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HORMONAL RESPONSES TO EXERCISE IN GIRLS DURING SEXUAL MATURATION

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The dependence of hormonal responses to exercise on sexual maturation was tested in three-year longitudinal experiment on 34 girls (11–12 years old at the beginning of the study). Sexual maturation of the girls was evaluated using Tanner scale. Girls were divided into three groups: maturation stages 1–2, 2–4 and 4–5. Children performed a 20-min cycle exercise at 60% of maximal oxygen uptake (VO max) once a year. Cortisol, insulin, somatotropin, β -estradiol, progesterone and testosterone were determined in venous blood by RIA procedures. High basal levels of β -estradiol and somatotropin appeared in stages 2–4 (387 ± 92 pmol \cdot l $^{-1}$ and 12.9 ± 2.85 ng \cdot ml $^{-1}$, respectively) and 4–5 (358 ± 54 pmol \cdot l $^{-1}$ and 14.3 ± 1.53 ng \cdot ml $^{-1}$, respectively). The basal progesterone level increased with maturation, testosterone appeared in the blood in stages 2–4 and 4–5. The exercise resulted in increased levels of cortisol and somatotropin, and a drop in insulin in all girls. The cortisol response was most pronounced in stage 1–2. Postexercise insulin concentration was the highest in stage 4–5. β -estradiol level increased by 23% in stages 1–2 and 4–5, while the response was insignificant in stages 2–4. Exercise-induced progesterone increase was significant in stage 4–5. In conclusion, sexual maturation associates with several quantitative changes in exercise-induced hormonal responses.

Key words: *cortisol, growth, insulin, longitudinal study, pubescent girls, sex hormones, sexual maturation, somatotropin*

INTRODUCTION

Several studies point on the possibility of alterations in exercise-induced hormonal responses during puberty. First of all, alterations are found in somatotropin response: in pubertal children of both gender the exercise-induced increase is more pronounced than in prepubertal ones (1, 2). In accordance, the somatotropin response has been found to increase with advanced sexual maturation (3). However, results of the study of Fahey *et al.*

(4) indicate that the amplitude but not the postexercise level of plasma somatotropin response to exercise is decreased in sexual maturation stage III due to the increased pre-exercise level of this hormone. Schwenk and Wennemann (5) reported in 1953 that cycling for 24 km causes increased excretion of corticosteroids in physically advanced 12- to 13-years old boys but not in physically retarded boys of the same age or in 10-year old boys. More recent study showed, however, that blood cortisol increases during 30-min physical activity in 10-years old fit boys (6). Corticotropin and β -endorphin responses (2) as well as catecholamine and insulin responses occurred to be similar in prepubertal, pubertal and postpubertal boys and girls (7). In one study significant decreases of serum insulin concentration were found only in subjects of the final stage of sexual maturation but not in less mature preadolescents (4).

The obtained information is not sufficient for generalizing conclusion. Therefore a study was conducted in order to establish relations between exercise-induced hormonal changes and sexual maturation stages in girls.

METHODS

Subjects

34 healthy girls (11 or 12 years old at the beginning of the three-year study) were examined. In the second and third year the number of studied children was 32 and 29, respectively. An informed consent was obtained from all children, their parents and the principal of the school. The design of the research was approved by the Ethical Commission of the Medical Faculty of the University of Tartu.

All children practiced twice a week in school physical education classes. In addition, 10 girls had training experience from the age of 9 years. Nine of them practiced basketball and 1 cross-country skiing. Basketball players exercised 1.5 h thrice a week, the skier 2 h four times a week. All children were under a regular medical care in school. The girls participating in sports training regularly underwent sportmedical examinations. In no case major health disorders were found during the longitudinal experiment or during the years preceding the experiment.

Procedure

During three consecutive years the exercise tests and other examinations were made in October or November. On the first day the children performed an incremental exercise test on a bicycle ergometer. In 11-year-old girls the test exercise started at 50 W, in 12-year-old and older children at 100 W. After every 2 min the exercise intensity was increased by 50 W. During the three steps of exercise intensity the pedalling rate was 60 r.p.m. Then during the last minute of exercise the children were asked to increase the pedalling rate up to the highest possible in order to attain the highest level of power output. During the last 30 s of each step of exercise the expired air was collected using Tissot gasometer. O_2 and CO_2 contents of expired air were determined with the aid of an electrochemical analyzer (AC 0011—0012, Factory of Gasoanalysers, Võru, Estonia). The highest level of VO_2 was considered to be the VO_{2max} .

On the second experimental day the 20-min exercise at 60% VO_2max was performed on the same bicycle ergometer. Before the exercise children rested in the laboratory 60 to 90 min. Then a Teflon catheter was inserted into the antecubital vein and the first blood sample was withdrawn. The second blood sample was taken 5 min after the end of exercise.

