Effects of Exercise on the Serum Concentrations of FSH, LH, Progesterone, and Estradiol


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Summary. The effects of 30 min of exercise (74.1 ± 3.0% \( VO_2 \)) on the responses of progesterone (P), estradiol \( (E_2) \), follicle stimulating hormone (FSH), and luteinizing hormone (LH) were investigated in 10 women. With such exercise significant increments occurred in P (37.6 ± 9.5%) and \( E_2 \) (13.5 ± 7.5%) \( (p < 0.05) \), whereas no changes were observed in FSH and LH \( (p > 0.05) \). Exercise in the luteal phase and during menses provoked similar changes in P, but \( E_2 \) concentrations remained unchanged when exercise occurred during menses \( (p > 0.05) \). With 8–11 weeks of training the menstrual cycles were quite irregular and retesting of subjects in the same phase of the cycle was not possible. Yet, when subjects were retested after training, no changes occurred in P, \( E_2 \) or LH \( (p > 0.05) \) but a decrement did occur in FSH \( (p < 0.10) \). Thus, heavy exercise in untrained subjects provokes significant increments in ovarian hormones, whereas no such increments are observed in trained subjects exercising at the same absolute workload.

Key words: Women — Exercise — Training — Hormones

Ovarian and gonadotropic hormones have well-defined, characteristic patterns during the normal menstrual cycle (Speroff and VandeWiele, 1971). Recently, it has been reported that intense training can interfere with the normal menstrual cycle (Erdelyi, 1976) and that teenage swimmers experience menstrual cycles with an abbreviated luteal phase (Bonen et al., 1978). Such cycles are characterized by lowered concentrations of follicle stimulating hormone (FSH) (Bonen et al., 1978), and no rise in progesterone (P) (Bonen and Belcastro, 1978).

Acute exercise provides a convenient model by which alterations in substrate and endocrine metabolism can be investigated (Bonen et al., 1979). However, in virtually all metabolic studies data are usually obtained from male subjects, presum-
ably since there is a suspicion that the large fluctuations in menstrual cycle hormones may exert an unknown effect on substrate metabolism. This has recently been reinforced by the evidence that oral contraceptives have a marked effect on carbohydrate and lipid metabolism (Beck, 1973; Bostofte et al., 1978; Hagenfeldt et al., 1977). However, the effects of exercise on circulating menstrual cycle hormones is only just being evaluated (Bonen et al., 1978; Bonen and Belcastro, 1978; Jurkowski et al., 1978). Moreover, neither substrate metabolism in different phases of the menstrual cycle nor the effects of training on such metabolism have been determined. Prior to such studies the responses of the menstrual cycle hormones to exercise must be clearly identified. Therefore, the purpose of this study was to investigate the effects of an acute bout of exercise on the circulating concentrations of luteinizing hormone (LH), FSH, P, and 17β-estradiol (E₂).

**Method**

Ten young women participated in this investigation. Their physical characteristics (± SEM) were as follows: age: 21.1 ± 0.6 years; height: 164.5 ± 1.4 cm; weight: 61.3 ± 1.8 kg; maximal oxygen intake (VO₂ max): 2.37 ± 0.12 l/min. Eight subjects had never used oral contraceptives, and two women had ceased treatment 2 months and 1 year, respectively, prior to this study.

Several days before the experimental sessions the subjects were familiarized with bicycle ergometry exercise, blood sampling, and oxygen consumption assessment. At this time they also completed three submaximal, 6-min exercise bouts and a load incremented (180 kpm/3 min) bicycle ergometer test to exhaustion, from which their maximal oxygen intake was determined with the open circuit method previously described (Belcastro and Bonen, 1975).

To assess the effects of exercise on the serum concentration of FSH, LH, E₂, P, each subject performed 30 min of intense exercise on a bicycle ergometer. Upon arrival in the laboratory, the subjects dressed for the exercise and then rested for 30 min before commencing exercise. Fifteen minutes prior to exercise, a 15-ml venous blood sample was obtained and designated as the pre-exercise sample. During the 30 min of exercise, the energy expenditure (Belcastro and Bonen, 1975) and heart rates were monitored from min 10–15, and on several occasions from min 25–30. Between min 15–17, the subjects were given a brief rest to obtain a blood sample. An additional blood sample was obtained immediately at the end of the 30-min exercise. In three subjects, these procedures were repeated at a lower exercise intensity two days later. Serum samples were stored at -20°C prior to analysis.

Seven of the subjects volunteered to train 30 min/day, 3 days/week for 8 weeks. However, during this phase of study, difficulties were encountered with retesting of the subjects (see discussion for details).

A simple, effective procedure for the extraction and separation of P and E₂ in the same serum sample was used. Briefly, steroids in 1.0 ml serum were extracted with 10.0 ml anhydrous ether. The individual steroids in the extract were then separated and purified by partition chromatography on a celite column. With ethylene glycol as stationary phase, P, 17α-hydroxyprogesterone (17P) and E₂ were successively eluted in iso-octane, 15% ethyl acetate in iso-octane, and 40% ethyl acetate in iso-octane, respectively. In addition, very sensitive and accurate radioimmunoassays for P and E₂ were established. The specific antibodies for these steroids were purchased from Dr. G. E. Abraham, for use with his assay method (Abraham et al., 1971). Under our assay conditions, the range for E₂ was 3–200 pg, and for P, it was 5–400 pg. Cross-reactions with other closely related steroids with anti-P and anti-E₂ were only significant for deoxycorticosterone (35%) and estrone (35%), respectively. This presented little problem in our assay, since these steroids separate quite distinctly from P and E₂ during partition chromatography.

The within and between assay variance was evaluated by duplicate measurements of the same samples in the same assay and in two different assay runs. The intra-assay and inter-assay coefficients of variation for P were 8.6% and 18.0%, respectively. Those for the E₂ assay were 5.5% and 12.6%, respectively.