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THE EFFECT OF EXERCISE TRAINING INTENSITY ON THYROID ACTIVITY AT REST

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The influence of exercise training intensity on thyroid activity at rest was studied in male Wistar rats, weighing 114g ± 24 (mean ± SD) at the beginning of the experiment. Animals were assigned to the following groups: untrained controls and rats trained on a treadmill at the speed of 20m/min over a 5-week period with different intensities: 2 × 60 min weekly, 4 × 60 min, 6 × 20 min, 6 × 40 min and 6 × 60 min weekly. Thyroid peroxidase (TPO) and hepatic iodothyronine 5'-monodeiodinase (5'DI) activities as well as plasma thyroxine (T_4), 3,3'5-triiodothyronine (T_3) and 3,3'.5'-triiodothyronine (rT_3) concentrations were determined. Training intensity was found to influence parameters under investigation. TPO activity was decreased in groups trained 240 min (4 × 60 min and 6 × 40 min) and 360 min (6 × 60 min) weekly in comparison to control, untrained group. Furthermore, a drop in T_4 plasma concentration in all trained groups and a decrease in T_3 plasma concentration in groups exercising for 120 min (2 × 60 min and 6 × 20min) weekly, as compared to control, untrained rats, was found. Hepatic 5'DI activity and rT_3 plasma concentration were not affected by training. Thus, exercise training in rats seems to elicit the fall in TPO activity and T_4 plasma concentration at rest but without changing hepatic 5'DI activity and rT_3 plasma concentrations. A decline in T_3 plasma concentration, observed in rats trained with the lowest exercise intensities, could be regarded as transitional effect in adaptation to chronic exercise.

Key words: *exercise, iodothyronine 5'-monodeiodinase, thyroid, thyroid peroxidase, thyroxine, triiodothyronine*

INTRODUCTION

Thyroid hormone metabolism is influenced by alterations in energy supply and expenditure. In states of energy restriction caused by fasting or undernutrition it has been found to undergo specific changes leading to a decrease in both production and clearance rates of triiodothyronine (T_3) and thyroxine (T_4) (1—4). Although exercise training represents a condition that can produce an energy deficit, it has been shown to enhance thyroid hormone metabolism both in humans with sufficient energy intake (5—7) and *ad libitum*

fed animals (8—12). However, little is known about the effect of chronic exercise on thyroid hormone metabolism at rest, since kinetic studies dealing with the impact of enhanced physical activity on thyroid hormone metabolism usually did not allow to assess independently the effects of acute and chronic exercise because they did not separate them distinctly. Particularly noteworthy seems to be an increased thyroid hormone turnover found in exercise trained male rats (9, 10) shown not to increase food intake to compensate for increased energy demand attributable to enhanced physical activity (13) and to reduce their body weight gain (10, 14, 15). Moreover, exercising male Wistar rats were found to display energy economizing adaptations such as reduction in total energy expenditure (15), suppression of cold induced thermogenesis (16, 17) and brown adipose tissue activity (18). On the other hand, the influence of training on thyroid hormone plasma levels in rats at rest is not quite clear. Although training has been shown not to affect total and free T_3 and reverse T_3 (rT_3) plasma concentrations at rest (10, 14, 19), data concerning T_4 plasma concentration are not consistent. Total T_4 plasma concentration was reported to be diminished (19) or remained unchanged (14), free fraction of plasma T_4 was demonstrated to be increased (9) or decreased (10) in trained rats compared to their control, sedentary counterparts.

This study was performed to evaluate the impact of exercise training intensity on thyroid activity at rest in male rats. The activities of two enzymes involved in thyroid hormone metabolism — thyroid peroxidase (TPO) and hepatic iodothyronine 5'-monodeiodinase (5'DI) were measured together with T_4, T_3 and rT_3 plasma concentrations. Thyroid peroxidase plays a key role in thyroid hormone biosynthesis because of its ability to catalyze oxidation of iodide, the iodination of tyrosyl residues of the thyroglobulin and their coupling (20). Hepatic 5'DI catalyzes monodeiodination of both outer and inner ring of T_4, yielding T_3 and rT_3. It acts also on rT_3, providing 3,3'diiodothyronine (21).

MATERIALS AND METHODS

Animals

Male Wistar rats (n = 56) 7 week old weighing 114g ± 24 (mean ± SD) at the beginning of the experiment were housed in standard environmental conditions individually in wire mesh cages and fed ad libitum with standard rodent food.