Exercise tests were performed between 9 and 11 a.m. Before the test children were allowed to consume their usual breakfast. The time interval between the breakfast and the first blood sample was at least 2 hours. The children were asked not to perform any exercise on the morning before the test as well as during the day before. Children were recommended not to change their habitual diet at least during a week before the exercise tests.

On the third experimental day the anthropometrical measurements and examination for evaluation of sexual maturation stages were performed.

Hormonal analyses

Blood was immediately centrifuged and samples of serum were immediately cooled in liquid nitrogen and stored at -20°C . Cortisol, insulin, somatotropin, β -estradiol, progesterone, and testosterone concentration were determined by RIA-tests. The commercial kits of ICN Micromedic Systems (Horsam, USA) was used for insulin and of Orion Diagnostics (Espo, Finland) for other hormones.

Analyses were carried out in duplicate. Quality control was included in all sets of determination. The coefficients of variation between duplicate analyses were within 3.4 to 6.6%. Pre- and post-exercise determinations for a particular hormone were made at the same time to avoid inter-assay variations.

Evaluation of sexual maturation

Tanner's (8) five-stage scale of development of sex characteristics was used. In most girls during each experiment the evaluations by pubic and axillary hair, development of mammary glands and the time of menarche overlapped over two or even three stages. Therefore, the children were divided into three groups. The first group was made up by children who belonged to the stage 1 at least by two or three indices of sexual maturation. Girls with evaluation of stages II or III by three indices or having various combination of evaluations II, III and IV were included into the second group. Third group constituted of children exhibiting evaluations of stages IV or V.

Statistical analysis

Two approaches were used for analysis of the obtained material. For the cross-sectional comparison of children of different maturation stages, results were added up taking into account the sexual maturation stage during each of the three years.

For evaluation of the outcomes of the longitudinal study, the girls were divided into 5 groups: 1 — girls who remained in stage 1—2 in two subsequent years, 2 — girls who transferred from stage 1—2 to stage 2—4 for the next year, 3 — girls who remained in stage 2—4 in two subsequent years, 4 — girls who transferred from stage 2—4 to stage 4—5, 5 — girls who were in stage 4—5 in two subsequent years.

The analysis of groups values was performed by one-way analysis of variance and by the Student-Fisher t-test. When significant F ratios were obtained, the differences between groups

values were evaluated. The 0.05 probability level was accepted as significant. The individual changes either during exercise or from one experiment to the subsequent experiment next year were evaluated with the paired t-test.

Possible differences in distribution of results obtained in children practicing sports and in their less active counterparts were tested using the Chi-square criterion.

RESULTS

General characteristics of maturation pattern

The distribution of girls by chronological age, sexual maturation stages, and menarche is presented in *Table 1*. All three sexual maturation groups included children of ages from 11—12 up to 13—14. Consequently, a strict dependence of sexual maturation from chronological age was not established.

Table 1. General characteristics of maturation pattern in studied girls

	First year	Second year	Third year
Distribution of children (in brackets the number of children practicing in sports)			
Chronological age			
11 years	13(2)	—	—
12 years	21(9)	13(2)	—
13 years	—	19(7)	12(1)
14 years	—	—	17(6)
Stages of sexual maturation			
1—2	15(4)	10(3)	—
3—4	11(5)	5(1)	12(4)
4—5	8(2)	17(5)	17(3)
Menarche			
pre	24(9)	14(5)	6(3)
post	10(2)	13(4)	23(1)

Children practising sports revealed a trend to a delayed menarche. The analysis of distribution of results between children of sports experience and in others with the aid of the Chi-square criterion indicated that on the third year of the experiment, the distribution of postmenarcheal and premenarcheal children was significantly different ($X^2 = 8.51$, $P < 0.05$) between groups formed by sport experience, proving the association of delayed menarche with sports training.

In stages 2—4 and 4—5 the height was greater than in stage 1—2. The weight and absolute levels of VO_2 max increased with sexual maturation while the values of VO_2 max relative to body mass remained constant (*Table 2*).

Table 2. Height, weight, aerobic power and basal hormone levels of girls in various stages of sexual maturation (means \pm SEM)

