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Energy availability and its association with health-related outcomes among national athletes at risk of relative energy deficiency in sports (REDs)

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ABSTRACT

Background Low energy availability (LEA) occurs when athletes' energy intake fails to match the energy expended during exercise, resulting in insufficient energy to support essential functions for optimal health, a condition known as relative energy deficiency in sports (REDs).

Objective This study aims to explore the prevalence of LEA among Malaysian national athletes and its associations with health-related outcomes.

Methods A total of 43 athletes (51.2% males, aged 18–40) identified previously as having moderate or high risk of REDs through a questionnaire underwent comprehensive clinical assessments. Resting metabolic rate (RMR) was measured using indirect calorimetry, with an RMR ratio of <0.90 indicating LEA. Weight and height were measured, and fasting blood samples were analysed for ferritin, free triiodothyronine (fT3), follicle-stimulating hormone (FSH), luteinising hormone (LH), estradiol (female athletes) and testosterone (male athletes). Bone mineral density (BMD) of the lumbar spine and total left hip, as well as body composition, were measured using dual-energy X-ray absorptiometry (DXA).

Results Out of the 43 athletes, 12 showed evidence of LEA, exhibiting at least one of the following characteristics: low estradiol levels (87.5%), low testosterone (75.0%), low fT3 (66.7%), low LH (58.3%), low FSH (58.3%), low ferritin (25.0%) and low BMD (8.3%). Notably, fT3, estradiol and testosterone were significant predictors for LEA.

Conclusions A low but noteworthy incidence of LEA among Malaysian national athletes was associated with hormone imbalances. Awareness about LEA among athletes and sports personnel is essential for early detection and appropriate intervention.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Low energy availability (LEA) commonly occurs among athletes and is associated with a range of physiological disturbances, collectively referred to as relative energy deficiency in sports (REDs).
⇒ REDs are prevalent among athletes, particularly females, and those in sports emphasise leanness and endurance.

WHAT THIS STUDY ADDS

⇒ Highlights the prevalence of LEA among Malaysian national athletes, providing region-specific data.
⇒ Free triiodothyronine, estradiol and testosterone are significant biomarkers associated with LEA in athletes.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Emphasises the need for increased awareness of LEA among athletes and sports personnel for early detection and intervention and incorporation of regular LEA screening in athletes.

INTRODUCTION

Elite athletes dedicate their lives to attaining peak physical performance across diverse disciplines, each with unique physiological demands and training regimens.[1] The rigorous training requires substantial energy intake to maintain optimal physiological function and meet high energy demands.[2] Energy availability (EA) is crucial, representing the energy available for physiological functions after accounting for energy expended in physical activity.[3] Low energy availability (LEA) occurs when insufficient energy to support all physiological functions threatens overall health.[4] LEA triggers various adaptations to conserve energy, leading to a cascade of physiological disturbances known as relative energy deficiency in sports (REDs).[5] LEA may arise intentionally, driven by pursuing a specific physique to enhance performance, or unintentionally during intense training or in high-demand sports.[6]

Recognising the diverse consequences of LEA, the International Olympic Committee (IOC) issued a consensus statement defining REDs.[3 7] REDs describe the multisystem consequences of energy deficiency beyond bone health and menstrual disruption, commonly observed in high-performing female athletes (the female athlete triad), with LEA as its core.[7] LEA affects metabolic function, bone

health, haematological parameters and endocrine regulation.[6 7] LEA-induced iron depletion can impair endurance performance, energy metabolism and overall athlete health.[8] LEA can also disrupt the hypothalamic-pituitary-thyroid (HPT) axis, reducing thyroid hormone levels, specifically triiodothyronine (T3).[9] LEA is also linked to disruption of reproductive hormone secretion, including luteinising hormone (LH), follicle-stimulating hormone (FSH), estradiol and testosterone.[10]

Recently, the IOC updated its consensus statement to define REDs as a syndrome of impaired physiological and psychological functioning in both female and male athletes due to problematic LEA.[11] This new position introduced the concepts of adaptable and problematic LEA.[11] Adaptable LEA involves mild and quickly reversible changes with minimal long-term impact. Conversely, problematic LEA leads to significant and potentially persistent disruptions in various body systems, severely affecting health, well-being and performance. While adaptable LEA may offer short-term benefits, problematic LEA results in REDs. Understanding this distinction is important for effective management and intervention.[11]

