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The Temporal Effects of Altitude and Low Energy Availability Manipulation on Resting Metabolic Rate in Female Race Walkers

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ABSTRACT

KUIKMAN, M. A., A. K. A. MCKAY, R. MCCORMICK, N. TEE, B. VALLANCE, K. E. ACKERMAN, R. HARRIS, K. J. ELLIOTT-SALE, T. STELLINGWERFF, and L. M. BURKE. The Temporal Effects of Altitude and Low Energy Availability Manipulation on Resting Metabolic Rate in Female Race Walkers. *Med. Sci. Sports Exerc.*, Vol. 57, No. 1, pp. 123–133, 2025. **Purpose:** This study aimed to investigate the temporal effects of ~1800 m altitude exposure and energy availability (EA) manipulation on resting metabolic rate (RMR). **Methods:** Twenty elite female race walkers underwent a 3-wk training camp at an altitude of ~1800 m. During the first 2 wk, athletes consumed a high EA (HEA) diet of 45 kcal·kg fat-free mass (FFM)$^{-1}$·d^{-1}. During the final week, half the athletes consumed a low EA (LEA) diet of 15 kcal·kg FFM^{-1}·d^{-1}, whereas the others continued on an HEA diet. Athletes followed individualized training plans throughout the study. To assess the effect of altitude on RMR, athletes in the HEA group had RMR measured at baseline (~580 m) before altitude exposure (Pre-alt), at 36 h (36h-alt), 2 wk (Wk2-alt), and 3 wk into altitude exposure (Wk3-alt), and at 36 h post-altitude exposure at ~580 m (36h-post). To assess the effect of LEA exposure on RMR while at altitude, athletes in the LEA group underwent RMR measurements at Pre-alt and before (Wk2-alt) and after the 7 d of LEA (Wk3-alt). **Results:** Compared with Pre-alt, the RMR of HEA athletes was increased at 36h-alt (+5.3% ± 3.1%; $P = 0.026$) and Wk2-alt (+4.9% ± 4.9%; $P = 0.049$), but was no longer elevated at Wk3-alt (+1.7% ± 4.2%; $P = 0.850$). The RMR of HEA athletes at 36h-post was lower than all timepoints at altitude ($P < 0.05$) but was not different from Pre-alt (−3.9% ± 7.2%; $P = 0.124$). The 7-d period of LEA exposure at altitude did not affect RMR ($P = 0.347$). **Conclusions:** RMR was transiently increased with ~1800-m altitude exposure in female athletes and was unaffected by short-term LEA. However, the altitude-induced increase was small (~25–75 kcal·d^{-1}) and was unlikely to have clinically significant implications for daily energy requirements. **Key Words:** RMR, ENERGY EXPENDITURE, HYPOXIA TRAINING

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Many athletes who undertake endurance-based training include natural/terrestrial altitude (hypobaric hypoxia) training, which typically involves a 2- to 4-wk period of living and training at altitudes ranging from "*low*" altitude (~500–2000 m) to "*moderate*" altitude (~2000–2500 m) (1,2). These "altitude camps" are strategically incorporated into an athlete's training and competition cycles (3), to take advantage of the hypoxic stress and hematological and nonhematological adaptations that may result in improved performance on return to sea level (4). Although nutrition plays a key role in optimizing adaptations to altitude training (5), many issues are unstudied. A question of particular concern is whether energy requirements differ during altitude exposure due to alterations in resting metabolic rate (RMR), which represents the minimal energy cost of living (6). Most research assessing changes in RMR with altitude

exposure have occurred at a higher altitude (>4000 m) (7–9) than the low to moderate levels (~1800–2400 m) that athletes commonly incorporate into a training cycle (1,2). Indeed, increases in RMR (~7%–27%) have been reported upon acute altitude exposure to high altitude (~4300 m) in men and women (7–9). However, in women at this altitude, this increase in RMR was transient, with RMR returning to sea level values by 6–7 d of altitude exposure (8,9). Only one study has assessed changes in RMR at a low to moderate altitude, finding an increase in the RMR (~19%; ~290 kcal·d^{-1}) of male and female middle-distance runners at the end of a 4-wk altitude training camp at ~2200 m (10). However, given the small sample size (3 M/2 F), this study may have been underpowered (10). Furthermore, it is possible that an even greater increase in RMR occurred with acute altitude exposure in this cohort of athletes, as has previously been seen at higher altitudes (7–9). However, RMR was measured only at baseline and the camp's end and failed to investigate the acute response to hypoxia (10). Determining any increases in basal energy requirements associated with altitude exposure is important when considering nutritional support of athletes.

When examining changes in RMR with altitude exposure, energy availability (EA) must also be considered. EA represents the dietary energy remaining to support the body's health and physiological basal functioning after exercise energy expenditure (EEE) has been subtracted (11). Low EA (LEA) exposure that results in persistent disruptions in body systems can lead to signs and symptoms of Relative Energy Deficiency in Sport (REDs), which includes a suppression in RMR (12). As such, when examining the effect of altitude on RMR, EA must be controlled to ensure that LEA is not confounding results. However, examining the effect of LEA exposure on RMR while at altitude is also of interest to understand how bodily systems are differently affected by LEA exposure and the contribution of altitude exposure as a moderating factor (13). For instance, concurrent increases in RMR from altitude exposure may be neutralized by LEA exposure, causing minimal overall effect on net changes in RMR. Examining the effect of LEA on RMR at altitude is also of relevance as altitude exposure may increase the risk of LEA as athletes may purposefully restrict energy intake (EI) during altitude training camps due to a desire to alter body composition or may inadvertently fail to consume sufficient energy due to changes in appetite (14). Reduced food availability in a new environment or increases in training load during altitude training camps may further perpetuate inadequate EI with altitude exposure. This is demonstrated by a case study involving elite male and female rowers that observed a trend for reduced RMR (~5%) and loss of fat mass on return from a 12-d training camp at altitude (~1800 m) (15). This reduced RMR was attributed to LEA exposure in the absence of a controlled EI during the camp (15). However, failure to measure RMR at altitude prevents the ability to discern the effects of altitude versus LEA exposure.

In order to better understand an athlete's energy requirements during altitude training camps, it is necessary to determine if RMR is altered with altitude exposure and the time course of such changes. Furthermore, determining if LEA alters this response is needed to better understand the specific effects of LEA exposure and moderating factors on REDs outcomes. As such, the purpose of this study was to investigate the temporal effects of altitude exposure and LEA manipulation on RMR in female athletes.

