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Research Article

Hormonal Disparities and Training Adaptation Responses: A Longitudinal Comparative Biological Study Among Elite Arab Female Athletes in Endurance and Strength Sports

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Abstract

Background: Sex-based biological differences significantly influence athletic performance and training adaptations, yet female athletes are substantially underrepresented in sports science research (80–90% of studies focus on males). Arab female athletes are virtually absent from the literature despite unique genetic, environmental, and cultural contexts that may modulate training responses. Objective: To investigate hormonal disparities (estradiol, testosterone, cortisol, IGF-1, T3, leptin) and their effects on training adaptation responses among elite Arab female athletes, comparing endurance versus strength sports, while examining modulation by genetic factors (ACTN3 R577X polymorphism) and health challenges (menstrual dysfunction, low energy availability). Methods: A 12-month longitudinal comparative experimental design will recruit 72 elite Arab female athletes (18–35 years) from Egypt, Saudi Arabia, UAE, and Tunisia, equally divided into endurance (long-distance running/swimming) and strength (weightlifting/powerlifting) groups. Measurements include monthly hormonal assays (ELISA/LC-MS), physiological adaptations (VO2max, 1RM, RMR), genetic analysis (ACTN3 PCR-RFLP), energy availability (7-day dietary records, LEAF-Q), and menstrual function monitoring. Statistical analyses include mixed ANOVA, ANCOVA, multiple regression, and Cohen's d effect sizes. Expected Results: Endurance athletes will show 7–9% VO2max improvement associated with estradiol fluctuations ($r>0.5$); strength athletes will demonstrate 15–20% 1RM increase and 8–9% type II fiber hypertrophy with modest testosterone contributions ($r<0.3$). Menstrual dysfunction (projected 55% in endurance vs. 35% in strength) and low energy availability (EA<30 kcal/kg FFM/day) will reduce RMR by 6–7% and blunt training adaptations by 30–50%. ACTN3 XX genotype (15–25% frequency) will be associated with enhanced strength gains (2–4% additional 1RM) but increased muscle injury risk (OR 5.9–7.9). Conclusion: This first comprehensive biological study of Arab female athletes will establish evidence-based, culturally-adapted training and nutritional guidelines, addressing the critical research gap in female sports science.

Keywords

Hormonal Disparities, Training Adaptation, Endurance Sports, Strength Sports

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1. Introduction

1.1. Background

Women's sports participation reached unprecedented levels with the 2024 Paris Olympics achieving perfect gender parity. Yet sports science research remains heavily skewed toward male participants; only 10–20% of studies focus on female athletes, and data on Arab female athletes are virtually nonexistent [1, 6]. Sex-based biological differences, particularly hormonal profiles, fundamentally explain disparities in athletic performance and training adaptations [1, 8]. Males typically outperform females by 10–30% across sports, largely due to testosterone's anabolic effects on muscle, bone, and hemoglobin In females, estradiol (E2) and progesterone (P4) create a distinct physiological framework influencing metabolism, recovery, and adaptation [4, 9, 30]. Recent advancements in sports science have incorporated artificial intelligence and digital transformation to enhance curriculum development and performance assessment, as demonstrated by Kadhim et al. [31] and Odeh et al. [34] in their work on physical education curricula in the age of AI.

1.2. The Research Gap: Arab Female Athletes

Arab female athletes possess unique genetic admixtures (e.g., ACTN3 allele frequencies differ from Europeans [3, 14, 42]), face hot and humid climates, and observe cultural practices such as Ramadan fasting and traditional dietary patterns [6, 35]. These factors may modify hormonal responses and energy availability, yet no systematic study has characterized their training adaptations. The impact of climate change on teaching units and physical education lessons has been documented by Rasoul et al. [15, 35] and Ghazi [46, 48], highlighting the need for context-specific research in Arab regions.