	Stage of sexual maturation		
	1—2 n = 28	2—4 n = 25	4—5 n = 42
Age, years	12.0 \pm 0.3	12.7 \pm 0.2	13.2 \pm 0.1
Height, cm	154 \pm 1.2	161 \pm 1.2	164 \pm 0.8
Weight, kg	39.5 \pm 0.9	47.0 \pm 1.0	51.9 \pm 0.7
VO ₂ max, l \cdot min ⁻¹ ml \cdot min ⁻¹ \cdot kg ⁻¹	1.665 \pm 0.069 42.4 \pm 1.8	1.990 \pm 0.067 42.2 \pm 1.6	2.315 \pm 0.059 44.9 \pm 1.1
β -estradiol, pmol \cdot l ⁻¹	197 \pm 25	387 \pm 92	358 \pm 54
Progesterone, nmol \cdot l ⁻¹	1.4 \pm 0.5	2.7 \pm 1.0	3.4 \pm 1.1
Testosterone, nmol \cdot l ⁻¹	\sim 0	< 1.0	1.6 \pm 0.2
Somatotropin, ng \cdot ml ⁻¹	8.1 \pm 1.6	12.9 \pm 2.9	14.3 \pm 1.5
Cortisol, nmol \cdot l ⁻¹	300 \pm 22	310 \pm 31	271 \pm 19
Insulin, μ U \cdot ml ⁻¹	18.6 \pm 1.7	15.8 \pm 1.3	21.9 \pm 1.4

Values are mean \pm SE. Statistically significant differences ($P < 0.05$) are denoted by horizontal lines with asterisks or by asterisks between columns.

Basal levels of hormones

The β -estradiol level was elevated in stage 2—4 and 4—5 in comparison with that in stage 1—2 (Table 2). Progesterone levels were low in stages 1—2 and gradually increased in stages 2—4 and 4—5. Testosterone level in the blood was detectable in 24% of girls of stage 1—2, 74% of stage 2—4 and 84% of stage 4—5. Somatotropin concentration was the highest in stage 4—5, revealing a statistically significant difference from values in stage 1—2 ($P < 0.05$), but not from values in stage 2—4. Differences in insulin levels were insignificant.

The longitudinal comparison of results confirmed the increase in β -estradiol level in stages 2—4 (Fig. 1). When stage 4—5 was achieved the β -estradiol level decreased ($P < 0.05$). A trend to reduced β -estradiol level appeared after an one-year period in stage 4—5.

