No effect of menstrual cycle on myofibrillar and connective tissue protein synthesis in contracting skeletal muscle

Benjamin F. Miller,1 Mette Hansen,1 Jens L. Olesen,1 Allan Flyvbjerg,2 Peter Schwarz,3 John A. Babraj,4 Kenneth Smith,4,5 Michael J. Rennie,4,5 and Michael Kjaer1

1Institute of Sports Medicine, Copenhagen, Bispebjerg Hospital, Copenhagen; 2Department of Endocrinology, Aarhus University Hospital, Aarhus; 3Osteoporosis and Metabolic Bone Unit, Department of Clinical Biochemistry, Copenhagen University Hospitals, Hvidovre, Denmark; 4Division of Molecular Physiology, Faculty of Life Sciences, University of Dundee, Dundee, Scotland; and 5University of Nottingham, Graduate Medical School, Derby City General Hospital, Derby, England, United Kingdom

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Miller, Benjamin F., Mette Hansen, Jens L. Olesen, Allan Flyvbjerg, Peter Schwarz, John A. Babraj, Kenneth Smith, Michael J. Rennie, and Michael Kjaer. No effect of menstrual cycle on myofibrillar and connective tissue protein synthesis in contracting skeletal muscle. Am J Physiol Endocrinol Metab 290: E163–E168, 2006.—We tested the hypothesis that acute exercise would stimulate synthesis of myofibrillar protein and intramuscular collagen in women and that the phase of the menstrual cycle at which the exercise took place would influence the extent of the change. Fifteen young, healthy female subjects were studied in the follicular (FP, n = 8) or the luteal phase (LP, n = 7, n = 1 out of phase) 24 h after an acute bout of one-legged exercise (60 min of kicking at 67% \( W_{\text{max}} \)). samples being taken from the vastus lateralis in both the exercised and resting legs. Rates of synthesis of myofibrillar and muscle collagen proteins were measured by incorporation of \([1^{3}]\text{Cl}	ext{eucine}, \text{Myofibrillar protein synthesis (means ± SD; rest FP: 0.053 ± 0.009%/h, LP: 0.055 ± 0.013%/h) was increased at 24-h postexercise (FP: 0.131 ± 0.018%/h, P < 0.05, LP: 0.134 ± 0.018%/h, P < 0.05) with no differences between phases. Similarly, muscle collagen synthesis (rest FP: 0.024 ± 0.017%/h, LP: 0.021 ± 0.006%/h) was elevated at 24-h postexercise (FP: 0.073 ± 0.016%/h, P < 0.05, LP: 0.072 ± 0.015%/h, P < 0.05), but the responses did not differ between menstrual phases. Therefore, there is no effect of menstrual cycle phase, at rest or in response to an acute bout of exercise, on myofibrillar protein synthesis and muscle collagen synthesis in women.

stable isotopes; estrogen; exercise

WE RECENTLY SHOWED THAT, in young healthy men, the rates of both myocellular proteins and intramuscular collagen (probably mostly peri- and epimysium) increased in response to an acute bout of exercise (24). Despite women having a smaller muscle mass than men (14), attempts to detect sex differences in protein metabolism have produced little evidence of their existence. There have been some reports of differences between the sexes in the rate of leucine oxidation at rest and during exercise (16, 22, 34) but there is no convincing evidence of major differences in whole body protein turnover and mixed-muscle protein synthesis (13, 16, 22, 26, 31, 34) between the sexes, even after corrections for different sizes of fat-free mass (13, 34). Furthermore, it is reported that men and women do not have different rates of muscle protein synthesis in response to exercise (31). The lower protein mass in women may therefore be the result of an accumulated sex-specific hormonal effect on synthesis or breakdown over a period of many years (32), or female sex hormones may influence protein synthesis in fractions of muscle, that are masked when measurements are made of mixed-muscle proteins in the whole body. Regarding the latter hypothesis, the synthetic rate of intramuscular connective tissue deserves scrutiny, because the frequency of several diseases linked to collagen-rich tissue, such as bone, connective tissue, and ligaments, seem to be sex specific (36). Women are at higher risk of suffering fibromyalgia, rheumatoid arthritis, and hypermobility and sustain more connective tissue injuries than men, and this may be related to exposure to different sex hormones. Estrogen and progesterone receptors have been localized in synoviocytes and fibroblast from muscle and tendon tissue samples from animals (7, 8, 30) and human beings (19, 30), and in vitro studies with increasing estrogen availability have shown a dose-dependent decrease in collagen synthesis rate and fibroblast proliferation in tissue samples from the human anterior cruciate ligament (20, 37, 38). Furthermore, in a well-designed study by Toth et al. (33) it was demonstrated that ovariectomized rats increased muscle protein synthetic rates and lean body mass compared with sham-operated controls. In addition, when estrogen and progesterone were separately added back to ovariectomized rats, each hormone returned synthetic rates and lean body mass to sham-operated values. Therefore, in rats at least, female sex hormones inhibited muscle growth.

