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Research Article

Recovery Of Respiratory Fatigue After Acute Exhaustive Exercise: Role Of Antioxidant Supplementation Versus Exercise Training

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ABSTRACT

Aim: To assess the effect of antioxidant supplementation versus exercise training on ventilatory hyperapnoea and respiratory rate retrieval time as an indicator of recovery of respiratory fatigue following acute exhaustive exercise.

Material and method: Thirty six medical undergraduate students participated in this study. The subjects were divided into three groups of twelve each. One group received placebo, second received antioxidant supplementation and third group received regular exercise training for twelve weeks. All subjects underwent first exercise challenge (CH1). After CH1 the work output (W1), post exercise respiratory rate (RR1) and the duration in which the post exercise respiratory rate retrieved to pre-exercise levels (TR1) was recorded. Three months later all subjects underwent second exercise challenge (CH2) after which the work output (W2), post exercise respiratory rate (RR2) and the duration in which the post exercise respiratory rate retrieved to pre-exercise levels (TR2) was recorded.

Results: The work output in both antioxidants supplemented and exercise trained group was significantly increased ($p < 0.001$). Subjects in both the groups could also perform the exhaustive exercise challenge for a significantly longer duration ($p < 0.001$ in antioxidant group and $p < 0.01$ in exercise trained group) as compared to the control group.

Conclusion: Antioxidant supplementation and exercise training separately can cause an increase in the capacity of work performance and also shorten the recovery time of exercise induced hyperapnoea.

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INTRODUCTION

Exercise-induced diaphragmatic fatigue is considered to represent an important factor limiting maximal ventilation and exercise performance.^[1-5] It has been well established that diaphragmatic fatigue as measured by trans diaphragmatic pressures lasts for at least 30 min after exhaustive exercise.^[6,7]

The data of Johnson et al^[1] suggests that the diaphragm is contributing less and accessory inspiratory and expiratory muscles more to the production of hyperventilation response as the duration of exercise proceeds. However they also found that diaphragmatic fatigue was not relieved until more than one hour of recovery from exercise as assessed by the average decrease in trans diaphragmatic pressure. They also concluded that trans diaphragmatic pressure values recovered partially after 30 minutes post exercise and almost completely by an average time of 70 minutes. Thus, the measurement of respiratory muscle fatigue by measurement of transdiaphragmatic pressures seems to be incomplete as it does not take into account the fatigue incurred by accessory expiratory muscles. Moreover, it remains an experimental approach which is difficult to apply in clinical setting.

Therefore, in this study we have considered two parameters as indirect indicators of ventilatory fatigue: 1) The ventilatory hyperpnoea at the end of exhaustive exercise, 2) Time taken for recovery of hyperapnoea to eupnoea. Initial reports regarding the role of oxidative stress in diaphragmatic fatigue came in 1990s.^[8] Since then many studies have reported that quenching these reactive oxygen species which are formed during submaximal contractions by using various enzymatic and non enzymatic antioxidants helps in delaying muscle fatigue.^[9-15] Although supplementation with antioxidants has been shown to decrease oxidative stress as indicated by a decrease in the markers of lipid per oxidation whether such supplementation also causes lesser ventilatory fatigue in response to an oxidative challenge is not known.^[16,17]

Exercise provides an excellent model to study the dynamic balance between oxidative challenge and antioxidant defense in biological systems. Strenuous exercise in a person who is unaccustomed to exercise will induce oxidative damage. However, high intensity endurance training is known to enhance the antioxidant capacity of the diaphragm.^[18,19]

In this study was plan to assess the effect of antioxidant supplementation and exercise training on the ventilatory hyperpnoea at the end of exhaustive exercise and the time taken for recovery of hyperapnoea to eupnoea as indicators of recovery of ventilatory fatigue following acute exhaustive exercise.

METHOD

The present study was conducted for three months on 36 healthy male medical student volunteers after approval from the research and ethics committee of the institute. Written informed consent taken from the participants. The mean age, height, weight and surface area of 21.08 ± 0.99 yrs, 173.75 ± 5.57 meters, 57.66 ± 6.05 Kg, 1.79 ± 0.10 m² respectively. All these subjects had not been involved in any previous exercise training and were not consuming any oral antioxidants. They were divided into three groups as follows.

Group PL: The subjects maintained their normal routine and received a placebo capsule containing dextrose each day throughout the study period.

Group AO: The subjects maintained their routine and received a capsule of antioxidant once daily (E-Carotin, Franco Indian Pharmaceuticals Pvt. Ltd.). Each capsule contained β carotene 10 mg, Vitamin E acetate IP 25 mg, Vitamin C IP 150 mg, Selenium dioxide monohydrate USP 61.8 mg.

Group EX: The volunteers while maintaining their routine and underwent high intensity exercise training on friction type bicycle ergometer, six days a week, at a frictional force (ΔT) of 0.75 kg and pedal revolution rate of 60-80 per minute for eight minutes.

Assessment at the beginning of study

After reporting to the laboratory the subjects were allowed to rest on a chair for half an hour and then their respiratory rate was recorded with the help of an electronic respiratory transducer carefully secured in front of one nostril connected to a single channel physiograph recorder. After this the subjects underwent an acute episode of exercise challenge (CH1) as described in detail in our pervious study. [20] The exercise challenge was terminated when the heart rate achieved was 75% of the maximum predicted heart rate. After terminating the exercise challenge the immediate post exercise RR was recorded every third minute until it approached the pre exercise value. This recovery time (TR1) was also recorded. The work output (W1) was calculated and the total duration of exercise was noted.

Assessment at the end of study: After the end of three months the pre exercise respiratory rate was again determined in all groups before

commencement of a second bout of acute exhaustive incremental exercise challenge (CH 2) after which the post exercise RR recording was made as after CH1. Also the work output (W2) and the duration of exercise (T2) were noted.