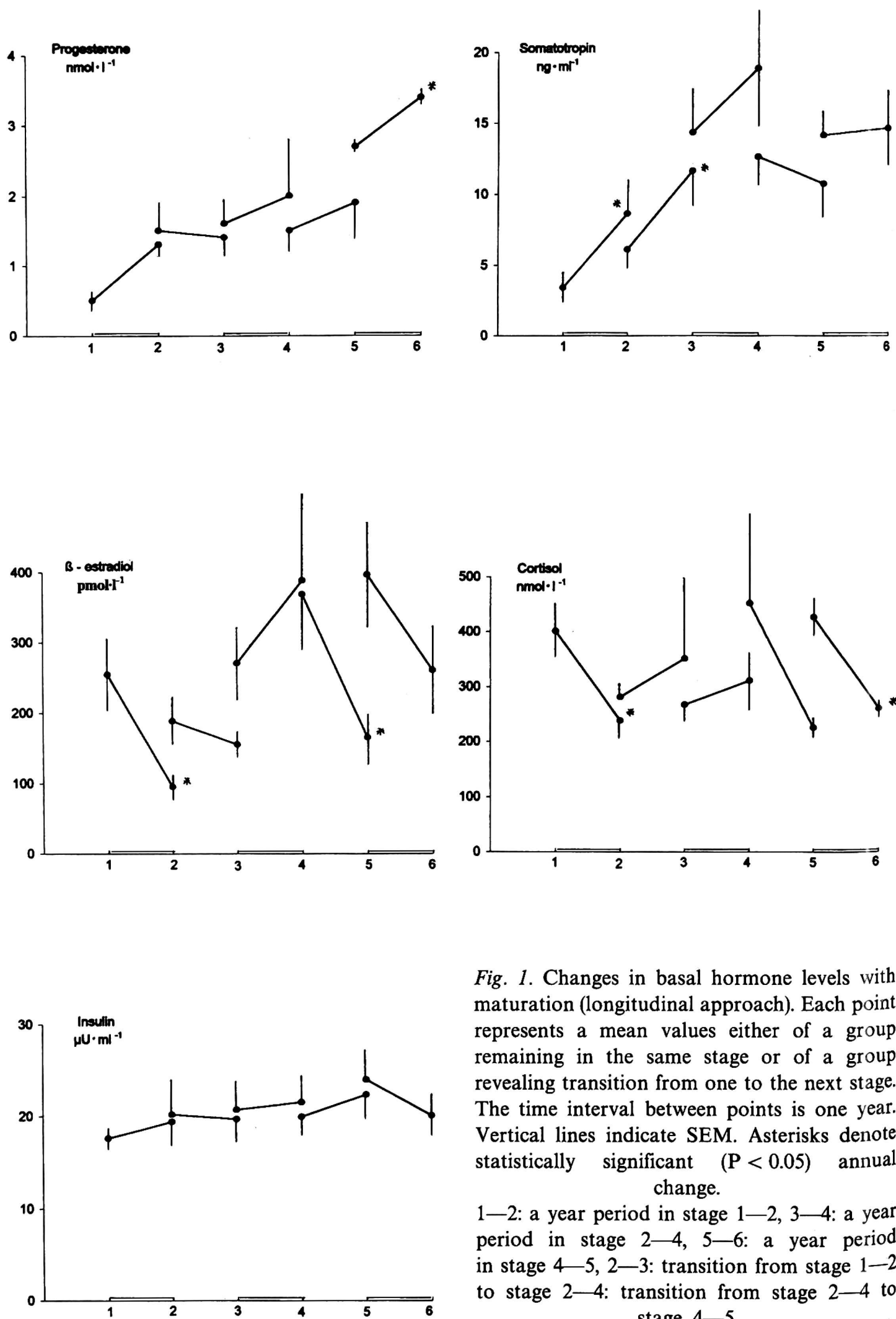


Fig. 1. Changes in basal hormone levels with maturation (longitudinal approach). Each point represents a mean values either of a group remaining in the same stage or of a group revealing transition from one to the next stage. The time interval between points is one year. Vertical lines indicate SEM. Asterisks denote statistically significant ($P < 0.05$) annual change.

1—2: a year period in stage 1—2, 3—4: a year period in stage 2—4, 5—6: a year period in stage 4—5, 2—3: transition from stage 1—2 to stage 2—4: transition from stage 2—4 to stage 4—5.

Progesterone concentration tended to increase during the stay in all stages.

Somatotropin concentration increased significantly ($P < 0.05$) during the stay in stage 1—2 and with entry into stage 2—4. The highest level of somatotropin ($18.8 \pm 4.1 \text{ ng} \cdot \text{ml}^{-1}$) appeared after a year in stage 2—4. The level reduced insignificantly to $10.7 \pm 2.4 \text{ ng} \cdot \text{ml}^{-1}$ after stage 4—5 was achieved.

Although the cross-sectional comparison indicated the stable basal level of cortisol (*Table 2*), longitudinal evaluation of results demonstrated pronounced variability of cortisol concentrations (*Fig. 1*). After a year in stage 1—2 or 4—5 the cortisol level decreased significantly ($P < 0.05$). The transition from stage 1—2 to stage 2—4 was accompanied with an insignificant increase ($P < 0.05$) in the cortisol level. The opposite change was found with achieving stage 4—5.

Exercise-induced hormonal responses.

In all stages of sexual maturation the common responses were an increase in the blood concentrations of cortisol and somatotropin, and a decrease in the blood insulin level (*Table 3*). There were no significant differences in post-exercise levels of cortisol and somatotropin between groups of sexual maturation (*Fig. 2*). The response of cortisol was in stage 1—2 higher than in other stages (*Tables 3*). In stage 2—4 the somatotropin response was

Table 3. Changes of hormone levels during 20-min cycling exercise in various stages of sexual maturation

Hormone	Sexual maturation stages		
	1—2	2—4	4—5
β -estradiol $\text{p mol} \cdot \text{l}^{-1}$	$+46 \pm 15^*$	$+16 \pm 24$	$+88 \pm 20^*$
Progesterone $\text{nmol} \cdot \text{l}^{-1}$	$+0.1 \pm 0.2$	$+0.4 \pm 0.6$	$+0.9 \pm 0.3^*$
Somatotropin $\text{ng} \cdot \text{ml}^{-1}$	$+13.9 \pm 4.9^*$	$+20.1 \pm 4.8^*$	$+15.9 \pm 1.8^*$
Cortisol $\text{nmol} \cdot \text{l}^{-1}$	$+224 \pm 28^*$	$+134 \pm 26^*$	$+138 \pm 17^*$
Insulin $\mu\text{U} \cdot \text{ml}^{-1}$	$-9.9 \pm 1.3^*$	$-7.1 \pm 1.2^*$	$9.5 \pm 1.1^*$

insignificantly higher than in other stages. The postexercise insulin level was significantly higher in the group of maturation stage 4—5 than in groups of stages 1—2 or 2—4 (*Fig. 2*), but no significant difference was found in the amplitude of response (*Table 3*).

