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EFFECTS OF VITAMINS C AND E ON STRESS-INDUCED LIPID PEROXIDATION AND ANTIOXIDANT-ENZYME ACTIVITIES IN RATS

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ABSTRACT

Immobilization stress induces the formation of reactive oxygen species and leads to the oxidative stress injury in various tissues. The present study was carried out in order to assess the effect of immobilization stress on the level of lipid peroxides and activities of antioxidant enzymes, and the protective role of the antioxidant vitamins E (α-tocopherol) and C (ascorbic acid) against the reactive oxygen species (ROS) produced by immobilization stress. Forty adult male albino rats were used in the present study; Animals were divided into four groups (10 rats each). (1)control group (2)stressed group (subjected to immobilization for 4 hours) (3)stressed group, treated by α-tocopherol, 10mg/kg, p.o daily for 2 weeks (4)Stressed group, treated by ascorbic acid, 80mg/kg, p.o daily for 2 weeks. Levels of thiobarbituric acid reactive substances (TBARS) level and activities of the antioxidant enzymes, superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GSHPx) in liver and heart were determined. In this study a significant dramatic increase in liver and heart TBARS level in immobilized group has been recorded, this may indicate the generation of ROS as a result of immobilization stress. A significant decrease in the activity of liver and heart SOD and GSHPx have been observed. The endogenous antioxidant mechanisms were fortified by administration of the free radical scavengers, vitamin C and E. A significant reduction in liver and heart TBARS level was recorded. Moreover, the antioxidant enzymes significantly restored their activities by administering the antioxidant vitamins, E and C. It is concluded that immobilization stress induces lipid peroxidation and alters the antioxidant enzyme activities, the protective role of vitamins C and E may be due to stimulation of the antioxidant enzyme synthesis or reduction of their consumption.

INTRODUCTION

Immobilization stress is a typical psycho-physiological stress which induces the formation of ROS that are capable of damaging various body components [1]. There is an increased production of superoxide radical or anion ($O_2^{\bullet-}$) under physiological stress in rats [2] and in human [3]. Oxidative stress is a cellular and physiological condition of elevated concentration of ROS [4] and is viewed as a continuous battle between inducers (pro-oxidants) and a vast array of different protective factors (antioxidants) [5].

Tissues have different oxidative loads and aggregated antioxidant capacity that differs as a result. It seems that each tissue has its own composition of antioxidants according to the oxidizing processes, it is most likely to encounter. Therefore, tissues respond differently to free radicals [6].

There are an expanding list of antioxidants; they can be divided into enzymatic and non-enzymatic antioxidants. Enzymatic antioxidants include SOD, CAT, GSHPx, glutathione reductase (Grd), glutathione transferase (Gt) [7]and DNA repair enzymes [8]. While non-enzymatic antioxidants contain a group of certain redox active low molecular mass molecules such as glutathione (GSH) [9], vitamin C [10], vitamin E [11], bilirubin [12] and albumin [13].

The most important water soluble antioxidant in the extracellular fluid is vitamin C. It has many cellular activities as an antioxidant as well. Vitamin C has been shown to scavenge $O_2^{\bullet-}$, hydrogen peroxide (H_2O_2), hydroxyl radical ($OH^{\bullet}$), peroxyl radical and singlet oxygen [14]. While vitamin E, which is a term that encompasss a small lipophilic group of related tocopherols, is operative in a membrane or lipoprotein particles. α-Tocopherol, the most active form of vitamin E, is probably the most efficient antioxidant in the lipid phase, which is believed to represent its major biochemical function. High levels are found in tissues such as liver and fatty tissues [14].

Understanding the tissue's antioxidant status may allow the prediction of the tissue undergoing oxidative stress. The aim of this work was to study the changes that take place in the enzymatic antioxidants' defense and lipid peroxidation of the heart and liver as a result of immobilization stress. The possible protection by the antioxidant vitamins C and E supplementation was investigated.

