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ACUTE EXERCISE TOLERANCE IN EXPERIMENTALLY-INDUCED HYPERTHYROIDISM IN ADULT MALE ALBINO RATS

By

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ABSTRACT

Background: Decreased muscle strength and exercise intolerance are prominent features of hyperthyroid patients. Skeletal muscle dysfunction may be severe in newly diagnosed hyperthyroid patients, compromising their abilities to perform daily activities. Thyroid hormones have shown to affect mitochondrial oxidative activity, synthesis and degradation of protein, differentiation of muscle fibers and capillary growth. **Objective:** Studying the effects of experimentally-induced hyperthyroidism on acute exercise tolerance in male albino rats. **Material and methods:** Thirty adult male albino rats of local strain were chosen to be the model of the present study. They were divided into three equal groups; control group, hyperthyroid group (subjected to induction of hyperthyroidism by daily IP injection of 10 ?g thyroxin /100 gm body weight for four weeks), and recovery group. Rats were weighed at the start and the end of the work. Blood samples were obtained for determination of serum free T_3, free T_4, TSH, CPK, TAC and MDA levels. **Results:** Induction of hyperthyroidism led to significant decrease in the body weight, swimming time, TSH level and total antioxidant capacity associated with significant increase in the FT_3, FT_4, CPK and MDA levels. These changes improved by allowing recovery of hyperthyroid state by thyroxin withdrawal over time. However, these improvements did not resume the basal level. **Conclusion:** Hyperthyroidism has a drawback effects on the body functions. These effects were improved by withdrawal of thyroxin (or treatment) without resuming the basal level of euthyroid state. Further studies are required to evaluate the effects of prolonged time of recovery or treatment of hyperthyroidism on resuming basal levels of disturbed body functions.

Key words: Hyperthyroidism, Oxidant-antioxidant capacity, Muscle performance.

INTRODUCTION

Hyperthyroidism is a pathologic syndrome characterized by overproduction of thyroid hormones and their excess in blood. Thyroid hormones accelerate the basal metabolic rate, oxidative metabolism and production of reactive oxygen species (ROS) by mitochondrial enzymes **(Mohamadin et al., 2006).**

Decreased muscle strength and exercise intolerance are prominent features of hyperthyroid patients. Although protein degradation as a result of accelerated proteolysis has been found in skeletal muscles of hyperthyroid patients, it seems unlikely that protein loss contributes only to muscle weakness **(Yamada et al., 2006).**

In hyperthyroid individuals, muscle strength has shown to be reduced by 40 % up to 100%, and the muscle mass is reduced by about 20% leading to decreased force of muscle contraction and easy fatigability **(Yamada et al., 2006).**

Thyroid hormones has shown to affect mitochondrial oxidative activity, synthesis and degradation of protein, differentiation of muscle fibers and capillary growth. It has been reported that thyroid hormones play a role in inducing oxidative stress **(Kale et al., 2007).** Evidence suggests that hypermetabolic state in hyperthyroidism is associated with increased production of free radicals and lipid peroxidation **(Yamada et al., 2006 and Chattopadhyay et al., 2010).**

The present work was designed to study the effects of experimentally-induced hyperthyroidism on acute exercise tolerance in adult male albino rats.

MATERIALS AND METHODS

Thirty adult male albino rats of local strain weighing 160-172 g were chosen to be the model of the present study. They were left for two weeks in the laboratory room in Medical Physiology Department, Al-Azhar Faculty of Medicine for acclimatization with free access to water and rat chow pellets. Rats were kept in a suitable cages (50 X 35 X 50 per 5 rats) at room temperature with the natural light-dark cycle. Rats were weighed and divided into three equal groups:

Group I (Control group): Normal rats received daily intraperitoneal injection of 0.5 ml normal saline / rat for four weeks.

Group II (Hyperthyroid group): Rats were subjected to induction of hyper-thyroidism by daily intraperitoneal injection of tetraiodothyronine (T_4) in a dose of 10 ?g /100 g body weight for four weeks **(Venditti et al., 2006).**

Group III (Recovery group): Rats of this group were subjected to induction of hyperthyroidism as group II. Then, they were allowed to recover from hyper-thyroid state by withdrawal of thyroxin over four weeks. Recovery were confir-med by measurement of thyroid hormones and thyroid stimulating hormone **(Rao et al., 2003).**

