

TESIS DOCTORAL



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Más allá del umbral: respuestas individuales a la baja
disponibilidad energética en deportistas

Autor:

Sergio Espinar Guerrero

Directores:

Dr. D. Carlos Javier Contreras Fernández

Dr. D. José Miguel Martínez Sanz

Dr. D. Lucas Jurado Fasoli

Murcia, Enero de 2026

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Beyond the Threshold: Individual Responses to Low Energy Availability in Athletes

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Fdo. Carlos J. Contreras Fernández.



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RESUMEN

La disponibilidad energética (del inglés, *Energy Availability*, EA) constituye un factor clave para mantener la salud, el rendimiento y la capacidad de adaptación del deportista. EA se define como la cantidad de energía que queda disponible para mantener las funciones básicas tras restarle el gasto energético derivado del ejercicio. Cuando la EA es insuficiente para cubrir estas funciones básicas, se produce un estado de baja disponibilidad energética (del inglés, *Low Energy Availability*, LEA), que puede alterar la función endocrina, el metabolismo y la capacidad de recuperación. Tradicionalmente, LEA se ha definido mediante un punto de corte fijo (<30 kcal/kg FFM/día); sin embargo, la evidencia reciente sugiere que las respuestas individuales a esta condición son variables y dependen de múltiples factores fisiológicos, metabólicos y conductuales.

El objetivo general de esta tesis doctoral fue analizar los determinantes fisiológicos, metabólicos y conductuales de LEA en deportistas élite y pre-élite, considerando sus posibles interacciones y respuestas individuales. Para ello, se adoptó un enfoque integrador basado en la evaluación de la composición corporal, el metabolismo en reposo, así como los patrones de actividad física y sedentarismo.

En relación con la composición corporal, se analizó la relación entre la composición corporal y los síntomas de déficit de testosterona (ADAM-Q) en hombres y el riesgo de Triada de la Atleta Femenina (LEAF-Q) en mujeres. En varones, la grasa corporal no se asoció a síntomas de déficit de testosterona, sin embargo, aquellos que reportaron síntomas presentaron valores mayores de masa libre de grasa y circunferencias. Por otro lado, en mujeres se observó que la puntuación total del LEAF-Q no se asoció con ninguno de los parámetros de composición corporal; no obstante, el porcentaje graso y la grasa visceral se correlacionaron positivamente con la subescala de lesiones del LEAF-Q.

Asimismo, se analizó la asociación entre la disponibilidad energética, la tasa metabólica en reposo (RMR) y el cociente RMR medido/predicho (RMRratio), así como la relación entre el RMRratio con variables subjetivas de bienestar y recuperación. Aunque no se observaron asociaciones significativas entre la EA y RMR o RMRratio, los deportistas con menor gasto en reposo presentaron una peor percepción subjetiva de bienestar.

Finalmente, se evaluó la relación entre patrones de actividad física y sedentarismo sobre la EA y la salud percibida. Los resultados mostraron que ninguna de las variables de actividad física se asoció de manera significativa con la EA. Este resultado se mantuvo al redistribuir el tiempo entre los diferentes tipos de actividad física. Sin embargo, se observó que niveles más elevados de sedentarismo se relacionaron con más síntomas menstruales e indicadores de peor bienestar, mientras que actividades de baja intensidad se asociaron con mejor sintomatología menstrual.

En conjunto, los hallazgos de esta tesis abogan por una reformulación de cómo se conceptualiza, detecta y gestiona la LEA. La naturaleza de la LEA exige una evaluación que integre medidas fisiológicas objetivas con indicadores subjetivos, perfiles conductuales y una contextualización específica del deporte. Esta tesis sostiene que ninguna variable, examinada de forma aislada, es suficiente para caracterizar el estado energético de los atletas ni para predecir la presencia de síntomas. En su lugar, estos marcadores ofrecen información complementaria, aunque incompleta.

Palabras clave

Disponibilidad energética; baja disponibilidad energética; deficiencia energética relativa en el deporte (REDs); actividad física; atleta; tasa metabólica en reposo; ratio de la tasa metabólica en reposo (RMRratio); composición corporal; LEAF-Q; LEAM-Q.

ABSTRACT

Energy availability (EA) is a key factor for maintaining athletes' health, performance, and adaptive capacity. EA is defined as the amount of energy remaining to sustain essential physiological functions after subtracting the energy expended during exercise. When EA is insufficient to support these basic functions, a state of low energy availability (LEA) arises, which may compromise endocrine function, metabolism, and recovery capacity. Traditionally, LEA has been defined using a fixed threshold (<30 kcal/kg FFM/day); however, recent evidence suggests that individual responses to this condition are highly variable and influenced by multiple physiological, metabolic, and behavioural factors.

The main aim of this doctoral thesis was to examine the physiological, metabolic, and behavioural determinants of LEA in elite and pre-elite athletes, considering their potential interactions and individual variability. To this end, an integrative approach was adopted, based on the assessment of body composition, resting metabolism, free-living physical activity and sedentary patterns, and validated symptom-based screening tools.

Regarding body composition, its association with symptoms of testosterone deficiency in men and with the risk of the Female Athlete Triad in women was examined. In male athletes, body fat was not associated with symptoms of testosterone deficiency; however, those who exhibited higher values of fat-free mass had higher risk of suffering symptoms. In female athletes, the total LEAF-Q score did not correlate with any body composition parameters; nevertheless, body fat percentage and visceral adipose tissue were positively associated with the LEAF-Q injury subscale.

The relationship between EA, resting metabolic rate (RMR), and the measured-to-predicted (RMRratio) was also examined, together with their association with subjective indicators of well-being and recovery. Although no significant associations were observed between EA and either RMR or RMRratio, athletes with lower RMRratio reported poorer subjective perceptions of well-being.

Finally, the relationship between physical activity and sedentary patterns with EA and perceived health was evaluated. The results showed that none of the physical activity variables were significantly associated with EA, and this finding

remained consistent when reallocating time across different activity intensities. However, higher levels of sedentary time were related to greater menstrual symptoms and poorer well-being indicators, whereas low-intensity ambulatory activity was associated with better menstrual symptomatology.

Taken together, the findings of this thesis advocate for a reformulation of how LEA is conceptualised, detected, and managed. The multi-systemic nature of LEA, demands an assessment paradigm that integrates objective physiological measures with subjective indicators, behavioural profiling, and sport-specific contextualisation. This thesis advocates that no single variable, when examined in isolation, is sufficient to characterise the energetic status of athletes or to predict the presence of symptoms. Instead, these markers offer complementary yet incomplete insights.

Keywords

Energy availability; low energy availability; relative energy deficiency in sport (REDs); physical activity; athlete; resting metabolic rate; RMRratio; body composition; LEAF-Q, LEAM-Q

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"Fill your heart with better memories, better actions, better friends. That's all any of us can do". Wolverine, X-23 #10 (2010)

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ADAM-Q , Androgen Deficiency in Aging Males Questionnaire
ASPS , Athlete's Subjective Performance Scale
BIA , Bioelectrical Impedance Analysis
BMC , Bone Mineral Content
BMD , Bone Mineral Density
DXA , Dual-energy X-ray Absorptiometry
EA , Energy Availability (Disponibilidad Energética)
EAT-26 , Eating Attitudes Test-26
EEE , Exercise Energy Expenditure
EI , Energy Intake (Ingesta Energética)
FFM , Fat-Free Mass
FHA , Functional Hypothalamic Amenorrhea
FM , Fat Mass
FMI , Fat Mass Index
FSH , Follicle Stimulating Hormone
HPG axis , Hypothalamic–Pituitary–Gonadal axis
IGF-1 , Insulin-like Growth Factor 1
LPA , Light Physical Activity
LEA , Low Energy Availability (Baja Disponibilidad Energética)
LEAF-Q , Low Energy Availability in Females Questionnaire
LEAM-Q , Low Energy Availability in Males Questionnaire
LH , Luteinizing Hormone
LM / LMI , Lean Mass / Lean Mass Index
MPA , Moderate Physical Activity
MVPA , Moderate to Vigorous Physical Activity
NEAT , Non-Exercise Activity Thermogenesis

NUKYA, Nutrition Knowledge for Young Athletes

PSQI, Pittsburgh Sleep Quality Index

REDs, Relative Energy Deficiency in Sport

REE, Resting Energy Expenditure

RMR, Resting Metabolic Rate

RMRratio, Resting Metabolic Rate ratio

RESTQ-76, Recovery-Stress Questionnaire for Athletes

SRS, Silhouette Rating Scale

SHBG, Sex Hormone–Binding Globulin

T3, Triiodothyronine

T4, Thyroxine

TDEE, Total Daily Energy Expenditure

VAT, Visceral Adipose Tissue

VPA, Vigorous Physical Activity

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I – INTRODUCTION

I - INTRODUCTION

Meeting the energetic demands of intense training presents a constant challenge for athletes and is a key determinant of both health and performance (1). When this balance is disrupted, even temporarily, adaptive mechanisms are triggered to conserve energy, often at the expense of endocrine, metabolic, and skeletal muscle function(2,3). To characterize these physiological interactions, Loucks introduced the concept of energy availability (EA), defined as the amount of dietary energy remaining for essential physiological processes after accounting for the energetic cost of exercise (4). Expressed relative to fat-free mass (FFM), EA reflects the energy accessible to sustain metabolic, hormonal, and cellular functions rather than simply describing total caloric intake or expenditure (5).

Although closely related, EA and energy balance represent distinct constructs. Energy balance describes the long-term relationship between energy intake (EI) and total energy expenditure (TDEE), typically inferred from long-term changes in body mass(6,7). In contrast, EA reflects energetic sufficiency, indicating whether the EI adequately supports ongoing biological functions once exercise demands are met (6). Consequently, an athlete may maintain a stable body weight while experiencing substantial hormonal and metabolic disturbances due to insufficient EA. Loucks and Thuma demonstrated this distinction by showing that reducing EA from 30 to 20kcal/kg FFM/day suppressed luteinizing hormone (LH) pulsatility, a sign of energy inadequacy, despite no significant differences in body weight between conditions (8). Such distinction is particularly relevant in sports emphasizing high training volumes or leanness, where the margin for maintaining energy equilibrium is narrow (9). Under these conditions, dietary intake may fail to fully meet the energetic demands of training, leading to periods of low energy availability (LEA) (10). LEA is typically defined as an EA <30kcal/kg FFM/day, a threshold below which compensatory endocrine and metabolic adaptations may occur (7,11). These alterations can compromise both the athlete's health and performance over time by disrupting recovery, adaptation, and overall training capacity (12).

In parallel to endocrine and metabolic considerations, athletes frequently pursue body composition (BC) optimisation to enhance performance, meet aesthetic expectations, or satisfy weight-category demands. BC refers to the proportion and distribution of adipose tissue (e.g., total fat mass, visceral adipose tissue), lean tissue (e.g., FFM, muscle mass), and bone structure (e.g., bone mineral density, bone mineral content). In athletic populations, the balance between fat mass (FM) and lean tissue is a key determinant of health status and performance, influencing metabolic regulation, cardiovascular efficiency, and musculoskeletal function (13–17). Consequently, athletes often seek to reduce FM to improve power-to-weight ratio, endurance capacity, and sport-specific performance outcomes (18,19). However, inappropriate reductions in FM or FFM can impair short-term performance and long-term health, including increased injury risk and compromised musculoskeletal integrity (20–24).