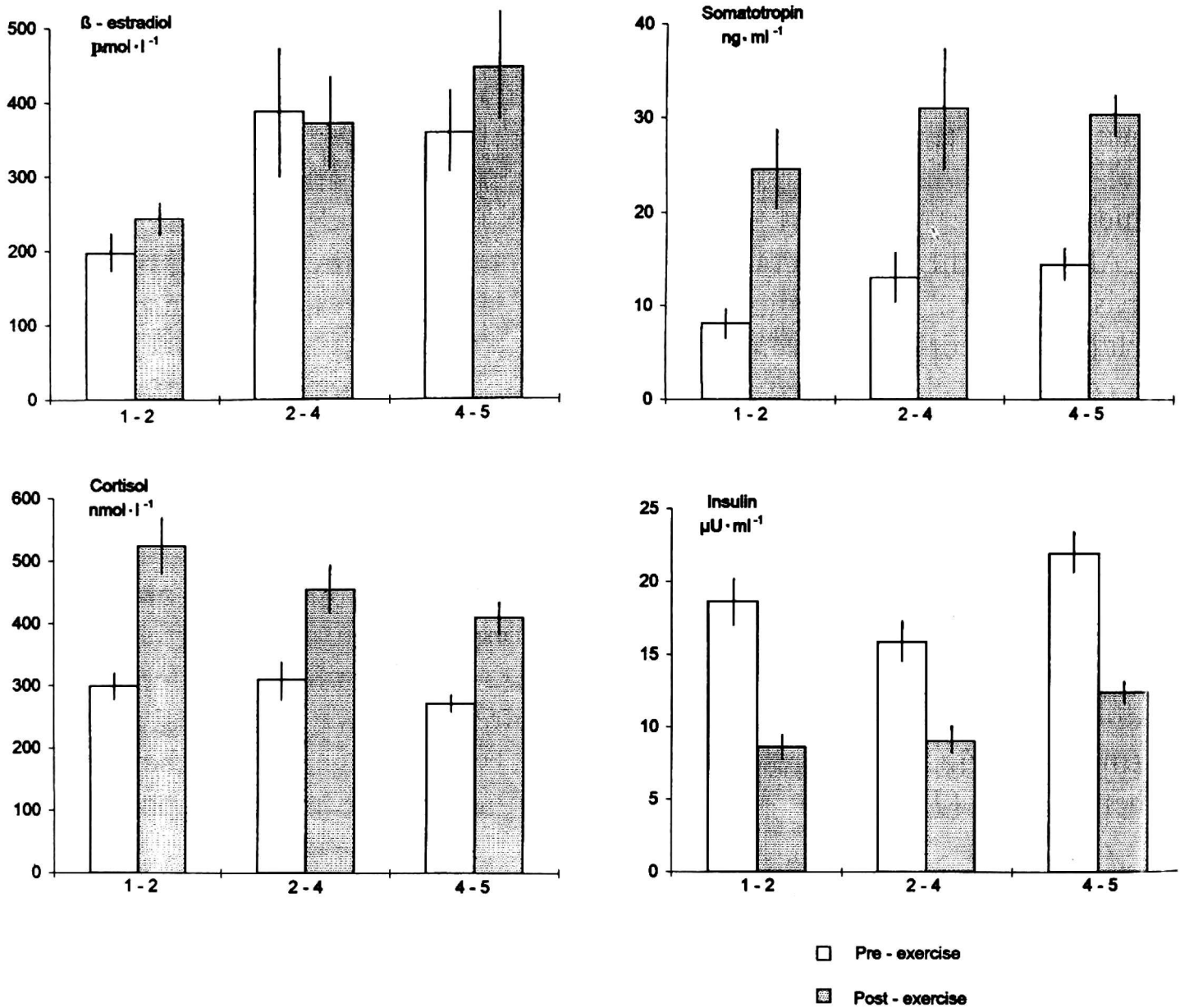


Fig. 2. Hormone levels before and after 20-min cycling exercise at 60% VO_2max in sexual maturation stages 1—2, 2—4 and 4—5. White columns — before exercise, striated columns — after exercise.

The longitudinal approach allowed us to confirm the decreased amplitude of cortisol response when girls transferred from stage 1—2 to stage 2—4 (Fig. 3). However the same was revealed in those girls who remained in stage 1—2 or 2—4 for a year. Postexercise levels of insulin and insulin responses did not alter. The highest somatotropin response ($+26.6 \pm 5.9$) was found in those girls of stage 1—2 who achieved stage 2—4 after a year. Post-exercise somatotropin levels was the highest in girls reached stage 2—4 ($37.8 \pm 6.8 \mu\text{g}\cdot\text{ml}^{-1}$).

Cross-sectional comparison (Fig. 2) demonstrated that β -estradiol level increased during exercise by 18% from a comparatively low level ($197 \pm 25 \text{pmol}\cdot\text{l}^{-1}$) in stage 1—2. The paired t-test allowed us to establish a significant increase of β -estradiol level with the exercise also in stage 4—5, but not in stage 2—4. These results were confirmed by the longitudinal approach of analysis (Fig. 3). The β -estradiol response became insignificant ($P > 0.05$) in stage 2—4 despite high mean values.