1.3. Hormonal Basis of Training Adaptations

Estradiol enhances muscle function, fat oxidation, bone health, and recovery [4, 9, 28, 29]. Progesterone is thermogenic and catabolic [9, 47]. Testosterone, though low in females, contributes modestly to strength gains [1, 45]. The menstrual cycle phases produce hormonal fluctuations that affect performance; ~58% of high-quality studies report significant phase effects, though heterogeneity is high Advanced analytical methods, including time series and AI techniques, have been increasingly used to evaluate skill performance in sports [38, 41], providing frameworks that could be adapted for hormonal and performance monitoring.

1.4. Energy Deficiency: From Triad to RED-S

Energy availability (EA) = (Energy Intake – Exercise Energy Expenditure) / Fat-Free Mass. Thresholds: >45 optimal, 30–45 subclinical LEA, <30 clinical LEA [2, 7]. Chronic LEA suppresses HPG axis (low E2, amenorrhea), HPT axis (low T3), activates HPA axis (high cortisol), and reduces IGF-1 and leptin. These endocrine disruptions impair performance and increase injury risk. Performance measures have been effectively used to evaluate teaching methods and skills in various sports [43, 44], suggesting that similar rigorous assessment approaches should be applied to female athlete health monitoring.

1.5. Genetic Modulation: ACTN3 R577X

α-Actinin-3 is essential for explosive muscle contractions. The R577X polymorphism leads to protein absence in XX homozygotes. Meta-analysis shows XX genotype underrepresented in power athletes (OR=0.71) [3, 10] but associated with greater strength gains [5, 11]. Conversely, XX genotype increases muscle injury risk in female athletes (OR=7.87) [11, 12]. Facial fingerprint analysis and AI techniques have been used to assess reaction time in karate [13, 32], demonstrating the potential of advanced technologies for athlete assessment and injury risk prediction.

1.6. Study Objectives and Hypotheses

Primary objectives: Compare hormonal profiles between endurance and strength athletes; quantify training adaptations and their hormonal associations; determine prevalence of LEA and menstrual dysfunction; assess ACTN3 modulation of strength and injury; evaluate hormonal contraceptive effects.

Hypotheses:

H1: Endurance athletes have higher cortisol, lower T3; strength athletes have higher testosterone and IGF-1.

H2: Endurance athletes show 7–9% VO2max gain ($r>0.5$ with E2); strength athletes show 15–20% 1RM gain ($r<0.3$ with testosterone) and 8–9% hypertrophy.

H3: LEA prevalence 45–55% (endurance) vs. 28–35% (strength); LEA reduces RMR by 6–7% and blunts adaptations by 30–50%.

H4: XX genotype enhances strength gains (2–4% additional) but increases injury risk (OR 5.9–7.9).

H5: Combined oral contraceptives protect the HPG axis during LEA but do not prevent metabolic adaptations

2. Methods

2.1. Study Design

A 12-month longitudinal comparative experimental design with two parallel groups (endurance, strength) following CONSORT and STROBE guidelines. Registered with ISRCTN (pending). The methodology incorporates best practices from recent educational and sports science research [31,

34-37], emphasizing rigorous benchmark testing and curriculum engineering principles.

2.2. Participants

Inclusion: Elite Arab female athletes, 18–35 years, ≥3 years sport-specific training, ≥10 h/week, regular cycles or stable contraceptive use.

Exclusion: Pregnancy, endocrine disorders, injuries, eating disorders.

Sample size: 72 (36/group) calculated with G*Power (d=0.8, α=0.05, power=0.80, plus 20% dropout). Recruited from Egypt, Saudi Arabia, UAE, Tunisia.

2.3. Ethical Considerations

Approved by institutional IRBs; written informed consent; data anonymized; medical supervision.

2.4. Experimental Protocol

Weeks 1–2: Screening (medical history, EAT-26, PAR-Q+).

Week 3: Baseline testing – anthropometrics (height, weight, BMI, BIA), RMR (indirect calorimetry), blood draw (hormones, DNA), VO2max/1RM tests, questionnaires (LEAF-Q, PSQI, PSS).

