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Power Loading–Induced Fatigue Is Influenced by Menstrual Cycle Phase

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ABSTRACT

PELTONEN, H., R. MIKKONEN-TAIPALE, T. UIMONEN, S. WALKER, A. C. HACKNEY, M. VALTONEN, H. KYRÖLÄINEN, and J. K. IHALAINEN. Power Loading–Induced Fatigue Is Influenced by Menstrual Cycle Phase. *Med. Sci. Sports Exerc.*, Vol. 54, No. 7, pp. 1190–1198, 2022. **Purpose:** This study aimed to examine the effects of fatiguing power loading on neuromuscular properties, force production, and metabolic capacities during four phases of the menstrual cycle (MC): menstruation (M), midfollicular (mid FOL), ovulation (OV), and midluteal (mid LUT). **Methods:** Sixteen eumenorrheic women performed sessions of maximal explosive leg press (2 × 10 at 60% one-repetition maximum load with 2-min recovery between sets). Serum hormones and neuromuscular responses were measured. **Results:** The loading protocol significantly decreased power (between −14.2% and −12.5%; $P < 0.001$) and maximal force production (between maximum voluntary force (MVC); −15.0% and −7.8%; $P < 0.001$–0.05), while decreasing activation level (between AL; −6.9% and −2.2%; $P < 0.001$–0.05) in all MC phases. The decreases in AL were greater during mid LUT ($P < 0.01$) compared with OV. Changes in MVC and AL were associated ($r^2 = 0.53$; $P < 0.01$) at all MC phases. The decrease in EMG during MVC did not differ between the MC phases; however, mean power frequency was higher during M (+7.7%; $P < 0.05$) and mid LUT (+3.1%; $P < 0.05$) compared with OV (−7.5%). Resting twitch force decreased during mid FOL (−6.9%; $P < 0.05$) and mid LUT (−16.2%; $P < 0.001$), and these values were significantly decreased ($P < 0.05$) compared with OV. In addition, resting twitch force at mid LUT was lower ($P < 0.01$) compared with M. Blood lactate levels increased more ($P < 0.05$) during M compared with mid LUT. Some serum hormone concentrations were associated with fatigue-induced changes in neuromuscular properties and force production, but these correlations behaved differently between the MC phases. **Conclusions:** OV may offer a more favorable hormonal milieu for acute neural responses, whereas mid FOL and mid LUT seem to be superior for acute muscular responses.
Key Words: EUMENORRHEIC, RESISTANCE EXERCISE, NEUROMUSCULAR, CENTRAL FATIGUE, PERIPHERAL FATIGUE

Neuromuscular fatigue can be defined as the inability to maintain a given loading intensity or required muscle force (1). As such, reductions in maximal isometric force, neural activation, and fast force production have been used as measurements of acute fatigue. Indeed, the ability to maintain maximal rapid force and power production after repeated muscle actions is a critical element in sport performance as well as many tasks of daily living. Several studies have shown that specific, fatigue-inducing training stimuli and resulting improved rapid force production are closely linked to changes in individual neural and hormonal profiles in both sexes (e.g., [2,3]). Previous, studies that have investigated acute neuromuscular fatigue following power and/or maximum strength loading protocols have been conducted primarily in men, whereas this topic is understudied in women (4). Furthermore, the scientific studies that have taken into consideration menstrual status, menstrual cycle (MC), and related hormonal concentrations are sparse.

The concentrations of sex hormones vary in a cyclical manner during a eumenorrheic MC where at least some of these hormones, for example, estrogens and progesterone, are associated with central nervous system function (5). The contribution of these hormones to neural function is complex and related to different mechanisms within the nervous system, where their

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effects may either excite or inhibit. Estrogens and progesterone may induce opposite effects on neural excitability and their kinetics change continuously throughout the MC (6). For example, an excitatory neuronal effect has been associated with estrogens and an inhibitory effect has been associated with progesterone (7). In terms of performance, the interaction of estrogens and progesterone with serotonin seems to increase force production at the beginning of exercise but compromise force production under fatigued conditions (8). Estrogen concentrations are low just before menstruation (M) at the beginning of the MC, which may lead to less optimal neuromuscular control, thus influencing performance (9). This decrease in estrogen may subsequently alter the contribution of peripheral and central factors to fatigability. The increased progesterone concentration during the latter half of the MC (luteal phase (LUT)), in turn, may inhibit the optimal function of the nervous system, as progesterone has antiestrogenic effects (10). Progesterone, however, has also been shown to enhance estrogenic effects and improve performance (11), so its effects are not one dimensional. Taken together, it is still unclear what effect MC and associated fluctuations in sex hormones might have on the acute responses of the central and peripheral neuromuscular system, especially during exercise targeting the development of speed–strength or power production capabilities.

Pereira et al. (12) evaluated studies examining loading-induced fatigability during different phases of the MC. Their review reported conflicting results in "time to task failure," "maximal strength," and "endurance." Regrettably, no rapid force production task was included in the studies, and it seems that the literature on this topic is also sparse (4). It is well known that specific acute responses and neuromuscular fatigue are among the key stimuli facilitating specific long-term adaptations in strength. By extension, it could be hypothesized that stimuli from identical loading protocols might cause different acute responses in the magnitude of experienced stress and the origin of fatigue based on the unique hormonal milieus observed during the MC. This study investigated the possible influence of MC on acute neuromuscular fatigue responses to a power-type loading protocol.