Preparation of L-thyroxin and induction of hyperthyroidism: L-thyroxin was purchased from Sigma Co. (USA) in the form of a bottle containing 100 mg of thyroxin powder. Fifteen mg of thyroxin powder were dissolved in 375 ml of normal saline with a concentration of 40 ?g L-thyroxin per one ml normal saline. From this solution, L-thyroxin was given to the rats in a dose of 10 ?g /100 g body weight by intraperitoneal injection once daily for four weeks **(Venditti et al., 2006).**

Exercise model: Rats were forced to swimming in a water tank for ten minutes/day for two days **(Matsakas et al., 2006).** Rat swimmed against load (5% of body weight) attached approximately two inches from the tail end **(Casimiro-Lopez et al., 2008).** Maximum swimming time was measured from the beginning of swimming with the weight until the point at which the rat could not return to the water surface (10 second after sinking), where the rat was taken out of water and returned to the cage for recovery **(Tanaka et al., 2003).**

At the end of the experimental period (four weeks for control and hyperthyroid groups, and eight weeks for the recovery group), rats were weighed and blood samples were withdrawn from the retro-orbital plexus into test tubes. Serum was separated and stored frozen at -20 ^{o}C until assayed for determination of free tri-iodothyronine (FT_3), free tetraiodothyronine (FT_4), thyroid stimulating hormone (TSH), creatine phosphokinase (CPK), total antioxidant capacity (TAC) and malondialdehyde (MDA) levels.

Biochemical assay: Serum free T_3 and T_4 levels **(Bowers et al., 1970),** Serum TSH level **(Chopra, 1971),** CPK level **(Brutis and Ashwood, 1999),** TAC level **(Koracevic et al., 2001),** and MDA level **(Yoshioka, et al., 1979).**

Statistical analysis: Data input and analysis were done using SPSS computer program. All results were expressed as mean ± standard error. Mean values of the different groups were compared using a one-way analysis of variance (ANOVA). Least significant difference (LSD) post hoc analysis was used to identify significantly different mean values. P value < 0.05 was accepted to denote a significant difference.

RESULTS

Changes in body weight (Table 1): At the end of the experimental period, there was significant increase in the mean final body weight of the control rats, hyperthyroid rats and hyperthyroid recovered rats where it increased by 28.64 %, 7.5 % and 19.77 % for the control, hyperthyroid and hyperthyroid recovered rats respectively. The increased body weight was more evident in the control group.

Induction of hyperthyroidism led to significant decrease in the mean final body weight by 16.23 % when compared to the control rats. Recovery from hyperthyroid state led to significant increase in the mean final body weight by 12.61 % when compared to the hyperthyroid rats. Recovery from hyperthyroid state led to enhanced body weight but, showed significant decrease in the mean final body weight by 5.66 % when compared to the control rats.

Changes in thyroid profile (Table 2): Induction of hyperthyroidism led to significant increase in the mean free T_3 level by 192.38 %, significant increase in the mean free T_4 level by 190.78 %, significant decrease in the mean TSH level by 93.5 % when compared to the control rats. Recovery from hyperthyroid state led to enhancement of these parameters with significant decrease in the mean free T_3 level by 61.15 %, significant decrease in the mean free T_4 level by 73.9 %, significant increase in the mean TSH level by 135.9 % when compared to the hyperthyroid rats.

Despite improvement of hyperthyroidism, recovery from hyperthyroid state showed insignificant increase in the mean free T_3 level by 13.58 %, insignificant decrease in the mean free T_4 level by 24.11 % and insignificant decrease in the mean TSH level by 5.32 % when compared to the control rats.

*** Changes in exercise tolerance and oxidative markers (Table 3):** Induction of hyperthyroidism led to significant decrease in the mean maximal swimming time by 39.76 %, significant increase in the mean CPK level by 484.26 %, significant decrease in the mean TAC level by 74.55 %, and significant increase in the mean MDA level by 256.8 % when compared to the control rats. Recovery from hyperthyroid state led to significant increase in the mean maximal swimming time by 54.13 %, significant decrease in the mean CPK level by 83.44 %, significant increase in the mean TAC level by 193.02 %, and significant decrease in the mean MDA level by 65.21 % when compared to the hyperthyroid rats.

Despite improvement of hyperthyroidism, recovery from hyperthyroid state showed insignificant decrease in the mean maximal swimming time by 7.16 %, insignificant decrease in the mean CPK level by 3.26 %, significant decrease in the mean TAC level 25.44 %, and insignificant increase in the mean MDA level 24.14 % when compared to the control rats. So, improved hyperthyroid state led to enhanced body functions without resuming basal level of euthyroid.