Despite the importance of maintaining optimal EA, current literature lacks universally accepted guidelines. Proposed thresholds include 45 kcal/kg fat-free mass (FFM)/day for sedentary women and 40 kcal/kg FFM/day for active men.[12 13] An EA below 30 kcal/kg FFM/day is considered critical for LEA.[7] Measuring EA presents several challenges, including reliance on self-reported data and inaccurately assessing energy intake.[3 7] An EA of 30 kcal/kg FFM/day is approximately equivalent to the average resting metabolic rate (RMR).[13] A suppressed RMR, indicated by a ratio of measured RMR to predicted RMR below 0.90, is a potential and emerging indicator of LEA.[14–16]

REDs affect athletes across various sports and demographics, posing significant health challenges, with impacts differing by gender, sport and EA status. Previously, only the prevalence of triad components was reported among elite female Malaysian athletes.[17] Thus, there is a need for a comprehensive understanding of LEA and its health consequences among Malaysian national athletes. Clinical biomarkers related to metabolism, bone health, haematological status and endocrine function are essential for elucidating the multifaceted impact of LEA on elite athletes. This study aims to address this gap by investigating the prevalence of LEA among Malaysian athletes, stratified by gender, sports and EA status, and exploring its associations with metabolism, bone health, haematological parameters and endocrine profiles.

MATERIALS AND METHOD

Study design and participants

This cross-sectional study involved 143 Malaysian national athletes from five sports categories: weight class, power, intermittent, endurance and skill. The athletes were training at the National Sports Institute of Malaysia and had previously been identified as medium or high-risk for REDs using the RED-S-specific screening tool,[18] adapted from Foley *et al*.[19] Eligibility criteria included being a Malaysian citizen and aged 18 years or older. Exclusion criteria were pregnancy or current injured individuals. Ethical approval was obtained from the university's Research Ethics Committee (reference code: UKM PPI/111/8/JEP-2022–303) and the National of Sports Institute Research Ethics Committee (reference code: RE/A/008/2022-003/2022). Written informed consent was obtained from all participants. Data collection occurred from January to August 2023.

Data collection

Participants fasted for 8 hours, avoiding food, caffeine, calcium supplements and alcohol, and refrain from strenuous activities for 12 hours before the assessment. These requirements were primarily to ensure accurate measurement of RMR. To standardise the protocol across all athletes and eliminate the possibility that the postprandial state might affect any of the hormones analysed, fasting blood samples were collected for all blood biomarkers.[20] Measurements were conducted between 7:00 and 9:00 am at the laboratory. After a briefing on study procedures, participants signed an informed consent form and completed a sociodemographic questionnaire. Height and weight were measured using standardised equipment, followed by RMR measurements, blood sample collection, and body composition and bone mineral density (BMD).

Anthropometric measurements

Height and weight were measured using a digital metre (SECA 284, Hamburg, Germany). Height was recorded to the nearest 0.1 cm and weight to the nearest 0.1 kg with minimal clothing. Each measurement was taken twice, and the average was recorded.

Resting metabolic rate (RMR)

RMR, reflecting oxygen consumption (VO_2) and carbon dioxide production (VCO_2), was measured using indirect calorimetry (TrueOne 2400, Parvo Medics, Sandy, USA). Calibration of the gas and flow metre was performed daily. Participants lay on a bed with heads covered by a ventilated canopy for 30 min, with data from the initial 10 min excluded. The measured RMR (mRMR) was calculated using the last-minute average for every 5 min of the final 20 min using the Weir equation.[21] The predicted RMR (pRMR) was calculated using the Cunningham equation.[22] An RMR ratio (mRMR/pRMR) less than 0.9 indicated LEA.[16]

Blood sample

Blood samples were collected from the antecubital arm vein into a 10-mL SST BD Vacutainer tube and allowed to coagulate for 10 min. The serum was separated by centrifugation (Eppendorf Centrifuge 5702R) at 3500 rpm for 10 min, then transferred into 1.5 mL Eppendorf tubes and stored at −80°C. Serum analyses

for ferritin, free-triiodothyronine (fT3), FSH, LH and estradiol were conducted using chemiluminescence microparticle immunoassay (Abbott Alinity ci-series). Testosterone was analysed using electrochemiluminescence immunoassay on the Cobas 8000 e801 system. All analyses were outsourced to an accredited laboratory.