MATERIALS & METHODS

The present study was conducted on 40 adult male albino rats, obtained from the National Institute of Drug Control and Research. Their average body weight was 150-200 g; food and water were allowed ad libitum. Animals were left to acclimatize to the environment for two weeks prior to the experiments. Rats were divided into 4 groups as follow:

Normal control group (10 rats): left freely moving in their cages throughout the experiment.

Stressed group (10 rats): Subjected to immobilization stress for 4 hours [17]. Stressed, vitamin E-supplemented group (10 rats): They received an oily solution of α-Tocopheral orally at a dose of 10 mg/kg body weight daily for two weeks, then subjected to immobilization stress for 4 hours [15].

Stressed, vitamin C-supplemented group (10 rats): 0.54 g vitamin C was dissolved in a liter of drinking water provided as the only available drink. Each rat drank

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30 ± 3 ml/day in a dose equivalent to approximately 80-100 mg/kg body weight of ascorbic acid supplied daily for each rat for 2 weeks, then subjected to immobilization stress for 4 hours [16].

Rats were stressed by fixing the four limbs of each rat to a wooden board and leaving it at room temperature (25-30°c) throughout the period of the experiment (four hours) [17]. The control group was not allowed to see, hear or smell the rats being stressed to avoid the possible psychological stress. At the end of the experimental period, each rat was anesthetized with ether and sacrificed by cervical dislocation.

The abdomen was immediately opened; the heart and liver tissues were rapidly excised. They were cleaned with saline and accurately weighted then minced into small pieces and suspended in a suitable volume of ice cold (0.05M) potassium phosphate buffer (pH 7.8) containing 1 mM EDTA to get a concentration of 10% (W/V). Homogenization was performed for 1 minute with the aid of a motor driven homogenizer at 5000 rpm on an ice background [18]. The homogenate was then centrifuged at 10000 rpm for 20 minutes at 4°c using cooling centrifuge to get supernatant that contains lipid peroxide and antioxidant enzymes, SOD, GSHPx and CAT. SOD was determined as described by Minami and Yoshikawa, [19], GSHPx activity was measured as mentioned by Paglia and Valentine, [20], CAT activity by Aebi, [21], lipid peroxide (TBARS) by Ohkawa et al,0 [22] and protein Content by Lowery et al, [23]. All samples were analyzed immediately.

RESULTS

In cardiac tissue homogenate, GSHPx activity showed a significant decrease in stressed group compared to normal control group (Stressed, 32 ± 1 vs Control, 75 ± 2, P<0.01, Figure 1-A,). On the other hand, in stressed groups supplemented with vitamin E or vitamin C GSHPx activity showed significant increase compared to stressed group (57 ± 2 and 95 ± 2, respectively, P1<0.01, Figure 1-A). While stressed group supplemented with vitamin E showed a significant decrease compared to normal control group (P<0.01), stressed group supplemented with vitamin C showed a significant increase in GSHPx activity compared to the control (P<0.01).

Figure 1-B shows that in cardiac tissue homogenate, SOD activity was decreased significantly in stressed group compared to the normal control group (Stressed, 5 ± 1.3 vs Control, 25 ± 1.2, P<0.01). However, stressed groups supplemented with vitamin E or vitamin C showed a significant increase in SOD activity compared to the stressed group (Vit. E, 17.1 ± 2.47, Vit. C, 35 ± 5 vs tressed, 5 ± 1.3, P1<0.01, Figure 1-B). On the other hand, stressed group supplemented with vitamin E showed a significant decrease compared to the normal control group, while stressed group supplemented with vitamin C showed a significant increase compared to the normal control group (P<0.01).

Lipid peroxide level showed a significant increase in the stressed group compared to the normal control group (Sressed, 237.11 ± 11.2 vs Control, 132.6 ± 5.21, P<0.01, Figure 1-C). On the other hand, stressed groups supplemented with vitamin E or vitamin C showed a significant decrease of lipid peroxides compared to the stressed group (166.9 ± 6.1 and 150.4 ± 14.6, respectively, P1<0.01, Figure 1-C), but these values were significantly higher than the control group (P<0.01). No significant difference in CAT activity has been observed among the studied groups.