The blood concentrations of female hormones change during the menstrual cycle, with low estrogen and progesterone during the early follicular phase (FP) and high concentrations during the luteal phase (LP). Measurements in both phases of the menstrual cycle should therefore allow the detection of any effects of female hormones on protein metabolism in muscle and its connective tissue. Comparisons of rates of protein synthesis between phases of the menstrual cycle are limited. Lamont et al. (15) reported an increase in nitrogen excretion in response to submaximal endurance exercise in the midluteal phase than during menstruation. Subsequently, Larièvère et al. (18) demonstrated greater whole body leucine flux, determined with \([1^{3}]\text{Cl}	ext{eucine}, during the luteal phase than in the follicular phase. No study has directly measured protein synthetic rates
in exercised skeletal muscle in different phases of the menstrual cycle. The current protocol was designed to discover any differences in rates of muscle collagen and myofibrillar protein synthesis between the menstrual cycle phases. We hypothesized that female sex hormones would decrease synthetic rates in the muscle protein constituents and diminish the exercise-induced responses in them; i.e., the fractional synthetic rates of myofibrillar and connective tissue proteins should be lower in the LP than in the FP at rest and after exercise.

SUBJECTS AND METHODS

Subjects. Sixteen young, healthy women with normal menstrual cycles were recruited to the study. All were nonsmoking, nulliparous, not currently taking oral contraceptives or other medications, and free of anatomic and metabolic disorders as judged by history and routine medical examination. The subjects gave informed consent to a protocol adhering to the Declaration of Helsinki, and approved by the Ethics Committee of Copenhagen and Frederiksberg Communities. Randomly, eight women were assigned to the FP and eight were assigned to the LP.

Menstrual cycle determination. Approximate cycle length and date of ovulation were determined at the interview session. The subjects were given a home ovulation kit (Clear-Plan; Unipath, Bedford, UK) to use for 5 days before predicted ovulation until a positive test was obtained. Home testing continued for 2 more days to confirm the positive test. Subjects assigned to testing during the follicular phase (FP) were tested 2–3 days after the onset of menses. Subjects assigned to testing during the luteal phase (LP) were tested 4 days after a positive ovulation. Blood was drawn on each day of testing for confirmation of menstrual cycle phase by chemiluminescent competitive immunoassay (estradiol; Diagnostic Product, Los Angeles, CA) and microparticle enzyme immunoassay (progesterone; Abbott Diagnostics, Wiesbaden, Germany). One subject from the LP group was subsequently eliminated from the comparisons between cycles because of progesterone values outside the normal range. However, this subject was included when data from all women were pooled. Therefore, the final subject groups consisted of 8 FP subjects, 7 LP subjects and 16 for all women (Table 1).

Study design. Our study design was very similar to that of our previous study of men (24) in which we obtained measurements of rates of synthesis of myofibrillar and collagen protein at rest and before and after exercise. In men, the rates of both myofibrillar muscle protein and intramuscular collagen protein synthesis were fastest 24 h after an acute bout of exercise, and we therefore chose this time for postexercise measurements in women.