METHODS

Participants

Twenty female race-walkers (26.5 ± 6.5 yr, $\dot{V}O_{2max}$: 58.2 ± 4.2 mL·kg^{-1}·min^{-1}) of Tier 3 (highly trained/national level) to Tier 5 (world-class) caliber (16) were recruited for this study. Naturally menstruating (defined as nonhormonal contraceptive using athletes with self-reported cycle lengths between 21 and 35 d; NM) athletes (n = 13) and hormonal contraceptive (HC) users (n = 6 oral contraceptive pill (OCP), n = 1 Implanon) were recruited. The OCP used by HC users included both combined (n = 1 Optilova, n = 1 Bellaface suave, n = 1 Harmonet, n = 1 Evaluna20, n = 1 Zoely) and progesterone only (n = 1 Slinda). See Supplemental Table 1 for details on OCP preparations (Supplemental Digital Content, http://links.lww.com/MSS/D76). It was not possible to standardize menstrual cycle or HC phase within RMR measurements because the research-embedded training camp study design required that all athletes needed to travel to altitude and begin the study at the same time. In addition, noting that the elite caliber of athletes in this study represents ~0.014% of the global population (16), it was not feasible to only include athletes of homogenous menstrual status (i.e., only NM athletes or HC users using one brand of OCP) as this would severely limit the sample size. Nevertheless, the potential influence of reproductive hormones in this study is likely small, given that we have previously shown that RMR appears to be unaffected by menstrual cycle phase and HC usage in athletic cohorts (17). As such, the ovarian hormone profiles were provided to describe the menstrual characteristics of athletes rather than to the control the hormonal profile and examine the effects of hormones on research outcomes. The menstrual status (MS) of each athlete was characterized twice (i.e., upon recruitment via self-reported means and at the end of the study when MS could be retrospectively verified via measured outcomes) with consideration of the Best Practice Guidelines (18). At recruitment, all NM athletes reported ≥9 periods in the preceding year. Thereafter, they tracked their menstrual cycle from 4 wk preceding the study until 1 wk after study completion using an online reporting system (REDCap), and tested for ovulation beginning on day 8 of the menstrual cycle using urinary luteinizing hormone (LH) surge testing (Advanced Digital Ovulation Test; Clearblue, Geneva, Switzerland). HC users reported bleeding using the same online reporting system. In addition, hormonal profiles of estradiol and progesterone were established at three time points throughout the training camp (pre-altitude exposure, at 2-wk altitude exposure, and post-altitude exposure) for both NM athletes and HC users. Data of one NM athlete were excluded from analysis because of

an injury sustained during the first week at altitude, thus preventing full completion of the study. Athletes were informed of the risks and requirements of the study before providing informed consent. Ethics approval was obtained from the Ethics Committee at Australian Catholic University.

Experimental Design

Baseline testing occurred at the Australian Institute of Sport (AIS) in Canberra, Australia (~580 m), over a 5-d period during which time all athletes had standardized dietary control. Athletes then traveled by vehicle to Perisher Valley, Australia (~1800 m) for a 3-wk altitude training camp before returning to Canberra for post-altitude testing that occurred over a 4-d period (see Fig. 1). The first 2 wk at altitude served as an acclimatization period during which all athletes consumed a fully provided diet providing an EA of 45 kcal·kg fat-free mass (FFM)$^{-1}$·d^{-1}. This was followed by a 7-d dietary intervention, which manipulated EA. During this dietary intervention, one group of athletes (n = 10) consumed a diet providing an EA of 15 kcal·kg FFM^{-1}·d^{-1} (LEA), whereas the remaining athletes (n = 9) continued to consume a diet providing an EA 45 kcal·kg FFM^{-1}·d^{-1} (high EA; HEA). Athletes were allocated into groups based on individual preferences for the EA intervention, with athletes who nominated no preference allocated strategically to ensure key characteristics (e.g., menstrual status, athlete caliber, etc.) were balanced between dietary groups.

In order to assess the time course of potential changes in RMR at altitude, athletes in the HEA group had RMR measured pre-altitude exposure during the baseline testing period (Pre-alt), after ~36-h exposure to altitude (36h-alt), 2-wk altitude exposure (Wk2-alt), 3-wk altitude exposure (Wk3-alt), and ~36 h post-altitude (36h-post). To assess the impact of LEA on RMR measurements, athletes in the LEA group had RMR measured at Pre-alt, and before and after the dietary intervention, which corresponded to an RMR measurement at Wk2-alt and Wk3-alt. In recognition of the burden already associated with the LEA diet, athletes in the LEA group were not required to undergo additional RMR measurements at 36h-alt and 36h-post. Body composition was also assessed using dual-energy x-ray absorptiometry (DXA) at Pre-alt and 36h-post.

Dietary Intervention

For 4 d before and 3 d after the altitude training camp, all participants consumed a standardized diet that provided ~8 g·kg^{-1} carbohydrate, ~1.5 g·kg^{-1} protein, and ~1.1 g·kg^{-1} fat, resulting in a daily EI of ~48 kcal·kg^{-1}. During the altitude training camp, daily energy requirements were determined prospectively for each athlete based on individualized training plans and calculated using the following equation: EI = (Target EA × FFM) + EEE. Daily protein intake was the same for both dietary interventions and provided ~2 g·kg^{-1}. When receiving a diet that contained an EA of ~45 kcal·kg FFM^{-1}·d^{-1}, ~20% of EI was from fat, whereas the LEA diet provided ~15% of EI from fat. Regardless of the target EA, the remaining energy came from carbohydrates. Individual meal plans were created for each athlete based on planned training for that day and personal preference, with a chef preparing all meals.

Training load (volume × intensity) was not controlled throughout the altitude training camp. Rather, athletes followed their individualized training plans throughout the duration of the study. Daily EEE was prospectively estimated from an athlete's planned training, which included race walking, running, cycling, and/or resistance training across 1–3 sessions a day. The EEE of a race walking training session was determined from the individualized gas exchange data collected during a four-stage submaximal race walking graded exercise test (GXT) completed on a treadmill during the Pre-alt period at the AIS. EEE during each GXT stage was determined using the Weir equation with Pre-alt RMR excluded from the same period as follows: $[(3.94 \times \dot{V}O_2 + 1.11 \times VCO_2) - (24\ h\ RMR/1440\ (min))]$ (19). EEE per km of outdoor race walk training was then estimated from each speed of the GXT as follows: $((EEE_{kcal/min} \times 60\ min))/Speed_{km/h})$. Walking EEE ranged from 0.88–1.07 kcal·km^{-1}·kg^{-1} (average ~1 kcal·km^{-1}·kg^{-1}). Running EEE was estimated as kilometer ran multiplied by an athlete's body mass (1 kcal·km^{-1}·kg^{-1}) (20), cycling using a metabolic equivalent (MET) of 8, and resistance training a MET of 4 (21). Pre-alt RMR was again excluded from the same time period when estimating EEE for running, cycling, and/or resistance training sessions.