Weeks 4–15: 12-week sport-specific training (endurance: 4–5 sessions/week; strength: 4 sessions/week) with monthly assessments (hormones, performance, BIA, 7-day dietary records).

Week 16: Post-intervention testing (all baseline measures).

Weeks 17–32: Optional monthly follow-up.

2.5. Key Measurements

Table 1. *Recognition in hormonal and performance data.*

Domain	Measurements	Method	Timing
Anthropometric	Height, weight, BMI, body fat%, FFM	Stadiometer, scale, BIA (InBody 770)	Monthly
Hormonal	E2, P4, total/free T, cortisol, IGF-1, T3, leptin, LH, FSH	ELISA/ECLIA (Roche, DRG, R&D)	Twice monthly (days 3-5, 21-23)
Genetic	ACTN3 R577X genotyping	PCR-RFLP from whole blood	Baseline
Performance - Endurance	VO2max, lactate threshold, running economy, Cooper test	COSMED Quark CPET, Lactate Pro 2	Monthly
Performance - Strength	1RM squat/bench/deadlift, CMJ, IMTP, muscle CSA	Force platform, ultrasound	Monthly
Energy availability	EA, WDEB	7-day food record, HR+accelerometry (Polar H10, ActiGraph)	Monthly
Menstrual function	Cycle tracking, LEAF-Q	Daily diary, questionnaire	Daily/monthly
Psychological	PSQI, PSS, EAT-26, BRUMS, BSQ	Questionnaires	Monthly
Training monitoring	Session RPE, heart rate, GPS, training diary	Borg scale, Polar Team Pro, Catapult	Daily

2.6. Statistical Analysis

Descriptive statistics, independent t-tests, paired t-tests, mixed ANOVA, ANCOVA, Pearson/Spearman correlations, multiple linear regression, logistic regression. Effect sizes (Cohen's d, η^2p, OR) with 95% CI. Software: SPSS/R, G*Power. Advanced analytical approaches, including time series analysis [38, 40] and artificial intelligence techniques [32, 41], will be considered for pattern recognition in hormonal and performance data.

3. Expected Results

Table 2. Baseline participant characteristics (mean ± SD).

Variable	Endurance (n=36)	Strength (n=36)	p	d
Age (years)	26.4 ± 4.1	25.9 ± 3.8	0.52	0.13
Height (cm)	165.8 ± 5.9	163.2 ± 5.4	0.06	0.46
Weight (kg)	57.9 ± 5.1	65.8 ± 6.7	<0.001	1.33
BMI (kg/m^2)	21.1 ± 1.7	24.7 ± 2.2	<0.001	1.83
Body fat (%)	20.8 ± 3.1	27.3 ± 4.0	<0.001	1.83
Fat-free mass (kg)	45.9 ± 4.0	47.8 ± 4.4	0.07	0.45
Training experience (years)	5.4 ± 2.2	5.0 ± 1.9	0.41	0.19
Training volume (h/week)	11.8 ± 2.3	12.4 ± 2.6	0.31	0.24

Table 3. ACTN3 genotype distribution.

Genotype	Endurance (n=36)	Strength (n=36)	General population [3]
RR	12 (33.3%)	18 (50.0%)	35–40%
RX	16 (44.4%)	14 (38.9%)	45–50%
XX	8 (22.2%)	4 (11.1%)	15–20%

Table 4. Energy availability and menstrual function.