In male athletes, aggressive reductions in FM and/or chronic dietary restriction may inadvertently promote sustained energy deficiency and endocrine disruption, including reductions in testosterone, with downstream implications for fatigue, recovery, performance, and bone health (25–28). In female athletes, persistent LEA constitutes the central aetiological factor of the Female Athlete Triad: LEA (with or without disordered eating), menstrual dysfunction, and impaired bone health, where hormonal disturbances increase the risk of stress fractures and compromise long-term skeletal integrity (29,30). Despite these conceptual links, the specific relationship between BC and the emergence of hypogonadal symptomatology in men or Triad-related features in women remains insufficiently characterised, underscoring the need to interpret physique-related metrics cautiously and within a broader clinical and behavioural framework (31,32). In both male and female athletes, these conditions appear to arise not from body composition per se, but from the interaction between high training loads, sustained energy restriction, and sociocultural pressures that shape dietary and training behaviours. Accordingly, the development of LEA commonly reflects a complex interplay between intensive training demands, inadequate EI, and reinforcing beliefs such as carbohydrate avoidance or the pursuit of extreme leanness as a performance strategy (33,34).

Accurate quantification of EA in real-world athletic settings remains challenging, largely due to the inherent complexity of measuring both dietary intake and exercise energy expenditure (EEE) outside controlled laboratory conditions (35). Given this multifactorial and behaviourally driven aetiology, accurately quantifying EA in applied athletic contexts is particularly challenging.

1.1. CONCEPTUAL FUNDAMENTALS OF ENERGY AVAILABILITY

Early research on EA aimed to explain why amenorrheic athletes exhibited alterations in reproductive and stress-related hormones compared with eumenorrheic athletes and sedentary women (36). Initial observational investigations into the health consequences of intensive training revealed consistent associations between disordered eating (DE), menstrual dysfunction, and impaired bone health in athletes (36–39).

Researchers subsequently questioned whether these disturbances resulted from the physical stress of exercise itself or from an energetic imbalance between EI and expenditure (40). Pioneering experimental studies by Professor Anne Loucks and colleagues demonstrated that restricting EI relative to EEE suppressed metabolic and reproductive hormones, reduced bone formation, and increased bone resorption (4,41,42). This body of evidence established that it was insufficient EA, rather than exercise stress alone, that triggered these physiological adaptations.

In 1992, the American College of Sports Medicine published the first Position Stand on the Female Athlete Triad (43), defining the syndrome as the coexistence of three interrelated conditions: DE, amenorrhea, and osteoporosis. However, dissatisfaction soon emerged because this model captured only the most severe clinical manifestations. De Souza and Williams later proposed a broader, spectrum-based model that recognized subclinical presentations across all three components (44). This perspective redefined the Triad as a continuum of conditions characterized by LEA (with or without DE), functional hypothalamic amenorrhea (FHA), and osteoporosis (45). However, it was later recognized that not all diagnostic criteria needed to be present, given substantial variability by sport type, highlighting the potential for underdiagnosis (46). To address these limitations, the International Olympic Committee (IOC) expanded the concept into Relative Energy Deficiency in Sport (REDs) in 2014 (29). This framework emphasized LEA

as a central factor underlying disruptions in multiple physiological systems, including reproductive, bone, endocrine, metabolic, hematological, growth and development, cardiovascular, gastrointestinal, and immune functions, ultimately affecting both athletic performance and overall health. Subsequent IOC consensus statements in 2018 (47) and 2023 (48) further refined the REDs model and its clinical applications, gaining increasing attention in both sport science and clinical practice.

Despite its widespread adoption, the scientific basis and clinical foundations of REDs remain subjects of debate. Jeukendrup and colleagues have argued that REDs should be regarded primarily as a theoretical construct rather than an established clinical syndrome (49). They highlight substantial limitations in accurately assessing LEA, the central construct of the model, due to methodological errors in estimating EI, EEE, and heterogeneous responses among athletes. They further contend that the signs and symptoms attributed to REDs (i.e., menstrual disturbances, osteoporosis, impaired immunity, and reduced performance) are nonspecific and can result from diverse causes, including psychological stress, sleep deprivation, illness, or injury. From this perspective, the current evidence base is largely correlational and insufficient to demonstrate causality. The authors caution that diagnosing REDs by exclusion may introduce confirmation bias, and thereby advocate for a broader, multifactorial integration of health determinants rather than focusing narrowly on LEA (i.e., Athlete Health and Readiness Checklist) (50).

Conversely, Mountjoy and colleagues maintain that REDs is a valid clinical syndrome, drawing upon extensive observational data, accumulated clinical experience, and the use of validated diagnostic tools such as the IOC REDs CAT2 (51). They argue that while controlled long-term trials are ethically unfeasible, the collective evidence supports the existence of REDs as a syndrome with tangible health and performance consequences. Moreover, clinicians across multiple disciplines routinely diagnose and manage REDs in elite athletes, underscoring their practical relevance. Finally, Mountjoy and colleagues emphasize that debate should be focused not on dismantling, but refining the model through the development of physiological submodels, enhanced education, and interdisciplinary collaboration.

1.2. SCREENING TOOLS

Accurately identifying athletes at risk of LEA and its associated health consequences remains a major challenge in both research and clinical practice. Direct quantification of EA requires precise measurement of EI, EEE, and body composition. This approach is rarely feasible outside controlled laboratory conditions. Consequently, several screening questionnaires have been developed as practical, cost-effective tools to detect early signs and symptoms suggestive of LEA.

1.2.1. Low Energy Availability in Females Questionnaire

The Low Energy Availability in Females Questionnaire (LEAF-Q), developed by Melin and colleagues in 2014, was the first validated instrument specifically designed to screen for LEA and the Female Athlete Triad in women. This tool was originally developed for endurance athletes and dancers, populations known for their susceptibility to energy deficiency (52,53). It consists of 25 items covering three main domains: menstrual function, gastrointestinal symptoms, and injury history. A total score of ≥ 8 classifies athletes as “at risk”. This questionnaire has been referenced in the IOC REDs CAT2 as a recommended screening instrument for identifying athletes requiring further evaluation (48).

Despite its widespread use, the LEAF-Q exhibits notable limitations. As it was originally developed for endurance athletes, its capacity to detect LEA-related symptoms in team, aesthetic, or strength-based disciplines remains uncertain, which may explain the variable diagnostic accuracy reported across sports and competitive levels (54,55). Besides, the questionnaire may also produce false-positive results when symptoms stem from conditions unrelated to energy deficiency, such as gastrointestinal disorders or polycystic ovary syndrome (56,57). Conversely, false negatives may occur in athletes using oral contraceptives, which may mask menstrual disturbances and obscure one of the most sensitive indicators of LEA (58–60). Thus, LEAF-Q results should always be interpreted in conjunction with clinical assessment, training history, and hormonal evaluation, rather than used in a stand-alone screening tool.

1.2.2. Androgen Deficiency in Aging Males Questionnaire

Before the development of male-specific screening instruments, the Androgen Deficiency in Aging Males Questionnaire (ADAM-Q) was frequently adopted in sports research as a proxy to detect symptoms related to androgen deficiency and potential LEA in male athletes (61,62). Originally developed by Morley and colleagues in 2000 for clinical use in middle-aged men, the ADAM-Q includes ten yes/no items addressing sexual function, mood, concentration, fatigue, and perceived physical performance (63). A positive result on the ADAM-Q is obtained if the participant answers “yes” to either of the two key questions regarding libido or erectile strength, or to three or more of the remaining items.

However, this questionnaire was not designed for athletic populations, and its applicability to sports settings is limited. While it demonstrates high sensitivity for identifying clinical hypogonadism, its specificity is low, often overestimating androgen deficiency prevalence in middle-aged adults. In its original validation, 37 % of men with normal bioavailable testosterone met the criteria for “androgen deficiency”, suggesting that the questionnaire may capture nonspecific manifestations such as fatigue, low mood, or reduced vitality that are not necessarily related to endocrine dysfunction. This lack of specificity limits its usefulness in sport, where those symptoms commonly reflect intensive training and energy imbalance rather than true hypogonadism. Furthermore, the questionnaire has not been validated against hormonal or metabolic markers of LEA in athletes, and its cut-off scores are unlikely to reflect the natural hormonal fluctuations observed in training populations. Consequently, the ADAM-Q should be interpreted with caution when applied in sports contexts and is best viewed as a general symptom checklist rather than a validated indicator of LEA.

1.2.3. Low Energy Availability in Males Questionnaire

Recognizing the previous gap, the Low Energy Availability in Males Questionnaire (LEAM-Q) was recently developed as the first male-specific instrument for screening LEA in male athletes. The questionnaire was initially designed based on expert consensus and existing LEA frameworks, and later validated in a cohort of more than 300 elite and sub-elite male athletes, primarily

from endurance and weight-sensitive sports (64). Item selection was informed by expert consensus and by validated questionnaires addressing physiological domains potentially affected by chronic energy deficiency, including fatigue, illness, dizziness, gastrointestinal function, and libido (64).

Early applications of the LEAM-Q have revealed promising associations with behavioural and psychological correlates of LEA (58,65). For example, male athletes classified as “at risk” frequently presented co-existing features of disordered eating, compulsive exercise behavior, and body-image dissatisfaction (58). Interestingly, LEAM-Q scores were not correlated with blood testosterone concentrations, suggesting that isolated hormonal measures may be insufficient to validate questionnaires (58).

As with its female counterpart, the LEAM-Q was validated in endurance and weight-sensitive sports (64). Therefore, its performance in team or power-based disciplines, where physiological adaptations and symptom presentation may differ, remains uncertain (66).

In summary, the LEAM-Q represents a meaningful advance toward sex-specific screening for LEA, offering a practical tool for early detection in male athletes. Future research should focus on cross validating the questionnaire across different sports and competitive levels, defining a reference threshold, and integrating it with objective biomarkers to strengthen its research and clinical utility.

1.3. METABOLIC AND PHYSIOLOGICAL MARKERS OF LOW ENERGY AVAILABILITY

Over the past two decades, research has increasingly examined the physiological mechanisms through which LEA (especially $<30\text{kcal/kg FFM/day}$) affects hormonal regulation and metabolic function in athletes. These endocrine and metabolic adaptations not only indicate energy stress but also directly influence recovery, performance, and adaptations in response to training (67,68). Among the most vulnerable systems to LEA are the thyroid, reproductive, and metabolic axes (69,70), with alterations that may persist throughout competitive seasons (71,72). Given their consistency across sports, and experimental conditions,

these markers have emerged as practical indicators of LEA in applied athletic settings (73).

1.3.1. Hypothalamic-Pituitary-Thyroid Axis and low energy availability

Alterations in the thyroid axis represent one of the most reproducible endocrine responses to LEA, particularly evident in the suppression of circulating triiodothyronine (T_3) concentrations (7). As a central regulator of resting metabolic rate (RMR), mitochondrial function, and thermogenesis (74,75), T_3 serves as a sensitive integrator of energetic status.

Loucks et al. demonstrated that circulating T_3 levels begin to decline when EA acutely drops to between 19.0 and 25.0 kcal/kg FFM/day, even though thyroxine (T_4) remains unchanged (76). Notably, when EA was ≤ 10 kcal/kg FFM/day, T_4 levels rose slightly, potentially reflecting a compensatory reduction in T_4 -to- T_3 conversion as part of an energy-conserving adaptation. These findings align with other controlled trials showing that caloric restriction typically induces a 10–25% decrease in circulating T_3 , whereas T_4 concentrations tend to remain stable (77–79). Cross-sectional research further supports these observations, reporting lower T_3 levels both in female athletes with FHA and male endurance athletes chronically exposed to energy deficits (36,52,80–82).

Collectively, these data suggest that reductions in T_3 are among the earliest and most reliable endocrine markers of LEA. Concentrations tend to remain relatively stable in the short term; however, prolonged or severe energy deficiency can eventually lead to a gradual decline in T_4 , further amplifying the suppression of metabolic rate (8).