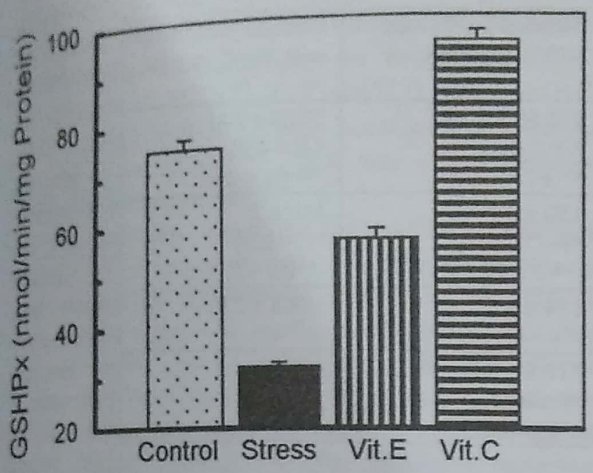
Table (1) shows means ± SD of lipid peroxides (TBARS) level, SOD activity, GSHPx activity and CAT activity in cardiac tissue homogenate. On the other hand, table (2) shows means ± SD of lipid peroxides (TBARS) level, SOD activity, GSHPx activity and CAT activity in hepatic tissue homogenate.

Table (1): Means ± SD of lipid peroxides (TBARS) level, SOD activity, GSHPx activity and CAT activity in cardiac tissue homogenate.

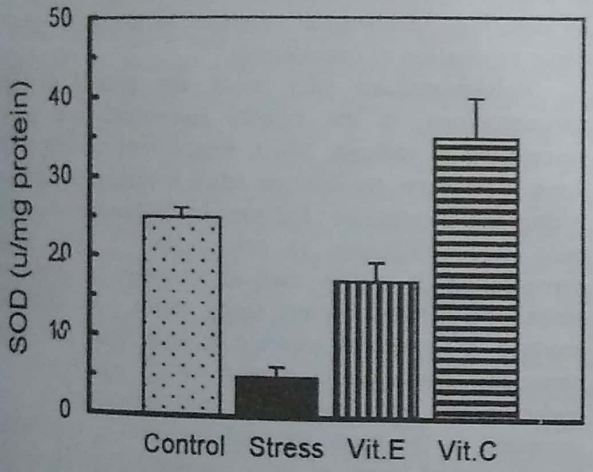
Studied gps / Biochem. Param.	Control group (n =10)	Stressed group (n = 10)	Stressed group suppl. with Vit. E (n = 10)	Stressed group suppl. with Vit. C (n = 10)
SOD (U/mg protein)	25 ± 1.2	5 ± 1.3 * P < 0.01	17.1 ± 2.47 * P < 0.01 ** P1 < 0.01	35 ± 5 * P < 0.01 ** P1< 0.01
GSHPx (nmol/min/mg protein)	75 ± 2	32 ± 1 * P < 0.01	57 ± 2 * P < 0.01 ** P1 < 0.01	95 ± 2 * P < 0.01 ** P1 < 0.01
CAT (U/mg protein)	26.1 ± 3.1	26.7 ± 2.7	24.2 ± 3.97	26.3 ± 2.79
Lipid peroxides (TBARS) (nmol/g wet wt)	132.6 + 5.21	237.11 ± 11.2 * P<0.01	166.9 ± 6.1 * P< 0.01 ** P1<0.01	150.4 ± 14.6 * P < 0.01 ** P 1< 0.01

P: compared with control group; P1 compared with stressed group
P< 0.01 significant.

A. changes in GSHPx in cardiac tissue homogenate.



B.Changes in SOD activity in cardiac tissue homogenate.