Body composition and bone mineral density (BMD)

Body composition and BMD were assessed using DXA (Hologic A, DXA Horizon System, USA). A qualified radiographer conducted the scans, with daily calibration using a phantom per manufacturer guidelines. For body composition, a whole-body scan was performed with participants lying supine. Data were recorded for fat mass and body fat percentage. BMD was assessed in the left hip and lumbar spine. A Z-score greater than −1.0 at all scanned sites indicated normal BMD, while a Z-score less than −1.0 in at least one scanned site indicated low BMD.[23]

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics software V.26.0 (IBM SPSS Statistics Corporation, Chicago, IL, USA). Data normality was assessed using the Shapiro-Wilk test. Normally distributed continuous data are presented as mean and SD, non-normally distributed data as median and IQR, and categorical data as frequency and percentage. Independent t-tests compared continuous data between two groups, and ANOVA with Tukey post-hoc tests compared data across three or more groups. Associations between categorical data were examined using the x^2 test. Significant associations guided binary logistic regression analysis. Statistical significance was set at $p<0.05$.

RESULTS

Sociodemographic profile

Out of 143 athletes invited, 43 completed the assessments (30% participation rate). Reasons for non-participation included programme discontinuation (n=48), injuries or rehabilitation (n=6), overseas tournaments (n=14) and intensive training schedules (n=75).

Half of the participants were male (51.2%) and Malay (55.8%) (table 1). The largest group was from the weight class sports category (41.9%). Training phases included general preparation (n=27.9%), specific preparation (n=39.5%) and pre-competition (n=32.6%). LEA, indicated by an rRMR <0.90, was observed in 12 athletes (27.9%), with no significant sociodemographic differences.

Table 1 Sociodemographic profile based on energy availability (EA) status

	N (%)	Low EA (rRMR<0.9) n=12	Optimal EA (rRMR>0.9) n=31	P value*
Gender				0.146
Male	22 (51.2)	4 (9.3)	18 (41.9)	
Female	21 (48.8)	8 (18.6)	13 (30.2)	
Ethnicity				0.549
Malay	24 (55.8)	8 (18.6)	16 (37.2)	
Chinese	13 (30.2)	2 (4.7)	11 (25.6)	
India	1 (2.3)		1 (2.3)	
Others	5 (11.6)	2 (4.7)	3 (7.0)	
Education level				0.547
Secondary school	15 (34.9)	5 (11.6)	10 (23.3)	
Pre-university	8 (18.6)	3 (7.0)	5 (11.6)	
Tertiary education	20 (46.5)	4 (9.3)	16 (37.5)	
Sport category				0.681
Weight class	18 (41.9)	5 (11.6)	13 (30.2)	
Power	11 (25.6)	2 (4.7)	9 (20.9)	
Intermittent	5 (11.6)	2 (4.7)	3 (7.0)	
Endurance	3 (7.0)	1 (2.3)	2 (4.7)	
Skill	6 (14.0)	2 (4.7)	4 (9.3)	
Training phase				0.132
General preparation	12 (27.9)	2 (4.7)	10 (23.3)	
Specific preparation	17 (39.5)	4 (9.3)	13 (30.2)	
Pre-competition	14 (32.6)	6 (14.0)	8 (18.5)	

*x^2 test.
EA, energy availability; RMR, resting metabolic rate.

Comparison of physical characteristics, bone mineral density, haematological and endocrine profile between sexes

Male athletes were taller and heavier and had lower body fat percentages than females (table 2). Males had significantly higher mRMR and pRMR. Female athletes had lower BMD at the lumbar spine (1.1 g/cm^2) and total hip (1.0 g/cm^2) compared with males (1.2 g/cm^2 for both sites). All male athletes had normal BMD, while two females (9.5%) had low BMD. Female athletes had lower fT3 levels (3.6 pmol/L) compared with males (4.3 pmol/L), and 28.6% had lower LH and FSH levels compared with males (4.5%).