Investigative protocols. Two weeks before the study, subjects visited the laboratory for determination of workload maximum (W\textsubscript{max}) on a 1-legged modified Krogh ergometer. After warming up for 5 min without resistance, the subjects began one-legged kicking (35 kicks/min) for 3 min at 0.5-kg load; this was increased by 0.3 kg every 3 min until the subjects could no longer maintain the cadence. The workload for each subject’s subsequent 1-h exercise bout was defined as 67% of this W\textsubscript{max}. The contralateral leg served as a resting control leg. The subjects were instructed to avoid physical activity and to maintain their habitual diet for the 2 days before, and during, all testing days. Diet was confirmed by weighed-diet records (3 days before the experiment and the 1st day of the experiment), which showed neither differences in energy intake and macronutrient composition between groups nor between the habitual diet and the diet on the day they were exercising (Table 2). On the day of investigation, the subjects fasted for 12 h and then received a commercial clinical nutrient drink (Semper, Fredriksberg, Denmark, 15% protein, 64% carbohydrate, and 21% fat) in divided doses every 30 min over the subsequent 4-h study period, a method that has previously been demonstrated to maintain steady-state plasma amino acid enrichments (24, 27). The drink provided the equivalent of 1.4× basal metabolic rate of each 30-min period, with a double-dose prime at initiation of feeding. As with the male subjects, basal metabolic rate was estimated from the subjects fat-free mass determined by the skinfold technique (6, 9). Therefore, subjects were fed according to fat-free mass.

Measurement of muscle protein synthetic rates. Measurement of rates of muscle collagen and noncollagen protein synthesis were performed according to our previously validated techniques (1–3, 24). Briefly, upon arrival, two cannulae were inserted into veins on opposite forearms for tracer infusion and blood sampling, and a blood sample was obtained for measurement of background isotope enrichments and insulin and hormone concentrations. Isotope-labeled leucine was then administered as a primed constant infusion of [1-13C]leucine (0.8 mg/kg prime, 1.0 mg·kg\textsuperscript{-1}·day\textsuperscript{-1} continuous). The tracer obtained from Cambridge Isotope Labs (Woburn, MA) was chemically pure (>99 atoms % in 13C) and certified to be sterile and without pyrogens. The tracer was dissolved on the morning of the infusion in 0.9% NaCl with a sterile technique and then passed through a 0.20-μm filter (Bic and Bernstein, Rosdovre, Denmark). Blood samples were drawn at 30-min intervals for determination of labeling (tracer-to-tracee ratio) of amino acids in plasma. Four hours after the start of tracer infusion, biopsies of vastus lateralis muscle (50–100 mg) of both legs were taken using a Bergström needle with suction after previous preparation of incision sites with local anesthetic (lidocaine 1%). Biopsies were cleared of external adipose tissue and blood, frozen in liquid nitrogen, and stored at −80°C for subsequent analysis.

Table 1. Subject characteristics for FP, LP, and all subjects combined

<table>
<thead>
<tr>
<th></th>
<th>FP (n = 8)</th>
<th>LP (n = 7)</th>
<th>All Women (n = 16)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>26±2</td>
<td>26±4</td>
<td>26±3</td>
</tr>
<tr>
<td>Height, cm</td>
<td>171±5</td>
<td>173±8</td>
<td>172±6</td>
</tr>
<tr>
<td>%BF</td>
<td>27.7±6.8</td>
<td>27.5±8.0</td>
<td>27.3±7.0</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>62.6±8.2</td>
<td>59.8±7.4</td>
<td>61.2±7.5</td>
</tr>
<tr>
<td>FM, kg</td>
<td>17.8±6.3</td>
<td>16.8±6.3</td>
<td>17.1±6.0</td>
</tr>
<tr>
<td>FFM, kg</td>
<td>44.9±3.5</td>
<td>43.0±3.0</td>
<td>44.1±3.5</td>
</tr>
<tr>
<td>Workload, W</td>
<td>47.1±9.4</td>
<td>44.6±5.3</td>
<td>45.9±7.5</td>
</tr>
<tr>
<td>Workload, %max</td>
<td>64.6±6.3</td>
<td>68.4±5.2</td>
<td>66.5±5.2</td>
</tr>
</tbody>
</table>

Values are means ± SD. FP, follicular phase; LP, luteal phase; %BF, percentage body fat; FM, fat mass; FFM, fat-free mass. One subject in the LP had hormone values out of range and was included only in the All Women group.
Free amino acid labeling. Plasma \(\alpha\)-ketoisocaproate (KIC) was prepared as previously described (1, 24) and analyzed as their \(t\)-BDMS derivatives by gas-chromatography mass-spectrometry (29).