Athletes reported their actual training daily to a member of the research team, and EI was adjusted if the difference in EEE between actual training and planned training exceeded the

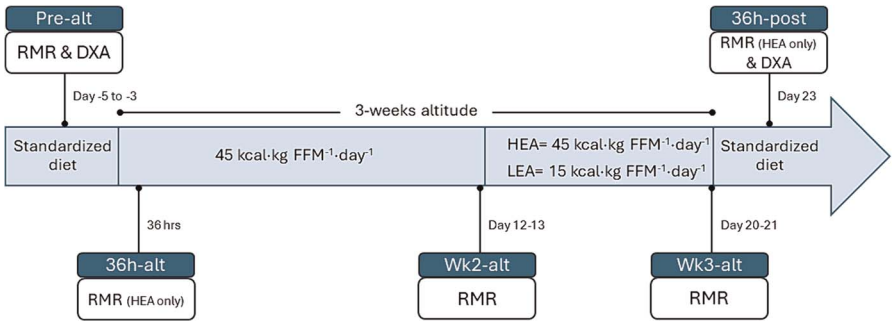

FIGURE 1—Schematic diagram of study design, detailing elevation, timeline, dietary protocols, and measurements taken.

EEE of 2 km of race walking. When increases in EI were needed, this was accomplished by increasing portion sizes at meals and/or providing additional snacks. When decreases in EI were needed, this was accomplished by decreasing the portion size of the day's final meal and/or removing snacks. Two days of *ad libitum* food intake were scheduled within the training camp: the day of ascent to altitude (day 1) and the day before commencing the 7-d dietary intervention after undergoing the Wk2-alt RMR measurement (day 13). These were implemented for logistical reasons and to provide participants a break from dietary control given the extensive nature and dietary compliance that this study involved.

Measurements

Body composition. DXA scans were done in accordance with Best Practice Guidelines (22) before and after the altitude training camp. Athletes presented for testing in an overnight fasted state and with no fluid intake before the scan. All scans were conducted by the same researcher with consistent positioning of participants on the DXA scanning bed using Velcro straps and positioning aids. Scans were performed in the same mode (GE Lunar iDXA) and analyzed using GE encore, which provided an assessment of FFM, lean body mass (LBM), and fat mass.

Resting metabolic rate. RMR was measured using the ParvoMedics TrueOne 2400 metabolic cart (ParvoMedics, Salt Lakes City, UT). Two metabolic carts were available for testing, with athletes having repeat RMR measurements on the same metabolic cart. Each ParvoMedics system was calibrated with gas concentrations (15.99% O_2, 4.00% CO_2) and ventilation using a 3-L syringe before testing. Testing occurred across two mornings with athletes presenting in an overnight fasted state and before morning training around the same time of day (±30 min) to account for circadian changes in RMR (23). Training was not controlled the day before RMR testing. Although training was not monitored before the first RMR measurement, distance walked or run, minutes of weight training, and minutes of cross-training did not differ for athletes the day before RMR measurements across testing time points (all $P > 0.05$). However, differences in training were seen between the HEA and LEA groups the day before RMR measurement. Athletes in the LEA group walked/ran more kilometers than athletes in the HEA group (14.0 ± 6.5 vs 20.0 ± 4.8 km; $P = 0.005$). Meanwhile, athletes in the HEA group engaged in more in weight training (25 ± 26 min; $P < 0.0001$) compared with the absence of weight training in the LEA group. At the AIS, athletes resided in a residence building next to where the RMR measurements occurred and while at altitude, RMR measurements occurred in the lodge where athletes resided. As such, upon waking, athletes were only required to walk a short distance to where the RMR measurement occurred. Upon arrival, athletes laid in a supine position in a dark, quiet room for 10 min to ensure a state of rest, and were then given a one-way mouthpiece that was connected to the ParvoMedics cart for a 10-min familiarization period. Expired air was then collected for a single 25-min period. Upon completion, data were exported into a Microsoft excel file. The first 2 min and last 2 min of each 25-min period were discarded, and a mean was calculated from the remaining minutes to estimate a 24-h absolute RMR ($kcal \cdot d^{-1}$) using the Weir equation (19).

Indicators of LEA. Indicators of LEA were measured throughout the training camp (24). Primary indicators included triiodothyronine (T3) concentrations. Secondary indicators included low-density lipoprotein (LDL) and total cholesterol (TC) concentrations (24). Potential and emerging indicators included insulin-like growth factor 1 (IGF-1) concentrations, cortisol concentrations, and RMR (24). RMR measurements were used to assess for a suppressed RMR by calculating an RMR ratio (measured RMR:predicted RMR) using the Cunningham 1990, Cunningham 1991, and Harris–Benedict (HB) equations to predict RMR (25–27) as well as relative RMR (measured RMR:FFM). These were selected given they have validated thresholds with a suppressed RMR being defined as an RMR ratio <0.90 when using the Cunningham 1980 or HB equation, RMR ratio <0.92 when using the Cunningham 1991 equation (28), and/or a relative RMR <30 $kcal \cdot kg\ FFM^{-1} \cdot d^{-1}$ (11). Athletes were not assessed as per the updated REDs Clinical Assessment Tool V.2 (REDs CAT2) (24) to ascertain their risk of REDs because the study was undertaken before its publication and did not capture data on all primary risk factors, increasing the risk of a false-negative assessment.

Blood samples. An 8.5-mL venous blood sample was collected from an antecubital vein into a serum separator tube by a trained phlebotomist at Pre-alt, Wk2-alt, and 36h-post. Blood tubes were left to clot at room temperature for 30 min, before centrifugation at 1500*g* for 10 min at 4°C. Remaining serum was split into aliquots and stored at −80°C until batch analysis could occur. Estradiol and progesterone were measured via an Access 2 Immunoassay System (Beckman Coulter, Brea, CA). Intra-assay coefficients of variation were 5% for estradiol and 11% for progesterone. Lipids, cortisol, IGF-1, and T3 were measured by chemiluminescent immunoassay through a commercial laboratory (Laverty Pathology, Bruce, ACT, Australia).