METHODS

Participants. Healthy women age 18–40 yr were recruited through advertisements and email lists as well as by word of mouth. Each participant candidate completed a health questionnaire and Low Energy Availability in Females Questionnaire (LEAF-Q) (13) before inclusion in the study. Participants included eumenorrheic women who had never used hormonal contraceptives (n = 16; age, 26 ± 4 yr; height, 167.1 ± 5.6 cm; body mass, 67.9 ± 7.0 kg; body fat, 21.8% ± 6.6%; length of MC, 28.3 ± 2.3 d; LEAF-Q, 3.8 ± 2.7 points). The inclusion criteria required that participants had to be highly recreationally active (three sessions of each strength and endurance training per week) with a body mass index between 18 and 25 $kg \cdot m^{-2}$ and a LEAF-Q score indicating energy balance. Participants were excluded if pregnant or lactating; if they had conditions affecting ovarian function, including known menstrual dysfunction, known endocrine disorders, or chronic diseases; or if they were taking medications that may affect loading responses. Participants received detailed information about the study design, measurements, and procedures before participation and the signing of an informed consent document. Participants were aware that they could withdraw their participation in the study at any time. The methodology of the present study was approved by the Ethical Committee at the University of Jyväskylä (October 22, 2018).

Study design. The study design included four experimental testing sessions. The first measured phase of MC was randomized, and participants were familiarized to the test situation, to mitigate any influence of learning effect. Testing was performed during menstruation (M; days 2–4 of bleeding), the midfollicular phase (mid FOL; 7–11 d from the onset of bleeding/menstruation), ovulation (OV; 24–48 h after detected positive ovulation test), and the midluteal phase (mid LUT; determined as 7 d after positive ovulation test). Daily identification of the luteinizing hormone (LH) surge as an indicator of ovulation was completed using an internationally marketed urine-based ovulation test kit (Dipro, LH Ovulation Strip; Aidian Oy). Each testing session was accompanied by venous blood samples in a fasted state. Venous blood samples were analyzed retrospectively. These procedures were performed according to currently established recommendations for best practice (14); however, it is important to note that phases in the present article are not described using the nomenclature, outlined in the study of Elliott-Sale et al. (14), because of differences in measurement timing and lack of "achieving" the defined hormonal cutoff points that would be expected in an idealized 28-d cycle. *A priori* exclusion criteria included lack of ovulation (detected by ovulation kit) rather than a posteriori exclusion based on hormonal cutoff points. The data presented are part of a larger endogenous and exogenous hormones and performance in women (MEndEx) study.

Serum hormones. Blood samples were collected into serum tubes (Venosafe Gel + Clot activator tubes; Terumo Medical Co., Leuven, Belgium) after a 12-h fast between 0700 and 0900 h by a qualified laboratory technician. The technician reviewed analyses of the basic blood count (collected in Venosafe EDTA Tubes (Terumo Medical Co.) and analyzed by Sysmex KX-21 N (Kobe, Japan)) for abnormalities that could indicate acute illness/infection that would preclude participation in strenuous loading. The samples were centrifuged with 2000*g* (Heraeus Megafuge 1.0 R; Thermo Scientific, Karsruhe, Germany) for 10 min at a refrigerated temperature of +4°C. Serum was kept at −80°C until analyzed for determination of estradiol (E2) and progesterone (P), LH, follicle-stimulating hormone (FSH), serotonin (SRTN), dehydroepiandrosterone (DHEA), sex hormone–binding globulin (SHBG), free testosterone (FT), cortisol (C), growth hormone (GH), and the E2/P ratio. Hormone analyses were performed using chemical luminescence techniques (Immulite 2000; Siemens Healthcare Diagnostics, Camberley, United Kingdom). The sensitivity for serum hormones was

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55.0 pmol·L^{-1} for E2, 0.95 nmol·mL^{-1} for P, 0.05 mIU·L^{-1} for LH, 0.10 IU·L^{-1} for FSH, 35.15 nmol·L^{-1} for SRTN, 0.24 nmol·L^{-1} for DHEA, 0.02 nmol·L^{-1} for SHBG, 0.14 pmol·L^{-1} for FT, 5.50 nmol·L^{-1} for C, and 0.01 μg·L^{-1} for GH. The interassay coefficients of variation were 6.7% for E2, 9.7% for P, 4.8% for LH, 3.4% FSH, 12.4% for SRTN, 7.9% for DHEA, 6.9% for SHBG, 12.4% for FT, 6.0% for C, and 5.7 for GH.

Nutrition. Participants were instructed to maintain their typical diet throughout the study. A 3-d food diary including the day before, day of, and day after aerobic testing was collected for each phase. Analysis of food diaries was completed with the Fineli program (National Institute for Health and Welfare, Helsinki, Finland). As reported earlier, no significant differences in energy intake were observed between MC phases (15). Participants were instructed to avoid caffeine and to eat a typical light meal or snack roughly 3 h before the test.