Extraction of muscle fractions and muscle collagen. Details regarding this procedure have been given elsewhere (1–3, 5, 24). Briefly, muscle (20–30 mg) was ground in liquid nitrogen to a fine powder and hand homogenized in a buffer containing 0.15 M NaCl, 0.1% Triton X-100, 0.02 M Tris-HCl, pH 7.4, and then centrifuged at 1,600 \(g\) at 4°C for 20 min to pellet the collagen and myofibrils, which were then subjected to differential salt extraction.

Gas chromatography-combustion-isotope ratio mass spectrometry. Isolated protein fractions were hydrolyzed in a slurry of 0.05 M HCl:Dowex 50WX8–200 (500 \(\mu\)l; Sigma, Poole, UK) at 110°C overnight (4), and the liberated free amino acids purified, were eluted from it using 1 M HCl. The amino acids were derivatized as their \(N\)-acetyl-\(n\)-propyl (NAP) ester (23). NAP amino acids were analyzed by capillary gas chromatography-combustion-isotope ratio mass spectrometry (GC-C-IRMS, Delta- plus XL; Thermofinnigan, Hemel Hempstead, UK); separation was achieved on a 25-m CP-SIL 19CB column (Chrompack). A leucine standard was prepared as its NAP derivatives and analyzed by GC-C-IRMS.

Calculations. The rates of protein synthesis were calculated using standard equations (27); thus, for the constant-infusion approach, fractional protein synthesis (FSR, \(\%/h\)) = \(\Delta E_{m}/E_{p} \times 1/t \times 100\), where \(\Delta E_{m}\) is the change in labeling (tracer/tracee) of leucine between the final muscle biopsy and a standard baseline natural enrichment, \(E_{p}\) is the mean enrichment over time of the precursor for protein synthesis taken as venous KIC enrichments, and \(t\) is the time (h) of tracer incorporation. As in previous studies (24, 25), a standard baseline enrichment for myofibrillar-bound protein leucine of \(-35.314 \pm 0.320\) per million with respect to Pee Dee Belemnite was used. This value was also used for muscle collagen, as it was obtained by analysis of skin and muscle protein-bound leucine from separate Danish subjects none of whom had previously received a tracer infusion. Any variation in baseline enrichment between subjects would be negligible compared with the increases after administration of labeled leucine. Furthermore, because control and exercise samples were taken from the same subject on the same day, we would not expect variability within subjects to have had any effect. Venous KIC was chosen to represent the immediate precursor for muscle noncollagen protein because fibroblasts, like myocytes, contain a branched-chain amino acid transaminase (35), and fibroblasts behave, with respect to branched-chain amino acid metabolism, like myocytes, rapidly equilibrating the branched-chain keto and amino acid labels (21, 24).

Additional analysis. Systemic IGF-I, IGF-II, and IGF-binding protein-3 (IGFBP-3) were measured from serum samples separated by centrifugation (4°C, 10 min, 2,000 \(g\)) and stored at \(-80^\circ\)C. Local tissue fluid concentrations of IGF-I, IGF-II, and IGFBP-1 through -4, were sampled by tissue microdialysis. High-molecular-mass cut-off (3,000 kDa, membrane length 30 mm, ID 0.50 mm) fibers were inserted under ultrasound guidance as previously described (24), into the vastus lateralis of the rested and exercised legs 24 h after exercise. The microdialysis relative recovery [as estimated by the loss of \([^{3}\text{H}]\)human type IV collagen calculated by the internal reference method (28)] exceeded 85%. Microdialysis fibers were placed within...
Table 3. Serum and muscle dialysate IGF-I, IGF-II, and IGFBPs