Experimental design

After one week of standardization rats were divided into 6 groups: sedentary controls (n = 11) and groups trained on a treadmill at the speed of 20m/min during 5 weeks: 2 × 60 min weekly (n = 6); 4 × 60 min (n = 9); 6 × 20 min (n = 10); 6 × 40 min (n = 9); 6 × 60 min (n = 11) weekly.

Rats were sacrificed at the age of 13 weeks after 5 weeks of training about 24 hrs after the last exercise bout. They were anesthetized with diethyl ether and blood was collected by cardiac puncture. Thyroid and liver were weighed and immediately frozen in liquid nitrogen. Then plasma was stored at −20°C and tissues at −80°C until determinations.

Analytical procedures

Thyroid peroxidase activity was determined in thyroid microsomal fraction based on Alexander (22), Neary *et al.* (23) and Hosoya *et al.* (24) methods. Briefly: thyroids were thawed, washed in physiological saline and homogenized on ice in 1.1% KCl in motor driven Potter Elvehjem homogenizer during 1 min and centrifuged (15 min × 1500g). Then the pellet was discarded and supernatant was centrifuged (1hr × 105000g). Pellet was suspended in 200 µl 20 mM phosphate buffer pH 7.4 with 10 µl 0.1% Triton × 100.

TPO activity was measured by iodide oxidation assay (24). The reaction mixture contained 50 mM phosphate buffer pH 7.4, 0.135 mM H_2O_2, 13.3 mM KJ and 50—80 µg of protein in 1.5 ml. The reaction was started by the addition of 10 µl of H_2O_2 and followed spectrophotometrically at 350nm at room temperature. The amount of enzyme which gave a change of 0.001 absorbance per sec was taken as 1 unit (U). Protein was determined by the method of Lowry *et al.* (25) with bovine serum albumin as a standard.

Liver microsomal fraction was prepared and 5'DI activity was estimated according to procedure described by Nauman *et al.* (26). The livers were thawed, washed with saline and homogenized on ice in 3 vol. 20 mM buffer Tris — HCl pH 7.2 with 3 mM EDTA, 10 mM DTT and 0.25 M sucrose using a motor driven teflon pestle/glass body homogenizer. The homogenate was filtered through gauze and centrifuged for 10min at 10000g. Then the pellet was discarded and supernatant was centrifuged for 60 min at 105000g. The microsomal pellet was then suspended in the same buffer without sucrose. The microsomal fraction was stored at −80°C untill further analysis.

5'DI activity was measured during 30 min incubation at 37°C. Incubation medium (400 µl) contained 50 mM Tris — HCl buffer pH 7.2, 3 mM EDTA, 1.3 nM T_4, 5 mM DTT and about 100 µg of microsomal protein. The reaction was stopped by placing test tubes on ice and adding of 800 µl of ice-cold 99% ethanol. After extraction and centrifugation (1500g, 20 min) T_3 concentration in ethanol extracts was determined immediately by radioimmunoassay. Enzyme activity was expressed as the amount of T_3 per min and mg of protein. Protein concentration was determined by the method of Bradford (27).

Thyroid hormone plasma concentrations were determined by radioimmunoassay commercial kits, for T_4 (cat. no MI 88) and T_3 (cat. no MI 86) produced by POLATOM, for rT_3 (cat. no 10834) by Serono Diagnostics.

Statistical analysis

Statistical analysis was performed by one — way analysis of variance (ANOVA) and Student t-test.

RESULTS

All results and statistically significant differences between groups according to Student t-test are summarized in *Table 1.*

Table 1. Effect of exercise training intensity on body weight gain and resting values of thyroid activity parameters.

	groups						
	C (n=11)	2×60 (n=6)	4×60 (n=9)	6×20 (n=10)	6×40 (n=9)	6×60 (n=11)	statistical significance
BW gain (%)	168.9[a] (17.2)	81.5[c] (9.5)	133.8[b] (13.9)	134.7[b] (14.0)	126.5[b] (27.5)	130.9[b] (10.5)	a vs b and b vs c – $p<0.01$; a vs c – $p<0.001$
TPO (U/sec/ mg)	45.7[a] (2.2)	41.9[a] (4.8)	31.8[b] (2.3)	48.4[a] (6.7)	21.4[c] (2.6)	23.7[c] (2.3)	a vs b and b vs c – $p<0.05$; a vs c – $p<0.001$
T_4 (ng/ml)	97.7[a] (4.4)	74.4[b] (7.8)	67.3[b] (8.2)	62.9[b] (8.1)	32.2[c] (2.7)	31.8[c] (2.3)	a vs b and b vs c – $p<0.01$; a vs c – $p<0.005$
T_3 (ng/ml)	0.99[a] (0.05)	0.63[b] (0.08)	0.83[a] (0.04)	0.71[b] (0.04)	1.00[a] (0.08)	0.89[a] (0.08)	a vs b – $p<0.05$
rT_3 (ng/ml)	0.131 (0.007)	0.132 (0.007)	0.120 (0.008)	0.109 (0.013)	0.114 (0.007)	0.117 (0.008)	NS
5'DI (pmol T_3/ min/mg)	4.40 (0.13)	4.38 (0.13)	4.57 (0.23)	4.62 (0.22)	4.63 (0.23)	4.71 (0.16)	NS