Statistics

Statistical analyses were performed using R Studio (v3.5.2) with statistical significance accepted at an α level of $P \leq 0.05$. The insulin results of two athletes ($n = 1$ LEA athlete, $n = 1$ HEA athlete) were considered outliers due to values being >3 SD above the mean and excluded from analyses. Histogram inspection revealed nonnormally distributed data for fat mass, which were then log transformed for analyses. Statistical analyses were completed using general linear mixed models where significance of fixed effects was tested using type II Wald F tests with Kenward–Roger degrees of freedom. For statistical analyses of RMR measurements, two separate models were used. One model assessed time course change in the HEA

TABLE 1. Mean daily training, EEE, and dietary intake during the 12-d acclimatization period and 7-d dietary intervention at altitude.

	Acclimatization		Dietary Intervention		*P*		
	HEA	LEA	HEA	LEA	Week	Intervention	Interaction
Race walk (km)	12.9 ± 2.8	15.1 ± 2.2	15.0 ± 2.3	16.7 ± 2.9	0.012	0.032	0.700
Run (km)	1.4 ± 1.1	2.4 ± 1.3	1.0 ± 2.1	1.8 ± 1.4	0.160	0.104	0.727
Weights (min)	13.6 ± 9.4	14.4 ± 6.1	7.2 ± 5.9	10.7 ± 7.4	0.002	0.469	0.407
Cross-training (min)	5.3 ± 8.2	2.2 ± 3.7	4.4 ± 7.4	1.5 ± 4.7	0.671	0.154	0.940
EEE (kcal)	824 ± 112	983 ± 93	915 ± 135	1062 ± 11	<0.001	<0.001	0.779
EI (kcal)	2764 ± 260	3018 ± 159	2811 ± 350	1732 ± 119[a]	<0.0001	<0.0001	<0.0001
EA (kcal·kg FFM^{-1})	46.2 ± 0.6	45.9 ± 0.5	45.1 ± 1.0	15.1 ± 0.6[a]	<0.0001	<0.0001	<0.0001
CHO (g·kg^{-1})	8.3 ± 0.7	9.0 ± 0.5	8.5 ± 0.7	4.6 ± 0.4[a]	<0.0001	<0.0001	<0.0001
Protein (g·kg^{-1})	2.1 ± 0.1	2.1 ± 0.1	2.0 ± 0.04	2.0 ± 0.03	<0.0001	0.659	0.293
Fat (g·kg^{-1})	1.2 ± 0.1	1.3 ± 0.1	1.2 ± 0.1[b]	0.6 ± 0.1[a]	<0.0001	<0.0001	<0.0001

Data presented as mean ± SD.
[a]Significant compared with acclimatization period and HEA during the dietary intervention period.
[b]Significant compared with LEA during the acclimatization period.
CHO, carbohydrate.

group only, which included test time point (Pre-alt, 36h-alt, Wk2-alt, Wk3-alt, 36h-post) as a fixed effect and subject as a random effect. The other model assessed the effect of EA manipulation, which included test time point (Pre-alt, Wk2-alt, Wk3-alt) and dietary intervention (HEA or LEA group) as a fixed effect. With this model, subject and body mass were used as a random effect except for the model assessing relative RMR, which only had subject as a random effect. For the models assessing diet, training, body composition, and LEA indicators, test time point and dietary intervention were fixed effects and subject was a random effect. For models assessing cortisol and T3, body mass was also included as a random effect. Where significant effects were evident, a Tukey's *post-hoc* comparison was performed.

RESULTS

Dietary analysis. As intended, energy and macronutrient intake during the standardized diet period did not differ between athletes in the HEA and LEA group or between the Pre-alt and 36h-post period (all $P > 0.05$; see Supplemental Table 2, Supplemental Digital Content, Mean daily intake of the standardized diets during pre-altitude and post-altitude testing, http://links.lww.com/MSS/D76). Table 1 outlines the daily training, EEE, EI, EA, and macronutrient intake during the acclimatization and dietary intervention period at altitude. Daily EEE was greater during the dietary intervention period compared with the acclimatization period ($P < 0.001$), and the EEE of athletes in the LEA group was higher than that of athletes in the HEA group ($P < 0.001$); however, no interaction was evident ($P = 0.779$). This was due to differences in the kilometers completed in daily race walking sessions, with this being greater during the dietary intervention period than the acclimatization period ($P = 0.012$), and greater for athletes in the LEA group than the HEA group ($P = 0.032$). Meanwhile, the minutes of weight training decreased from the acclimatization period to the dietary intervention period ($P = 0.002$). There were no differences in the kilometers completed in running sessions or minutes of cross-training across the altitude period or between groups ($P > 0.05$). As intended, the EA, energy, carbohydrate, and fat intake was lower for athletes in the LEA group during the dietary intervention compared with their intake during acclimatization period and compared with athletes in the HEA group during both the acclimatization period and dietary intervention period ($P < 0.0001$). The protein intake did not differ between athletes in the HEA and LEA groups ($P = 0.659$), but protein intake during the acclimatization period was marginally higher (+0.1 g·kg^{-1}·d^{-1}) compared with the dietary intervention period for both groups ($P < 0.0001$).

Menstrual status. For OCP users, only one athlete had testing during a placebo pill day of the OCP cycle with the remaining testing occurring during the active pill days. For the single athlete with an implant, all testing occurred on days without bleeding. In accordance with Best Practice Guidelines (18), detailed information on the MC characteristics can be found in Supplemental Table 3 (Supplemental Digital Content, Retrospectively verified menstrual cycle characteristics, http://links.lww.com/MSS/D76). Individual estradiol, progesterone levels, and the corresponding ratio at Pre-alt, Wk2-alt, and 36h-post can be found in Supplemental Table 4 (Supplemental Digital Content, Estradiol, progesterone, and the ratio of estradiol to progesterone with menstrual status at pre-altitude, Wk2-alt, and 36h-post for athletes in the HEA and LEA groups, http://links.lww.com/MSS/D76).