Table (1): Changes in body weight.

Parameters / Groups	Mean ± S.E.		P value	% change
	Initial weight (g)	Final weight (g)		
Group I (n = 10)	**166.2 ± 3.84**	**213.8 ± 2.08***	**P< 0.001 ▲**	**+ 28.64 %**
Group II (n = 10)	**166.6 ± 3.13**	**179.1 ± 2.07***	**P< 0.005 ▲**	**+ 7.5 %**
Group III (n = 10)	**168.4 ± 2.8**	**201.7 ± 2.53***	**P< 0.001 ▲**	**+ 19.77 %**
			P< 0.001 ●	**- 16.23 %**
			P< 0.001 ♦	**+ 12.61 %**
			P< 0.05 ◙	**- 5.66 %**

- Group I: control group.
- Group II: hyperthyroid group.
- Group III: recovery group.
- n: No. of rats in each group.
- ▲: compared to itself.
- ●: group II compared to group I.
- ♦: group III compared to group II.
- ◙: group III compared to group I.
- * Significant.

Table (2): Changes in thyroid profile.

Groups / Parameters	Mean ± S.E		
	Group I (n = 10)	Group II (n = 10)	Group III (n = 10)
Free T_3 (pg/dl)	3.02 ± 0.36	8.83 ± 0.8	3.43 ± 0.26
P value		P < 0.01 ●	P < 0.01 ♦ P > 0.05 ◙
% changes		+ 192.38 % ●	- 61.15 % ♦ + 13.58 % ◙
Free T_4 (ng/dl)	1.41 ± 0.12	4.1 ± 0.63	1.07 ± 0.13
P value		P < 0.01 ●	P < 0.01 ♦ P > 0.05 ◙
% changes		+ 190.78 % ●	- 73.9 % ♦ - 24.11 % ◙
TSH (mIU/ml)	0.94 ± 0.29	0.061 ± 0.005	0.89 ± 0.012
P value		P < 0.05 ●	P < 0.05 ♦ P > 0.05 ◙
% changes		- 93.5 % ●	+ 135.9 % ♦ - 5.32 % ◙

- Group I: control group.
- Group II: hyperthyroid group.
- Group III: recovery group.
- n: No. of rats in each group.
- ●: group II compared to group I.
- ♦: group III compared to group II.
- ◙: group III compared to group I.

Table (3): Changes in exercise tolerance and oxidative markers.

Groups / Parameters	Mean ± S.E		
	Group I (n = 10)	Group II (n = 10)	Group III (n = 10)
Swim time (min)	23.89 ± 0.73	14.39 ± 1.32	22.18 ± 0.6
P value		P < 0.01 ●	P < 0.01 ♦ P > 0.05 ◙
% changes		- 39.76 % ●	+ 54.13 % ♦ - 7.16 % ◙
CPK (u/l)	117.5 ± 11.39	686.5 ± 44.92	113.67 ± 10.31
P value		P < 0.01 ●	P < 0.01 ♦ P > 0.05 ◙
% changes		+ 484.26 % ●	- 83.44 % ♦ - 3.26 % ◙
TAC (Mm/l)	1.69 ± 0.07	0.43 ± 0.08	1.26 ± 0.06
P value		P < 0.01 ●	P < 0.01 ♦ P < 0.05 ◙
% changes		- 74.55 % ●	+ 193.02 % ♦ - 25.44 % ◙
MDA (mmol/l)	1.023 ± 0.22	3.65 ± 0.34	1.27 ± 0.2
P value		P < 0.01 ●	P < 0.01 ♦ P > 0.05 ◙
% changes		+ 256.8 %	- 65.21 % + 24.14 %

- Group I: control group.
- Group II: hyperthyroid group.
- Group III: recovery group.
- n: No. of rats in each group.
- ●: group II compared to group I.
- ♦: group III compared to group II.
- ◙: group III compared to group I.

DISCUSSION

The present work was designed to investigate the effects of experimentally-induced hyperthyroidism on acute exercise tolerance in adult male albino rats. Results of the present work showed that induction of hyperthyroidism led to significantly increased serum fT_3, fT_4, CPK and MDA levels associated with significantly decreased body weight, TSH, exercise tolerance and total antioxidant capacity.