These alterations indicate that the thyroid axis undergoes a progressive, energy-conserving adjustment. This downregulation represents an adaptive physiological mechanism that lowers basal energy expenditure and supports metabolic homeostasis during sustained energy restriction

1.3.2. Hypothalamus-Hypophysis-Gonadal Axis and low energy availability

The hypothalamic–pituitary–gonadal (HPG) axis plays a central role in coordinating reproductive and metabolic homeostasis. Under LEA conditions, pulsatile gonadotropin-releasing hormone (GnRH) secretion is suppressed, leading to reduced LH and follicle-stimulating hormone (FSH) release, with subsequent decreases in sex steroid production (estradiol in women and testosterone in men) (83). This endocrine response reflects an adaptive mechanism that reallocates limited energy resources toward essential short-term metabolic processes at the expense of nonessential functions, such as reproduction (84–86).

1.3.2.1. *Functional Hypothalamic Amenorrhea (FHA)*

In female athletes, LEA disrupts normal GnRH pulsatility, causing downstream ovarian suppression (87). Classic studies by Loucks and colleagues demonstrated that EA <30kcal/kg FFM/day decreases LH pulsatility, a sign of hypothalamic suppression (8). However, Lieberman and colleagues reported that menstrual disturbances such as luteal phase defects, anovulation, and oligomenorrhea occur across a continuum of EA values, with each 1 kcal/kg FFM/day reduction increasing the odds of menstrual dysfunction by ~9% (88). This continuum underscores substantial interindividual variability in sensitivity to energetic stress and highlights that reproductive suppression may occur even at EA levels traditionally considered “safe” (89,90).

Beyond reproductive disruption, FHA is closely linked to bone health deterioration. Chronic hypoestrogenism reduces bone formation and increases resorption, predisposing athletes to low bone mineral density (BMD) and stress fractures (91–97). Longitudinal studies show that women with FHA have up to 4.5-fold greater bone injury risk than eumenorrheic athletes, while adolescent female athletes with LEA exhibit compromised peak BMD (98–100).

LEA and FHA are increasingly recognized for their negative impact on athletic performance, neuromuscular recovery, and psychological well-being (52,101,102). Reduced T_3 , suppressed estradiol, and elevated cortisol could affect metabolic efficiency, muscle protein synthesis, and tissue repair, ultimately blunting training adaptations and delaying recovery (103–106). Amenorrheic endurance athletes show lower muscle strength and power than eumenorrheic

peers (107), while chronically energy-deficient athletes often experience fatigue, low mood, and sleep disturbances driven by neuroendocrine stress and altered serotonergic activity (22,26). Together, these findings highlight that LEA and FHA compromise both the physiological and psychological aspects of performance.

1.3.2.2. *Male Hypogonadism*

Reproductive suppression in men manifests differently from that seen in women. Whereas menstrual disruption provides a potential clinical sign of energy deficiency in women, men often exhibit no overt symptoms, despite measurable alterations in the HPG axis. Chronic LEA leads to a subtle but sustained reduction in GnRH pulsatility and LH secretion, resulting in lower testicular steroidogenesis and decreased circulating testosterone levels (26).

This condition, known as “*Exercise Hypogonadal Male Condition (EHMC)*”, is now recognized within the broader REDs framework (28). Rather than reflecting a pathological failure of the gonads, EHMC represents a functional, reversible downregulation of the HPG axis (108–111). This suppression can develop rapidly; short-term fasting and caloric restriction have been found to decrease LH pulsatility within 48 hours, underscoring the acute sensitivity of the male reproductive axis to energetic deficit (112). Additionally, evidence from endurance athletes and military cohorts indicates that hypogonadotropic hypogonadism induced by LEA is largely reversible once adequate EA is restored (109,113). These adaptations represent a coordinated energy-conserving response that blunts skeletal muscle protein synthesis, intracellular anabolic signaling, and bone formation and remodeling. Such alterations compromise tissue repair and recovery, ultimately heightening the risk of injury, stress fractures, and performance decline (103,114–116).

The endocrine suppression associated with LEA exerts wider systemic effects that extend beyond the musculoskeletal system. Long-term neuroendocrine alterations (including elevated cortisol and reduced anabolic hormones) can negatively affect mood, motivation, and sleep regulation, contributing to fatigue, irritability, and depressive symptoms frequently reported in REDs contexts (117–120).

In summary, exercise-induced hypogonadism represents a continuum of energy-dependent adaptations ranging from transient suppression to chronic recalibration of the HPG axis. Early recognition is essential not only to restore hormonal function but also to prevent the cumulative consequences of prolonged LEA on bone integrity, psychological well-being, and overall athletic performance.

1.3.3. Metabolic Adaptations to Low Energy Availability

LEA also disrupts the broader neuroendocrine network responsible for sensing and communicating energetic status. Leptin, insulin, and IGF-1 are pivotal hormones linking EI, BC, and metabolic function. Their collective response reflects an orchestrated regulatory mechanism through which the body prioritizes survival and energy conservation overgrowth and performance.

1.3.3.1. *Leptin*

Leptin acts as a key signal linking peripheral energy stores to central neuroendocrine regulation. It reflects both long-term fat stores and short-term fluctuations in EA (121–123), and its circulating levels decline proportionally to the energy deficit, independently of body mass changes (124,125). By modulating hypothalamic control of GnRH pulsatility, leptin influences reproductive regulation (126); reduced leptin during sustained energy deficiency contributes to hypothalamic downregulation and the reproductive suppression characteristic of REDs (48).

Leptin levels differ by sex, with women consistently exhibiting higher concentrations than men for a given fat mass. This difference is largely explained by greater overall adiposity and estrogen-driven leptin synthesis (127,128). Such sex-specific regulation may help explain the greater susceptibility of female athletes to FHA (48). Laughlin and Yen reported that both eumenorrheic and amenorrheic endurance athletes displayed approximately threefold lower 24-hour serum leptin concentrations than sedentary controls, although only eumenorrheic athletes maintained a normal diurnal rhythm of leptin secretion (129).

Experimental evidence further supports leptin's causal role in reproductive restoration. Administration of recombinant leptin in women with FHA has been

shown to restore gonadotropin secretion, increase estradiol concentrations, and reinstate menstrual function (130). These findings identify leptin as a permissive signal of energy sufficiency, coordinating metabolic and reproductive axes in response to nutritional status.

1.3.3.2. *Insulin and Hepatic-Endocrine Adaptations to Low Energy Availability*

LEA is consistently associated with suppressed fasting insulin and a broader shift toward an energy-conserving endocrine profile in female athletes (131). Comparable endocrine adaptations have also been observed in healthy men exposed to controlled energy restriction. In a military cohort subjected to a severe caloric deficit of approximately 1,200 kcal/day, accompanied by high physical demands, participants developed a transient, subclinical hypogonadism characterized by reductions in insulin, T_3 , insulin-like growth factor 1 (IGF-1), and testosterone. These alterations returned to baseline after a period of controlled refeeding, confirming that the endocrine suppression was primarily driven by the energy deficit rather than by exercise stress alone (113).

Reduced insulin levels during LEA also promote hepatic upregulation of sex hormone-binding globulin (SHBG), thereby reducing free testosterone without altering total testosterone (132). These changes likely reflect an adaptive endocrine response to LEA rather than a purely pathological dysfunction. Insulin further regulates substrate partitioning by stimulating glucose uptake via GLUT4 translocation and activating glycogen synthase (133,134). When insulin remains chronically low, glycogen resynthesis after exercise is impaired, shifting metabolism toward greater fat oxidation and reducing the capacity for high-intensity, glycolytic performance (135,136).

Although women oxidize proportionally less carbohydrate and more fat at comparable exercise intensities, maintaining adequate glycogen stores remains essential for optimal performance. Walker et al. (2000) demonstrated that carbohydrate loading increases muscle glycogen content and prolongs time to fatigue in well-trained female athletes, underscoring the importance of glycogen availability for both sexes (137). From a practical standpoint, the combination of low energy and low carbohydrate availability increases the likelihood of beginning

subsequent training sessions with suboptimal glycogen stores, thereby amplifying typical symptoms of energy deficiency such as early fatigue, slower recovery, and reduced training quality (138).

1.3.3.3. *RMR and RMR Ratio*

RMR constitutes the largest component of total daily energy expenditure and represents the energy needed to sustain vital physiological functions at rest. RMR is highly responsive to energetic status, and in athletes, LEA can induce a reduction in RMR that conserves energy during chronic underfuelling, but further exacerbates the overall energetic deficit (3,139). Therefore, the assessment of RMR through indirect calorimetry has been proposed as a marker of an athlete's energy status. Persistently low RMR values may reflect the physiological adaptations associated with REDs, including hormonal disturbances, impaired bone metabolism, and blunted training adaptations (10,67).

To enhance interpretability, researchers have proposed the RMR ratio (RMRratio), defined as *measured RMR divided by predicted RMR*. Values below 0.90 are typically interpreted as indicative of metabolic suppression and are often used as a surrogate marker of chronic LEA (140,141). Nevertheless, evidence remains inconsistent. Some studies have reported that LEA is associated with a reduced RMRratio (140,142), while others have found no significant relationship between these variables (143,144). Indeed, some athletes exhibit suppressed RMR despite apparently adequate EI, whereas others with confirmed LEA show minimal metabolic downregulation. These findings suggest that metabolic suppression does not occur uniformly among athletes with LEA and that reduced RMR or RMRratio reflects a physiological adaptation to energetic stress rather than a direct indicator of EA.

Moreover, the practical classification of “metabolic suppression” is highly sensitive to the predictive equation used to estimate RMR, and recent consensus statements emphasise that RMR-derived indices should not be interpreted as standalone indicators of LEA but rather contextualised alongside clinical history, symptom profiles, and complementary physiological markers (48,141).

In summary, LEA triggers a cascade of hormonal, metabolic, and physiological adaptations aimed at conserving energy and maintaining

homeostasis. Although such changes reflect a coordinated, adaptive response to energy deficiency, prolonged or recurrent exposure can compromise bone health, recovery capacity, and overall performance. The magnitude and pattern of these responses differ among individuals, sports, and training conditions, supporting the notion that LEA represents a continuum of physiological adaptations rather than a fixed state. **Figure 1** illustrates the main endocrine and systemic responses associated with LEA, summarising the key hormonal alterations and their potential impact on multiple organ systems.