Body composition. Body composition across the altitude training camp is summarized in Table 2. Athletes in the LEA group ($P < 0.001$), but not the HEA group ($P = 0.250$), had a reduction in body mass from Pre-alt to 36h-post. For athletes in both groups, FFM ($P = 0.408$) and LBM ($P = 0.421$) did not change, but fat mass decreased ($P < 0.0001$) from Pre-alt to 36h-post.

RMR with altitude exposure. Absolute RMR was increased from Pre-alt to 36h-alt (+5.3% ± 3.1%; $P = 0.026$) and Wk2-alt (+4.9% ± 4.9%; $P = 0.049$), but was no longer elevated by Wk3-alt (+1.7% ± 4.2%; $P = 0.850$) or 36h-post (−3.9% ± 7.2%; $P = 0.124$; Fig. 2). Absolute RMR at 36h-post was decreased compared with measurements taken at 36h-alt (−10.0% ± 7.1%; $P < 0.0001$), Wk2-alt (−9.4% ± 5.3%; $P = 0.0001$), and Wk3-alt (−6.1% ± 6.0%; $P = 0.012$). Changes in relative RMR followed the same trends with increased values at 36h-alt (+5.3% ± 3.1%; $P = 0.016$) and

TABLE 2. Body composition before and after the 3-wk altitude training camp for athletes in the HEA and LEA groups.

	Pre-Alt		Post-Alt		Change		*P*		
	HEA	LEA	HEA	LEA	HEA	LEA	Visit	Intervention	Interaction
BM (kg)	52.9 ± 6.0	54.8 ± 5.0	52.6 ± 5.8	53.9 ± 5.1[a]	−0.35 ± 0.61	−0.89 ± 0.47	<0.0001	0.532	0.030
FFM (kg)	41.8 ± 4.8	44.3 ± 2.9	42.0 ± 4.7	44.4 ± 2.9	0.16 ± 0.65	0.12 ± 0.81	0.408	0.160	0.909
LBM (kg)	39.5 ± 4.6	42.0 ± 2.9	39.7 ± 4.5	42.1 ± 2.8	0.16 ± 0.66	0.12 ± 0.81	0.421	0.155	0.901
FM (kg)	11.1 ± 2.4	10.4 ± 3.5	10.6 ± 2.4[a]	9.4 ± 3.3[a]	−0.54 ± 0.57	−1.00 ± 0.60	<0.0001	0.354	0.053

Data presented as mean ± SD.
[a]Significant compared with Pre-alt.
BM, body mass; FM, fat mass.

Wk2-alt (+4.9% ± 4.9%; $P = 0.034$) compared with Pre-alt, but no longer elevated at Wk3-alt (1.2% ± 3.5%; $P = 0.931$). Relative RMR at 36h-post was decreased compared with all values at altitude (all $P < 0.01$), and there was a trend for a decrease in relative RMR at 36h-post compared with Pre-alt (−4.3% ± 6.9%; $P = 0.052$). RMR variables with altitude and LEA exposure can be found in Supplemental Table 5 (Supplemental Digital Content, http://links.lww.com/MSS/D76).

RMR with LEA exposure. The 7 d of LEA exposure at altitude did not affect absolute RMR ($P = 0.347$) or relative RMR ($P = 0.547$; Fig. 3). Two of the ten athletes in the LEA group had a decrease in RMR that exceeded 60 kcal (>4% variation in baseline RMR) from Wk2-alt to Wk3-alt. Greater interindividual variation was noted in the HEA group with five of the nine athletes having a decrease in RMR >60 kcal from Wk2-alt to Wk3-alt.

Given the unexpected change in body composition, we reanalyzed changes in RMR while at altitude between athletes who did ($n = 5$ HEA + $n = 7$ LEA) and did not ($n = 4$ HEA + $n = 3$ LEA) have a decrease in fat mass over the training camp that exceeded the least significant change of 4.7% (29) regardless of dietary intervention allocation. Like LEA exposure, we found no effect of fat mass reduction ($P = 0.282$) on changes in RMR at altitude (Fig. 4).

To explore the interindividual variation for changes in RMR during the final week at altitude, a Pearson correlation was used to assess the association between change in RMR from Wk2-alt to Wk3-alt and changes in determinants of RMR across the altitude training camp. There was a negative correlation between change in RMR from Wk2-alt to Wk3-alt and change in fat mass over the training camp for athletes in the HEA group ($r = −0.735$; $P = 0.024$), but not for athletes in the LEA group ($r = 0.102$; $P = 0.778$). No correlation was seen for change in RMR from Wk2-alt to Wk3-alt and change in FFM over the training camp for athletes in the HEA group ($r = 0.583$; $P = 0.099$) or athletes in the LEA group ($r = −0.081$; $P = 0.823$). There was also no correlation for change in RMR from Wk2-alt to Wk3-alt and change in T3 concentrations over the training camp for athletes in the HEA group ($r = 0.145$; $P = 0.710$) or for athletes in the LEA group ($r = −0.367$; $P = 0.297$).

Indicators of LEA. No athlete had an RMR measurement that was considered suppressed over the course of the study using RMR ratio or relative RMR thresholds. The RMR ratio (using each predictive equation) was increased at 36h-alt ($P < 0.03$) and Wk2-alt ($P < 0.05$) compared with Pre-alt, but was no longer increased at Wk3-alt ($P > 0.05$) or 36h-post ($P > 0.05$) (Fig. 5). The RMR ratio at 36h-post was lower than all RMR ratios at altitude (all $P < 0.01$). The 7 d of LEA exposure did not affect the RMR ratio calculated from the HB ($P = 0.286$), Cunningham 1980 ($P = 0.868$), or Cunningham 1991 equations ($P = 0.953$).

In the LEA group, T3 concentrations were lower at 36h-post compared with both Pre-alt ($P = 0.002$) and Wk2-alt ($P = 0.025$); cortisol concentrations were greater at Wk2-alt ($P < 0.0001$) and 36h-post ($P < 0.001$) compared with Pre-alt (Fig. 6). LEA and HEA groups both had lower TC concentrations at Wk2-alt compared with Pre-alt ($P = 0.041$). Although there was an interactive effect of LDL ($P = 0.001$), IGF-1 ($P = 0.015$), and insulin ($P = 0.036$), *post-hoc* testing was nonsignificant ($P > 0.05$). There was a trend for differences in LDL between Wk2-alt and 36h-post for athletes in the HEA group ($P = 0.05$).