Values are means with SE indicated in parantheses. Statistically significant differences are denoted by different letters, NS — lack of significance. Groups: C — control, untrained rats; 2×60, 4×60, 6×20, 6×40, 6×60 — rats trained, first number — exercise frequency during the week, second — daily exercise duration in minutes; n — number of rats. BW — body weight, TPO — thyroid peroxidase activity, T_4, T_3, rT_3 — thyroxine, triiodothyronine and reverse triiodothyronine plasma concentration (respectively), 5'DI — iodothyronine 5'-monodeiodinase activity in liver.

Body weight gain in all trained groups was significantly reduced compared to the control rats. Group trained twice weekly for 60 min gained significantly less than the other trained groups. ANOVA: effect of daily exercise duration: $p<0.02$; effect of frequency during the week: $p<0.0001$.

Thyroid peroxidase activity was influenced by daily exercise duration (ANOVA: $p<0.0001$), but not by its frequency during the week (ANOVA: $p<0.1$). In rats trained 6×60, 6×40 and 4×60 min weekly TPO activity was significantly reduced compared to sedentary controls and rats trained 6×20 and 2×60 min. In groups trained 6×40 and 6×60 min it was significantly lower than in group trained 4×60 min.

Plasma T_4 concentration in all trained groups was lower than in sedentary animals. ANOVA revealed significant influence of daily exercise duration ($p<0.0002$) and frequency ($p=0$). In groups trained 6×20, 2×60 and

4×60 min values were statistically not different, but significantly higher than in groups trained 6×60 and 6×40 min.

Plasma T_3 concentration was diminished in groups trained 2×60 and 6×20 min in relation to sedentary rats and the other trained groups. ANOVA indicated significant influence of daily exercise duration ($p < 0.0002$).

Plasma rT_3 concentration and 5' DI activity were neither influenced by exercise frequency nor by duration. In all trained groups the values did not differ statistically significantly from those of control group.

DISCUSSION

This study has demonstrated that chronic enhancement of physical activity in rats can elicit a diminish in TPO activity and T_4 plasma concentration in resting state without changing plasma T_3 and rT_3 concentrations and hepatic 5'DI activity.

The decrease in body weight gain, which occurred in all trained rats compared to sedentary controls, could be attributed to inadequate energy intake in relation to increased energy demand which has been stated in chronically exercising male Wistar rats (13, 15). Suprisingly, body weight gain did not differ between groups trained with various exercise intensities except the group exercised twice weekly. It could be supposed, that the lower body weight gain in group trained with the least frequency could be the result of an emotional stress.

Thyroxine plasma concentration and TPO activity were reduced depending on the weekly training dose. However, T_4 level was decreased in all trained rats while TPO activity was diminished in rats trained at least 240 min. weekly. With the 240 min. of training performed weekly both T_4 plasma level and TPO activity were more reduced when exercise was applied with shorter daily duration but more frequently.

The effect of exercise on TPO activity, according to the author's knowledge, has not been examined as yet. Some insight into the effect of exercise on thyroid gland activity could yield results of studies on thyroidal iodine uptake, although they were conducted both during rest and periods of enhanced daily activity. They revealed decrease in iodine uptake in trained rats and humans (18, 29). Rats exercising spontaneously were found to store only half as much iodine in the thyroid as did the nonexercising controls with the amount of exercise and amount of iodine being negatively correlated (28). This effect has been suggested to be connected with depression of thyroid activity or increase in thyroid hormone production and release. However, a significant difference in the rate of renewal of thyroidal iodine between sedentary and exercising rats was not stated (28). Thyroidal iodine uptake as well as TPO synthesis and activity are stimulated by TSH (30, 31)). In trained rats, both unstimulated