These results were in agreement with **Messarah et al. (2011)** who reported that despite increased food consumption in hyperthyroid rats by 27%, there was a significant loss of body weight. **Postler et al. (2009)** has also reported that hyperthyroid animals failed to gain weight compared with the control rats over 14 days experimental period, and concluded that weight gain in the hyperthyroid rats was about 1/16 of the control rats despite the highly increased food intake. Also, **Santini et al. (2014)** has reported that administration of thyroxin results in a decrease in body weight due to decline in both lean and fat mass despite increased appetite.

Results of the present work were also in agreement with **Yamada et al. (2006)** who reported that hyperthyroid animal maintains 2.8 fold increase in serum-free T_3 levels compared with control animals. **Liu et al. (2007).** Also, **Ahmed et al. (2010)** reported that thyroxin administration for three weeks resulted in significantly increased serum T_3 and T_4 levels and reduced TSH level. **Ray et al. (2013)** has reported that treatment of euthyroid individuals with thyroxin significantly raises serum free T_3 and T_4 levels with significant reduction of TSH level. **Chang et al. (2013)** has reported that patients with toxic manifestations of hyperthyroidism had higher plasma levels of free thyroid hormones than the clinically euthyroid patients.

Reduced exercise tolerance observed in hyperthyroid rats was compatible with **Casimiro-Lopez et al. (2008)** who reported that in hyperthyroid rats, there is a markedly reduced exercise tolerance due to effects of hyperthyroid state on glycogen metabolism, leptin level and increased corticosterone level. Taken together, these metabolic disturbances impair exercise capacity in hyperthyroid state. **Yamada et al. (2006)** has reported that the twitch force developed by soleus muscles treated with T_3 was less than that of controls with significant reduction in tetanic force.

Disturbed muscle functions and exercise intolerance in hyperthyroid state could be explained by protein oxidation which modify the structure and function of protein associated with disturbed excitation-contraction coupling, and accelerated mitochondrial oxidative metabolism leading to augmented production of reactive oxygen species **(Moopanar and Allen, 2005).**

It has been reported that experimentally-induced hyperthyroidism leads to down- regulation of oxidative and glycolytic enzymes in skeletal muscle, remodeling of muscle tissue, and loss of muscle mass **(Ray et al.,, 2013).** Hyperthyroidism produces conversion of muscle fibers from fast to slow fiber types, and a more efficient energy metabolism producing a reversible transi-

tion of myosin heavy chain isoform compared with healthy individuals. (**Haizlip et al., 2015**).

In the present work, CPK level increased in hyperthyroid rats. This result was in agreement with **Popova et al. (2008)** who reported significant increase in serum CPK level in hyperthyroid rats compared with euthyroid indicating tissue injury including skeletal muscles which could be due to increased lipid peroxidation and free radicals level in hyperthyroid rat tissue and serum.

Hyperthyroid individual manifests limb weakness, myalgia, pain and/or spasms associated with elevated creatine kinase levels up to 1500 U/l and do not correlate with the severity of weakness **(Douglas, 2010 and Messarah et al., 2011).**

In the present work, there was disturbed oxidant-antioxidant levels indicated by increased MDA and reduced TAC levels. These results were supported by **Mohamadin et al. (2006)** who reported that hyperthyroidism accelerates generation of ROS concomitant with disturbed antioxidant activity in various tissues. **Venditti et al. (2009)** has reported that hyperthyroidism is associated with impaired oxidant-antioxidant capacity and lipid peroxidation indicated by increased MDA and hydroperoxides in mouse skeletal muscles, and decreased antioxidant activities indicated by significantly decreased glutathione reductase activity and total antioxidant levels. Also, **Vargas et al. (2006)** has reported that oxidative stress in hyperthyroidism may be due to a primary down-regulation of antioxidant enzymes indicated by elevated MDA levels and reduced its urinary excretion in patients and T_4 -treated rats.

Results of the present work showed that recovery of hyperthyroidism by thyroxin withdrawal led to return back of thyroid profile to euthyroid state, enhanced weight gain, exercise tolerance, and decreased CPK and MDA levels. These indicated that the effects of hyperthyroidism were reversible with prompt control and treatment.

These results were in agreement with **Rao et al. (2003)** who reported that serum T_3, T_4 and TSH levels were restored to normal values when allowing rats to recover by discontinuation of thyroxin treatment for three weeks. **Santini et al. (2014)** has reported that achieving euthyroidism resulted in increased body weight, body mass index, fat mass and fat free mass due to reduced energy expenditure and/or greater energy intake than required to maintain body weight. **Santos et al. (2006)** has reported that the muscle mass and muscle strength increased after achievement of euthyroid state. Enhanced muscle mass is reflected on peak strength which considered a key indicator of muscle performance and endurance. Also, **Inal et al. (2015)** has reported that muscle weakness in hyperthyroidism is severe and evolves rapidly, but recovery after treatment of hyperthyroidism results in clinical improvements in muscle mass and strength.