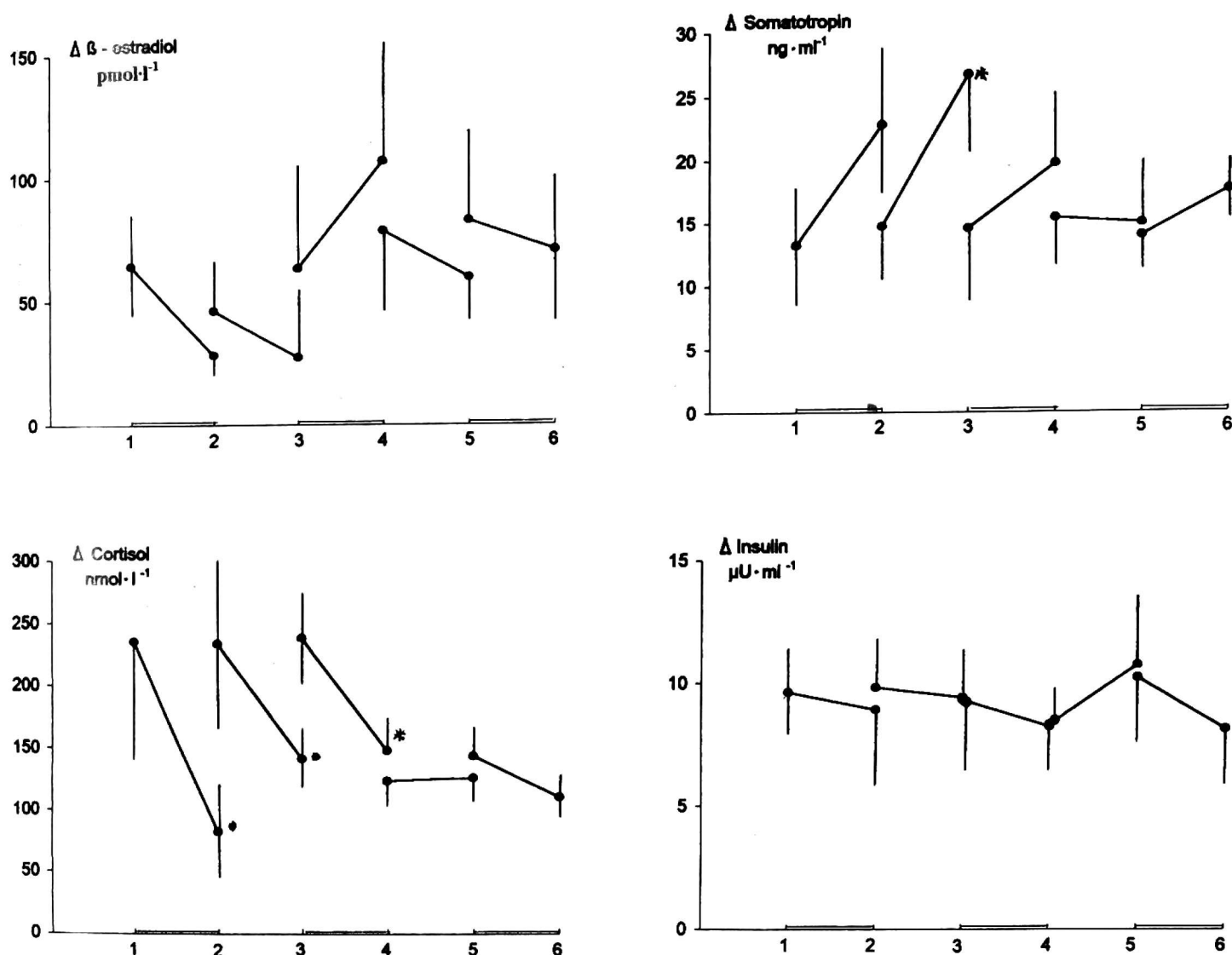


Fig. 3. Changes in hormone responses to exercise with maturation (the longitudinal approach). For further explanation see Fig. 1.

The progesterone level increased during exercise in 63% of the girls in stages 1—2 and in 50% in stage 2—4. In stage 4—5 the progesterone response to exercise was statistically significant (*Table 3*). In postmenarcheal girls progesterone levels tended to be higher than in premenarcheal girls before exercise, (2.6 ± 0.41 and 1.5 ± 0.69 nmol·l⁻¹, respectively).

After exercise the difference between plasma progesterone concentration between post- and premenarcheal girls became significant (3.2 ± 0.37 and 1.8 ± 0.40 nmol·l⁻¹, respectively).