Familiarization. Before the first acute loading, the height of each participant was measured using a stadiometer and standard methods. Body composition was assessed with a multifrequency bioelectrical impedance analyzer (InBody 720; Biospace, Seoul, Korea) in a fasted state (12 h). Participants were not given feedback regarding body composition until all four testing sessions were completed to avoid potentially influencing eating behavior. During this first visit to the laboratory, participants were also familiarized with the testing equipment including proper techniques for each test. This familiarization session included determining settings for each dynamometer/strength training device as well as determination of electrode placement for measurement of muscle activation via EMG according to the SENIAM guidelines (16). Electrode placements were marked with small permanent marker ink dots before skin preparation. The marks were renewed throughout the testing period to ensure that electrodes were placed on the same location of the muscle for every measurement.

Maximal bilateral isometric force production. Isometric force production of the leg extensors was measured using an isometric horizontal bilateral leg press (Neuromuscular Research Center, Faculty of Sport and Health Sciences, University of Jyväskylä, Finland). The knee angle was 107° determined from the greater trochanter, lateral tibiofemoral joint space, and lateral malleolus. Participants were instructed to produce as much force as possible as fast as they could for ~3 s. Isometric maximum voluntary force (MVC), the average force over the first 100 ms (F100), and the rate of force development (RFD) during steepest 10-ms data were collected at a sampling frequency of 2000 Hz, and then filtered (20-Hz low-pass filter) and analyzed using customized scripts (Signal 4.10; CED, Cambridge, United Kingdom). A minimum of three trials was performed. If the maximum force during the last trial was greater than 5% compared with the previous trial, an additional trial was performed up to a maximum of five trials. The best performance, in terms of maximal force, was used for statistical analysis.

Maximal bilateral dynamic strength. Maximum bilateral concentric leg extension strength (one-repetition maximum (1RM)) assessment was performed on a modified David 210 (David Health Solutions Ltd., Helsinki, Finland) horizontal leg press device equipped with foot plate displacement and force sensors (University of Jyväskylä). For determination of 1RM and the subsequent loads to be used during loading protocol, participants were in a seated position (110° hip angle). The concentric movement started from a 60° ± 2° knee joint angle and ended with a fully extended leg (180° knee angle). Measurement of 1RM was preceded by three warm-up sets that were based on the previously (familiarization or previous phase of MC) determined 1RM. These warm-up sets consisted of 10 repetitions at 30% 1RM, 6 repetitions at 50% 1RM, and 3 repetitions at 70% 1RM. Each set was followed by 1 min of recovery. For determination of 1RM in each MC phase, participants performed 3–5 trials with a rest period of 2 min between trials. The highest load successfully lifted was selected for further analysis.

Loading protocol. The dynamic power-type loading protocol (loading protocol) included two sets of 10 repetitions at 60% 1RM with 2-min rest between sets that were performed on a modified David 210 (David Health Solutions Ltd.) horizontal leg press device. Each repetition of the two sets was accompanied by verbal commands "Ready" and "Now!" and performed as fast as possible. Immediately before the loading protocol and immediately after both sets of 10 repetitions, maximal isometric strength tests and muscle stimulation were performed, and a blood lactate sample was taken from the fingertip. Participants were required to move from the David 210 leg press device to other testing devices during and after loading.

Surface electromyography. EMG of the vastus lateralis (VL) and vastus medialis (VM) muscles during contractions was recorded (NeuroLog 824; Digitimer Ltd., Welwyn Garden City, United Kingdom) from the right leg using bipolar Ag/AgCl surface electrodes (10-mm pickup area and 20-mm interelectrode distance, common mode rejection ratio >100 dB, input impedance >100 MU, baseline noise <1 μV EMG). Signals were sampled at a frequency of 2000 Hz, preamplified at a gain of 500 (sampling bandwidth, 10–500 Hz) and were passed real-time through an analog-to-digital board converter (Power 1401) and recorded to a computer using Signal 2.16 software (Cambridge Electronic Design, Cambridge, United Kingdom). The EMG signal data was band-pass filtered (20–350 Hz) and transformed to root mean square EMG amplitude offline before being normalized to peak EMG measured during isometric MVC for each measurement session. Maximal EMG activity was analyzed from the plateau phase of isometric MVC over a 500-ms time window. EMG activity during rapid force production was analyzed manually over the initial 100 ms of isometric MVC (EMG100). Also, Fast Fourier Transformation (Hamming, 1024 data points) was performed to obtain EMG median power frequency (MPF) over a 500-ms epoch from the isometric MVC trials. EMG activity of the VL and VM muscles was combined and averaged during the analyses. Changes in neuromuscular efficiency were analyzed based on loading-induced changes in isometric MVC and EMG as follows: (MVC/((VL + VM)/2)).