C. Changes in TRARS in cardiac tissue homogenate.

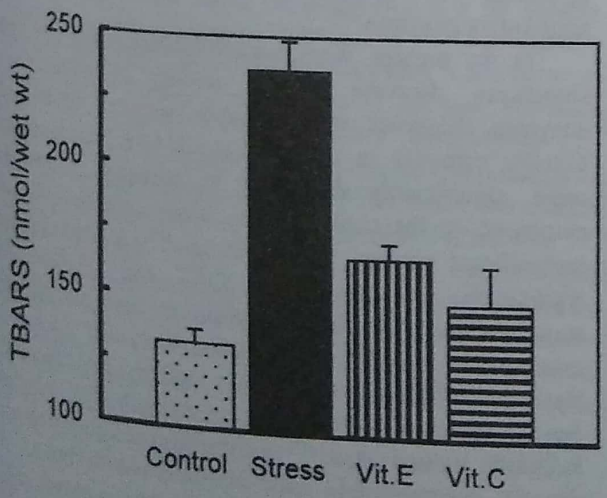
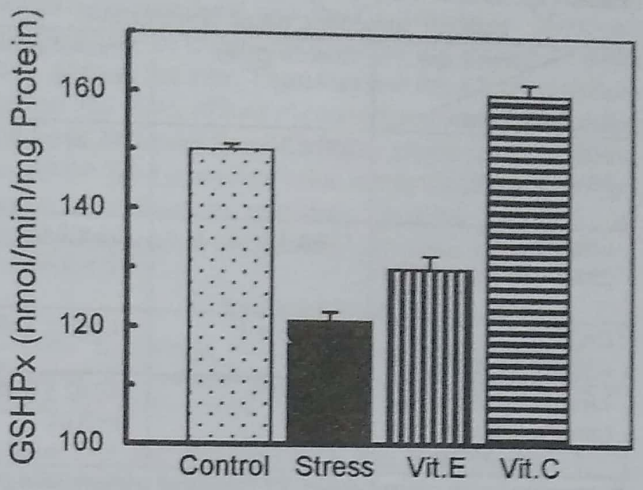
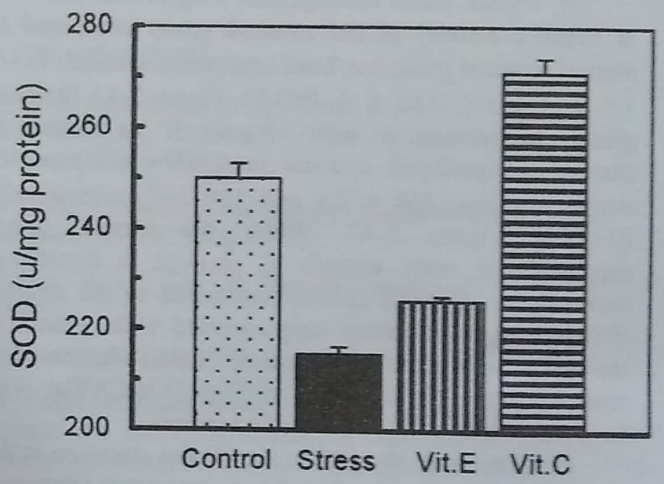


Figure 1: Effects of vitamins E(10 mg/kg, p.o, daily for 2 weeks) and C supplement (0.54 g/l drinking water) on the levels of A) glutathione peroxidase, B) superoxide dismutase and C) thiobarbituic substances in cardiac tissue homogenate in rats.

A. changes in GSHPx in hepatic tissue homogenate.



B.Changes in SOD activity in hepatic tissue homogenate.



