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PII S0024-3205(97)01114-4

Life Sciences, Vol. 62, No. 4, pp. 327–332, 1998

0024-3205/98 $19.00 + .00

THE EFFECTS OF CHRONIC EXERCISE ON METABOLIC AND REPRODUCTIVE FUNCTIONS IN MALE RATS

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(Received in final form November 10, 1997)

Summary

Previous studies concerning the effects of swimming on various endocrine gland functions have been performed(4,6). Our study was thus designed to analyze the effects of chronic exercise (swimming) on the resting metabolic rates (RMR) of adult rats. Most of the protocols used a water temperature of 33 C. It is our contention that such a protocol is not exclusively an exercise stress, but also a hypothermic stress (10). The protocol of our study was designed in a way that hypothermic stress was not part of the exercise stress. Male rats were swam in 36 C water for 3 hours a day, 5 days a week for 4 months. RMR of the animals were determined 24 h after the next to last swim session. Plasma hormone levels and epididymal sperm concentrations were determined in animals sacrificed 24 h after the last swim period. Exercising animals had a RMR 16% greater than that of control animals ($p < 0.02$), yet total and free thyroxine and total and free triiodothyronine were not significantly elevated. Neither plasma testosterone nor epididymal sperm counts were significantly reduced in the exercising animals. It appears that chronic exercise produces an elevation in RMR which is unrelated to thyroid gland activity and does not suppress the hypothalamic-pituitary-gonadal axis.

Key Words: exercise, swimming, thyroxine, triiodothyronine, TSH, testosterone, sperm

With the increased popularity of exercise training for both the maintenance of good health and the treatment of existing

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disease, hormonal changes associated with exercise have become an important clinical issue (6). There have been numerous studies in both humans and animals that have described changes in plasma hormone levels associated with exercise (4,6). When examining the effects of exercise on plasma hormone levels, the acute effects of exercise (i.e., the changes in hormone levels seen during or immediately after the exercise period) must be distinguished from the effects of training (i.e., chronic exercise programs).

The effects of training on plasma hormone levels have been studied in several laboratories. Studies have focused on reproductive dysfunctions associated with many female athletes (3), but the effects of exercise on male reproductive functioning have not been done (6). Studies on the effects of exercise on TSH or thyroid hormones are inconclusive (4,6). The present study was designed to examine the effects of chronic exercise on resting plasma testosterone, thyroid hormone levels, and spermatogenesis in male rats.

Methods

Exercise Protocol

Seven, 200 - 300 g male Sprague-Dawley rats were placed in a 21 x 16 inch diameter tank of water 14 inches deep and maintained at 36 C. The rats were allowed to swim 30 minutes the first day and then increased 30 minutes a day until the animals were swimming three continuous hours. The exercise was maintained at 3 hr/day, 5 days/week for four months. Seven control animals were placed in the swim tank for 1 min/day, 5 days/week for four months(10).

Determination of Resting Metabolic Rate

Three days prior to the completion of the swim protocol, and 24 hr after the completion of an exercise period, each rat was placed in a metabolism chamber containing soda lime as a CO_2 absorbent. The time required to consume 60 ml of O_2 was determined following a 10 min acclimatization period. This procedure was repeated two additional times. Liters of O_2 consumed/hr/Kg body weight was then determined for each animal.

Determination of Epididymal Sperm Content

Animals were sacrificed by decapitation and trunk blood was collected for hormone analysis, 24 hrs after the final exercise period. Both epididymi from each rat were excised and gently homogenized in 1 ml semen buffer (0.12 M NaCl, 4.3 mM KCl, 1.2 mM $MgSO_4$, 2 mM glucose, and 16 mM Hepes at pH 7.4). The total number of sperm present in 4 large grids of a standard hemocytometer was then determined and utilized for comparison of epididymal sperm content between control and experimental animals (7).

Determination of Plasma Hormone Levels

Plasma levels of total testosterone, total and free T_3, total and free T_4, plasma TBG capacity, and plasma TSH levels were determined using commercially available RIA kits purchased from Diagnostic Products Corporation (Los Angeles, CA).

Calculations

Percent T_3 uptake, free T_3 index and free T_4 index were calculated as follows.

$$\%\ T_3\ \text{uptake} = \frac{\text{Average cpm of unknown}}{\text{Average cpm of calibrator}} \times \text{Lot-specific value of calibrator}$$

$$\text{free } T_3 \text{ index} = \frac{\%\ T_3 \text{ uptake}}{100} \times \mu g\ T_3/dl$$

$$\text{free } T_4 \text{ index} = \frac{\%\ T_3 \text{ uptake}}{100} \times \mu g\ T_4/dl$$

Statistical Analysis

All data are reported as mean ± standard deviation with n = 7. The student's t-test was used to establish statistical significance with p values <0.05 being accepted as statistically significant.

Results

Resting Metabolic Rate

The resting metabolic rate of each control and experimental rat was determined three days prior to the completion of the four month exercise regimen and 24 hrs following the three hr exercise period. As can be seen in TABLE I, swimming increased the RMR 16%; L O_2/hr/Kg BW. This difference was statistically significant ($p<0.02$).