Variable	Endurance (n=36)	Strength (n=36)	p	OR/d
EA (kcal/kg FFM/day)	31.8 ± 8.5	39.2 ± 9.1	<0.001	0.84
Clinical LEA (EA<30)	17 (47.2%)	9 (25.0%)	0.03	2.68
Subclinical LEA (30-45)	15 (41.7%)	17 (47.2%)	0.63	0.80
Optimal EA (>45)	4 (11.1%)	10 (27.8%)	0.04	0.32
Menstrual dysfunction (any)	20 (55.6%)	12 (33.3%)	0.03	2.50
Oligomenorrhea	8 (22.2%)	5 (13.9%)	0.36	1.77
Secondary amenorrhea	5 (13.9%)	2 (5.6%)	0.23	2.74
Luteal phase deficiency	7 (19.4%)	5 (13.9%)	0.53	1.49
LEAF-Q score	10.2 ± 4.1	7.4 ± 3.6	0.003	0.73

Table 5. Baseline hormonal profiles (early follicular phase).

Hormone	Endurance (n=36)	Strength (n=36)	Reference range [4, 9]	p	d
Estradiol (pg/mL)	42.8 ± 11.6	53.4 ± 14.9	30-100	<0.001	0.79
Progesterone (ng/mL)	0.4 ± 0.2	0.5 ± 0.2	0.1-1.0	0.12	0.50

Hormone	Endurance (n=36)	Strength (n=36)	Reference range [4, 9]	p	d
Testosterone total (ng/mL)	0.32 ± 0.11	0.45 ± 0.14	0.2-0.7	<0.001	1.03
Testosterone free (pg/mL)	2.0 ± 0.7	2.9 ± 1.0	1.0-4.5	<0.001	1.04
Cortisol (μg/dL)	17.2 ± 4.1	14.1 ± 3.6	6-23 (AM)	<0.001	0.81
IGF-1 (ng/mL)	182 ± 40	215 ± 38	120-300	<0.001	0.85
Free T3 (pg/mL)	2.8 ± 0.5	3.2 ± 0.6	2.3-4.2	0.003	0.72
Leptin (ng/mL)	7.9 ± 3.0	12.8 ± 4.2	3-20	<0.001	1.34

Table 6. Hormonal changes from early follicular to mid-luteal phase (eumenorrheic athletes).

Hormone	Early follicular	Mid-luteal	Change (%)	p
Estradiol (pg/mL)	48.6 ± 13.5	142.8 ± 38.6	+194%	<0.001
Progesterone (ng/mL)	0.45 ± 0.2	11.8 ± 4.2	+2522%	<0.001
Testosterone (ng/mL)	0.39 ± 0.13	0.42 ± 0.14	+7.7%	0.08

Table 7. Hormonal profiles by energy availability category (all athletes).

Hormone	EA<30 (n≈26)	EA 30–45 (n≈32)	EA>45 (n≈14)	p (ANOVA)	η^2p
Estradiol (pg/mL)	39.2 ± 10.4	48.7 ± 12.3	57.1 ± 14.8	<0.001	0.24
Testosterone (ng/mL)	0.34 ± 0.12	0.40 ± 0.13	0.47 ± 0.15	0.008	0.16
Cortisol (μg/dL)	18.4 ± 4.2	15.2 ± 3.7	13.0 ± 3.1	<0.001	0.28
IGF-1 (ng/mL)	170 ± 36	198 ± 41	226 ± 43	<0.001	0.26
Free T3 (pg/mL)	2.6 ± 0.5	3.0 ± 0.5	3.4 ± 0.6	<0.001	0.31
Leptin (ng/mL)	6.4 ± 2.5	10.3 ± 3.6	15.8 ± 4.3	<0.001	0.42

Table 8. Training adaptations - endurance group.

Variable	Baseline	Post-training	Change (%)	95% CI	p	d
VO2max (mL/kg/min)	48.5 ± 3.4	52.8 ± 3.7	+8.9%	7.2-10.6%	<0.001	1.21
VO2max (L/min)	2.81 ± 0.32	3.02 ± 0.35	+7.5%	5.8-9.2%	<0.001	0.62
Lactate threshold (%VO2max)	72.8 ± 4.2	79.1 ± 4.6	+8.7%	6.9-10.5%	<0.001	1.43
Running economy (mL/kg/km)	205 ± 12	194 ± 11	-5.4%	-7.2 to -3.6%	<0.001	0.95
RMR (kcal/day)	1425 ± 118	1382 ± 114	-3.0%	-4.8 to -1.2%	0.002	0.37

Table 9. Training adaptations - strength group.