C. Changes in TRARS in hepatic tissue homogenate.

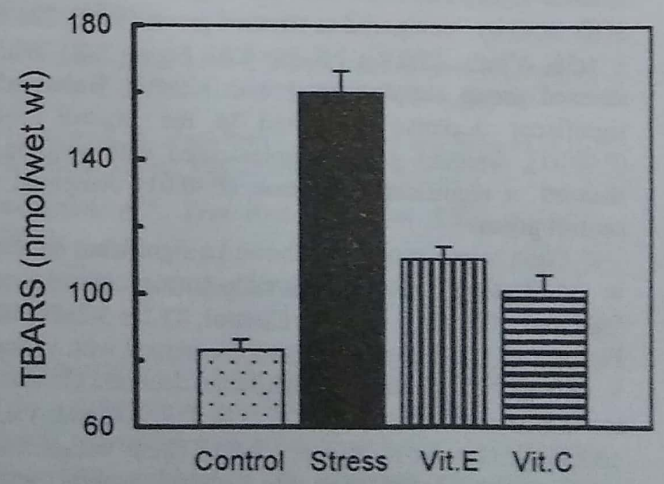


Figure 1: Effects of vitamins E(10 mg/kg, p.o, daily for 2 weeks) and C supplement (0.54 g/l drinking water) on the levels of A) glutathione peroxidase, B) superoxide dismutase and C) thiobarbituic substances in hepatic tissue homogenate in rats.

Table (2): Means ± SD of lipid peroxides (TBARS) level, SOD activity, GSHPx activity and CAT activity in hepatic tissue homogenate.

Studied gps / Biochem. Param.	Control group (n = 10)	Stressed group (n = 10)	Stressed group suppl with Vit. E (n = 10)	Stressed group suppl with Vit. C (n = 10)
SOD (U/mg protein)	250 ± 3	215.16 ± 1.36 * P < 0.01	225.60 ± 1.02 * P < 0.01 ** P1 < 0.01	270.9 ± 2.8 * P < 0.01 ** P1 < 0.01
GSHPx (nmol/min/mg protein)	150 ± 1	121 ± 1.6 * P < 0.01	130 ± 2.3 *P < 0.01 ** P1 < 0.01	160 ± 2 *P < 0.01 **P1 < 0.01
CAT (U/mg protein)	116.1 ± 4.4	112.05 ± 3.60	114 ± 3.8	118.8 ± 2.60
Lipid peroxides (nmol/g wet wt)	83.2 ± 3.2	160.02 ± 6.48 *P<0.01	110.4 ± 3.8 *P< 0.01 **P1<0.01	100.8 ± 4.8 *P< 0.01 **P1<0.01

P: compared with control group, P1 compared with stressed group
P< 0.01 significant.

In hepatic tissue homogenate, a significant decrease in GSHPx activity in the stressed group compared to normal control group has been observed (Stressed, 121 ± 1.6 vs Control, 150 ± 1, P<0.01, Figure 2-A). Stressed groups supplemented with vitamin E or vitamin C showed a significant increase in GSHPx compared to stressed group (130 ± 2.3 and 160 ± 2, respectively, P1<0.01, Figure 2-A). While the stressed group supplemented with vitamin E showed a significant decrease in GSHPx activity compared to the control (P<0.01), stressed group supplemented with vitamin C showed a significant increase (P < 0.01) compared to control group. No significant difference in CAT activity has been noted between the studied groups.

SOD activity showed a significant decrease in the stressed group compared to the control group (Stressed, 215.16 ± 1.36 vs Control, 250 ± 3, P<0.01, Figure 2-B).On the other hand, stressed groups supplemented with vitamin E or vitamin C showed a significant increase in SOD activity compared to stressed group (Vit.E, 225.60 ± 1.36, Vit.C, 270.9 ± 2.8, P1<0.01, Figure 2-B). While stressed group supplemented with vitamin E showed a significant decrease compared to the control group (P<0.01), stressed group supplemented with vitamin C showed a significant increase (P<0.01) compared to control group.

Lipid peroxides level showed a significant increase in the stressed group compared to normal control group (Stressed, 160.02 ± .48 vs Control, 83.2 ± 3.2, P<0.01, Figure 2-C). Stressed groups supplemented with vitamin E or vitamin C showed a significant decrease compared to the stressed group (Vit.E, 110.4 ± 3.8 and Vit.C, 100.8±48, P1<0.01, Figure 2-C). These values were significantly higher than the normal control group (P<0.01). No significant difference in CAT activity has been found among the studied groups.

