the direct effect of ethanol on

Chennaiah, K., Khalindar Basha, K., Sivasankar, R., Ramaiah, K. and Sathyavelu Reddy, K. (2011). Exercise prevents ethanol-induced oxidative stress : regulation of selected dehydrogenase activities in the skeletal muscle fibers of male albino rat with reference to aging. *Asian J. Animal Sci.,* **6**(1): 8-13.