DISCUSSION

An essential marker of sexual maturation is the increased level of sex hormones. The results obtained in this study showed that a high level of β-estradiol appears in girls in stage 2—4 and persists also in stage 4—5 of sexual maturation. The progesterone level increased gradually in association with sexual maturation. Blood testosterone level became detectable

(> 1.0 nmol·l⁻¹) in most girls in stage 2—4. These patterns as well as the quantitative values of hormone levels in various stages of sexual maturation fit with results of other studies (9, 10). However, the longitudinal approach used in the present study allowed us to indicate some additional feature of the pattern. During the time period of stage 2—4 the β -estradiol level tended to increase, but after transition to stage 4—5, a temporary reduction of β -estradiol level occurred. After a year in stage 4—5 a trend to reduced β -estradiol level appeared again. The existence of these trends makes it possible to explain why we did not find in stage 4—5 a higher β -estradiol level in comparison with stage 2—4, as it was demonstrated in the study of Ducharme *et al.* (9). Comparison of pre- and postmenarcheal girls in β -estradiol concentration did not reveal significant difference.

The exercise resulted in an increase in the β -estradiol level from a low initial level in stage 1—2 and again in stage 4—5 although in this stage the basal level was high. The response disappeared in stage 2—4 when the basal β -estradiol level began to increase. These results indicate that in stage 1—2, despite the low basal level, there is a possibility to increase secretion of the hormone during exercise. However, this possibility disappears in association with the increased basal level in stage 2—4, and was restored after transition to stage 4—5, probably due to the functional maturation of the pituitary-gonadal system. A question remains whether these changes of β -estradiol response to exercise are related to the functional capacity of estrogen producing cells or to the regulatory influences from the adenohipophysis. The β -estradiol response disappeared after an one-year period in stage 4—5. This fact speaks for the significance of regulatory influences. However, this suggestion needs further support.

The significance of periodic formation of the corpus luteum may be considered as the apparent reason why the progesterone concentrations was in stage 4—5 (93% of postmenarcheal girls) significantly higher than in stage 1—2 (no postmenarcheal girls). However in stage 4—5 the progesterone concentration only tended to be higher than in stage 2—4, although in the stage 2—4 the percent age of postmenarcheal girls was only 26. Moreover, a comparison of progesterone level in pre- and postmenarcheal girls indicated only an insignificantly higher concentration in the latter group (2.6 ± 0.38 mmol·l⁻¹ vs. 1.5 ± 0.69 mmol⁻¹). Obviously the significance of *corpus luteum* was matched (1) by progesterone originating from synthesis of other steroids as a byproduct, (2) by the time relation between blood sampling and formation of the *corpus luteum*, after the ovulation.

An ability to increase the plasma levels of progesterone and testosterone during exercise appeared in association with elevation of their basal levels. In the postexercise blood samples testosterone was detectable at an earlier age than in the pre-exercise samples. Plausibly, this is related to the

exercise-induced increase in the production of adrenal steroids. In association with the increased cortisol secretion a modest amount of androgens is released into the blood stream and can be converted to testosterone (11).

Several studies confirm the function of somatotropin in somatic growth and demonstrated an increase in somatotropin secretion in association with the growth spurt (12,13). Accordingly, we observed the most pronounced increase in basal somatotropin concentration during transition from maturation stage 1—2 to stage 2—4 in conjunction with a pronounced increase in height.

Exercise-induced increase of blood somatotropin level in 10 to 15-year-old preadolescents has been reported in several studies (14—17). Exercise tests are advised for screening of somatotropin deficiency in children (18, 19). Several studies indicate that in pubertal and postpubertal teenagers the blood somatotropin level increases during exercise to higher levels than in prepubertal children (1—3). Our results confirmed that in stage 1—2 the average postexercise somatotropin level is lower than in stages 2—4 and 4—5, but the differences were insignificant ($P > 0.05$). The longitudinal comparison allowed us to demonstrate that the highest somatotropin response appears after transition to stage 2—4.

The basal cortisol levels in girls of various stages of sexual maturation fit well the data of Ducharme *et al.* (9). Taking into account the amplitude of individual changes, the exercise-induced cortisol increase was the highest in stage 1—2. This may be related to the altered interrelations between various endocrine systems caused by sexual maturation. If this suggestion is correct, one must assume that increased activity of pituitary-gonadal system exerts a certain inhibitory influence on the exercise-induced glucocorticoid response. The alternative explanation is that the same relative exercise intensity exerts a stronger influence on prepubescent than pubescent children. The latter possibility is supported by the facts that in the course of sexual maturation the heart rate response to submaximal exercise decreases (1).

Insulin may play an important role in growth stimulation. A question arises whether the comparatively high insulin level in the final maturation stage ($21.9 \pm 1.4 \mu\text{g} \cdot \text{ml}^{-1}$) is related to growth control. In all stages of sexual maturation insulin level drops during the exercise. This is in accordance with previous findings showing that prolonged exercise causes a decrease in insulin level in pre- and postpubertal teenagers of both genders (7, 15, 16). The response occurred to be more pronounced in boys than in girls (7) However, Fahey *et al.* (4) reported that the drop in insulin level appears only in the final stage of sexual maturation in males.