DISCUSSION

It is believed that factors which cause major emotional and / or physical stress, such as fear, grief, immobilization and others can affect the antioxidant status (the balance between antioxidants and pro-oxidants in living organism), either by reducing appetite and / or by causing biochemical reactions in the body which cause production of free radicals [10].

Polyunsaturated fatty acids are present in high concentrations in the cellular membrane and most susceptible to radicals attack. Reaction of radicals, with these membrane constituents leads to lipid peroxidation within the membranes followed by disintegration of the cells and ultimately to cell death [10]. This lipid peroxidation is widely used as an index of oxidative stress [10]. In the present study, stress was found to produce a significant increase in both heart and liver peroxides. These results agreed with [11] who found that under cold immobilization, lipid peroxidation was increased in the heart, stomach and liver homogenates. The increased levels of lipid peroxides could be due to increased activity of hormonal component of the sympatho-adrenal system, accumulation of excessive quantities of free fatty acids and reduced activity of antioxidant enzymes [10].

In the present work, immobilization stress induced significant decrease in the activity of antioxidant enzymes, however with variable degrees. SOD and GSHPx activities in both cardiac and liver homogenates were significantly decreased in stressed groups as compared to the control group while catalase showed insignificant change. Murray et al. [10] reported that the antioxidant enzymes, SOD, CAT and GSHPx are complementary in their function to get rid of an excessively formed free radicals. Moreover, Das and Banerjee, [12] stated that GSHPx activity may decrease due to excessive accumulation of hydroxyl radicals. Recently Velken et al. [13] deduced that restraint stress in rats is accompanied with an increased formation of oxygen free radicals that account for the increased lipid peroxidation and with a marked decrease of the antioxidant defense mechanisms represented in the antioxidant enzymes. Because lipids, DNA, proteins and enzymes are susceptible to oxidative damage by the endogenous generation of ROS, a well balanced antioxidant defense system protects against such

damage[31]. If hydrogen peroxide is not removed effectively enough by catalase and glutathione peroxidase, it may in turn inactivate SOD [32]. Moreover, immobilization stress increases the production of $O_2^{\bullet-}$, the substrate of SOD [2]. $O_2^{\bullet-}$ and H_2O_2 generate $OH^{\bullet}$ which interact with SOD with consequent damage and decrease in its activity [33]. This may, at least partially, explain the present results.

The naturally occurring free radical scavengers, ascorbic acid (vitamin C) and α-tocopherol (vitamin E) were exogenously administered to find out their role in prevention of changes in cardiac and hepatic antioxidant enzymes during stress. These compounds were selected because they react directly, but not catalytically with free radicals. These scavengers proved to be protective against several human diseases [34]. The significant protection offered by these free radical scavengers was accompanied with a significant decrease in both heart and liver peroxides indicating an efficient scavenging effect. Vitamin C is water soluble, passes readily through cell membranes, has a high affinity to react with free radicals and the products of the reaction are inert and can easily regenerate ascorbic acid once again. Therefore, vitamin C is considered as an efficient free radical scavenger both intra- and extracellularly[35]. Furthermore, vitamin C is a catalyst for a number of biochemical reactions involved with the stress response. These include hydroxylation of proline for collagen synthesis required for the integrity of cell membranes and steroidogenesis in the adrenal cortex [29]. It is also used as a cofactor for catecholamine biosynthesis; in particular the conversion of dopamine to norepinephrine catalyzed by dopamine-B-hydroxylase [36]. While corticosteroids can induce the synthesis of antioxidant defensive enzymes [37]. In addition, vitamin C also could reduce consumption of antioxidant enzymes during stress and exert an antioxidant effect [38].