<table>
<thead>
<tr>
<th>Serum</th>
<th>Phase</th>
<th>Rest</th>
<th>24 h Postexercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>IGF-I, µg/l</td>
<td>FP</td>
<td>237±61</td>
<td>203±39</td>
</tr>
<tr>
<td></td>
<td>LP</td>
<td>211±45</td>
<td>213±53</td>
</tr>
<tr>
<td>IGF-II, µg/l</td>
<td>FP</td>
<td>1,109±108</td>
<td>1,066±120*</td>
</tr>
<tr>
<td></td>
<td>LP</td>
<td>1,105±197</td>
<td>1,043±179</td>
</tr>
<tr>
<td>IGFBP-1, pixel intensity</td>
<td>FP</td>
<td>33±10</td>
<td>35±8</td>
</tr>
<tr>
<td></td>
<td>LP</td>
<td>31±9</td>
<td>28±9</td>
</tr>
<tr>
<td>IGFBP-2, pixel intensity</td>
<td>FP</td>
<td>72±37</td>
<td>85±35</td>
</tr>
<tr>
<td></td>
<td>LP</td>
<td>60±22</td>
<td>53±22</td>
</tr>
<tr>
<td>IGFBP-3, µg/l</td>
<td>FP</td>
<td>5,476±696</td>
<td>5,251±772</td>
</tr>
<tr>
<td></td>
<td>LP</td>
<td>5,489±844</td>
<td>5,978±1,451</td>
</tr>
<tr>
<td>IGFBP-4, pixel intensity</td>
<td>FP</td>
<td>56±17</td>
<td>51±14</td>
</tr>
<tr>
<td></td>
<td>LP</td>
<td>54±12</td>
<td>46±13</td>
</tr>
</tbody>
</table>

Values are means ± SD; FP, n = 8; LP, n = 7. IGFBP, IGF-binding protein.

2 cm of the appropriate tissue sample sites with analysis dialysis fluid collected in the third and fourth hours after insertion. IGF-I concentration was determined after acid-ethanol extraction with a noncompetitive, time-resolved, monoclonal immunofluorometric assay, as previously described (11). Serum IGFBP-3 was measured by immunoradiometric assay (Diagnostic System Laboratories, Webster, TX). Small volumes of muscle dialysates were analyzed by Western ligand blotting, as previously described (10).

Statistics. Results are presented as means ± SD. Resting and exercise values were compared by a t-test between phases and from rest to exercise within phases. The level of significance was set at \( P < 0.05 \), with subsequent adjustment for repeated comparisons by the Bonferroni method.

RESULTS

Female sex hormones. Estrogen and progesterone plasma concentrations were both higher in LP compared with FP (\( P < 0.05 \)) in correspondence with the definition of the menstrual cycle phases (Fig. 1).

Muscle protein synthesis. Resting myofibrillar protein synthesis (FP: 0.053 ± 0.009%/h; LP: 0.055 ± 0.013) increased ~2.5-fold 24 h postexercise (FP: 0.131 ± 0.018, \( P < 0.05 \); LP: 0.134 ± 0.018, \( P < 0.05 \); Fig. 2A) with no differences at rest or after exercise between menstrual phases. Similarly, resting muscle collagen protein synthesis (FP: 0.024 ± 0.017; LP: 0.021 ± 0.015) increased approximately threefold 24 h postexercise in both phases (FP: 0.073 ± 0.016, \( P < 0.05 \); LP: 0.072 ± 0.015, \( P < 0.05 \); Fig. 2B). However, there were no differences in the rates between phases at rest or after exercise.

IGF and IGFBPs. Serum IGF-II significantly decreased from rest to 24 h postexercise (rest: 1,109 ± 108; 24 h postexercise: 1,066 ± 120, \( P < 0.05 \)) in FP but not LP (Table 3). Additionally, IGFBP-1 in muscle dialysate decreased significantly from rest to 24 h postexercise (rest: 43 ± 20, 24 h postexercise: 33 ± 17, \( P < 0.05 \)) during LP but not FP. There were no other differences in serum and muscle dialysate IGF-I, IGF-II, and IGFBP-1 through -4.