In conclusion, the obtained results indicate that in pubescent girls the exercise-induced hormonal responses are qualitatively the same as in adults (20, 21). However, several quantitative aspects differences in these responses exist between girls of various stages of sexual maturation.

REFERENCES

1. Wirth A, Träger E, Scheele K, Mayer D, Diehm K, Reischle K, Weicker H. Cardiopulmonary adjustment and metabolic response to maximal and submaximal physical exercise of boys and girls at different stages of maturation. *Eur J Appl Physiol* 1978; 39: 229—240.
2. Bouix O, Brun JF, Fedou C *et al.* Plasma beta-endorphin, corticotropin and growth-hormone responses to exercise in pubertal and prepubertal children. *Horm Metab Res* 1994; 26: 195—199.
3. Marin G, Domene HM, Barnes KM, Blackwell BJ, Cassorla FG, Cutler GB. The effects of estrogen priming and puberty on the growth-hormone response to standardized treadmill exercise and arginine-insulin in normal girls and boys. *J Clin Endocrin Metab* 1994; 79: 537—541.
4. Fahey DT, Valle-Zuris AD, Oehlsen G, Trieb M, Seymour J. Pubertal stage differences in hormonal and hematological responses to maximal exercise in males. *J Appl Physiol* 1979; 46: 823—827.
5. Schwenk A, Wennemann J. Die Harnsteroidausscheidung bei früh und spätereifenden Jungen im Pubertätsalter unter körperlicher Belastung. *Z Kinderheilkd* 1953; 73: 407—420.
6. Zuliani U, Cacciari E, Fiorella PL *et al.* L'attività sportiva in età prepubere. *Medicina dello Sport* 1992; 45: 109—117.
7. Delamarche P, Gratas-Delamarche A, Monnier M, Mayet MH, Koubi HE, Favier R. Glucoregulation and hormonal changes during prolonged exercise in boys and girls. *Eur J Appl Physiol* 1994; 68: 3—8.
8. Tanner JM. *Growth at Adolescence*. Blackwell 1962.
9. Ducharme J-R, Forest MG, De Peretti E, Sempre M, Collu R, Bertrand J. Plasma adrenal and gonadal sex steroids in human pubertal development. *J Clin Endocrinol Metab* 1976; 42: 468—476.
10. Jenner MR, Kelch RP, Kelch SL, Kaplan SL, Grumbach MM. Hormonal changes in puberty. IV Plasma estradiol, LH, FSH in prepubertal children, pubertal females, and in precocious puberty, premature menarche, hypogonadism, and in child with a feminizing ovarian tumor. *J Clin Endocrin* 1972; 34: 521—530.
11. Rivarola MA, Saez JM, Meyer WJ, Jenkins ME, Migeon CJ. Metabolic clearance rate and blood production rate of testosterone and androst-4-ene-3, 17-dione under basal conditions, ACTH and HCG stimulation. *J Clin Endocrinol Metabol* 1966; 26: 1208—1218.
12. Kaplan S, Abrams C, Bell J, Conte F, Grumbach M. Growth and growth hormone. *Pediatr Res* 1968; 2: 43—63.
13. Kosyto JL, Isaakson O. Growth hormone and regulation of somatic growth. *Int Rev Physiol* 1977; 13: 255—274.
14. Buckler JM. The effect of age, sex and exercise on the secretion of growth hormone. *Clin Sci* 1969; 37: 765—774.
15. Oseid S, Hermansen L. Hormonal metabolic changes during and after prolonged muscular work in pre-pubertal boys. *Acta Paediatr Scand Suppl* 1971; 217: 147—153.
16. Eriksson BO, Persson B, Thorell JI. The effects of repeated prolonged exercise on plasma growth hormone, insulin, glucose, free fatty acids, glycerol, lactate and γ -hydroxybuturic acid in 13-year old boys and in adults. *Acta Paediatr Scand Suppl* 1971; 217: 142—146.
17. Eriksson BO, Thorell J. The effect of two different types of maximal exercise on the plasma level of growth hormone in 13-year-old boys. *Acta Paediatr Belgica* 1974; 28 (Suppl): 274—286.
18. Keenan B, Killmer L, Sode J. Growth hormone response to exercise. A test of pituitary function in children. *Pediatrics* 1972; 50: 760—764.

19. Lacey KA, Hewison A, Parkin JM. Exercise as a screening test for growth hormone deficiency in children. *Arch Dis Child* 1973; 48: 508—512.
20. Galbo H. Hormonal and Metabolic Adaptation to Exercise. G. Thieme Verlag, 1983.
21. Viru A. Hormones in Muscular Activity. Hormonal Ensemble in Exercise Vol. 1. CRC Press, 1985.

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