Muscle stimulation. Unilateral isometric knee extension torque response to electrical stimulation at rest (resting stimulation) was determined at a 107° knee joint angle measured on

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a dynamometer chair (University of Jyväskylä) also determined from the greater trochanter, lateral tibiofemoral joint space, and lateral malleolus. Participants were stabilized by inelastic straps at the hip and ankle. Four carbon film muscle stimulation electrodes (V-Trodes; Mettler Electronics Corp., Anaheim, CA; diameter, 70 mm) were placed on the mid and proximal portion of the quadriceps femoris muscle belly of the right leg. The stimulation electrode pairs were galvanically separated while the skin under the electrodes was abraded and cleaned with ethanol. The constant current was increased progressively in 20-mA steps between stimulations until a torque response plateau was observed. When maximal torque response was reached, 50% of the stimulation current was added. This supramaximal stimulus (150%) was used for all subsequent stimulations. The electrical stimulator (Digitimer Ltd., Model DS7AH) delivered single rectangular pulses (1 ms, 400 V). Resting stimulations were performed twice with 1 min between these twitches. Resting twitch force (RT) was analyzed from each twitch to determine the fatigue of peripheral components (17). The deficit of central drive (activation level (AL)) was assessed by the interpolated twitch technique (18), including resting twitches before and 2 s after MVC, and two twitches were delivered over the plateau phase of MVC. The latter twitch response during the plateau phase was used in the calculations. The participants were able to reach MVC within 5 s, and they were instructed to avoid brief torque peaks when they increased torque progressively toward the maximum. The level of voluntary activation was calculated according to the formula by Bellemare and Bigland-Ritchie (19):

$$\mathrm{AL}\ (\%) = [1 - (\mathrm{SIT}/\mathrm{RT})] \times 100,$$

where SIT is the difference between the voluntary torque and twitch torque from the superimposed twitch, and RT is the resting twitch torque after MVC. Postactivation potentiation (PAP) was analyzed by comparing resting twitches before and after MVC. EMG of the biceps femoris muscle indicated that the prior mentioned procedure did not stimulate this antagonist muscle.

Statistical analysis. Statistical analyses were completed with IBM SPSS Statistics 26.0 (IBM SPSS Statistics for Windows; IBM Corporation, Armonk, NY). Means and SD were calculated using standard methods. Force and neuromuscular data were found to be normally distributed; hence, a two-way ANOVA with repeated measures and *post hoc* tests were performed with Bonferroni adjustments. Where significant main effects were observed, pairwise comparisons were used to identify the location of the differences between phases. Hormonal data were not normally distributed; thus, a bivariate correlation (Spearman) analysis was completed with AL, RT, F100, EMG100, and RFD for all MC phases. A subgroup analysis was performed for mid LUT for participants with "higher" (>16 nmol·L^{-1}) and "lower" P concentrations (<16 nmol·L^{-1}) in line with the cutoff point for P (14). Statistical significance was set at $P \leq 0.05$.

Intersession intraclass correlation coefficients and percent coefficient of variation for main neuromuscular parameters were acceptable levels: isometric force, 0.981% and 3.4%; maximum RFD, 0.997% and 3.2%; EMG amplitude, 0.918% and 7.2%; median frequency, 0.957% and 6.8%; maximum resting twitch force, 0.994% and 1.3%; and calculated voluntary activation, 0.732% and 1.9% (20).

RESULTS

Urinary LH testing results indicated that ovulation had occurred in all included participants, whereas basal hormone levels fluctuated as might be expected throughout MC (Table 1). In the nonfatigued condition, power production, maximal isometric force, and dynamic force did not differ between the MC phases. Furthermore, neither voluntary muscular activity (EMG, MPF) nor electrical stimulation parameters (RT, AL, PAP) differed between the MC phases before loading protocol.

The present loading protocol led to similar significant decreases in power production in the leg press (from −14.2% to −12.5%; $P < 0.001$) in all MC phases. In addition, MVC (−15.0% to −7.8%; $P < 0.001$–0.05) and AL (−6.9% to −2.2%; $P < 0.001$–0.05) decreased significantly during all phases. The decreases in AL were greater during mid LUT ($P < 0.01$) and M ($P < 0.07$; trend) compared with OV (Fig. 1). A significant correlation between changes in MVC and AL was observed ($r^2 = 0.53$; $P < 0.01$) throughout the MC phases. Neuromuscular efficiency was maintained

TABLE 1. Basal hormone concentrations and power production during MC.