Comparison of physical characteristics, bone mineral density, haematological and endocrine profile between sport categories

Weight-class athletes had significantly lower fat mass (11.6±2.7 kg) and body fat percentage (18.6±3.5%) compared with skill athletes (16.9±7.6 kg and 30.2±8.5%) (table 3). Endurance athletes had lower BMD at the total hip (1.0 g/cm^2) than skill athletes (1.2 g/cm^2). No significant differences were observed in other physical characteristics, bone health or haematological and endocrine profiles between sports categories.

Table 2 Physical characteristics, bone health, haematological and endocrine profiles based on sex

	Male Mean±SD n=22	Female Mean±SD n=21	P value
Physical characteristics			
Age (years)*	23.0±5.1	21.9±5.5	0.264†
Weight (kg)	68.4±10.8	58.8±12.2	0.009‡
Height (cm)	169.9±7.2	161.4±7.2	<0.001‡
Fat mass (kg)	12.5±3.8	16.6±1.5	0.022‡
Body fat percentage (%)	17.7±0.7	27.3±6.2	<0.001‡
Measured RMR (mRMR) (kcal/day)	1618±210	1328±179	<0.001‡
Predicted RMR (pRMR) (kcal/day) (Cunningham equation)	1676±179	1391±168	<0.001‡
Bone health			
BMD lumbar (g/cm^2)	1.2±0.1	1.1±0.1	0.016‡
Z-score lumbar	1.7±1.0	0.9±1.2	0.079‡
BMD total hip (g/cm^2)	1.2±0.1	1.0±0.1	<0.00‡
Z-score total hip*	2.1±1.4	1.0±2.2	0.087‡
Low BMD§ (Z-score<−1.0 at any scanned site)	0	2 (9.5)	0.138¶
Normal BMD§ (Z-score>−1.0 at any scanned site)	22 (100)	19 (90.5)	
Haematological and endocrine profiles			
Ferritin (µg/L)	140.1±68.2	116.6±64.2	0.104‡
Free triiodothyronine (fT3) (pmol/L)*	4.2±0.2	3.6±1.8	0.009†
Luteinising hormone (LH) (IU/L)	3.4±1.5	2.9±3.1	0.520‡
Follicle-stimulating hormone (FSH) (IU/L)	3.9±1.9	4.1±4.0	0.295‡
Estradiol (pmol/L)	n/a	156.0±233.5	n/a
Testosterone (nmol/L)	18.9±7.1	n/a	n/a
Low ferritin level§	1 (4.5)	2 (9.5)	0.522¶
Normal ferritin level§	21 (95.5)	19 (90.5)	
Low fT3 level§	2 (9.1)	6 (28.6)	0.101¶
Normal fT3 level§	20 (90.9)	15 (71.4)	
Low LH level§	1 (4.5)	6 (28.6)	0.033¶
Normal LH level§	21 (95.5)	15 (71.4)	
Low FSH level§	1 (4.5)	6 (28.6)	0.033¶
Normal FSH level§	21 (95.5)	15 (71.4)	
Low estradiol level§	–	7 (33.3)	
Normal estradiol level§	–	14 (66.7)	
Low testosterone level§	3 (13.6)	–	
Normal testosterone level§	19 (86.4)	–	

*Median (IQR).
†Mann-Whitney U test.
‡Independent t-test.
§n (%).
¶x^2 test.
BMD, bone mineral density; RMR, resting metabolic rate.

Comparison of physical characteristics, bone mineral density, haematological and endocrine profile between EA status

Athletes with LEA had significantly lower mRMR (1328 kcal/day) compared with those with optimal EA (1534 kcal/day) (table 4). There was a trend toward lower lumbar BMD in the LEA group (1.1 g/cm^2) compared with the optimal EA group (1.2 g/cm^2), though not statistically significant (p=0.08). LEA athletes also had significantly lower levels of haematological and endocrine hormones compared with the optimal EA group.