Statistical evaluation: The data was statistically analysed at 95% level of confidence ($p < 0.05$) using students 't' test.

RESULT

The mean duration for which subjects could perform exercise challenge at the beginning of the study was 10.25 ± 1.98 , 10.45 ± 1.57 and 10.33 ± 0.98 minutes in PL, AO and EX groups respectively. However at end of study period, the subjects in AO and EX groups could perform the exercise challenge for a significantly longer duration ($p < 0.001$ in EX group and $p < 0.01$ in AO group) (Table 1). Also the work output in CH 2 was significantly increased in both AO and EX groups in comparison to work output in CH1.

Table 1: Work output (W), duration of exercise (T) and normalization time of respiratory rate (TR) after first and second exercise challenge

	1 st exercise challenge			2 nd exercise challenge		
	W1 (Watt) (Mean \pm SD)	T1 (min) (Mean \pm SD)	TR1 (min) (Mean \pm SD)	W2 (Watt) (Mean \pm SD)	T2 (min) (Mean \pm SD)	TR2 (min) (Mean \pm SD)
Placebo group	213.3 ± 60.3	10.3 ± 2	34.3 ± 11.8	255.1 ± 67.5	11.1 ± 1.6	37.8 ± 3.9
Antioxidant group	206.6 ± 61.5	10.5 ± 1.6	34.8 ± 11.8	311.8 ± 65.2	13.0 ± 2.1	27.8 ± 9.1
Exercise group	227.7 ± 38.7	10.3 ± 1	33.8 ± 13.4	370.2 ± 86	14.5 ± 1.7	25.8 ± 11.0

In all groups the increment in RR after CH1 and CH2 showed insignificant difference, ($p > 0.05$) (Table 2).

Table 2: Increment in respiratory rate after first and second exercise challenge in various groups

	Change in respiratory rate after first exercise challenge (Mean \pm SD)	Change in respiratory rate after second exercise challenge (Mean \pm SD)
Placebo group	11.7 ± 5	13.1 ± 4.7
Antioxidant group	13.3 ± 2.6	11.8 ± 4.5
Exercise group	13.5 ± 3.7	15.7 ± 4.8

The respiratory rate normalization time in PL group after CH1 was insignificantly different from that after CH2 (Table 1). However in the volunteers taking antioxidant regularly the normalization time for respiration was 27.75±9.14 minutes after CH2 which was significantly less than the normalization time in placebo group at the end of the study ($p < 0.05$). In the exercise training group the normalization time of RR after CH2 was 25.75±11.03 which was significantly lower than the corresponding normalization time in placebo group after CH2. However no significant difference in TR1 and TR2 was seen between AO and EX groups.

DISCUSSION

It is well known that respiratory muscle fatigue is due to reactive oxygen species (ROS). A review^[15] mentions that during skeletal muscle contractions production of ROS increases which causes muscle fatigue. The latter is aggravated by antioxidant depletion and diminished by administration of exogenous antioxidants. It has also been reported that vitamin C supplementation is beneficial in recovery from unaccustomed exercise.^[21]

Earlier studies^[22,23] have shown that exercise training leads to an augmented antioxidant system and a decrease in lipid peroxidation. It has also been stated that endurance exercise enhances the oxidative and antioxidant capacity of the diaphragm.^[24] It has been shown previously that fatigued respiratory muscles need more than one hour for recovery.^[25] In our study the mean duration of recovery from exercise induced hyperpnoea in AO and EX groups was 27.75±9.14 and 25.75±11.03 minutes respectively (Table 2) which was much quicker than in the placebo group which showed respiratory recovery at 37.75±13.92 minutes after CH2. This could presumably be because of an increased protection offered by antioxidants to the respiratory muscles in AO group or as a result of enhanced endogenous antioxidant system in EX group.

Two ways have been suggested in which whole body exercise may contribute to diaphragmatic fatigue^[26]

1) Increasing levels of circulating metabolites originating in contracting and fatiguing loco motor muscles i.e. increased lactate, H^+ , K^+ , Pi. The recovery period of diaphragmatic fatigue of more than one hour after whole body endurance exercise may be associated with a slow return of blood lactate and acid base status to normal in the post exercise period. Potassium is an important humoral factor in the regulation of exercise ventilation. It has been reported that during incremental exercise testing the patterns of change in ventilation and arterial K^+ are similar suggesting that change in arterial K^+ may lead to the phenomenon of the ventilatory threshold through the action on peripheral chemoreceptors.^[27] However it had been earlier shown that the time constant of the exponential decline in potassium was 30 seconds faster than the changes in ventilation at the end of incremental exercise.^[28] It could be possible that antioxidant administration or exercise training could have decreased the post exercise rise in levels of or more of these metabolite, or could have caused faster return of the increased levels of metabolites to normal, thereby decreasing the RR retrieval time in the present work. This however needs further exploration.

2) Exercise leads to a compromised blood flow to the diaphragm since now it has to compete with exercising loco motor muscles. Thus there might be reduced oxygen for the given amount of ventilatory work during exercise and therefore, inadequate oxygen transport in relation to the metabolic demand placed on the diaphragm.

It has been suggested that the flux of carbon dioxide to the lungs is an important determinant of ventilatory drive during recovery and that neither central command nor neural afferents from contracting muscles are requisite for the control of ventilation during recovery from exercise.^[29]

CONCLUSION

We found that for the same degree of rise in post exercise respiratory rate after first and second exercise challenge, the duration of performance of the exhaustive exercise test in both antioxidant supplementation and exercise training group was enhanced at the end of the study. There was faster recovery of respiratory hyper apnea to eupnoea in subjects taking antioxidants and exercise training in when compared to placebo group.

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