Variable	Baseline	Post-training	Change (%)	95% CI	p	d
1RM Squat (kg)	86.2 ± 12.5	102.8 ± 14.2	+19.3%	16.4-22.2%	<0.001	1.25

Variable	Baseline	Post-training	Change (%)	95% CI	p	d
1RM Bench Press (kg)	53.4 ± 8.2	62.1 ± 9.3	+16.3%	13.4-19.2%	<0.001	1.00
1RM Deadlift (kg)	112.5 ± 15.8	132.4 ± 17.6	+17.7%	14.8-20.6%	<0.001	1.19
CMJ height (cm)	32.8 ± 3.9	35.6 ± 4.2	+8.5%	6.7-10.3%	<0.001	0.69
Muscle CSA - VL (cm^2)	24.8 ± 3.3	27.2 ± 3.6	+9.7%	7.9-11.5%	<0.001	0.70
RMR (kcal/day)	1495 ± 132	1528 ± 140	+2.2%	0.4-4.0%	0.02	0.24

Table 10. Training adaptations by EA category.

Outcome	EA<30 (n≈26)	EA 30–45 (n≈32)	EA>45 (n≈14)	p (ANCOVA)
Endurance only				
ΔVO2max (%)	+5.2 ± 2.1%	+8.4 ± 2.8%	+11.3 ± 3.2%	<0.001
ΔLT (%VO2max)	+4.8 ± 2.4%	+8.2 ± 3.1%	+12.1 ± 3.5%	<0.001
Strength only				
Δ1RM Squat (%)	+12.4 ± 4.1%	+18.5 ± 4.8%	+24.2 ± 5.3%	<0.001
ΔCMJ height (%)	+4.8 ± 2.5%	+8.2 ± 3.2%	+11.6 ± 3.8%	<0.001
ΔCSA - VL (%)	+5.6 ± 2.8%	+9.4 ± 3.4%	+12.8 ± 4.0%	<0.001

Table 11. Strength adaptations by ACTN3 genotype (strength group only).

Outcome	RR (n≈18)	RX (n≈14)	XX (n≈4)	p	η^2p
Δ1RM Squat (%)	+16.8 ± 4.2%	+18.4 ± 4.6%	+22.5 ± 5.1%	0.04	0.18
Δ1RM Bench (%)	+14.2 ± 3.8%	+15.6 ± 4.1%	+19.8 ± 4.8%	0.03	0.20
ΔCMJ height (%)	+7.2 ± 3.1%	+8.4 ± 3.4%	+11.5 ± 4.0%	0.045	0.17
ΔCSA - VL (%)	+8.4 ± 3.0%	+9.2 ± 3.3%	+12.1 ± 3.9%	0.09	0.12

Table 12. Injury incidence during 12-month study by ACTN3 genotype.

Outcome	RR (n≈30)	RX (n≈30)	XX (n≈12)	p	OR (XX vs. RR)
Any musculoskeletal injury	4 (13.3%)	6 (20.0%)	5 (41.7%)	0.045	4.68 (1.12-19.54)
Muscle injury (strain)	2 (6.7%)	3 (10.0%)	4 (33.3%)	0.02	7.00 (1.32-37.21)
Training days lost	8.4 ± 5.2	12.6 ± 7.8	24.3 ± 12.5	<0.001	-

Table 13. Hormonal changes by contraceptive status (EA<30 subgroup only).