	M			Mid FOL			OV			Mid LUT		
Hormones	**Mean**	**SD**	**Range**	**Mean**	**SD**	**Range**	**Mean**	**SD**	**Range**	**Mean**	**SD**	**Range**
P (nmol·L^{-1})	1.9	1.7	0.4–6.7	1.0	0.4	0.5–2.2	4.1	2.7	0.9–8.0	14.8	8.7	0.9–28.2
E2 (pmol·L^{-1})	478.3	581.3	110–940	592.2	498.0	130–1604	798.7	736.5	99–2037	684.5	234.9	118–951
LH (mIU·L^{-1})	5.8	3.4	1.2–13.7	6.9	2.9	3.2–13.1	14.2	13.5	2.3–54.5	9.5	22.0	0.6–91.3
FSH (IU·L^{-1})	5.6	2.6	1.9–8.7	6.6	2.7	2.4–11.9	6.8	3.0	2.4–11.7	2.8	1.3	1.2–5.5
SRTN (nmol·L^{-1})	1084.9	564.1	217–2144	961.2	396.3	234–1720	953.0	479.5	197–1693	983.9	476.6	237–1923
DHEA (µmol·L^{-1})	30.8	13.5	9.6–54.4	38.1	16.7	13.1–67.4	42.5	20.3	17.7–90.0	37.0	23.0	8.84–93.0
SHBG (nmol·L^{-1})	69.9	38.3	31.8–170	70.7	41.3	32.2–174	74.3	42.0	32.3–179	72.3	40.1	29.0–169
FT (pmol·L^{-1})	7.4	5.1	1.8–22.5	8.8	5.5	2.5–25.8	9.4	5.2	3.0–24.0	8.0	4.7	1.8–20.6
C (nmol·L^{-1})	466.8	91.6	353–717	482.6	109.5	320–643	469.1	135.4	255–670	453.0	139.4	252–723
GH (µg·L^{-1})	4.7	3.0	0.1–10.1	3.9	4.5	0.1–13.6	4.2	4.4	0.2–14.7	5.8	2.5	0.9–9.4
E2/P ratio	246.4	120.4		673.6	593.9		300.1	403.6		121.0	209.6	

Power Production	**M**		**Mid FOL**		**OV**		**Mid LUT**	
Load 60% 1RM (kg)	88.8	14.6	86.9	13.7	87.1	14.8	87.1	14.0
Mean power (W)	569.1	112.8	554.6	108.7	569.4	120.0	551.7	86.4

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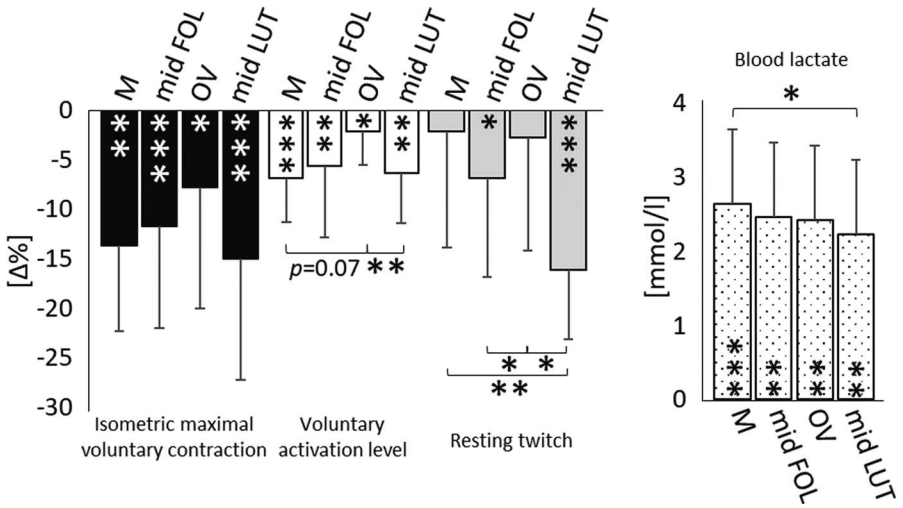

FIGURE 1—Mean (±SD) relative changes in MVC, VA, and RT due to power-type loading and the absolute blood lactate values after loading protocol. *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

because of loading-induced changes in MVC and EMG during M (0.0% ± 6.7%) compared with mid LUT (+8.7% ± 5.6%; $P < 0.01$) and mid FOL (+5.2% ± 9.5%; $P = 0.06$, trend). The decrease in EMG during MVC due to the loading protocol did not differ between MC phases; however, mean power frequency in the fatigued state was higher during M (+7.7%; $P < 0.05$) and mid LUT (+3.1%; $P < 0.05$) compared with OV (−7.5%) (Table 2, Fig. 2A). The changes due to loading protocol in RFD (−22.3% to −11.0%) and the first 100-ms force production (−22.4% to +1.9%) as well as EMG (−32.1% to −14.3%) did not differ between MC phases. Nevertheless, a significant correlation between the changes in force and EMG during the first 100 ms from the onset of force production was observed ($r^2 = 0.34$; $P < 0.001$), when all MC phases were combined (Fig. 2B).

Electrical evoked resting twitch force responses decreased significantly because of the loading protocol during mid FOL (−6.9%; $P < 0.05$) and mid LUT (−16.2%; $P < 0.001$), a decrease significantly greater than that observed during OV (−2.7%; $P < 0.05$). Electrical evoked resting twitch force responses also decreased ($P < 0.01$) more during mid LUT compared with M (−2.2%). PAP did not change as a result of the loading protocol (Table 2). Blood lactate concentrations increased from resting values as a result of the loading protocol (1.46–1.53 to 2.23–2.66 mmol·L^{-1}) in all phases of MC. The highest blood lactate concentrations were reached during M, which differed significantly ($P < 0.05$) compared with mid LUT phase (Fig. 1).