TSH values and TRH — stimulated TSH responses have been found to be decreased compared to ad libitum fed nonexercising rats (14). It must be also taken into consideration that insulin, known to stimulate TPO gene expression (32), has been shown to be reduced by training both in humans (33, 34) and rats (35). Effect of training on thyroid activity could be mediated by brain systems known to inhibit TRH and/or TSH secretion, such as dopaminergic and NPY systems (36, 37) and those suggested to stimulate, such as serotoninergic (38). Exercise training was stated to stimulate the hypothalamic NPY (39) and dopaminergic (40) systems. On the other hand, a downregulation of brain serotonin receptors by training was suggested (41). All of these effects could lead to decline in TRH/TSH secretion and reduction in stimulation of thyroid.

Decrease in TPO activity could contribute to a fall in T_4 plasma concentration found in exercising rats. In previously reported investigations training — induced decrease in T_4 plasma concentration was associated with changes in T_4 metabolism and distribution. A decline in T_4 half life and elevation in T_4 secretion rate were reported in horses together with decrease in protein bound iodine plasma concentration (8). In rats, an increase in T_4 metabolic clearance rate, as well as decrease in T_4 plasma half life and total distribution space have been observed (9). However, a decline in T_4 plasma concentration was also stated in trained men to appear with decrease in degradation rate of T_4 (6). Increase in T_4 plasma concentration demonstrated by Katzeff and Selgrad (42) in exercising rats compared to nonexercising controls was connected with increase in T_4 total body pool and total volume of distribution and decrease in its plasma clearance rate.

Decrease in T_3 plasma concentration demonstrated in the present experiment could be treated as transitional stage in adaptation to enhanced physical activity, since it was found in rats trained with the lowest intensity. Unaltered plasma T_3 concentration, found in this experiment in rats trained at least 240 min weekly compared to untrained controls, has been also reported by others in trained rats (10), mice (12) and men (6, 7). Despite plasma T_3 concentration being unaffected by training, changes in T_3 metabolism have been found in trained men (6, 7) and laboratory rodents (10, 12). In trained men elevation in T_3 degradation rate together with increase in metabolic and urinary clearance rates have been shown (6, 7). Total volume of distribution, disposal rate and total body pool were found to be positively correlated with aerobic capacity (7). In trained rats an increase in T_3 metabolic clearance rate was demonstrated (10), while in mice with chronic enhancement of physical activity an increase in T_3 production rate without changes in clearance rate was noted (12).

Lack of changes in T_3 plasma concentration together with decrease in T_4 plasma level was stated to be associated with elevation in T_4 concentration in liver and diminution in T_3 concentration in heart and kidney in trained rats

compared to non — exercising controls (19). In another study, exercising rats were found to increase both T_4 and T_3 concentration in liver and their total distribution space (10).

It should be mentioned, that only in experiment performed by Rone *et al.* (7) in men effects of training and acute exercise were separated since subjects did not continue exercise during turnover study. Results obtained in this study clearly demonstrate, that enhanced T_3 metabolism represents event existing in resting conditions. Contrary to this, studies reporting enhanced T_4 and T_3 metabolism in trained rats (9, 10) did not enable the distinction between periods of activity and rest.

Despite elevation in T_4 and T_3 concentration in liver and increased metabolism of T_4 in trained rats reported by others (9, 10), hepatic 5'DI activity at rest was found to be unaffected by training in the present study, which is in agreement with the previous data obtained in rats (9) and mice (12). In addition, 5'deiodinating activity was found not to be altered in kidneys and muscles in trained rats (9). Unchanged plasma rT_3 level found in exercising rats in this study according to data obtained by others in men (43, 44) and rats (14) could confirm lack of training effect on hepatic 5'deiodinating activity in the resting state, since rT_3 plasma concentration is considered to be an index of 5'DI activity in liver (45). In the study conducted on mice after four weeks of spontaneous enhanced physical activity, decrease in hepatic 5'DI activity was observed in animals submitted to feeding restriction (12). Therefore, decrease in 5'DI activity could appear above the certain stage of energy deficit and/or shortage of its specific stimulant, such as glucose or T_3 (46, 47).

A decline in TPO activity at rest could contribute to the energy conservation trends observed in trained male rats (15, 18). It could be supposed that exercise training induced adaptations in thyroid hormone metabolism found in rats known not to increase their food intake to compensate for the energy expenditure attributable to exercise could also occur in humans submitted to the energy restriction, as in the treatment of obesity.

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