DISCUSSION

The main finding of this study, implemented as a research-embedded training camp, was a transient increase in RMR with exposure to ~1800-m altitude but no change in RMR in

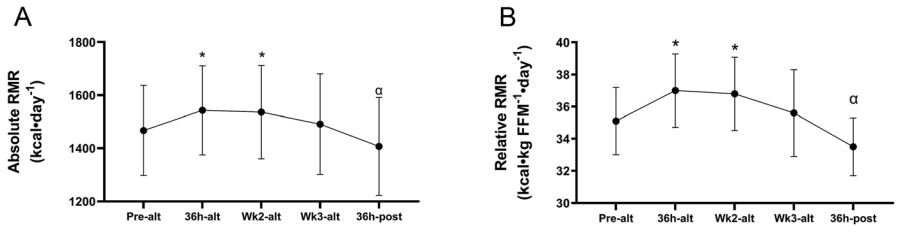

FIGURE 2—Absolute RMR (A) and relative RMR (B) at baseline (Pre-alt), 36-h altitude exposure (36h-alt), 2-wk altitude exposure (Wk2-alt), 3-wk altitude exposure (Wk3-alt), and 36 h post-altitude (36h-post). Data are presented as mean ± SD. *Different compared with Pre-alt. αDifferent compared with all measurements at altitude.

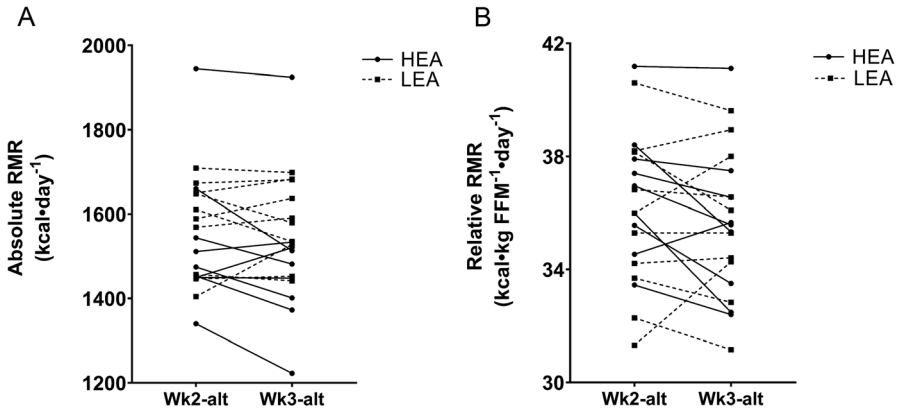

FIGURE 3—Absolute RMR (A) and relative RMR (B) before (Wk2-alt) and after (Wk3-alt) the 7-d dietary intervention for athletes in the HEA group and LEA group. Each line represents an individual athlete.

association with a 7-d period of LEA at this altitude. The increase in RMR (~5.3% or ~75 kcal·d^{-1}) was greatest with acute (36-h) exposure, but differences across 3 wk of altitude exposure were not significant (~1.7% or ~24 kcal·d^{-1}). These findings are novel and build on previous athlete research pertaining to RMR changes with low to moderate altitude exposure (10,15), as we examined a time course for RMR change at altitude, and also investigated if EA alters this response.

RMR with altitude exposure. Our observed ~2%–5% increase in RMR was smaller than the ~19% increase in RMR previously reported in highly trained middle-distance runners (n = 3 males/2 females) at the end of a 4-wk altitude training camp at ~2200 m, where baseline measures also occurred at ~580 m (10). The smaller RMR increase that we observed may be due to a smaller elevation increase between the studies (1220 vs 1620 m) (10). However, the ~19% increase reported at ~2200 m (10) is greater than the ~7% increase in RMR reported with acute exposure to an even higher altitude of ~4300 m in women (8), but smaller than the ~27% increase in RMR reported in men also with acute exposure altitude to ~4300 m (7). We also observed a return in RMR back to baseline values with more prolonged altitude exposure, with the ~5% increase in values at 36 h being reduced to ~2% after 3 wk of altitude exposure. A decrease in RMR back to sea level values has been observed at higher altitudes with RMR returning to baseline after 5 d of high-altitude exposure in women (8), although in male subjects, RMR still remained elevated ~17% above sea level values with 3 wk of high-altitude exposure (7). Notably, our study included a female-only cohort, and it is possible that sex-based differences exist for the effect of altitude on RMR. Although the origins of increases in RMR are unclear, hypoxia-inducible factor (HIF) is thought to play a role in the increased RMR seen at altitude by increasing Cori cycle activity and energy inefficiency (30), with evidence that estrogen may downregulate HIF activity in rodent models (31), providing some support for sex-based differences in RMR at altitude. Increased sympathetic activation is also thought to play a role in the increased RMR with altitude exposure (32), and there may be lower sympathetic support of RMR in women compared with men (33). Further studies are needed to investigate the presence of sex-based differences in RMR changes in response to mild and moderate altitude exposure in athletic cohorts. While reaching statistical significance, the magnitude of RMR change seen in our study must be considered. Indeed, the upper limit of the generally accepted 3%–5% day-to-day variation in RMR (34) equates only to ~25–75 kcal·d^{-1}; thus, our findings are unlikely to have clinically significant implications for an athlete's total daily energy requirements.

Although not reaching statistical significance (P = 0.052), relative RMR at 36h-post was decreased by 1.6 kcal·kg FFM^{-1}·d^{-1} compared with Pre-alt. This is similar to the 1.5 kcal·kg FFM^{-1}·d^{-1} reduction measured following 12 d of altitude exposure in a case study of male (n = 2) and female (n = 2) rowers that was attributed to LEA during the altitude training camp (15). The ~0.5 kg decrease in fat mass for athletes in the HEA group cannot explain this ~60 kcal·d^{-1} decrease in RMR from pre- to post-altitude, as this would result in an absolute reduction in RMR of ~2.3 kcal·d^{-1} (35). As such, it appears that the physiological adaptations that occurred with altitude

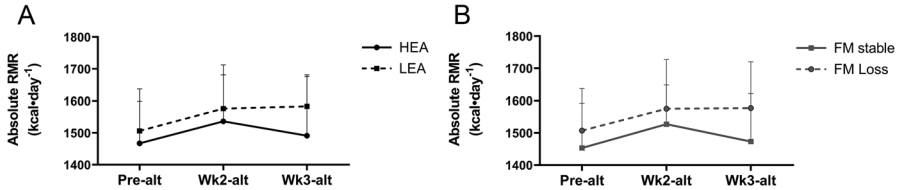

FIGURE 4—Absolute RMR at baseline (Pre-alt), 2-wk altitude exposure (Wk2-alt), and 3-wk altitude exposure (Wk3-alt) for athletes in the HEA group and LEA group (A) and for athletes who had a reduction in fat mass (n = 5 HEA, n = 7 LEA) or no change in fat mass (n = 4 HEA, n = 3 LEA) across the training camp (B). Data are presented as mean ± SD.

