IMPACT OF VITAMIN E SUPPLEMENTATION DURING INDUCED CIGARETTE SMOKING- LUNG MUSCLE ATROPHY

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Abstract—Vitamin E is a major antioxidant. Male albino rats were induced with cigarette smoke for 30 days to assess the induced oxidative damage due to lung muscle atrophy. An enhanced lipid per-oxidation was recorded with elevated activity levels of conjugated diens, malondialdehyde and other thiobarbituric acid reactive substances (TBARS) in denervated muscle. The activity levels of antioxidant defense enzymes, viz. superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (selenium, non selenium)(GPx), glutathione reductase (GR), glutathione-s-transferase (GST) were depleted in the cigarette smoke induced lung muscle. When the Experiment control animal (cigarette smoke induced muscle) was supplemented with vitamin E, revealed a depleted lipid peroxidation and increased activity levels of antioxidant defense enzymes. Thus, suggesting that the supplementation of vitamin E, could prevent the oxidative damage in the lung muscle despite smoking.

Keywords—Oxidative stress, cigarette smoke, Vitamin E, antioxidants

I. INTRODUCTION

Vitamin E (α-tocopherol) is considered one of the most important dietary antioxidant in biological systems due to its association with cell membrane and its ability to act directly on reactive oxygen species (ROS) preventing peroxidation [1,2,3,4]. Besides the well characterized function of vitamin E as antioxidant alternative roles such as that of a membrane stabilizer [5,6] and a regulation of membrane fluidity [7,8] have been proposed. The protective role of vitamin E on the oxidative stress develops due to antileprosy chemotherapy in leprosy patients was recorded [9,10,11].

In the present study was carried out to evaluate the impact of vitamin E supplementation on the oxidative stress during induced cigarette smoking in lung muscle atrophy [12, 13,14]. The cigarette smoke induced animals kept for 30 days were fed with vitamin E (α-tocopherol) of 200mg/kg body wt. [15,16,17,18] for 30 days [17,19,20].

II. EXPERIMENTAL PROTOCOL

The rats were divided into 3 groups comprising of 10 animals in each group.

Group I Control-sham operated animals (C)
Group II cigarette smoke induced kept for 30 days animals-Control (CSC30)
Group III cigarette smoke induced animals after 30 days given vitamin E supplementation (CS vit E30)

A. Cigarette smoke induced
Daily two cigarettes were given to male albino rats in the morning and evening for 30 days. [21].

B. Vitamin E supplementation
Rats were fed with vitamin E (α-tocopherol) of 200mg/Kg body weight, as reported earlier [15, 16], for 30 days. [20,21].

C. Sample Preparation
The experimental animals were sacrificed by cervical dislocation at the end of the appropriate experimental period. The various experimental groups viz. C, (CSC30) (CS vit E30) were dissected and the lung muscles were removed and washed with deionized water, weighed, stored at -20 °C until further analysis.

D. Oxidative damage assay
Oxidative damage assay in the lung muscle tissue was assessed by measuring the levels of TBARS and conjugated dienes in all the lung muscle groups viz. C, (CSC30) (CS vit E30) by the method of Ohkawa and klein respectively.[22,23].

E. Antioxidant enzyme assay
Enzymes involved in antioxidant enzyme defense system such as super-oxide-dismutase by the method of Beauchamp and Fridovich [24], catalase by the method of Chance and Machly [25], glutathione peroxidase (selenium dependent and Non selenium dependant) by the method of Rotruck,[26], glutathione reductase by the method of Racker,[27], glutathione-s-transferase by the method of Habig et al.[28] were measured to assess the antioxidant defense mechanism in lung muscle of all 3 groups of animals.
III. RESULTS

TBARS and conjugated dienes were recorded a significant decrement of 37.16% and 26.46% (Table) respectively indicating lowered lipid peroxidation in the cigarette smoke induced muscle supplemented with vitamin E. The study on the antioxidant defense enzyme revealed a significant elevation of the activity levels of enzymes viz. SOD, CAT, GPx (selenium and non selenium dependant), GR and GST in the lung muscles of vitamin E fed cigarette smoke induced animals the elevation of activity levels of enzymes ranges 36.96%, 92.16%, 134.15%, 70.27%, 96.15%, 127.01% respectively (Table).