Possible sex differences. Previously, we (24) reported the intramuscular collagen protein and myofibrillar synthesis rates in two cohorts of men at rest and at 6, 24, 48, and 72 h postexercise. In the present study, we chose to compare men and women only at rest and 24 h after exercise for muscle collagen and muscle myofibrillar protein synthesis. Although data were collected identically, they were not collected simultaneously with the study in males; thus there was no direct matching between subjects of different sexes. Nevertheless, the data did allow us to make some comparisons. Because no differences between cycle phases in protein synthesis rates were observed, all data for women in this study can be combined (Table 4). When this was done, it was seen that women and men worked at similar relative workloads (women, 66.5 ± 5.2%; men, 63.2 ± 4.6%), which resulted in a lower absolute workload in women for the 1-h exercise period (women, 45.9 ± 7.5 W; men, 60.4 ± 9.1 W). Myofibrillar protein synthesis rates were the same 24 h after exercise in both women and men. Lower resting synthesis rates in the men made the average exercise induced increase appear somewhat larger in men (148 ± 39% in women vs. 229 ± 49% in men). Muscle collagen protein synthesis rates were similar in both sexes at rest and 24 h after exercise, with an average increase from rest to after exercise of 250 ± 80% for women and 241 ± 50% for men (Table 4).

DISCUSSION

To our knowledge, this is the first attempt to directly measure muscle collagen and noncollagen protein synthesis in women at rest or after an acute bout of exercise in the two different phases of the menstrual cycle. Although there is some overlap of estrogen concentrations between samples taken in different phases, an average twofold difference in circulating estrogen and clearly differentiated progesterone values were insufficient to affect synthetic rates of muscle collagen or myofibrillar proteins. Therefore, menstrual cycle phase, and its associated changes in female sex hormones, does not seem to
have any effect on either resting or postexercise protein synthesis rates of muscle myofibrillar protein or intramuscular collagen.

One explanation for our findings could be that the inhibiting effect of estrogen observed in vitro and in animal studies (20, 37, 38) is counteracted by a stimulating effect of progesterone in whole human beings. In one recent study, when overectomized rats were administered progesterone, the protein synthetic rate of cardiac muscle tissue measured in vitro almost doubled; when the overectomized rats received progesterone and a progesterone receptor antagonist, the rate fell to control values (12). In support of this idea, a stimulating effect of progesterone on the collagen protein synthesis in tissue from the anterior cruciate ligament has been observed by others (38); certainly in the present study, a 25-fold increase in progesterone was seen in LP compared with FP (Table 2). However, counter to this argument, an inhibiting effect of progesterone on muscle protein synthesis has also been observed (33), so the effect of progesterone is not completely clear. Thus we cannot completely rule out whether (1) in our study the two female sex hormones were counteracting each other, 2) the inhibiting effect of female sex hormones on protein synthesis in vitro is not present in vivo, or 3) the inhibitory effect simply requires larger changes in hormonal concentrations than the ones occurring naturally in the menstrual cycle.

A potential limitation of our study is its cross-sectional design, whereby two different groups of subjects were used for the FP and LP. As the procedures described herein were part of a protocol that required significant tissue sampling, we felt that the additional biopsies required to establish baseline labeling values was not ethically justified. In any case, our subjects were matched for age, height, weight, percent body fat, fat-free mass (Table 1), and dietary intake (Table 2), and one subject who had questionable hormone concentrations was excluded from analysis. The careful matching of subjects should have increased the power to distinguish the effect of female sex hormones.

We have to emphasize that we have not determined the breakdown rates for the myofibrillar or collagen protein in the current study, making it impossible to express any net protein balance. Therefore, the observed increase in collagen and myofibrillar protein synthesis after exercise could indicate an increase in protein turnover or even a net gain of tissue protein, if we assume an unchanged breakdown rate.

This study demonstrates that women, like men, increase muscle collagen synthesis after an acute bout of strenuous exercise and that this response persists for at least 24 h after completion of the activity. Furthermore, the magnitude of this increase is similar to what we found recently in men (24).

Only minor changes were found in the IGF system, but IGFBP-1 was different between LP and FP (Table 3). The difference was not manifested in different rates of muscle protein synthesis between menstrual cycle phases, although increases in IGFBP-1 are associated with catabolic conditions and have been demonstrated to decrease skeletal muscle protein synthesis rates in rats (17).

In conclusion, the present study shows that, in women, myofibrillar protein synthesis and muscle collagen synthesis are elevated in parallel in response to acute exercise and, furthermore, that menstrual cycle phase, and its associated changes in female sex hormones, had no effect on resting or postexercise synthetic rates of the proteins.

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