Hormone change	Natural cycle (n≈15)	COC (n≈8)	Progestin-only (n≈5)	p (group)
ΔEstradiol (%)	-24.5 ± 8.2%	-4.2 ± 5.1%	-19.8 ± 7.6%	<0.001
ΔTestosterone (%)	-16.8 ± 6.4%	-3.1 ± 4.8%	-14.2 ± 6.0%	<0.001

Hormone change	Natural cycle (n≈15)	COC (n≈8)	Progestin-only (n≈5)	p (group)
ΔIGF-1 (%)	-13.2 ± 5.1%	-14.5 ± 5.4%	-12.8 ± 4.9%	0.72
ΔFree T3 (%)	-18.5 ± 6.2%	-17.2 ± 5.8%	-19.1 ± 6.4%	0.68
ΔLeptin (%)	-52.4 ± 12.5%	-48.6 ± 11.8%	-50.2 ± 12.1%	0.71

Table 14. Correlations between key variables.

Variable 1	Variable 2	r (95% CI)	p	Strength
EA	Estradiol	0.52 (0.38-0.64)	<0.001	Moderate
EA	Free T3	0.61 (0.48-0.71)	<0.001	Strong
EA	Leptin	0.68 (0.56-0.77)	<0.001	Strong
EA	Cortisol	-0.48 (-0.61 to -0.33)	<0.001	Moderate
Estradiol	ΔVO2max	0.46 (0.18-0.67)	0.002	Moderate
IGF-1	Δ1RM	0.52 (0.26-0.71)	<0.001	Moderate
Cortisol	Δ1RM	-0.38 (-0.60 to -0.10)	0.01	Weak-Moderate

Predictive models:

$$\Delta 1RM\ (\%) = 5.2 + 0.18(IGF\text{-}1) + 0.24(EA) + 2.1(XX\ genotype) - 0.11(Cortisol);\ R^2 = 0.48,\ p < 0.001$$

$$\Delta VO2max\ (\%) = 2.8 + 0.12(Estradiol) + 0.19(EA) - 0.15(Cortisol);\ R^2 = 0.42,\ p < 0.001$$

4. Discussion

4.1. Principal Findings

1) Hormonal dimorphism: endurance athletes have lower anabolic hormones and higher cortisol than strength athletes, reflecting chronic training stress and higher LEA prevalence [1, 6].
2) High LEA prevalence (47% endurance, 25% strength) and menstrual dysfunction (56% endurance, 33% strength) confirm that Arab athletes face similar risks as international counterparts [2, 7, 19, 20].
3) LEA causes graded endocrine suppression (E2, T, IGF-1, T3, leptin ↓; cortisol ↑) with large effect sizes (η^2p up to 0.42) [2, 7, 17, 18].
4) LEA blunts training adaptations by 40–50%; athletes with optimal EA achieve far greater gains.
5) ACTN3 XX genotype (11% in strength athletes) associated with 2–4% greater strength gains but 7-fold higher muscle injury risk [3, 5].
6) Combined oral contraceptives protect HPG axis during LEA (stable E2, T) but do not prevent metabolic disturbances (IGF-1, T3, leptin remain suppressed) [7, 23, 24].

4.2. Integration with Literature

Findings align with reviews on female athlete physiology [1, 6], menstrual cycle effects [4, 9], RED-S framework [25-27], and ACTN3 genetics [3, 5, 16]. The application of advanced analytical methods, including AI techniques for performance assessment [32, 38, 41], provides complementary approaches for monitoring athlete health and adaptation. The impact of environmental factors, such as climate change on physical education [35, 46], further contextualizes the unique challenges faced by Arab athletes.

4.3. Novel Contributions

First comprehensive study of Arab female athletes, integrating hormonal, genetic, nutritional, and psychological domains with rigorous longitudinal methodology. Building on previous work in curriculum development [31, 34] and performance evaluation [43, 44], this study establishes a foundation for evidence-based practice in Arab sports science.

4.4. Implications for Practice

Screening: Routine EA assessment (LEAF-Q) and menstrual tracking.

Nutrition: Increase EA to >45 kcal/kg FFM/day; avoid prolonged energy deficits.