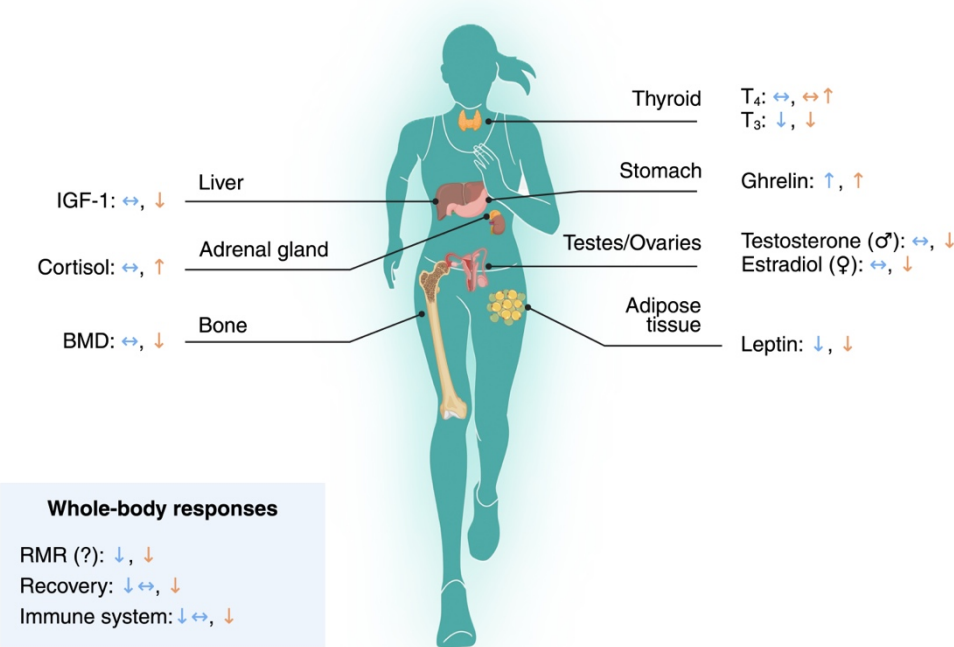


Figure 1. Schematic representation of the endocrine and physiological adaptations to LEA. LEA affects multiple systems, leading to a coordinated downregulation of endocrine and metabolic functions aimed at conserving energy. Blue arrows represent acute changes, while red arrows indicate chronic adaptations that may emerge with sustained energy deficiency. Abbreviations: T₄, thyroxine; T₃, triiodothyronine; IGF-1, insulin-like growth factor 1; BMD, bone mineral density; RMR, resting metabolic rate.

1.4. METHODOLOGICAL CHALLENGES IN THE ASSESSMENT OF ENERGY AVAILABILITY

The quantification of EA remains one of the major methodological challenges in this field. Accurate measurement requires the accurate assessment of multiple dynamic components (EI, EEE, and FFM), which are subject to substantial error and interindividual variability. Consequently, inconsistencies in measurement protocols and settings (e.g., controlled vs. free-living settings) have led to a lack of a single protocol to assess EA and to inconclusive findings that hinder the establishment of universally accepted diagnostic thresholds (**Figure 2**).



Figure 2. The accurate quantification of EA in athletes is limited by several methodological constraints.

These include the absence of a standardized assessment protocol, inaccuracies in the measurement of individual components of the EA equation (energy intake, exercise energy expenditure, and fat-free mass), and the inherent differences between controlled laboratory conditions and free-living athletic environments. Additionally, inconsistencies in defining the threshold for low energy availability (LEA) and the wide interindividual variability in physiological responses could affect the interpretation and comparison of findings across studies

In most studies, EI is estimated through indirect methods such as prospective dietary records, which rely on self-reported intake over several consecutive days (98,145). Although these methods are valid when properly implemented, their accuracy depends heavily on participant compliance and portion-size estimation. Factors such as underreporting, omission of snacks, and social desirability bias frequently lead to under- or overestimation of EI and, consequently, to inaccurate EA calculations (146). On average, self-reported food records typically underestimate EI by 5–21% compared to actual intake (147). As a result, findings from field studies using self-reported nutritional data are difficult to interpret and have not yielded consistent thresholds or clear associations between EA and objective indicators of energy conservation or health, such as alterations in metabolic hormones (98) or menstrual function (52).

Estimation of EEE presents similar challenges. Most studies rely on indirect methods, including accelerometry (148–150), heart rate monitors (151,152), or activity logs, such as the Bouchard Activity Record (153–155). These approaches are prone to error, as their accuracy depends on algorithmic assumptions and participant compliance (156). Although the doubly labeled water method provides an accurate measure of TDEE, it is rarely used in sports research because it cannot distinguish EEE from other activity components and is limited by high cost, logistical complexity, and reduced feasibility in free-living athletic settings. Consequently, there is currently no consensus on the most valid or standardized approach for quantifying EEE, leading to substantial variability and limiting the comparability of reported EA values across studies.

Another methodological limitation is the lack of assessment of physical activity performed outside structured training sessions. The classical EA equation was derived from controlled laboratory studies where physical activity remained unchanged (42,157), thereby not capturing the variability of real-world athletic environments. In free-living settings, a meaningful proportion of TDEE arises from non-exercise activity thermogenesis (NEAT) (158), which encompasses unstructured and incidental movements such as walking, standing, fidgeting, and occupational or household tasks. NEAT can represent a variable yet substantial component of total TDEE (158), but it is seldom quantified in applied research.

Neglecting this component may lead to systematic inaccuracies in LEA estimation and obscure interindividual variability in physiological adaptations.

Importantly, TDEE in athletes is not solely determined by structured training, but also by how time is distributed across movement behaviours throughout the day. Beyond NEAT, TDEE is shaped by the distribution of 24-hour movement behaviours, including sedentary behaviour, light physical activity, and sleep (159–162). NEAT itself varies markedly between individuals and may contribute substantially to daily energy expenditure (162,163). In athletic cohorts assessed under free-living conditions, non-training activity patterns have been characterised and shown to fluctuate depending on lifestyle and recovery (163). Moreover, some evidence suggests that athletes may increase sedentary time during periods of high training load as a compensatory strategy to conserve energy (164,165), and prolonged sedentary behaviour may adversely influence recovery-related processes (165). However, experimental work indicates that short-term exposure to LEA does not necessarily induce an immediate reduction in NEAT, underscoring the complexity of behavioural compensation in energy-deficient states (166). Despite these insights, relatively few studies have examined how reallocation between movement behaviours within the 24-hour continuum interacts with EA in free-living athletic populations, limiting ecological inference and highlighting the need for integrated monitoring approaches (167).

Because EA is typically normalized to FFM, accurate BC assessment is also essential. Dual-energy X-ray absorptiometry (DXA) is commonly the most used method for assessing body composition and FFM in athletes. However, alternative techniques such as bioelectrical impedance analysis (BIA) (150) or skinfold measurements (149,155) are frequently employed in field settings due to their accessibility and lower cost. These methods are not interchangeable, and there is no consensus on the optimal approach. Compared with DXA, BIA, and skinfold methods typically underestimate fat mass and overestimate lean mass, introducing systematic bias in FFM assessment (168–170). This methodological variability further limits the comparability of EA values across studies and complicates the interpretation of findings across studies.

1.5. FROM RESEARCH TO PRACTICE

Translating the concept of LEA and REDs from theoretical models into applied practice requires an integrated, comprehensive approach to early detection and prevention. Since no single biomarker or diagnostic criterion can reliably identify LEA, effective screening should combine clinical, physiological, and behavioural indicators within a coordinated framework.

Although no universal protocol exists for identifying LEA or REDs, current consensus statements recommend an initial evaluation that includes a detailed clinical assessment encompassing dietary habits, menstrual history, training patterns, injury profile, fatigue, and recent fluctuations in performance or body weight (48,171,172). Validated self-report instruments, such as the LEAF-Q (173), REDs Specific Screening Tool (174), and the REDs CAT2(175), provide practical means to identify athletes at risk. However, these tools should be considered as screening rather than diagnostic instruments, given their reliance on self-report and variable psychometric validation.

The most effective approach integrates subjective questionnaires with objective physiological and performance indicators, including RMR, hormonal profiling, BMD, and longitudinal monitoring of recovery and training load (48,171,172). Such multimodal strategies acknowledge that LEA-related manifestations often emerge gradually and heterogeneously, and that reliance on single-point assessments may fail to capture early or subclinical presentations. This integrated framework may enhance sensitivity and specificity and enable earlier identification of athletes at risk before overt clinical consequences develop.

Importantly, timely recognition may prevent the progression from transient, adaptive responses to LEA toward chronic endocrine and metabolic suppression. Acute LEA may preserve relative hormonal and immune stability, whereas prolonged LEA leads to sustained reductions in triiodothyronine (Figure 3).

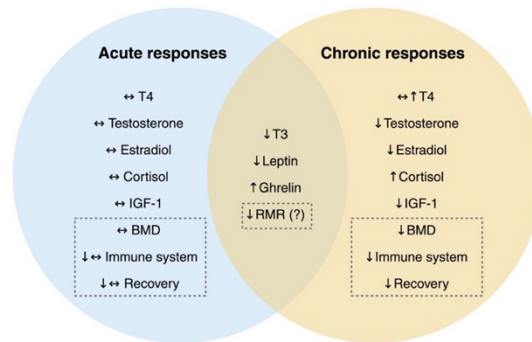


Figure 3. Overview of acute versus chronic physiological adaptations to LEA.

While acute LEA may maintain hormonal and immune stability, prolonged LEA leads to significant endocrine, metabolic, and immunological disruptions that compromise recovery and performance. Abbreviations: T4, thyroxine; T3, triiodothyronine; IGF-1, Insulin-like growth factor 1; RMR, resting metabolic rate; BMD, bone mineral density.

These physiological changes reinforce the importance of early detection and intervention to prevent the consolidation of chronic maladaptations. Preventing REDs extends beyond simply identifying energy deficiency; it requires a proactive, interdisciplinary approach embedded within the daily practice and culture of sport. Education remains the cornerstone of prevention, empowering athletes, coaches, and supporting staff with the knowledge to recognize, discuss, and respond to early signs of LEA. Yet, substantial knowledge gaps persist: less than half of the surveyed coaches (176–178) can identify the three components of the Female Athlete Triad, or are even aware of REDs. Although educational interventions targeting DE, nutrition, and REDs awareness have improved literacy and attitudes (179–181), these programs are often short-term and insufficiently integrated into sports organizations.

A sustainable prevention framework must extend beyond information delivery to foster environments that discourage weight-centered cultures and promote open communication about health and performance. Embedding routine medical screening, nutritional education, and recovery monitoring into daily

training can facilitate early recognition and timely intervention. Effective management also relies on multidisciplinary collaboration, with physicians, dietitians, psychologists, physiologists, and coaches contributing complementary expertise within an athlete-centered care (172).

In summary, although the concept LEA has emerged as one of the most interesting concepts in the sport science, important questions remain regarding its assessment, its variability across different sport disciplines, and the influence of behavioural and contextual factors. These uncertainties highlight the need for a broader, multidimensional perspective that integrates physiological, metabolic and free-living athletes. This gap in current knowledge provides the foundation for the justification and objectives of the present Doctoral Thesis.

II – JUSTIFICATION

II - JUSTIFICATION

LEA represents one of the main factors capable of compromising both the health and performance of athletes. Although its impact has been well documented, particularly in relation to hormonal disruptions, metabolic adaptations and methodological challenges continue to limit its accurate detection and effective management.

The commonly applied threshold of 30 kcal/Kg FFM/day although widely used as a diagnostic benchmark for identifying states of LEA, has come under increasing scrutiny in recent literature. Several studies have reported that athletes can present EA below this proposed threshold without manifesting the expected physiological or clinical markers typically associated with LEA. These findings challenge the universality and diagnostic validity of this criterion and suggest that additional contextual factors may modulate the expression of energy deficiency symptoms across individuals and sport disciplines.

A key factor contributing to this variability is the nature of the energetic demands imposed by certain sports. In numerous athletic disciplines, especially those that emphasize leanness or strict body composition targets for performance or aesthetic purposes, athletes are frequently exposed to a combination of elevated training volumes and sustained dietary restriction. Such practices often lead to prolonged energy mismatches that, although not always immediately symptomatic, may compromise physiological function over time. These subclinical states further complicate the early identification of LEA and hinder the implementation of preventive strategies.

Moreover, current estimations of EA are typically derived from the subtraction of exercise energy expenditure from dietary energy intake, offering only a partial view of an athlete's true energetic state. A major limitation of this model is its omission of NEAT, which encompasses all forms of unstructured, low-intensity physical activity such as walking, fidgeting, and occupational movement. Although NEAT is not currently factored into standard EA calculations, it may represent a substantial portion of total daily energy expenditure. In athletes with elevated NEAT, even modest underreporting of intake or overestimation of

training load may result in an underappreciated cumulative energy deficit, increasing their susceptibility to the physiological effects of LEA despite meeting conventional energy benchmarks.