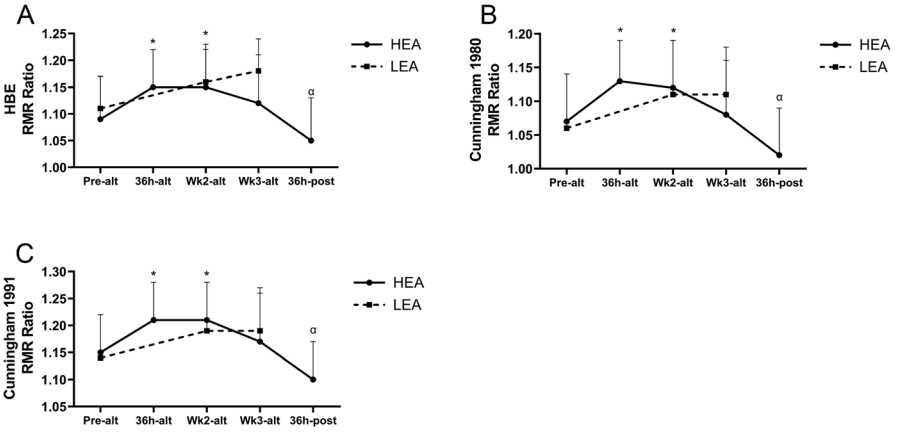

FIGURE 5—RMR ratio with the HB equation (A), Cunningham 1980 equation (B), and Cunning 1991 equation (C) at baseline (Pre-alt), 36-h altitude exposure (36h-alt), 2-wk altitude exposure (Wk2-alt), 3-wk altitude exposure (Wk3-alt), and 36 h post-altitude (36h-post). Data are presented as mean ± SD. *Different compared with Pre-alt. αDifferent compared with all measurements at altitude. HBE, Harris–Benedict equation.

training may be responsible for this 1.6 kcal·kg FFM^{-1}·d^{-1} reduction in RMR. An improved mitochondrial efficiency with altitude training (4,36) could contribute to a reduced RMR given that mitochondrial parameters have been linked to RMR in humans (37). Furthermore, in rodents, weight loss–induced decreases in RMR have been attributed to improved mitochondrial efficiency in skeletal muscle (38). Given this finding, it is possible that this previously reported reduction in RMR was due to adaptations that occurred with altitude exposure rather than LEA during the 12 d at altitude (15). Alternatively, increases in training load during the altitude training camp may have altered RMR as this has been seen following periods of intensified training, although this may have been due to concurrent LEA as an increased training load may not have been matched with an increased EI (39). Future studies are needed to determine if there is a reduction in RMR upon return to sea level following altitude training camps independent of EA status and changes in training load, and if so, the duration of this suppression and the mechanism for this change.

RMR with LEA exposure. Despite a reduction in RMR independent of changes in body composition being an outcome within the REDs model (12), we did not find any effect of 7 d of LEA on RMR while at altitude. Interestingly, the

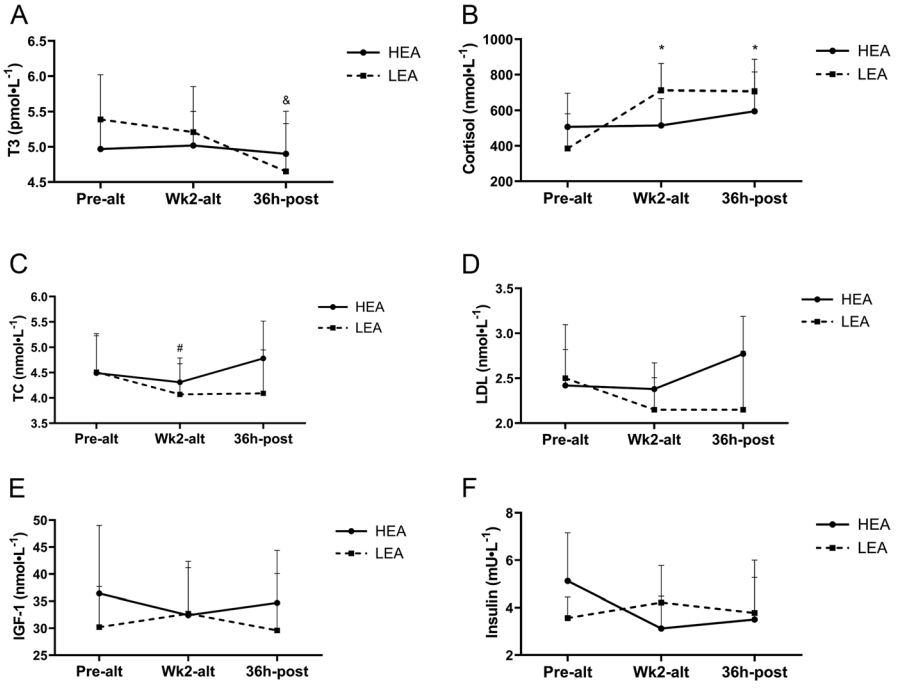

FIGURE 6—T3 (A), cortisol (B), TC (C), LDL (D), IGF-1 (E), and insulin (F) levels at baseline (Pre-alt), 2-wk altitude exposure (Wk2-alt), and 36 h post-altitude (36h-post). Data are presented as mean ± SD. &Different compared with Pre-alt and Wk2-alt for athletes in the LEA group. *Different compared with Pre-alt for athletes in the LEA group. #Different compared Pre-alt for both groups.