TABLE 2. Changes in mean power and EMG between the best (mean power) and last dynamic repetitions during power loading protocol (2 × 10 × 60% 1RM with 2-min rest between sets).

	M		Mid FOL		OV		Mid LUT	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Dynamic								
Mean power (%)	87.0	10.0	85.8	6.5	87.5	7.7	86.8	6.7
EMG (VL + VM) (%)	108.2	13.8	102.4	10.4	97.5	13.3	96.5	10.4
Isometric								
MVC (Δ%)	−13.7	8.6	−11.7	10.3	−7.8	12.2	−15.0	12.3
EMG (VL + VM) (Δ%)	−19.6	12.2	−19.2	14.0	−14.9	7.6	−15.3	11.8
MPF (VL + VM) (Δ%)	7.7	24.8	−1.1	19.0	−7.5	11.1	3.1	10.6
Force 0–100 ms (Δ%)	−4.7	21.3	−18.3	29.7	−11.9	29.4	1.9	34.3
EMG 0–100 ms (Δ%)	−13.7	27.9	−14.8	48.7	−3.8	52.8	−10.2	39.8
PAP (Δ%)	−1.4	8.3	2.9	9.7	−0.1	7.9	1.3	10.8

In addition, the relative changes during maximal (MVC, EMG, and MPF), fast (F100 and EMG100) force production, and PAP (Δ%).

Basal concentrations of serum hormones and hormone ratios were associated with the fatigue-induced changes in voluntary AL, resting twitch response, initial 100-ms EMG and force production, and RFD, but these correlations behaved differently between the phases of MC (Table 3). High SRTN concentration was "associated with lower loading-induced fatigue in voluntary fast force production (RFD, F100, and EMG100), especially in mid LUT. In addition, a higher individual E2/P ratio and higher E2 were associated positively with smaller loading-induced decreases in RFD, F100, and EMG100 during mid FOL, OV, and mid LUT, whereas these correlations were negative with higher P. Higher P ($r = 0.76$, $P = 0.001$) and lower E2/P ratios ($r = −0.85$, $P < 0.001$) were associated with lower loading-induced central fatigue (AL) at mid LUT. In mid LUT, participants with lower P (<16 nmol·L^{-1}) had greater decreases in AL (−12.3% ± 3.3% vs. −5.2% ± 5.7%; $P < 0.01$) than those with a higher P (>16 nmol·L^{-1}; 5.75 ± 5.46 vs 21.13 ± 3.82 nmol·L^{-1}; $P < 0.001$). Nevertheless, a statistically significant difference between P subgroups' E2 or E2/P ratio levels was not observed. SRTN concentrations were associated significantly with sex hormones (SRTN and E2: $r = 0.3$, $P = 0.023$; SRTN and T: $r = −0.4$, $P = 0.004$).

DISCUSSION

Physically active eumenorrheic women were able to perform maximal and rapid power output at the same level before the loading protocol in all four MC phases we assessed. The present dynamic power-type loading protocol-induced fatigue profiles were, however, different between MC phases, which were observed in peripheral, central, and metabolic origins

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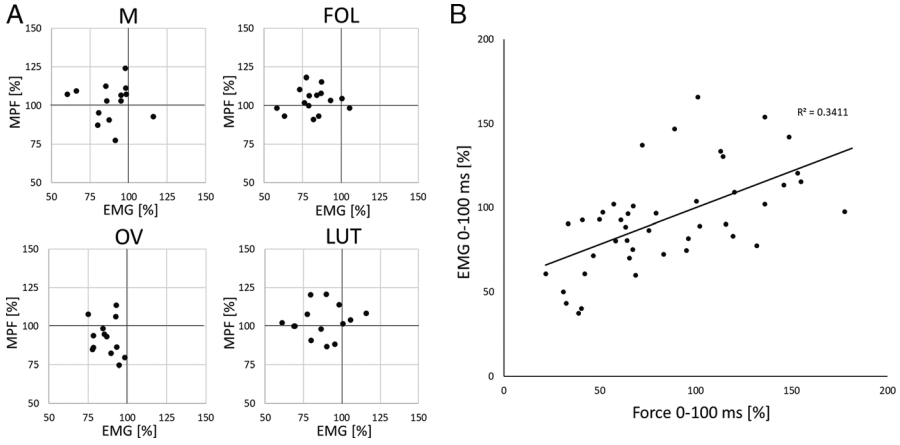

FIGURE 2—Individual changes due to loading in (A) MPF and EMG during isometric MVC and (B) the correlation between EMG and force production during the first 100 ms after force onset.

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of fatigue. These findings suggest that variation in individual basal hormonal profiles, neuromuscular capacity, and subsequent loading-induced fatigability interact, at least partly.