Table 3 Physical characteristics, bone health, haematological and endocrine profiles based on sport categories

	Weight class Mean±SD n=18	Power Mean±SD n=11	Intermittent Mean±SD n=5	Endurance Mean±SD n=3	Skill Mean±SD n=6	P value
Physical characteristics						
Age (years)*	22.4±4.1	23.8±4.7	26.4±6.8	19.9±0.0	21.2±7.5	0.173†
Weight (kg)	63.1±12.2	61.8±14.2	73.9±12.7	58.5±4.7	63.1±10.1	0.383‡
Height (cm)	166.2±9.6	164.8±88	169.2±7.9	162.3±1.4	164.8±6.5	0.826‡
Fat mass (kg)	11.6±2.7*	14.2±8.1	14.5±6.0	16.6±1.8	16.9±7.6*	0.015‡
Percent body fat (%)	18.6±3.5*	22.2±7.9	23.6±4.3	28.3±1.3	30.2±8.5*	<0.001‡
RMR (mRMR) (kcal/day)	1491±251	1415±215	1674±327	1549±198	1345±243	0.192‡
Predicted RMR (pRMR) (kcal/day) (Cunningham equation)	1573±208	1476±252	1723±285	1457±107	1423±108	0.152‡
Bone health						
BMD lumbar (g/cm^2)	1.2±0.1	1.2±0.1	1.1±0.2	1.1±0.1	1.0±0.2	0.446‡
Z-score lumbar	1.3±1.3	1.6±0.8	1.3±1.5	1.1±1.0	0.5±1.4	0.515‡
BMD total hip (g/cm^2)	1.0±0.2	1.1±0.1	1.3±0.2	1.0±0.0*	1.2±0.2*	0.033‡
Z-score total hip*	1.6±2.7	1.5±2.5	2.9±2.8	0.3±0.0	1.2±5.8	0.321†
Low BMD¶ (Z-score<−1.0 at any scanned site)	1 (5.6)	n/a	n/a	n/a	1 (16.7)	0.572§
Normal BMD¶ (Z-score>−1.0 at any scanned site)	17 (94.4)	11(100)	5 (100)	3 (100)	5 (83.3)	
Haematological and endocrine profiles						
Ferritin (µg/L)	102.6±59.6	99.1±59.1	221.8±50.5	100.1±43.3	143.6±51.4	0.113‡
Free triiodothyronine (fT3) (pmol/L)	4.0±0.9	4.2±0.6	3.9±0.9	3.4±0.7	3.4±0.9	0.311‡
Luteinising hormone (LH) (IU/L)	3.7±4.7	3.0±1.5	2.2±1.3	3.2±2.2	3.8±1.3	0.551‡
Follicle-stimulating hormone (FSH) (IU/L)	3.4±2.8	4.1±2.0	5.2±3.1	5.5±2.3	4.7±1.8	0.694‡
Estradiol (pmol/L)	396.5±325.1	182.1±95.9	88.0±0.0	117.3±43.2	219.5±153.4	0.261‡
Testosterone (nmol/L)	19.6±8.2	21.8±2.1	17.9±6.5	n/a	10.8±2.9	0.349‡
Low ferritin level¶	2 (11.1)	n/a	n/a	n/a	1 (16.7)	0.598§
Normal ferritin level¶	16 (88.9)	11 (100)	5 (100)	3 (100)	5 (83.3)	
Low fT3 level¶	4 (22.2)	n/a	1 (20.0)	1 (33.3)	2 (33.3)	0.411§
Normal fT3 level¶	14 (77.8)	11 (100)	4 (80.0)	2 (66.7)	4 (66.7)	
Low LH level¶	1 (5.6)	2 (18.2)	2 (40.0)	1 (33.3)	1 (16.7)	0.373§
Normal LH level¶	17 (94.4)	9 (81.8)	3 (60.0)	2 (66.7)	5 (83.3)	
Low FSH level¶	1 (5.6)	2 (18.2)	1 (20.0)	1 (33.3)	2 (33.3)	0.475§
Normal FSH level¶	17 (94.4)	9 (81.8)	4 (80.0)	2 (66.7)	4 (66.7)	
Low estradiol level¶	5 (83.3)	5 (71.4)	1 (100)	2 (66.7)	2 (50.0)	0.506§
Normal estradiol level¶	1 (16.7)	2 (28.6)	n/a	1 (33.3)	2 (50.0)	
Low testosterone level¶	2 (16.7)	n/a	1 (25.0)	n/a	n/a	0.687§
Normal testosterone level¶	10 (83.3)	4 (100)	3 (75.0)	n/a	2 (100)	