On the other hand, α-tocopherol is a fat soluble vitamin; it acts as a free radical trap and would prevent the formation of lipid peroxides in membranes. It acts better in high oxygen tension conditions as in the lungs and heart and better associated with membrane phospholipids [39]. The drawback of α-tocopherol is that the end products of the scavenging reaction are excreted and hence, the vitamin is not recycled and has to be supplemented continuously [29]. The protective effect of α-tocopherol against lipid peroxidation of susceptible cardiac tissue was reported by Donzel et al [40], this is in agreement with the present results.

The current study shows that the heart has a limited defensive antioxidant enzymes that guard against the toxic effects of peroxides compared to the liver; this has been concluded by Rossowska et al [41] and Oskarsson et al [42].

From the previous data, it is apparent that the antioxidant defensive mechanisms are impaired during exposure to restraint stress. Oxygen free radicals are formed in excess under such conditions and are responsible at least partially for the development of cardiac and hepatic stress lesions. They attack all membranes producing peroxidation of phospholipids followed by membrane damage. Scavenging free radicals by the exogenously administered scavengers, ascorbic acid (vitamin C) and α-tocopherol (vitamin E) offered a significant effect on the antioxidant defensive mechanisms and lipid peroxidation. Moreover, the activities of antioxidant enzymes in the heart are lower than that in the liver. Thus, the heart has a limited defense against the toxic effects of peroxides when compared with the liver. Vitamins E and C dietary supplementation seems to be of good protective value in this tissue due to the low levels of endogenous enzymatic activities present in the mammalian heart in comparison with those of the liver.

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تأثيرات فيتامين هـ ، جـ علي أكسدة الدهون المصاحبة للإجهاد ونشاط الإنزيمات المضادة للأكسدة في الجرذان

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**قسم الأدوية و السموم–كلية الصيدلة– جامعة قناة السويس– الإسماعيلية – مصر

وجد أن الإجهاد عن طريق تقييد حركة الحيوان يؤدي إلي تكوين وحدات الأوكسجين النشطة و الذي بدوره يؤدي إلي إصابة الأنسجة المختلفة بما يعرف بالإجهاد التأكسدي.

وكان الغرض من إجراء البحث الحالي هو دراسة تأثير تقييد حركة الحيوان علي مستوي الدهون المؤكسدة و نشاط الإنزيمات المضادة للأكسدة ، و معرفة دور الفيتامينات المضادة للأكسدة (فيتامين هـ ، جـ) ضد وحدات الأوكسجين النشط.

استخدمت عينات من الكبد و القلب لتحديد مستوي المواد النشطة لحامض الثيوباربيتيوريت و نشاط الإنزيمات المضادة للأكسدة و تشمل سوبر أكسيد دسميوتيز، كاتاليز ، فوق أكسيد جلوتاثيون.

في هذه الدراسة لوحظ زيادة واضحة في مستوي المواد النشطة لحامض الثيوبربيتيوريت في مجموعة الجرذان المعرضة للإجهاد عن طريق تقييد الحركة، مما يشير إلي تكوين وحدات الأكسجين النشطة. كذلك لوحظ نقص كبير في نشاط الإنزيمات سوبر أكسيد دسميوتيز ، فوق أكسيد جلوتاثيون.

ساعد العلاج لفيتامين هـ ، جـ علي رفع فعالية الإنزيمات الطبيعية المضادة للأكسدة، حيث سجل نقص كبير في مستوي المواد النشطة لحامض الثيوبربيتيوريت، بالإضافة إلي زيادة في نشاط الإنزيمات المضادة للأكسدة. و خلصت الدراسة إلي أن إجهاد الحيوان عن طريق تقييد حركته تؤدي إلي زيادة أكسدة الدهون و تغيير نشاط الإنزيمات المضادة للأكسدة، و يعزي الأثر الوقائي لفيتامين هـ ، جـ لزيادة تكوين الإنزيمات المضادة للأكسدة أو لتقليل استهلاك هذه الإنزيمات.