TABLE I. Resting Metabolic Rate

Group	Mean RMR	S.D.
Controls	0.9725	0.1177
Swimmers	1.1306*	0.1512

Thyroid Gland Activity

There were no significant increases in free or total T_3, free or total T_4, percent T_3 uptake, the free T_3 index, the free T_4 index, or plasma TSH levels as shown in TABLE II.

Gonadal Function

The exercise protocol used in this study did not result in significant changes in epididymal sperm counts (TABLE III) or in plasma testosterone levels (TABLE IV).

TABLE II. Plasma Thyroid Hormone and TSH Levels

Hormone	Controls*	Swimmers*
Free T_3 (pg/ml)	1.3 ± 0.4	1.8 ± 0.9
Total T_3 (ng/dl)	70.7 ± 6.8	67.1 ± 10.7
Free T_4 (ng/dl)	1.3 ± 0.5	1.2 ± 0.2
Total T_4 (μg/dl)	3.4 ± 0.7	3.5 ± 0.5
% T_3 Uptake	56.4 ± 1.3	56.3 ± 1.4
Free T_3 Index	39.9 ± 4.0	37.7 ± 5.5
Free T_4 Index	1.9 ± 0.4	2.0 ± 0.3
TSH (nIU/dl)	8.0 ± 7.2	6.2 ± 3.2

*Mean ± S.D.

TABLE III. Epididymal Sperm Counts

Group	Mean* ± S.D.
Controls	536 ± 129
Swimmers	463 ± 59

*Total sperm in four large hemocytometer grids/rat

TABLE IV. Total Plasma Testosterone Levels

Group	Mean* ± S.D.
Controls	1.170 ± 1.157
Swimmers	0.881 ± 0.581

*ng/ml

Discussion

MacConnie *et al.*(8) determined plasma levels of various reproductive hormones in well conditioned male athletes who were identified as marathon runners that averaged 125 - 200 km/week for 5 years. These individuals had resting plasma testosterone levels that were found to be comparable to sedentary controls. Studies in rats(5) also found that plasma testosterone levels were not effected by chronic exercise. Our findings also demonstrated that chronic exercise had no significant effect on

either plasma testosterone levels (an acute indicator of gonadal function) or epididymal sperm counts (a chronic indicator of gonadal function). One would conclude that the various changes found during exercise where not associated with changes in gonadal activity and must therefore be associated with a variable yet to be identified.

While exercise was associated with a significant increase in RMR and, as shown previously, an elevation in lysophospholipase activity(1), there were no concomitant increases in thyroid gland activity. There was no significant increase in free or total T_3, free or total T_4, percent T_3 uptake, the free T_3 index, the free T_4 index, or plasma TSH levels. These results agree with those of Rupp et. al (9) who also showed no increase in thyroid activity following chronic exercise in rats, despite finding significant changes in myosin V1 levels.

These data would support the hypothesis that the elevation in RMR is due to a post-transcriptional modification of individual cells. Simply stated, the cells produce an increase in certain key enzymes, thereby increasing the metabolic demands of the individual cells and, hence, the RMR of the entire animal. What triggers this increase in enzyme synthesis, and which enzymes are affected, has yet to be identified. Obviously, more information will be needed to prove this hypothesis.

Conclusion

Following a four month period of swimming 3 hr/day, 5 days/week, there was not a significant alteration in rat gonadal activity. Neither plasma testosterone levels nor epididymal sperm counts were significantly different from control animals. In addition, despite exercising rats having a 16% elevation in resting metabolic rate, we found no significant changes in either plasma TSH levels or thyroid gland activity.

Acknowledgements

The authors would like to thank the Nova Southeastern University College of Medical Sciences for providing financial support for this project.

References

1. P. AMINI-ALASHTI, V. INGRA-WILLEY, C.J. WOODY, S.L. WEBER, B.A. STURBAUM, H.E.LAUBACH, Biochem. Genet.(Life Sci. Adv.) **11** 13-18 (1993).
2. J.C. ARCOS, R.S. SOHAL, S. SUN, M.F. ARGUS, G.E. BURCH, Exptl. Molec. Pathol. **8** 49-65 (1968).
3. L.J. DEGROOT (Ed), *Endocrinology*, 2338-2347, W.B. Saunders Co, Philadelphia, PA (1987).
4. H. GALBO, Diabetes/Metabol. Rev. **1** 385-408,(1986).
5. M. HARKONEN, H. NAVERI, K. KUOPPASALMI, I. HUHTANIEMI, J. Steroid Biochem. **35** 127-132 (1990).
6. T.A. HOWLETT, Clin. Endocrinol. **26** 723-742 (1987).
7. W.G. KEMPINAS, T.L. LAMANO-CARVALHO, Lab. Animals **22** 154-156, (1988).

8. S.E. MACCONNIE, A. BARKAN, R.M. LAMPMAN, M.A. SCHORK, I.Z. BEITINS, N.E.J.M. **315** 411-417 (1986).
9. H. RUPP, R. WAHL, J. Appl. Physiol. **68** 973-978 (1990).
10. B.A STURBAUM, S.L. WEBER, H.E. LAUBACH, V. INGRAM-WILEY, P. AMINI-ALASHTI, C.J. WOODY, FASEB **6** C113 (1992).