*Median (IQR).
†Kruskal-Wallis test.
‡ANOVA (Tukey's post-hoc test).
§x^2 test.
¶n (%).
BMD, bone mineral density; RMR, resting metabolic rate.

Association between rRMR and REDs health-related biomarkers

Binary logistic regression identified fT3, estradiol and testosterone as significant predictors of LEA (table 5). Each unit increase in fT3, estradiol and testosterone was associated with a 55.4%, 45.1% and 27.1% decrease in the odds of having LEA, respectively.

DISCUSSION

This study found that 27.9% of athletes identified as moderate or high risk for REDs exhibited LEA based on rRMR. Gender differences were evident, with female athletes having lower BMD, fT3, LH and FSH levels than males. In terms of sports categories, endurance athletes had lower BMD than skill athletes. LEA was associated

Table 4 Physical characteristics, bone health, haematological and endocrine profile based on EA status

	Low EA (rRMR<0.9) Mean±SD n=12	Optimal EA (rRMR>0.9) Mean±SD n=31	P value
Physical characteristics			
Age (years)*	21.0±5.0	23.5±5.5	0.914†
Weight (kg)	61.7±13.4	64.5±12.0	0.524‡
Height (cm)	163.9±9.5	166.5±7.9	0.368‡
Fat mass (kg)	16.1±7.1	13.9±5.5	0.288‡
Percent body fat (%)	25.7±7.8	21.1±6.2	0.131‡
RMR (mRMR) (kcal/day)	1328±201	1534±235	0.011‡
Predicted RMR (pRMR) (kcal/day) (Cunningham equation)	1513±228	1546±226	0.668‡
Bone health			
BMD lumbar (g/cm^2)	1.1±0.2	1.2±0.1	0.080‡
Z-score lumbar	0.8±1.5	1.4±1.0	0.091‡
BMD total hip (g/cm^2)	1.1±1.2	1.2±0.2	0.263‡
Z-score total hip*	0.8±2.6	1.5±2.0	0.626†
Low BMD§ (Z-score<−1.0 at any scanned site)	1 (8.3)	1 (3.2)	0.104¶
Normal BMD lumbar§ (Z-score>−1.0 at any scanned site)	11 (91.7)	30 (96.8)	
Haematological and endocrine profiles			
Ferritin (µg/L)	85.1±81.7	115.0±64.9	0.042‡
Free triiodothyronine (fT3) (pmol/L)*	2.6±1.3	3.9±0.9	0.006†
Luteinising hormone (LH) (IU/L)	2.5±1.7	3.5±1.8	0.012‡
Follicle-stimulating hormone (FSH) (IU/L)	2.9±2.1	4.1±2.7	0.050‡
Estradiol (pmol/L)*	88.0±0.0	237.0±207.0	<0.001†
Testosterone (nmol/L)	13.6±13.1	20.0±5.0	0.039‡
Low ferritin level§	3 (25.0)	–	0.004¶
Normal ferritin level§	9 (75.0)	31 (100)	
Low fT3 level§	8 (66.7)	–	<0.001¶
Normal fT3 level§	4 (33.3)	31 (100)	
Low LH level§	7 (58.3)	–	<0.001¶
Normal LH level§	5 (41.7)	31 (100)	
Low FSH level§	7 (58.3)	–	<0.001¶
Normal FSH level§	5 (41.7)	31 (100)	
Low estradiol level§	7 (87.5)	–	<0.001¶
Normal estradiol level§	1 (12.5)	13 (100)	
Low testosterone level§	3 (75.0)	–	<0.001¶
Normal testosterone level§	1 (25.0)	18 (100)	

*Median (IQR).
†Mann-Whitney U test.
‡Independent t-test.
§n (%).
¶x^2 test.
BMD, bone mineral density; EA, energy availability; RMR, resting metabolic rate.

with reduced ferritin, fT3 and sex hormones, with fT3, estradiol and testosterone being significant predictors of LEA.