Results of the present work showed that hyperthyroidism affected muscle function and oxidative markers which improved by treatment without resuming basal level. These results were in agreement with **Yamada et al. (2006)** who reported that even after normalization of thyroid hormones level, impairment of

muscle function occasionally lasts for prolonged periods. **Inal et al. (2015)** reported that achievement of euthyroid state does not improve all aspects of muscle function and muscular endurance does not reach the level of healthy individuals following medical treatment.

CONCLUSION

Hyperthyroidism affected muscle function and oxidative markers which are improved by treatment without resuming basal level, indicating that despite treatment of hyperthyroidism, it has a drawback effects on the body functions. Further studies are required to evaluate the effects of prolonged time of recovery or treatment of hyperthyroidism on resuming basal levels of disturbed body functions.

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تحمل التمارين الرياضية الحادة في فرط نشاط الغدة الدرقية المحدث تجريبيا في ذكور الجرذان البيضاء البالغة

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خلفية البحث: يعتبر ضعف القوة العضلية وعدم القدرة على تحمل التمارين الرياضية من العلامات المميزة لمرضى فرط نشاط الغدة الدرقية، ومن الممكن أن يكون ضعف الأداء العضلي حادا بدرجة تؤثر على أداء النشاط اليومى فى المرضى المشخصين حديثا. ويؤثر إنحراف الغدة الدرقية عن معدل نشاطها الطبيعى على تكوين الشوارد الحرة التى تؤدى إلى حدوث أكسدة فى الخلايا وما يترتب على ذلك من ضرر للأنسجة. كما تؤثر على تخليق البروتين وإنحلاله بالإضافة إلى التمييز بين الألياف العضلية ونمو الشعيرات الدموية.

الهدف من البحث: دراسة تأثير فرط نشاط الغدة الدرقية المحدث تجريبيا على القدرة على تحمل التمارين الرياضية الحادة فى ذكور الجرذان البيضاء البالغة.

مواد وطرق البحث: إستخدم فى هذا العمل ثلاثون ذكرا من السلالة المحلية من الجرذان البيضاء تم تقسيمهم إلى ثلاث مجموعات متساوية: المجموعة الأولى (مجموعة ضابطة)، والمجموعة الثانية (مجموعة محدث بها زيادة نشاط الغدة الدرقية)، والمجموعة الثالثة (مجموعة محدث بها زيادة نشاط الغدة الدرقية ثم عودتها إلى الحالة الأولى بسحب عقار الثيروكسين المسبب لزيادة نشاط الغدة الدرقية).وقد تم وزن الجرذان فى بداية العمل ونهايته. وفى نهاية مدة العمل، تم سحب عينات دم لقياس مستويات هرمونى الغدة الدرقية، و الهرمون المنشط للغدة الدرقية، و فسفوكينيز الكرياتين، و إجمالى مضادات الأكسدة ، والمالوندالدهيد.

النتائج: أدى فرط نشاط الغدة الدرقية إلى نقص ذى دلالة إحصائية فى كل من وزن الجرذان، و تحمل التمارين الرياضية، و الهرمون المنشط للغدة الدرقية، و إجمالى مضادات الأكسدة. فيما أدى ذلك إلى زيادة ذات دلالة إحصائية فى مستويات هرمونى الغدة الدرقية، وفسفوكينيز الكرياتين، والمالوندالدهيد. وقد لوحظ تحسن هذه التغيرات بسحب عقار الثيروكسين المسبب لفرط نشاط الغدة الدرقية عن طريق الوقت. وبالرغم من التحسن فى القياسات بسحب العقار إلا أنه لم يصل للحد الطبيعى.

الإستنتاج: فرط نشاط الغدة الدرقية يؤثر على القدرة على تحمل التمارين الرياضية الحادة و دلالات الإجهاد التأكسدى والتى تتحسن بالعلاج أو سحب المسبب دون الوصول للحد الطبيعى مما يشير إلى الآثار السلبية التى يخلفها فرط نشاط الغدة الدرقية على وظائف الجسم. وعليه، فمن الممكن عمل دراسات تالية لبيان تأثير مدة أطول على العودة للقياسات الطبيعية لإضطراب وظائف الجسم الناتج عن فرط نشاط الغدة الدرقية.