Peripheral and metabolic fatigue. The focus of this study was power-type leg press loading protocol, where contraction velocities were maximal, but the load and number of repetitions per set (2 × 10 × 60% 1RM load) were submaximal. Typically, this type of loading protocol causes both fatigue and PAP with interindividual differences compensating for each other (21,22). Regardless of the peripheral effects (e.g., enhanced calcium (Ca^{+}) kinetics in the sarcoplasmic reticulum) in some participants' enhanced performance due to PAP, peripheral fatigue as measured by RT was observed both at mid LUT ($P < 0.001$) and, to a lesser degree, at mid FOL ($P < 0.05$) at the group level. This is in line with decreased muscle endurance observed during LUT compared with OV in the study of Petrofsky et al. (23). Recently, the study of Ansdell et al. (11) showed reduced muscular endurance with high-intensity contractions at M compared with the LUT. In the present study, blood lactate was increased after all loadings, with the greatest increase observed during M compared with mid LUT ($P < 0.05$). Maintaining maximal power for repetitions during sets (>20 s) depends mainly on the resynthesis of phosphocreatine and followed by anaerobic glycolysis (24). Thus, elevated blood lactate levels may indicate less use of phosphocreatine as an energy source during M. Nicklas et al. (25) showed that glycogen content in the muscle is decreased during M compared with mid LUT, which increases the potential for glycogen sparing due to increase lipid metabolism (26). In addition, higher blood lactate levels indicate increased anaerobic glycolysis–induced acidosis and decreased pH in the muscular milieu, suggesting that greater metabolic fatigue has occurred during M.

Peripheral fatigue due to anaerobic metabolism is also associated with decreased conduction velocities and lengthening of intracellular action potentials (27,28). These changes may affect EMG signals by increasing EMG amplitude as was observed during the loading protocol in the end of the last set during M compared with mid LUT. Nevertheless, no differences were observed in decreased power production between MC phases. The magnitude of decreases in EMG during post loading isometric MVC were similar between all MC phases, but calculated neuromuscular efficiency was enhanced after the loading protocol only at mid LUT (+8.7%, $P < 0.01$). This finding may indicate impaired excitation–contraction function and greater peripheral fatigue in mid LUT (29).

Central fatigue. The present power-type loading protocol seemed to induce central fatigue during all MC phases, but the interpolated twitch technique revealed less central fatigue during OV (AL −2.2%) compared with M (AL −6.9%, $P = 0.07$, statistical trend) and mid LUT (AL −6.4%, $P = 0.02$). This could be associated with better cortical excitability during OV compared with M (30) and LUT (7). In support, the study of Ansdell et al. (11) observed enhanced neural capacity due to increased AL with decreased fatigability in mid LUT.

TABLE 3. Statistically significant correlations between serum hormone concentrations or hormone ratios and fatigue-induced changes in neuromuscular properties and fast force production.

		M			Mid FOL			OV			Mid LUT	
		r	*P*		*r*	*P*		*r*	*P*		*r*	*P*
AL%	DHEA	−0.717	**0.03**	DHEA	−0.65	0.06	E2/P	−0.667	0.07	E2/P	−0.767	**0.02**
							SHBG	0.738	**0.04**	P	0.700	**0.04**
RT%	E2	−0.552	**0.04**							GH	0.673	**0.02**
	GH	0.556	**0.03**							E2/P	0.587	**<0.05**
EMG100%										SRTN	0.608	**0.04**
										LH	0.804	**0.002**
	FSH	−0.514	0.05	GH	0.476	0.06				E2/P	0.591	**0.02**
F100%	SHBG	−0.639	**0.01**							P	−0.521	**0.04**
										SRTN	0.639	**0.01**
RFD%				P	−0.522	**<0.05**				DHEA	0.475	0.07
				SRTN	0.534	**<0.05**				SRTN	0.507	0.05

Significant group difference, $P < 0.05$ (*P* value in bold).

In general, the fatigue-induced changes in neuromuscular efficiency and EMG are related to central fatigue and synchronization of motor units (31). Although there are limitations to conclusions based on surface EMG (32), the decreases in both EMG amplitude and MPF may indicate fatigue primarily in fast twitch motor units during OV, whereas fatigue seems to be more pervasive between different motor unit types during M, mid FOL, and mid LUT. This may be a consequence of enhanced neural capacity during OV, as was observed by Ansdell et al. (11), who suggested that power loading–induced stress might remain relatively lower in slow twitch motor units during OV. Similarly, Sarwar et al. (33) showed greater fatigue levels during late FOL and OV compared with other MC phases after isometric exercise caused by electrical stimulation (33), which is observed to activate primarily the fast twitch motor units (34). Such issues require further clarification but would improve understanding of the potential factors influencing motor unit firing and consequent central fatigue over the MC.