LEA often results in a decreased RMR as the body conserves energy,[24] a phenomenon known as metabolic suppression, which is well-documented in the literature.[14–16] For instance, a study of female endurance elite athletes in Sweden and Denmark found that 53% had low rRMR, with those in the LEA group showing lower metabolic rates (rRMR=0.87) compared with those with

Table 5 Factors associated with low EA (rRMR<0.90)

	Wald	Exp(B)	95% CI for EXP(B)		P value**
			Lower	Upper	
Ferritin (µg/L)	0.167	1.003	0.989	1.102	0.683
Free triiodothyronine (fT3) (pmol/L)	6.364	0.554	0.036	0.658	0.012
Luteinising hormone (LH) (IU/L)	2.101	0.559	0.254	1.227	0.147
Follicle-stimulating hormone (FSH) (IU/L)	0.484	0.838	0.508	1.380	0.487
Estradiol (pmol/L)	6.319	0.451	0.907	0.997	0.015
Testosterone (nmol/L)	4.424	0.271	0.732	1.036	0.046

*Binary logistic regression.

optimal EA (rRMR=0.93).[15] Similarly, a study of male football players in Korea reported that the LEA group had a lower rRMR (0.91) than the optimal EA group (1.01).[14] In a study of ballet dancers in Denmark, 45% of females and 25% of males had a low rRMR (<0.90), indicating LEA, when assessed using the Harris-Benedict equation.[16] A higher prevalence of suppressed rRMR was observed when using the Cunningham equation, with all females and 80% of males affected, highlighting the importance of choosing an appropriate equation to avoid overestimating or underestimating rRMR and misclassification of athletes at risk for energy deficiency.[16] The Cunningham equation is particularly relevant for athletes as it incorporates lean body mass, which accounts for about 70% of the variation in predicting RMR.[22] While rRMR is a reliable measure of energy status, its requirement for laboratory assessment may limit its broader application.

In this study, female athletes had lower BMD, consistent with known hormonal differences between genders.[25] Bone density and bone area are similar in growing males and females until around age 16 when gender disparities become evident as males continue to accrue bone mass more rapidly than females.[26] Anatomical and hormonal factors also lead to a higher injury rate among female athletes than males.[27] While both genders are vulnerable to low BMD, males may have some protective resilience due to factors such as lower reproductive energy expenditure and the bone-protective effects of androgens.[28]

Endurance athletes in the current study had lower hip BMD than skill athletes. A comprehensive review suggests that athletes focused on endurance, lean physique and aesthetics are more likely to exhibit components of the female athlete triad, including low BMD.[29] Specific groups of endurance athletes, such as runners and cyclists, are at a higher risk for low BMD.[30 31] Despite this, only 4.7% of athletes in our study had a low BMD, indicating regular physical activity may positively impact bone formation and structure.[32] Supporting this, Hoch *et al* found that only 16% of athletes had low BMD, compared with 30% of sedentary individuals, emphasising the protective role of regular exercise on bone health.[33]

In this study, athletes with LEA had significantly lower ferritin levels (85.1 µg/L) than those with optimal EA (115.0 µg/L). Iron deficiency is prevalent among athletes and can lead to direct and indirect energy deficiencies.[3 7] Iron is essential for oxygen transport, particularly for athletes with high blood cell turnover.[34] Inadequate calorie intake can reduce intake of essential micronutrients, including iron. Previous research has linked LEA to haematological issues, including low ferritin levels.[35] Ferritin serves as a key marker of iron storage, highlighting the impact of LEA on iron deficiency. A previous study showed that 55.6% of athletes at high risk for the female athlete triad had poor iron status, compared with only 9.5% in the low-risk group.[36] The lower ferritin levels in the LEA group of the current study emphasise the importance of assessing ferritin levels when evaluating LEA.