In addition, most of the existing literature has been derived from endurance-based sports, leaving substantial gaps in the understanding of LEA in athletes from strength, power, and intermittent disciplines. Compounding this limitation is the relative scarcity of research addressing multidimensional factors such as psychological stress, recovery quality, disordered eating behaviors, and sleep-related disturbances, all of which may interact with energy status to influence both health outcomes and performance capacity.

To address these limitations, the present Doctoral Thesis proposes a multidimensional framework for the assessment of LEA. This model integrates objective physiological markers (including body composition indices, RMR, and objectively measured physical activity and sedentary behavior) with a suite of validated psychometric instruments evaluating perceived fatigue, sleep quality, eating attitudes, and psychological stress. Such an integrative approach allows for a more comprehensive characterization of the athlete's energetic and behavioural profile, supporting a reconceptualization of LEA as a dynamic and individualized continuum rather than a binary clinical state. The overall aim is to generate evidence that contributes to more precise, context-sensitive strategies for the early detection, prevention, and management of LEA in a broad range of sporting populations.

III – AIMS

III - AIMS

3.1. GENERAL AIM

The overall aim of this Doctoral Thesis is to examine the physiological, metabolic, and behavioural dimensions of LEA in competitive athletes, adopting an integrative perspective that reflects individual variability in adaptation and response.

Specifically, this work aims to explore how body composition, resting metabolism, and daily movement behaviors interact with EA, and how these factors collectively influence health-related outcomes in both male and female athletes.

3.2. SPECIFIC AIMS

Specific Aim I: To analyse the relationship between body composition and the risk of LEA in competitive athletes, using validated screening tools (LEAF-Q and ADAM-Q) to identify symptoms or physiological alterations associated with energy deficiency.

Specific Aim II: To examine the association between RMR, RMR ratio, and calculated EA, and to determine whether reductions in metabolic function reflect chronic energy deficiency and its potential effects on recovery, bone health, and well-being.

Specific Aim III: To evaluate the influence of daily movement behaviours, including physical activity, sedentary time, and NEAT, on calculated EA and health-related indicators such as menstrual function, fatigue, and perceived well-being in athletes under free-living conditions.

IV - METHODS

IV -METHODS

This Doctoral Thesis employed a cross-sectional observational design to examine the physiological, metabolic, and behavioural determinants of LEA in competitive athletes under free-living conditions.

4.1. ASSESSMENT OF BODY COMPOSITION AND LEA-RELATED SYMPTOMS

4.1.1. Study design and participants

This research was conducted as part of the PRECORED project, a cross-sectional initiative aimed at characterizing endocrine and metabolic health in competitive Spanish athletes.

A total of 71 athletes, consisting of 48 men and 23 women, were included in this cross-sectional study. Participants were recruited through non-probabilistic convenience sampling and represented a variety of sports disciplines, including swimming, cross-training, endurance-based sports, and mixed or intermittent modalities such as football or basketball. All the participants were competing at regional, national, or international levels. Their competitive level was verified based on validated criteria for classifying athletes, as described by McKay A et al(182) Participants were required to attend the Sport and Health University Research Center (iMUDS, University of Granada, Spain) on two separate occasions. During the initial visit, a preliminary meeting was conducted, during which the study procedures were thoroughly explained, and informed consent was obtained. Following this, a comprehensive assessment was carried out, including measurements of body composition, low testosterone symptoms in male athletes, and the risk for the Female Athlete Triad in female athletes.

The study protocol was reviewed and approved by the Human Research Ethics Committee of the "Junta de Andalucía" (Nº 0909-N-22). All participants were fully informed of the study procedures, and potential risks, and provided written consent prior to their participation. The inclusion criteria were as follows: (1) participants had to be between 18 and 35 years of age, (2) actively competing and federated in any sports discipline at the national level for at least 2 years, and

(3) capable of comprehending the instructions, objectives, and protocol of the study. Exclusion criteria included the following: (1) a history of a pathological event during physical exercise, (2) a history of major adverse cardiovascular events, renal insufficiency, cirrhosis, eating disorders, polycystic ovary syndrome, surgical intervention for weight control, type 2 diabetes mellitus, or HIV/AIDS, (3) any chronic pathology, (4) any condition that, in the judgment of the investigator, would impair the participant's ability to engage in the study or represent a personal risk to the participant, (5) the use of medications that could affect the study results, (6) pregnancy or breastfeeding, and (7) active tobacco or illicit drug use, or a history of treatment for alcohol abuse. All exclusion criteria were verified through a structured face-to-face interview conducted by qualified research staff.

Participants were instructed to abstain from intense physical activity for 24 hours before the assessments and to arrive at the center in a fasting state, having refrained from consuming alcohol or stimulants, to ensure the accuracy of the measurements.

4.1.2. Anthropometric and body composition measurements

Body mass was measured to the nearest 0.1 kg and stretch stature to the nearest 0.1 cm, using a model 799 scale and a stadiometer, respectively (Seca model 799, Electronic Column Scale, Hamburg, Germany), without shoes and with light clothing. BMI was calculated from weight and height (kg/m^2).

Body composition was assessed using three distinct methods: DXA, BIA, and anthropometry. These methods provided comprehensive measurements of body fat, lean mass, and other key body components.

Lean, bone, fat, and visceral adipose tissue (VAT) masses were measured by DXA using a Discovery Wi device (Hologic, Bedford, Massachusetts, USA) equipped with analysis software (APEX version 4.0.2). Lean and fat mass indices were expressed as kg/m^2 ; FM was also expressed as a percentage of body weight. Regarding bone outcomes, bone mineral content (BMC), BMD, and BMD Z-score were also obtained. During the scan, participants laid motionless on a table while a scanning arm moved over the body, capturing detailed cross-sectional images. The DXA scan was performed under standardized conditions, with strict adherence to the manufacturer's guidelines for calibration and quality control.

Bioelectrical measurements of resistance and reactance were taken at 50 kHz using a Tanita (MC-980 MA, segmental, Tokyo, Japan). Electrodes were strategically placed on the participants, hands and feet, and a small safe electrical current was passed through the body. The resistance to this current, combined with factors such as height, weight, age, and sex, was used to estimate body composition. The following outcomes were obtained from the procedure: fat mass (kg), fat mass (%), FFM (kg), and muscle mass (kg).

Anthropometry involved a series of manual measurements to assess body size, shape, and composition. This method included the use of calipers to measure skinfold thickness at specific sites on the body (biceps, triceps, subscapular, iliac crest, supraspinal, abdominal, thigh, and calf) which provided estimates of subcutaneous fat. Additionally, girths of various body parts, including the ten circumferences (head, forearm, arm relaxed, arm flexed and contracted chest, waist, hips, thigh middle, and calf), were measured using an inextensible and flexible tape, while bone breadths diameters at key anatomical sites (biacromial, biiliocrystal, antero-posterior chest depth, transverse chest, humerus, and femur) were recorded using sliding calipers. These measurements were conducted following the standardized protocols of the International Society for the Advancement of Kinanthropometry (ISAK) (183) ensuring consistency and accuracy across all assessments. The data obtained from these measurements were used to estimate body fat percentage, bone mass, and lean mass through well-established anthropometric equations (184).

DXA served as the primary method for estimating body composition in this study, given its comprehensive assessment capabilities. The results from BIA and anthropometry were used to cross-validate these findings, offering a more detailed and multi-faceted understanding of the participants' body composition.

4.1.3. Low testosterone symptoms

The presence of low testosterone symptoms was assessed using the ADAM-Q (63). This 10-item yes/no tool evaluates symptoms such as reduced libido, energy, strength, mood alterations, and decreased morning erections. Although the LEAM-Q has been proposed as a screening tool for LEA in male athletes, the ADAM-Q remains more specifically targeted to identifying clinical symptoms associated with

testosterone deficiency and hypogonadism. Moreover, it continues to be widely used and validated in both clinical and physically active populations (64).

4.1.4. Risk for the female triad

Risk for the Female Athlete Triad was assessed using the LEAF-Q, which includes 25 items covering menstrual function, gastrointestinal symptoms, and injury history. A total score ≥ 8 has been validated as the optimal cut-off point to identify athletes at risk of LEA, with demonstrated sensitivity of 78% and specificity of 90% (173). Additionally, subscale cut-offs of ≥ 2 for the injury and gastrointestinal domains have been applied to explore symptom-specific risk profiles.

4.1.5. Statistical Analysis

Descriptive data are expressed as mean and standard deviation (SD) unless otherwise indicated. Data normality was checked using histograms, Q-Q plots, box plots, and the Shapiro-Wilk test. As ADAM-Q and LEAF-Q scores did not follow a normal distribution, we performed Spearman correlations to examine the association between these questionnaire scores, body composition, and muscular strength. Furthermore, Student's t-tests or Mann-Whitney U tests were conducted to compare normally distributed and non-normally distributed body composition outcomes, respectively, between men with and without symptoms of low testosterone levels, and between women at no risk and risk for the Female Athlete Triad. We assessed variance equivalence using Levene's test and applied the corresponding correction when required. Statistical significance was set at $P < 0.05$. Figures were built with GraphPad Prism software v.8 (GraphPad Software, San Diego, CA, USA), and all analyses were performed using the Statistical Package for the Social Sciences v.27.0 (IBM Corporation, Pittsburgh, PA, USA).

4.2. ASSESMENT OF ENERGY AVAILABILITY AND RESTING METABOLIC RATE

To address the objective of examining the relationship between EA and metabolic function under free-living conditions, a cross-sectional methodological

approach was employed, focusing on RMR, RMRratio, and their association with EA and phenotypical traits in competitive athletes.

4.2.1. Study design and participants

A total of **49 competitive athletes** (33 men and 16 women) from diverse sport disciplines were included in this analysis. Athletes competed at regional, national, or international levels, and competitive status was determined using validated classification criteria (182). All participants attended the Sport and Health University Research Center (iMUDS, University of Granada, Spain) on two scheduled occasions.

The study protocol was approved by the Human Research Ethics Committee of the Junta de Andalucía (approval number: 0909-N-22) and conducted in accordance with the Declaration of Helsinki. All participants received detailed information regarding the study objectives, procedures, and potential risks and provided written informed consent prior to participation.

Eligibility criteria included: (1) age between 18 and 35 years; (2) active competition with national registration for at least two consecutive years; (3) for women, not being pregnant or breastfeeding; (4) absence of diagnosed cardiovascular conditions or chronic diseases; and (5) no use of tobacco, recreational drugs, excessive alcohol, or medications with potential interference with study outcomes.

4.2.2. Resting metabolic rate

RMR was assessed using indirect calorimetry under standardized conditions to ensure accuracy and reproducibility. Participants arrived at the laboratory following an overnight fast of at least 8 hours and were instructed to refrain from strenuous physical activity, caffeine, and alcohol consumption for at least 24 hours prior to the assessment. Upon arrival, participants rested in a supine position in a thermoneutral environment for a minimum of 20 minutes before data collection.

Gas exchange measurements were obtained using a ventilated canopy hood system connected to a metabolic cart (Quark RMR, COSMED, Rome, Italy). Data were collected over a 30-minute period, with oxygen consumption (VO_2) and

carbon dioxide production (VCO_2) continuously recorded at 5-second intervals. The first 5 minutes of data were discarded, and mean VO_2 and VCO_2 values from the remaining 25 minutes were used for analysis. Resting energy expenditure was calculated using Weir's abbreviated equation, assuming negligible nitrogen excretion.