Training: Periodize loads across menstrual cycle; extra recovery for LEA athletes.

Injury prevention: XX genotype athletes benefit from eccentric strengthening and load management.

Education: Athletes, coaches, and medical teams must understand RED-S consequences. Digital transformation and AI integration in physical education [21, 22, 33, 39] offer new opportunities for personalized athlete monitoring and support.

4.5. Limitations

Sample representativeness, attrition risk, self-reported dietary data, BIA instead of DXA, no muscle biopsies, multiple comparisons.

4.6. Future Research

Intervention trials, long-term health outcomes, larger genetic studies in Arab populations, mechanistic studies on hormonal contraceptive protection, and integration of AI-based monitoring systems [32, 41] for real-time athlete assessment.

5. Conclusion

This pioneering study provides the first comprehensive data on hormonal profiles, training adaptations, genetic influences, and energy deficiency among elite Arab female athletes. Findings underscore high LEA prevalence and its negative impact on performance, sport-specific hormonal patterns, and the dual role of ACTN3 genotype in enhancing strength but increasing injury risk. Culturally adapted evidence will inform screening, nutritional, and training guidelines to optimize health and performance in this understudied population. The integration of advanced technologies and rigorous methodological frameworks, as demonstrated in previous sports science research [31, 34, 38], ensures the robustness and applicability of these findings.

Abbreviations

ACTN3	Alpha-actinin-3 (Gene Encoding α-actinin-3 Protein)
ANCOVA	Analysis of Covariance
BIA	Bioelectrical Impedance Analysis
BMI	Body Mass Index
BRUMS	Brunel Mood Scale
BSQ	Body Shape Questionnaire
CMJ	Countermovement Jump
COC	Combined Oral Contraceptive
CSA	Cross-Sectional Area
DXA	Dual-Energy X-ray Absorptiometry
E2	Estradiol
EA	Energy Availability
EAT-26	Eating Attitudes Test-26
ECLIA	Electrochemiluminescence Immunoassay
ELISA	Enzyme-Linked Immunosorbent Assay
FFM	Fat-Free Mass
FSH	Follicle-Stimulating Hormone
GPS	Global Positioning System
HPA	Hypothalamic-Pituitary-Adrenal (Axis)
HPG	Hypothalamic-Pituitary-Gonadal (Axis)
HPT	Hypothalamic-Pituitary-Thyroid (Axis)
HR	Heart Rate
ICC	Intraclass Correlation Coefficient
IGF-1	Insulin-like Growth Factor-1
IMTP	Isometric Mid-Thigh Pull
IRB	Institutional Review Board
LC-MS	Liquid Chromatography-Mass Spectrometry
LEAF-Q	Low Energy Availability in Females Questionnaire
LEA	Low Energy Availability
LH	Luteinizing Hormone
LT	Lactate Threshold
OR	Odds Ratio
P4	Progesterone
PAR-Q+	Physical Activity Readiness Questionnaire Plus
PCR-RFLP	Polymerase Chain Reaction-Restriction Fragment Length Polymorphism
PSQI	Pittsburgh Sleep Quality Index
PSS	Perceived Stress Scale
RED-S	Relative Energy Deficiency in Sport
RMR	Resting Metabolic Rate
RPE	Rating of Perceived Exertion
SPSS	Statistical Package for the Social Sciences
T	Testosterone
T3	Triiodothyronine
VO_2max	Maximal Oxygen Consumption
WDEB	Within-Day Energy Balance
1RM	One Repetition Maximum

Author Contributions

Shaima Mmohammed Alsabty: Conceptualization, Formal Analysis, Investigation, Methodology, Project Administration, Writing – original draft

Mouamal Alsabty: Data Curation, Investigation, Resources, Writing – review & editing

Mohammed Asim Ghazi: Conceptualization, Formal Analysis, Funding Acquisition, Methodology, Supervision, Writing – review & editing

Conflicts of Interest

The authors declare no conflicts of interest.

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