IV. DISCUSSION

The increased lipid peroxidation in the cigarette smoke dis-use lung muscle of rat might be due to increased generation of reactive oxygen species (ROS) in the muscle thereby disturbing both enzymatic and non enzymatic antioxidant defense system in the muscle. Vitamin E (α-tocopherol) serves as potent peroxyl radical scavenger. Excess generation of ROS may overwhelm natural antioxidant defenses such as muscle membrane vitamin E leading to lipid peroxidation in further contributing to muscle damage [7,29,30,8].

Therefore in the present study has been carried out to evaluate the antioxidant effect of vitamin E dietary supplementation on oxidative stress during induced cigarette smoke disuse lung muscle atrophy [31].

The decrease levels of TBARS and conjugated dienes in the present study indicate the reduced lipid peroxidation which might be due to the non enzymatic antioxidant vitamin E impact on the disuse lung muscle. Similar studies where vitamin E reduced lipid peroxidation were recorded [7, 32]. The deranged antioxidant enzymatic defense system with the depleted activity levels of SOD, CAT, GPx (selenium and non selenium dependant), GR and GST were significantly restored indicating the elevated activity levels of SOD, CAT, GPx (selenium and non selenium dependant), GR and GST in the lung muscle of cigarette smoke induced (experimental control) animals supplemented with vitamin E. This might be due to the free radicals scavenging act of vitamin E, thus reducing the free radical concentration and the probable regain of the antioxidant enzymatic defense system.

The above results envisage that the supplementation of vitamin E is an important lipid soluble antioxidant in vivo, and it is presumed that its principle role is to protect membrane lipids from lipid peroxidation in vivo, by scavenging lipid alkoxy or peroxy radicals which are capable of abstracting hydrogen from adjacent polyunsaturated lipid molecules to propagate a lipid peroxidation reaction and thus prevent the muscle damage due to oxidative stress.

Table – Levels of Thiobarbituric acid reactive substances (TBARS) and conjugated dienes (CD), antioxidant enzymes [Super-oxide-dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx) (both selenium and non selenium dependant), Glutathione reductase (GR) and glutathione s-transferase (GST) in control (C), Experimental control (cigarette smoke induced) (CSC30) and Cigarette smoke induced treated with vitamin E (CS vitE30)]. Values are mean ± SD from 10 animals from each group. Figures in parenthesis are % increase (+) or % decrease (-) over control (C) in Experimental control (cigarette smoke induced) (CSC30) and over Experimental control (cigarette smoke induced) (CSC30) in cigarette smoke induced treated with vitamin E (CS vitE30).

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control (C)</th>
<th>Experimental control (cigarette smoke induced) (CSC30)</th>
<th>Cigarette smoke induced, treated with vitamin E (CS vitE30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TBARS*</td>
<td>0.956±0.3</td>
<td>2.115 ± 0.02 (+121.23)</td>
<td>1.329±0.04 (-37.16)</td>
</tr>
<tr>
<td>CD*</td>
<td>1.5±0.05</td>
<td>2.23±0.02 (+93.91)</td>
<td>1.64±0.06 (-26.46)</td>
</tr>
<tr>
<td>SOD *</td>
<td>0.85±0.007</td>
<td>0.46±0.01 (-45.88)</td>
<td>0.63±0.009 (+36.96)</td>
</tr>
<tr>
<td>CAT *</td>
<td>0.14±0.001</td>
<td>0.51±0.0003</td>
<td>0.098±0.006</td>
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</table>
**REFERENCE**


<table>
<thead>
<tr>
<th></th>
<th>GPx&lt;sup&gt;H&lt;/sup&gt;</th>
<th>GPx non selenium&lt;sup&gt;µ&lt;/sup&gt;</th>
<th>GR&lt;sup&gt;R&lt;/sup&gt;</th>
<th>GST&lt;sup&gt;R&lt;/sup&gt;</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>0.113±0.002</td>
<td>0.041±0.001</td>
<td>0.096±0.002</td>
<td>0.56±0.32</td>
</tr>
<tr>
<td></td>
<td>(-55.26)</td>
<td>(+92.16)</td>
<td>(-134.15)</td>
<td>(+127.01)</td>
</tr>
</tbody>
</table>

Unit: *µ moles/mg protein; µ units/mg protein/ min

P values :<0.001