4.2.3. RMR_{ratio} calculation

To determine the RMR_{ratio}, we first obtained the measured RMR via indirect calorimetry. Predicted RMR values were then estimated using a suite of published equations, including Cunningham (185), Ten Haaf (both fat-free mass- and body weight-based variants) (186), Tinsley (body weight and fat-free mass versions), Wong (187), Freire (188), Van Hooren (189), Harris–Benedict, Pontzer (body weight and FFM versions) (190,191) and De Lorenzo (192).

Among these, Ten Haaf et al.'s equation (186) was selected to examine the relationship between RMR_{ratio} and phenotypic traits, based on recent work by O'Neill et al. (2023) indicating its superior accuracy (especially in athletic cohorts) relative to other commonly utilized predictive models (193). In addition, the Ten Haaf equation was specifically validated in physically active populations and incorporates FFM as a key determinant (186), making it more suitable for sports performance and athlete monitoring contexts.

The RMR_{ratio} was determined by dividing the measured RMR by the predicted RMR. A threshold of 0.90 was used as the cut-off point to indicate metabolic suppression, as commonly referenced in the literature for detecting metabolic adaptations associated with LEA (141). This approach allows for a more precise assessment of potential metabolic adaptations that may arise in competitive athletes due to energy deficiency, training load, or other physiological stressors.

4.2.4. Energy availability assesment

EA was estimated following the method described by Loucks et al. (1998)(4), which integrates dietary energy intake and FFM to provide a physiologically relevant index. The formula applied was: $\text{EA} = [\text{EI (kcal)} - \text{EEE (kcal)}] / \text{FFM (kg)}$.

FFM was derived from a whole-body Dual-Energy X-ray Absorptiometry (DXA) scan.

Dietary intake was assessed through three non-consecutive 24-hour dietary recalls. Participants were systematically guided through the 24-hour dietary recall, strategically distributed to include two weekdays and one weekend or holiday. All interviews were conducted in person or via video call by qualified dietitians trained in standardized data collection techniques. To enhance the accuracy of portion size estimation, standardized photographic aids depicting commonly consumed food portions were employed, allowing for more precise and consistent reporting of both food and beverage intake. Nutritional analysis, including total energy intake and macronutrient distribution, was performed using EvalFINUT® software, which incorporates compositional data from both the United States Department of Agriculture (USDA) and the Spanish Food Composition Database (BEDCA).

Participants maintained a daily log documenting the duration (in hours) and frequency of various physical activities. Energy expenditure for each activity was calculated using the following equation: Energy Expenditure (kcal) = MET (kcal/kg/h) × Body Weight (kg) × Duration (hours) (194). These daily values were then averaged over seven consecutive days to derive a weekly mean, integrating both resting metabolic demands and activity-specific energy costs to provide a comprehensive estimate of total energy expenditure.

Importantly, the three non-consecutive 24-hour dietary recalls were conducted within the same 7-day period in which physical activity logs were recorded for the estimation of EE. This ensured temporal alignment between EI and EE measures. While the recalls did not cover all seven days, they were distributed strategically (two weekdays and one weekend/holiday) to reflect habitual intake and to overlap with the week of activity monitoring. Thus, both dietary and training data represent the same period, providing a coherent basis for the calculation of EA.

4.2.5. Assessment of phenotypical traits

A broad set of phenotypical traits relevant to LEA was evaluated. Dietary outcomes (energy, macronutrient, and fiber intakes) were evaluated through 24-

hour recalls as aforementioned. We assessed physical activity and sleep quality through accelerometry as detailed elsewhere (66). Subjective health and wellness were assessed through validated questionnaires. Symptoms of androgen deficiency by the ADAM-Q (195), eating behaviours and attitudes linked to eating disorders by the Eating Attitudes Test-26 (EAT-26)(196), body image perceptions by the Silhouette Rating Scale (SRS) (197), athlete's self-perceived performance by the Athlete's Subjective Performance Scale (ASPS), subjective quality and quantity of sleep by the Pittsburgh Sleep Quality Index (PSQI)(198), indicators of low energy availability in female athletes by LEAF-Q (Melin et al. 2014a), athletes' nutritional knowledge by the Nutrition Knowledge Questionnaire for Young Athletes (NUKYA) (199), the risk of overtraining by the Overtraining Score (200), and the balance between stress and recovery by the Recovery-Stress Questionnaire for Athletes (RESTQ-76) (201).

4.2.6. Statistical analysis

Data normality was checked using histograms, Q-Q plots, box plots, and the Shapiro–Wilk test. Differences in the RMRratio depending on the equation used to obtain the RMR predicted were analyzed using repeated measures analysis of variance (ANOVA) with post-hoc Bonferroni pairwise comparisons. Sphericity was determined by Mauchly's test, and the Greenhouse–Geisser correction was applied when required. Differences in the prevalence of participants with a ratio lower or higher than 0.9 depending on the predictive equation used were analyzed by the Chi-Square Test.

Then, we conducted Pearson correlation analyses to examine the relationship between RMR and RMRratio with EA. Differences in RMR and RMRratio between participants with LEA and adequate EA were assessed using independent samples t-tests.

We also used Pearson correlation analyses to study the relationship between the RMRratio and phenotypical traits (i.e., body composition, dietary outcomes, physical activity, sleep quantity and quality, and subjective well-being). Lastly, we conducted independent samples t-tests to compare body composition, dietary outcomes, sleep quality and quantity, and subjective well-being between individuals with an RMRratio higher or lower than 0.9. To represent differences

between individuals with an RMRratio higher or lower than 0.9, we further calculated the Z-scores and 95% confidence interval of the mean differences between groups. To account for multiple testing in the Pearson correlation analyses and independent samples t-test analyses, we used the two-stage step-up method of the false discovery rate (FDR) (202).

The level of statistical significance was set at $P < 0.05$. Analyses were performed in Statistical Package for the Social Sciences v.27.0 (IBM Corporation, Pittsburgh, PA, USA) and R software version 4.1.2 (<https://cran.r-project.org/>, The R Project for Statistical Computing, Vienna, Austria). Plots were built using GraphPad Prism software v.8 (GraphPad Software, San Diego, CA, USA) and R software version 4.1.2 (<https://cran.r-project.org/>, The R Project for Statistical Computing, Vienna, Austria).

4.3. ASSESSMENT OF MOVEMENT BEHAVIOURS AND PERCEIVED HEALTH

To address the objective of examining the relationship between daily movement behaviours, EA, and perceived health, an observational cross-sectional methodological approach was employed, integrating objective accelerometry-based assessments with dietary data and validated self-report questionnaires.

4.3.1. Study design and participants

A total of 49 competitive Spanish athletes (33 men and 16 women; 25.2 ± 5.6 years) were included in this analysis. Participants were recruited through non-probabilistic convenience sampling from endurance, combat, and team-based sports, all competing at regional, national, or international levels. Competitive level was determined using validated classification criteria (182).

Eligibility criteria required participants to be between 18 and 35 years of age and actively competing for a minimum of two consecutive years. Exclusion criteria included a history of pathological events during exercise; major adverse cardiovascular events; renal insufficiency; cirrhosis; eating disorders; polycystic ovary syndrome; surgical intervention for weight control; type 2 diabetes mellitus; HIV/AIDS; any chronic pathology; use of medications affecting study outcomes; pregnancy or breastfeeding; and active tobacco or illicit drug use, or a history of

treatment for alcohol abuse. All exclusion criteria were confirmed through structured face-to-face interviews.

4.3.2. Procedure

Participants were recruited through social media platforms and official communication channels of national and regional sports federations in Spain. Information regarding study objectives, eligibility criteria, and procedures was disseminated online to reach potential volunteers. Athletes meeting inclusion criteria were contacted and scheduled for laboratory visits.

Each participant attended two visits to the Sport and Health Research Center (iMUDS, University of Granada, Spain). During the first visit, participants underwent a comprehensive phenotypic assessment and were provided with triaxial accelerometers to be worn continuously for nine consecutive days. During the second visit, accelerometers and corresponding physical activity logs covering the same period were returned.

4.3.3. Anthropometry and body composition

Anthropometry and body composition were assessed following the same standardized protocol described previously in Section 4.1.2. Body mass and standing stature were measured to the nearest 0.1 kg and 0.1 cm, respectively, using a calibrated digital scale and stadiometer (Seca model 799, Hamburg, Germany), with participants barefoot and wearing light athletic clothing. Body mass index (BMI) was calculated as weight divided by height squared ($\text{kg}\cdot\text{m}^{-2}$).

Whole-body composition was determined by dual-energy X-ray absorptiometry (DXA) using a Discovery Wi device (Hologic, Bedford, MA, USA) and APEX software (version 4.0.2), yielding measures of lean mass and fat mass. Participants remained motionless in a supine position while the scanning arm acquired sequential cross-sectional images under standardized conditions.

4.3.4. Anthropometry of movement behaviours

Objective physical-activity patterns, sedentary behavior and sleep variables were captured by wrist-worn accelerometry (wGT3X-BT, ActiGraph LLC, Pensacola, FL, USA), programmed at 100 Hz. Devices were delivered in the first laboratory visit, remained in place day and night for nine uninterrupted days, and were removed only for prolonged aquatic activities. After retrieval, the raw files (.gt3x) were imported directly into R and processed with the GGIR package (v 2.8-0). The GGIR workflow comprised: (i) automatic calibration of the raw triaxial signal with the van Hees algorithm (203); (ii) identification of non-wear by evaluating axis-specific standard deviation and range (updated van Hees method) (204,205); (iii) replacement of gaps created by Idle-Sleep Mode and detection of clipping artefacts characterised by implausibly sustained high accelerations; (iv) computation of Euclidean-Norm-Minus-One (ENMO) (205), the device's z-axis angle relative to gravity, and step counts employing the Verisens algorithm refined by Rowlands et al.; (v) determination of the principal nocturnal sleep window each calendar day via the HDCZA heuristic applied to z-angle variability; and (vi) classification of movement intensity from ENMO using Hildebrand-validated thresholds for adults (206) (light ≥ 35 mg, moderate ≥ 100 mg, vigorous ≥ 400 mg).

For each 24-h period, the pipeline produced total time accumulated in sedentary behavior, light, moderate and vigorous activity, step counts, peak cadence (fastest 1-, 30- and 60-min epochs), the number and duration of inactivity bouts (30–60 min and ≥ 60 min), and objective sleep metrics (total sleep time). Daily outputs were averaged across the seven-day recording to generate an integrated 24-h movement-behaviour profile for subsequent statistical analyses.

4.3.5. Statistical analysis

Unless otherwise indicated, descriptive data are expressed as mean and standard deviation (SD). Data normality was checked using histograms, Q-Q plots, box plots, and the Shapiro–Wilk test. Then, we conducted Pearson correlation analyses to examine the relationship between physical activity and EA. Differences in physical activity between participants with LEA and adequate EA were assessed using independent samples t-tests. To represent differences between individuals with LEA or adequate EA, we further calculated the Z-scores and 95% confidence

interval of the mean differences between groups. All the analyses were repeated, adjusting for sex and sport classification.

Compositional data analysis was applied to evaluate the relationships between movement behaviours (i.e., MVPA, LPA, sedentary behaviour, and sleep) and EA. This approach considers the co-dependent and relative nature of 24 h time use, and addresses multicollinearity (207). We firstly calculated isometric log-ratios in sequential binary partition as proposed (208) and they were subsequently included as explanatory variables. The nonstandardized B coefficient shows the strength and direction of the relationship of each behavior relative to the rest of behaviors in the composition with the outcome. The models' coefficients were used to predict the effect of reallocating time proportionally across behaviors (i.e., increasing MVPA while reducing the remaining behaviors) and pairwise (i.e., increasing MVPA while reducing LPA) on the outcomes. The results can be interpreted as the hypothetical change in the outcome associated with reallocating time between behaviors for a hypothetical average participant in our sample as the model coefficients are relative to the mean time-use composition. A variance matrix was used to summarise data variability, with lower values indicating greater interdependence between behaviour pairs.