Female athletes in the current study had lower fT3 levels than male athletes. Furthermore, those with LEA had lower fT3 levels than those with optimal EA. LEA affects the HPT axis, leading to reduced levels of T3 and thyroxine (T4).[37] Although most EA studies do not measure T4, it is well-documented that LEA lowers both fT3 and total T3 levels.[38] The observed decrease in T3 levels among females with LEA aligns with previous studies.[39] Studies indicate that EA, rather than exercise-induced stress, primarily regulates thyroid hormone levels.[40] For instance, maintaining EA at around 38 kcal/kg FFM/day during exercise did not affect thyroid levels.[39] However, reducing EA to approximately 11 kcal/kg FFM/day for 4 days led to decreased free and total T3 levels, regardless of exercise intensity.[41] Recent studies consistently show low fT3 and total T3 levels during LEA,[8 40–42] supporting the significant impact of LEA on thyroid hormones and suggesting that T3 could be a reliable biomarker for LEA in athletes.[43]

Analysis of reproductive hormones in the present study revealed significant differences in LH, FSH, estradiol and testosterone levels between athletes with LEA and those with optimal EA. These hormonal differences highlight the complex relationship between EA and the endocrine system, particularly the hypothalamic-pituitary-gonadal (HPG) axis. In a state of LEA, the HPG axis reduces energy expenditure, leading to a 'sick euthyroid' profile where the body prioritises minimal energy function.[36] As

a result, energy is diverted from the reproductive axis, affecting hormonal balance and overall metabolic function.[39]

Both female and male athletes with LEA may experience changes in reproductive hormone levels and functions.[44] Studies have reported lower LH levels in female athletes with LEA compared with those with optimal EA.[15 45] However, some research, like Kyte *et al*,[45] found no significant difference in FSH levels between groups. Sygo *et al*[46] observed lower LH and FSH levels in 60% of elite female sprinters after 5 months of training, highlighting the impact of LEA on reproductive hormones. However, Loucks and Thuma[12] found no significant changes in LH and FSH levels in females with severe LEA. These inconsistencies underscore the complex relationship between LEA and reproductive hormones, emphasising the need for further research. Reduced FSH and LH levels often reduce estradiol release from the ovaries.[47] Despite variability in LH and FSH findings, athletes with LEA consistently show lower estradiol levels, indicating a strong association between EA and estradiol.[12 47 48]

Male athletes, similar to their female counterparts, are susceptible to impaired reproductive function, a phenomenon known as exercise hypogonadal male syndrome, marked by reduced testosterone levels.[39 49] Research on hormonal variations in male athletes, particularly LH and FSH, is limited, with most studies focusing on testosterone. For instance, Hooper *et al* found that athletes with an average EA of 27.2 kcal/kg FFM/day had significantly lower testosterone levels than non-athletes with a sufficient EA of 45.4 kcal/kg FFM/day. However, LH and FSH levels showed no significant differences.[44] This aligns with the current study and other research linking low EA with reduced testosterone levels in athletes.[44] The exact mechanisms underlying these lower testosterone levels are not fully understood. Still, current theories suggest that inadequate calorie intake and the physiological stress induced by intense exercise may disrupt the HPG axis, leading to suppressed testosterone production.[49] More research is required to comprehend the impact of LEA on the male HPG axis and improve EA assessment to better understand hormonal responses and their implications.

Strengths and limitations

This study is the first to assess EA among Malaysian national athletes, using robust methods for evaluating RMR and BMD. However, the small sample size and cross-sectional design limit the generalisability of the findings. The low participation rate, particularly from athletes in endurance sports and those with intensive training schedules, may obscure the true prevalence of REDs within the broader cohort of national athletes. Future research should include larger, more diverse samples and employ longitudinal studies to explore the long-term effects of EA on hormonal health and athletic performance.

CONCLUSION

A low but significant incidence of LEA was observed among Malaysian national athletes, which is associated with hormonal imbalances. This highlights the need for routine screening to address LEA and its potential health impacts. Future research should expand hormonal profiling related to metabolism and stress response to better understand the complex relationship within the endocrine system in athletes with LEA.

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