majority of athletes in the LEA group had an unchanged RMR following the 7-d period of LEA, whereas among athletes in the HEA group, there was greater interindividual variation when examining changes in RMR across this final week (see Fig. 3). Notably, despite an RMR ratio commonly being used as an indicator of LEA (40), most of the evidence supporting the use of an RMR as an indicator of LEA comes from cross-sectional studies demonstrating differences in RMR between athletes with and without indicators of LEA (28,41–46). Indeed, evidence of LEA suppressing RMR in athletic populations is limited. This includes a −257 kcal·d^{-1} reduction in RMR in a male combat athlete following 7 wk of ~20 kcal·kg FFM^{-1}·d^{-1} followed by 5 d of further restrictions in EA to −4–9 kcal·kg FFM^{-1}·d^{-1} (47), a −65 kcal·d^{-1} reduction in the RMR of female athletes following 10 d of ~25 kcal·kg FFM^{-1}·d^{-1} (48), and a −101 kcal·d^{-1} reduction in RMR in a cohort of nonathletic women following just 3 d of ~15 kcal kcal·kg FFM^{-1}·d^{-1} (49). On the other hand, several other studies have reported no changed in RMR with periods of reduced EA ranging from 8 to 30 kcal·kg FFM^{-1}·d^{-1} for 3–14 d (50–53). When assessing why different outcomes of LEA exposure occur, both the characteristics of LEA exposure and moderating factors must be considered (13). Altitude exposure may be a moderating factor that alters the physiological outcomes of LEA. For instance, reductions in sympathetic nervous system activity are thought to contribute to reductions in RMR with LEA (54). However, altitude exposure is thought to increase sympathetic nervous system activity (55) and contribute to increases in RMR seen with altitude exposure (32). As such, it is possible that the altitude exposure altered the response to LEA exposure and a decrease in RMR would have been observed if the same LEA exposure occurred at sea level. Alternatively, a more prolonged and/or severe exposure of LEA may be needed to impact RMR. It must be noted that we did not control exercise on the day before RMR testing, allowing athletes to engage in their individualized training plans throughout the study. We acknowledge that this design feature could have contributed to variability in RMR measurements, because there were subtle differences in the pre-RMR training between athletes in the LEA and HEA groups. However, within groups, there were no differences over time in the training undertaken on the day before RMR measurements providing confidence in the reliability of these measurements.

Markers of LEA. The updated International Olympic Committee consensus statement on REDs provides new guidelines for diagnosing and assessing the risk of REDs using a mixture of primary and secondary LEA indicators, as well as emerging indicators that require more research before being fully endorsed as indicators of LEA (24). Among the LEA indicators that we assessed, T3 was the only one that was affected by the 7-d period of LEA, strengthening its use as a primary indicator of REDs (24). Interestingly, there was no association between change in T3 levels over the training camp and change in RMR over the 7-d period of LEA. Other measured indicators showed inconsistent changes and seemed altered by altitude exposure and/or training rather than EA (see Figs. 4 and 5). However, a limitation of this study is that blood biomarkers could have been impacted by altitude induced shifts in plasma volume, but plasma volume changes were not quantified in this study (56). Despite other LEA indicators being present (24), no athlete presented with an RMR measurement considered suppressed across the training camp. Notably, a suppressed RMR is listed only as an emerging indicator in the updated REDs CAT2 because of current concerns with specificity and sensitivity of measurement (24). Our results demonstrate that altitude exposure may be contributing to noise in this measurement and must be considered when measuring RMR in athletic cohorts. For instance, athletes undergoing RMR measurements at laboratories or institutions located at low to moderate altitude may present with an increased RMR if unacclimatized, leading to an artificially inflated RMR ratio or relative RMR. In addition, measuring RMR in the periods following an altitude training camp should be used with caution until more research examining RMR following periods of altitude training is conducted.

Energy needs at altitude. The diet provided to athletes in the HEA group was aimed at providing optimal EA. However, meaningful reductions in fat mass occurred for some athletes in the HEA group ($n = 5$), suggesting that study diets provided insufficient energy for these athletes. Differences in duration spent at altitude (15) and methods to determine body composition (10) make it difficult to compare our observations of body composition changes with data from other studies that have allowed *ad libitum* intake. It is possible that an even greater weight loss would have occurred in the present study if an *ad libitum* dietary intake protocol been implemented; indeed, several athletes within the HEA cohort had difficulty consuming the volume of food required to achieve an EA of 45 kcal·kg FFM^{-1}·d^{-1}. Given this, it is possible that athletes were not compliant with the dietary intervention despite the best efforts of the research team to ensure adherence, such as weighing and monitoring meals and taking into consideration individual food preferences. Of the five athletes in the HEA group that had a reduction in fat mass that exceeded the least significant change, four maintained an elevated RMR during the final week. The remaining athlete was unique in also recording a reduction in FFM in addition to fat mass, potentially explaining the observed reduction in RMR. On the other hand, the remaining four athletes in the HEA group that maintained fat mass had a return in RMR back to Pre-alt levels at Wk3-alt. This, along with the negative correlation between changes in fat mass over the training camp and changes in RMR over the final 7 d at altitude ($r = -0.735$; $P = 0.024$), suggests that athletes in the HEA group who maintained an increased RMR with altitude exposure were more likely to experience reductions in fat mass. This loss in fat mass may be due to an underestimation of their energy requirements due to increases in RMR with altitude exposure altering the EA (40–45 kcal·kg FFM^{-1}·d^{-1}) that is recommended to support all physiological systems at sea level (57). However, even if an "optimal" EA threshold could be determined for each athlete within this cohort at altitude, there are known complexities and nuances with the EA equation (58). In addition, the

estimation of EEE from training at altitude was determined from metabolic testing data conducted at sea level, with the possibility of EEE being increased at altitude due to changes in metabolic pathways (59). Finally, it is possible that physiological adaptations at altitude increase energy needs via mechanisms outside of RMR that were not accounted for in the EA equation, such as an increased excess postexercise oxygen consumption (60). Early studies at high altitudes in women reported an increase in total energy requirements beyond what could be accounted for by changes in RMR or EEE, which was termed "energy requirement excess" (8). Given this, further research is needed to assess if physiological adaptations with altitude alter another component that contributes to daily energy needs.

CONCLUSIONS

In conclusion, RMR was transiently increased in female endurance athletes while living and training at altitude but was unaffected by LEA exposure. The increase in RMR observed was small (50–75 kcal·d^{-1}) and is unlikely to have clinically significant implications for an athlete's total daily energy requirements. However, RMR represents only one component of daily energy requirements, and physiological adaptations that occur with altitude may alter other components that contribute to daily energy needs. Given the downward trend in RMR that was seen upon return to sea level, care should be taken when measuring and interpreting the RMR of athletes immediately post-altitude. Future studies are needed to determine if other components of total daily energy expenditure are altered with altitude exposure, what the impact of EA status on these alterations may be, and if there are further sex-based differences in RMR changes in response to altitude exposure in athletic cohorts.

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