Lastly, we investigated the association between physical activity and subjective well-being using Pearson correlation analyses.

The level of statistical significance was set at $P < 0.05$. For Pearson correlation analyses and independent samples t-test analyses, we further corrected P values by controlling the false discovery rate (FDR). Analyses were performed in Statistical Package for the Social Sciences v.27.0 (IBM Corporation, Pittsburgh, PA, USA) and R software version 4.1.2 (<https://cran.r-project.org/>, The R Project for Statistical Computing, Vienna, Austria). Plots were built using GraphPad Prism software v.8 (GraphPad Software, San Diego, CA, USA) and R software version 4.1.2.

An overview of the methodological approaches, sample sizes, main variables assessed, and statistical analyses employed to address the different research objectives of this Doctoral Thesis is provided in Table 1.

Table 1. Methodological overview of the studies included in the doctoral thesis.

Study	Design	Participant's characteristics	Independent variable	Dependent variables	Statistical analysis
STUDY I	Cross-sectional	71 competitive athletes (48 men, 23 women), 18–35 years	Body composition (FM %, LM, VAT)	LEAF-Q scores (women) and ADAM-Q scores (men)	• Pearson correlation analyses - Independent samples t-test • Binary logistic regression
STUDY II	Cross-sectional	49 competitive athletes (33 men, 16 women), 18–35 years	EA (kcal/kg FFM/day), RMR and RMRratio	BMD, sleep quality/quantity, perceived stress, and well-being indicators	• Pearson and partial correlation analyses • Independent samples t-test • Linear regression analyses
STUDY III	Cross-sectional	49 competitive athletes (33 men, 16 women), 18–35 years	Free-living movement behaviours (physical activity, sedentary time)	EA (Kcal/Kg FFM/day) Menstrual health, fatigue, and perceived well-being	• Pearson correlation analyses • Independent samples t-tests • FDR correction

Abbreviations: FM%, fat mass%; LM, lean mass; VAT, visceral adipose tissue; EA, energy availability; RMR, resting metabolic rate; BMD, bone mineral density; FDR, false discover rate

V – RESULTS

V - RESULTS

5.1. RESULTS RELATIONSHIP BETWEEN BODY COMPOSITION AND LOW TESTOSTERONE SYMPTOMS OR RISK FOR THE FEMALE ATHLETE TRIAD IN ELITE AND PRE-ELITE ATHLETES

5.1.1. **Body composition is not related to the risk of low testosterone symptoms in male athletes**

Body weight was not significantly correlated with ADAM-Q scores ($r = 0.151$, $P = 0.305$; **Figure 4A**). Similarly, adipose indices (i.e., fat mass, fat mass percentage, FMI, and VAT), measured by DXA, showed no significant relationship with ADAM-Q scores (all $P \geq 0.079$; **Figure 4B-D** and **Table S1**). Likewise, adipose indices derived from BIA and anthropometry were not correlated with ADAM-Q scores (i.e., fat mass, fat mass percentage, adipose mass, and the sum of skinfolds; all $P > 0.05$; **Table S1**). Then, we compared men with and without symptoms of low testosterone levels and observed no differences in adipose indices measured by DXA or BIA (i.e., fat mass, fat mass percentage, android fat mass, gynoid fat mass, and the sum of skinfolds; all $P > 0.05$; **Table S2**).

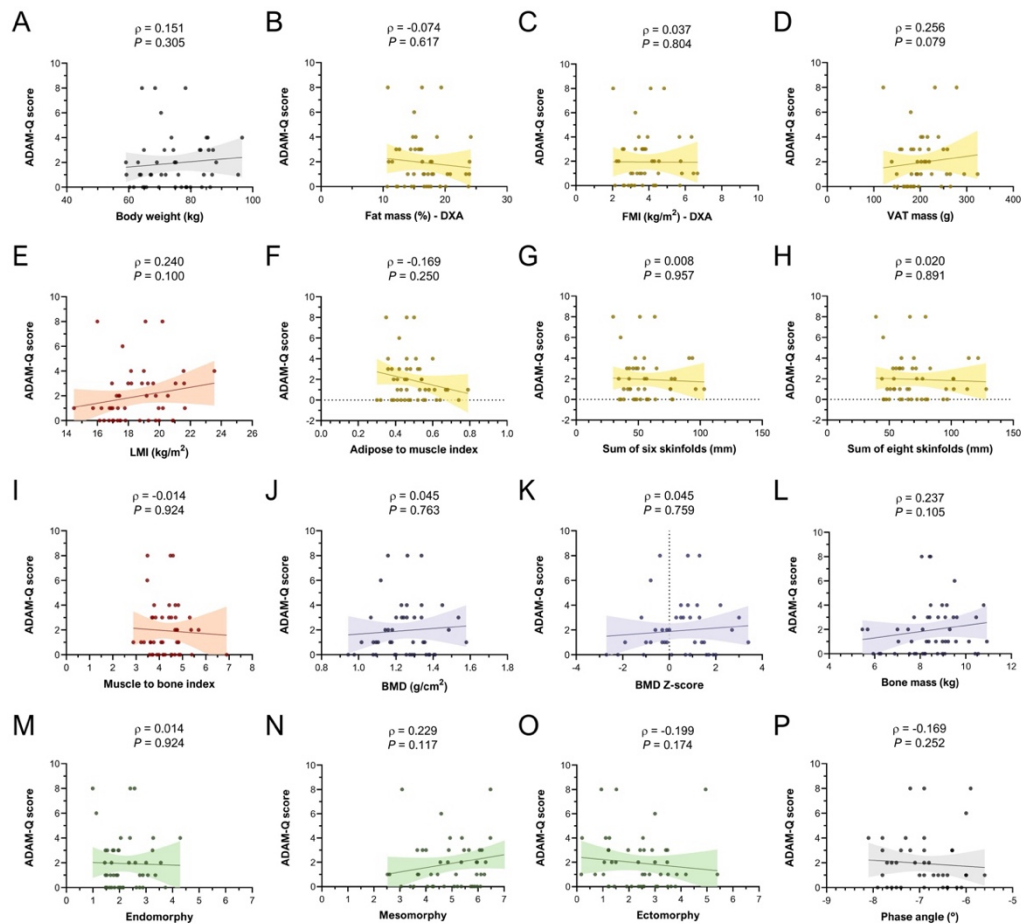


Figure 4. Correlation between body composition and the ADAM-Q score in male athletes.

Body composition outcomes included body weight (Panel A, grey), fat mass and adipose indices (Panels B-D, and F-H, yellow), lean indices (Panels E and I, red), bone outcomes (Panels J-L, blue), somatotype (Panels M-O, green) and phase angle (Panel P, grey). Each plot represents individual points, a trend line, and its corresponding 95% confidence interval. P -values were obtained from Spearman's correlation analyses between the questionnaire scores and body composition measures. Bold P -values indicate statistically significant associations. Abbreviations: ADAM-Q, Androgen Deficiency in the Aging Male Questionnaire; BMC, bone mineral content; BMD, bone mineral density; DXA, dual-energy X-ray absorptiometry; FMI, fat mass index; LMI, lean mass index; VAT, visceral adipose tissue. ρ = Spearman's correlation coefficient.

Lean indices, measured by DXA, were not correlated with ADAM-Q score [i.e., lean mass, FFM, and lean mass index (LMI); all $P \geq 0.100$; **Figure 4E** and **Table**

S1]. However, a positive and significant correlation was observed between FFM and muscle mass measured by BIA and ADAM-Q scores (both $\rho = 0.306$, $P = 0.035$; **Table S1**). When comparing lean indices between athletes with and without symptoms of low testosterone (according to the ADAM-Q), we observed a significantly higher total lean mass ($P=0.012$) and LMI ($P=0.027$) in athletes in the symptoms group compared to the no symptoms group (**Figure 5A and B**). Similarly, we observed that men with symptoms of low testosterone levels showed higher FFM measured by DXA ($P = 0.015$), FFM measured by BIA (FFM-BIA), and muscle mass measured by BIA (both $P=0.007$) than those without symptoms (**Table S2, Figure 5C and D**). We also found that men with symptoms of low testosterone levels showed higher thorax and thigh circumference than those without symptoms ($P=0.043$ and $P=0.044$ respectively; **Figure 5G and H**).

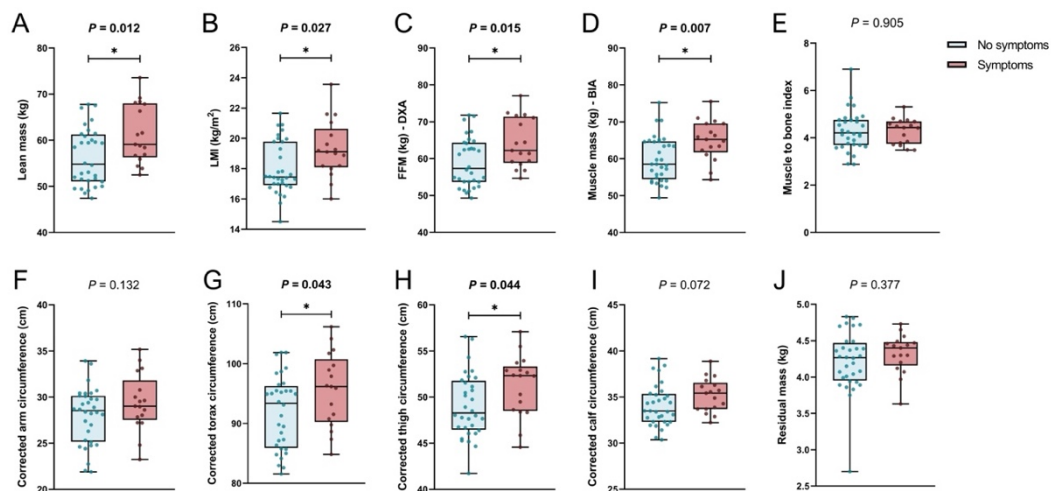


Figure 5. Differences in lean mass and lean indices in male athletes with (red color; $n = 17$) and without (blue color; $n = 31$) symptoms of low testosterone levels.

Box plots represent data as the median, interquartile range, and whiskers (minimum and maximum values). P -values from panels A-H and J were obtained from independent Student's t -tests, whereas P -value from panel I was obtained from Mann-Whitney U tests. * Symbols depict statistically significant differences between groups ($P < 0.05$). Abbreviations: BIA, bioelectrical impedance analysis; DXA, dual-energy X-ray absorptiometry; FFM, fat-free mass; LMI, lean mass index.

Bone outcomes, including BMC, BMD, BMD Z-score, and bone mass, were not significantly correlated with ADAM-Q scores (all $P \geq 0.105$; **Figure 4J-L** and

Table S1). Similarly, somatotypes (endomorph, mesomorph, and ectomorph) and phase angle did not show significant associations with ADAM-Q scores (all $P \geq 0.117$; **Figure 4M-P** and **Table S1**). In addition, no significant differences were observed between men with and without symptoms of low testosterone levels in bone outcomes (BMC, BMD, BMD Z-score, and bone mass; all $P \geq 0.105$), somatotypes (endomorph, mesomorph, and ectomorph; all $P \geq 0.117$), or phase angle ($P = 0.252$; **Table S2**).