Hormones and fatigue. The study assessed several hormones and their ratios during four different phases of the MC. The group-level hormonal fluctuations (notably E2, P, and LH) fluctuated as might be expected, although phases were not entirely in line with E2 and P cutoffs for estrogen and progesterone (Table 1 and Supplemental Table 1, Supplemental Digital Content, http://links.lww.com/MSS/C539) as defined by Elliott-Sale et al. (14). Regrettably, it is difficult to pinpoint specific hormonal milieus in advance of fatiguing testing while maintaining adequate rest between tests/loadings and concomitantly avoiding a training effect. Furthermore, it is important to recognize the difficulty of recruiting women with idealized 28-d cycles that consistently achieve specific hormonal cutoffs. As such, we report the observed hormonal concentrations and responses according to our study design to clarify that included measurement time-points were not in line with the idealized MC phases as defined by Elliott-Sale et al. (14). Power-type loading protocols are known to cause acute decreases in EMG and force production, especially during the early phase of rapid contractions. However, it seems that individual variation in women is remarkable, especially in EMG (21), although it is important to note that, for example, Linnamo et al. (21) did not assess hormones or take into consideration MC phase. The present study observed correlations ($r^2 = 0.41–0.26$, $P = 0.01–0.05$) between basal SRTN and power-type loading-induced decrements in voluntary fast force production (RFD, F100ms, and EMG100) during mid LUT. The neurotransmitter or neuromodulator roles of SRTN seem to enhance performance in the short-term (8), as in this loading protocol. Although changes in SRTN levels are reported to affect, for example, motor unit firing rate (35), the present study did not find this relationship, even though MPF and basal SRTN concentrations demonstrated a similar pattern on a group level. Nevertheless, the MPF spectrum is influenced by other factors along with different motor unit activation (e.g., synchronization). The function of SRTN in the present study seems to be linked somewhat to sex hormones (T ($r = −0.4$) and E2 ($r = 0.3$)) that also seem to have a relationship with the level of central fatigue in MC phases where sex hormones like E2 and P are known to fluctuate (36). However, the reasons for the associations detected are unclear and need to be pursued in future research.

Previously, E2 has been associated with enhanced neuronal sensitivity at several sites within the neural system (e.g., [37]). Moreover, P concentrations in the blood of eumenorrheic women tend to increase from OV to mid LUT, which may impair the effects of E2 (D'Eon et al) (48). Power loading–induced central fatigue seems to be significantly ($P < 0.01$) higher in participants with lower P concentrations in mid LUT, although P levels were elevated at a group level. A greater decrease in AL (−5.2% ± 5.7% vs −12.3% ± 3.3%; $P < 0.01$) was clearly observed when the participants were divided into high and low P subgroups (21.1 ± 3.8 vs 5.7 ± 5.5; $P < 0.001$) using the "phase 4" cutoff (16 nmol·L^{-1}) defined by Elliott-Sale et al. (38), as described previously. It should be noted that the size of these subgroups is quite small in total, which may obscure the statistical meaningfulness between the change in AL and subgroups' E2/P concentrations alone (high E2/P vs low E2/P, 241.7 ± 289.1 vs 34.6 ± 11.9; P = not significant).

In the present study, E2/P was associated with fatigue-induced decreases in fast (F100ms, $P = 0.02$; EMG100ms, $P < 0.05$) and maximal force production (AL $P = 0.02$) in mid LUT. In addition, correlations between SRTN and fatigue-induced decrements in fast force production were also observed, which is logical because E2 and P affect numerous functional properties of the SRTN neural system (39). These findings highlight that fast and maximal sustained (even short) force production is governed by different neuromuscular factors (36). Nevertheless, it is important to remember that there is individual variation in hormonal profiles (40,41) and that correlations and/or hormone ratios may oversimplify hormonal interactions and/or effects.

Performance under unfatigued condition. The present study did not identify any differences between MC phases in power production, 1RM, or isometric MVC before the loading protocol (PRE), which is in line with several studies (38,42). Previously, only a limited number of studies have focused on central and peripheral "readiness to train" with electrical stimulation-evoked force production during different phases of MC, which should mitigate the changes in force production due to possible fluctuation in motivation. Stability in electrical evoked RT results over the MC is in line with previous studies (11,42,43), although few studies have reported contradictory results (e.g., [33]). In the present study, individual hormonal changes at mid LUT, when P concentrations were high, showed that the E/P ratio was associated with enhanced RT ($r = 0.85$, $P < 0.001$) and PAP ($r = 0.63$, $P = 0.01$) at PRE. PAP responses have been linked to recruitment of the fast twitch motor units, and PAP is likely affected by modified Ca^+ kinetics in the sarcoplasmic reticulum (44) of muscles with predominantly fast motor units (45).

We did not show any significant changes in baseline voluntary AL between the MC phases, but the interpolated twitch technique has been shown to be insensitive to small changes (46). However, Ansdell et al. (11) showed slightly enhanced

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AL during OV (14th day in MC) (11). Although this could be explained by differences in warm-up or measurement protocols (47), conflicting results allow for speculation, whether or not the observed enhancement in baseline voluntary activation could reflect a higher capacity of the neural system concomitant with lower loading-induced central fatigue, at least in some individuals.

CONCLUSIONS

The present study showed that a power-type loading protocol induced differences in fatigue profiles between different phases of the MC. The fluctuation in female sex hormones was observed throughout the MC, even though differences in performance parameters in the unfatigued condition were not observed. Interestingly, correlations between hormones, like SRTN, and neuromuscular fatigue varied throughout the MC. Peripherally located fatigue during mid LUT and M may offer a beneficial milieu for improving contractile properties and enzyme activities on a muscular level, whereas lower neural fatigability during OV could allow for more quality repetitions with rapid force and power production (48).

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