5.1.2. Body composition is not related to the risk of the Female Athlete Triad in female athletes

Body weight did not significantly correlate with LEAF-Q total score and sub-scores ($r = -0.089$, $P = 0.685$; **Figure 6A** and **Table S1**). Similarly, adipose measurements obtained through DXA (including FM, FM%, VAT, gynoid fat mass, android fat mass, and total adipose mass) showed no significant associations with LEAF-Q total score (all $P \geq 0.277$; **Figures 6B-D** and **Table S2**). Similarly, adipose indices derived from BIA and anthropometry, such as fat mass by BIA, fat mass percentage by BIA, sums of skinfolds, and the adipose-to-muscle index, were also not correlated with LEAF-Q scores (all $P > 0.05$; **Table S1**).

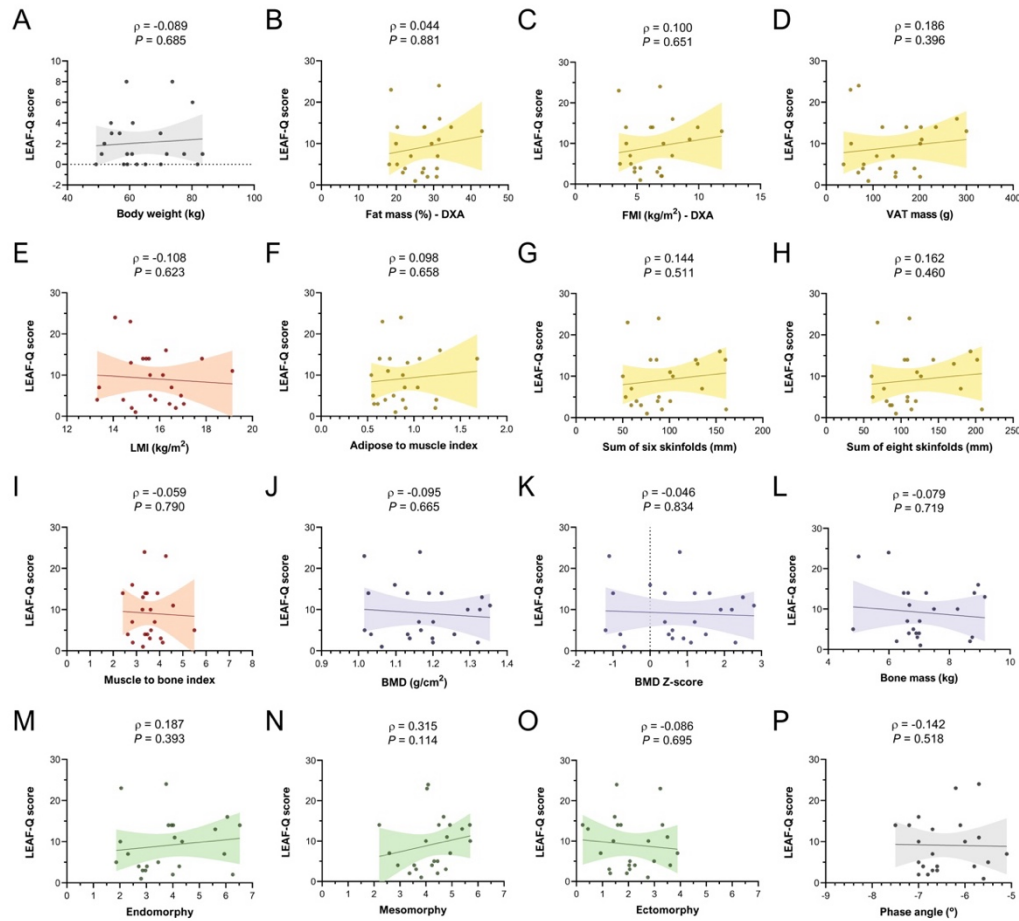


Figure 6. Correlation between body composition and the LEAF-Q score in female athletes.

Body composition outcomes included body weight (Panel A, grey), fat mass and adipose indices (Panels B-D, and F-H, yellow), lean indices (Panels E and I, red), bone outcomes (Panels J-L, blue), somatotype (Panels M-O, green) and phase angle (Panel P, grey). Each plot represents individual points, a trend line, and its corresponding 95% confidence interval. P-values were obtained from Spearman's correlation analyses between the questionnaire scores and body composition measures. Abbreviations: BMC, bone mineral content; BMD, bone mineral density; DXA, dual-energy X-ray absorptiometry; FMI, fat mass index; LEAF-Q, Low Energy Availability in Females Questionnaire; LMI, lean mass index; VAT, visceral adipose tissue. ρ = Spearman's correlation coefficient.

Nevertheless, although fat mass percentage and VAT were not significantly associated with overall LEAF-Q scores, we observed that FM% DXA ($\rho = 0.427$, $P = 0.042$), and VAT mass ($\rho = 0.438$, $P = 0.037$) were positively correlated with the LEAF-Q injury score (Figure 7B and F, and Table S1).

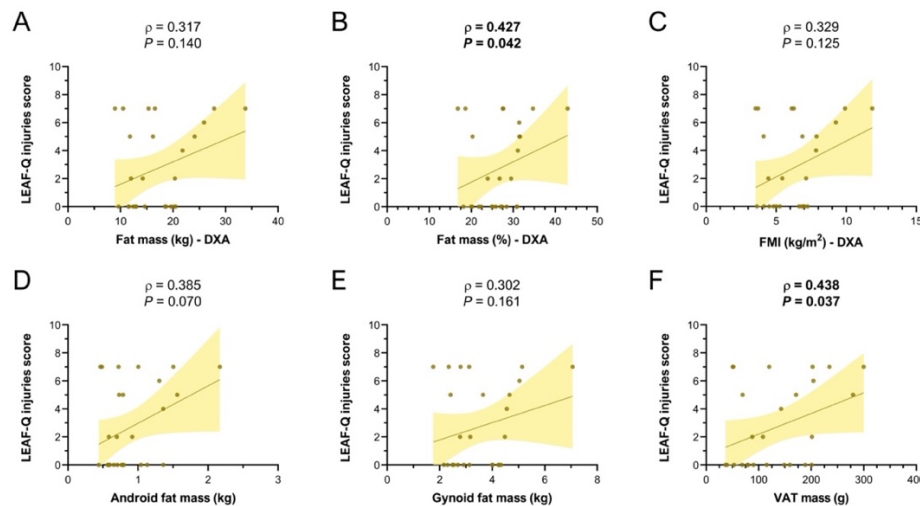


Figure 7. Correlation between fat mass and adipose indices, and the LEAF-Q injuries score in female athletes.

Fat mass and adipose indices included fat mass (DXA; Panel A), fat mass percentage (DXA; Panel B), FMI (Panel C), android fat mass (Panel D), gynoid fat mass (Panel E), and VAT mass (Panel F). Each plot represents individual points, a trend line, and its corresponding 95% confidence interval. *P*-values were obtained from Spearman's correlation analyses between the questionnaire scores and fat mass and adipose indices measures. Bold ρ and *P*-values represent statistically significant correlations ($P < 0.05$). Abbreviations: DXA, dual-energy X-ray absorptiometry; FMI, fat mass index; LEAF-Q, Low Energy Availability in Females Questionnaire; VAT, visceral adipose tissue. ρ = Spearman's correlation coefficient.

Lean indices measured by DXA, including FFM, LMI, and FMI, did not display significant correlations with LEAF-Q total score and subscores (all $P \geq 0.170$; **Figure 6E** and **Table S1**). Similarly, lean measurements from BIA and anthropometry (such as FFM and muscle mass) showed no significant associations with LEAF-Q scores (all $P \geq 0.140$; **Table S1**).

Bone-related parameters, including BMC, BMD, BMD Z-score, and bone mass, also did not have significant relationships with LEAF-Q total score and subscores (all $P \geq 0.584$; **Figures 6J-L** and **Table S1**). Additionally, somatotypes (endomorphs, mesomorphs, and ectomorphs) and phase angle were not

significantly correlated with LEAF-Q scores (all $P \geq 0.114$; **Figures 6M-P** and **Table S1**). Skinfold measurements (sum of six and eight skinfolds) likewise showed no significant associations (all $P \geq 0.460$; **Table S1**). Comparative analysis between female athletes at risk and not at risk for the Female Athlete Triad, as determined by LEAF-Q, revealed no significant differences in body composition variables. This included adipose indices (fat mass, fat mass percentage, VAT mass, android fat mass, gynoid fat mass, and skinfold sums), lean indices (FFM by DXA, FFM by BIA, lean mass, LMI), bone outcomes (BMD, BMD Z-score, bone mass), somatotypes, and phase angle (all $P > 0.05$; **Table S2**).

5.2. ENERGY AVAILABILITY AND METABOLIC FUNCTION

5.2.1. Participant characteristics

A total of 49 Spanish competitive athletes (33 males and 16 females), with a mean age of 25.2 ± 5.6 years, participated in the study (Table 2). The cohort included athletes competing at regional (57.1%), national (36.7%), and international (6.1%) levels. The average body mass index (BMI) was 23.6 ± 3.0 kg/m², and the mean training experience was 9.0 ± 6.4 years. No significant sex-based differences were observed in RMRratio categories or EA status (both $P > 0.05$; **Table 2**); consequently, the analyses are presented together for both sexes.

Table 2. Descriptive characteristics of the participants according to resting metabolic rate suppression status (RMRratio <0.9 vs. ≥0.9).

	All (n=49)		RMR measured/predicted (n=34)		ratio <0.9	RMR measured/predicted (n=15)		ratio ≥0.9
	Mean	SD	Mean	SD		Me	SD	
Age (years)	25.2	5.6	26.1	6.1		23.3	3.7	
Sex (n; %)								
Male (n, %)	33	67.3	22	64.7		11	73.3	
Female (n, %)	16	32.7	12	35.3		4	26.7	
Weight (kg)	71.4	11.7	68.7	11.3		77.6	10.5	
Height (cm)	173.6	8.2	172.4	8.2		176.4	7.8	
BMI (kg/m ²)	23.6	3.0	23.0	2.8		24.9	3.2	
FFM (kg)	56.0	10.1	54.4	10.4		59.7	8.7	
FM (%)	20.0	7.0	19.5	6.2		21.3	8.6	
Training experience (years)	9.0	6.4	9.8	7.0		7.4	4.7	
Competition level								
Regional (n, %)	28	57.1	21	61.8		7	46.7	
National (n, %)	18	36.7	10	29.4		8	53.3	
International (n, %)	3	6.1	3	8.8		0	0	

Data presented as mean and standard deviation (SD), unless otherwise indicated. The RMRratio measured/predicted was calculated using the ten Haaf (FFM) equation (186) for the predicted RMR. Abbreviations: BMI, body mass index; FFM, fat-free mass; FM, fat mass; RMR, resting metabolic rate.

5.2.2. Differences in the RMRratio based on predictive equations

Statistical differences in the RMRratio values were identified depending on the predictive equation used for RMR estimation ($P < 0.001$; **Figure 8** and post-hoc comparisons shown in **Table S3**). The mean RMR ratio ranged from 0.74 ± 0.09 using the Van Hooren equation to 0.95 ± 0.10 with the Cunningham equation ($P < 0.001$; **Table S3**). In line with these findings, the proportion of participants classified as having a suppressed RMR ratio (<0.9) also varied significantly across predictive equations ($P < 0.001$; **Table 3**). Notably, the Freire and Van Hooren equations identified the highest proportion of participants as metabolically suppressed, with 93.9% falling below and only 6.1% upper the 0.9 threshold (**Table 3**). Meanwhile, other models, such as both versions of the Pontzer equation,

resulted in more balanced classifications, with over half of the athletes exhibiting $\text{RMR}_{\text{ratio}} \geq 0.9$ (Table 3).

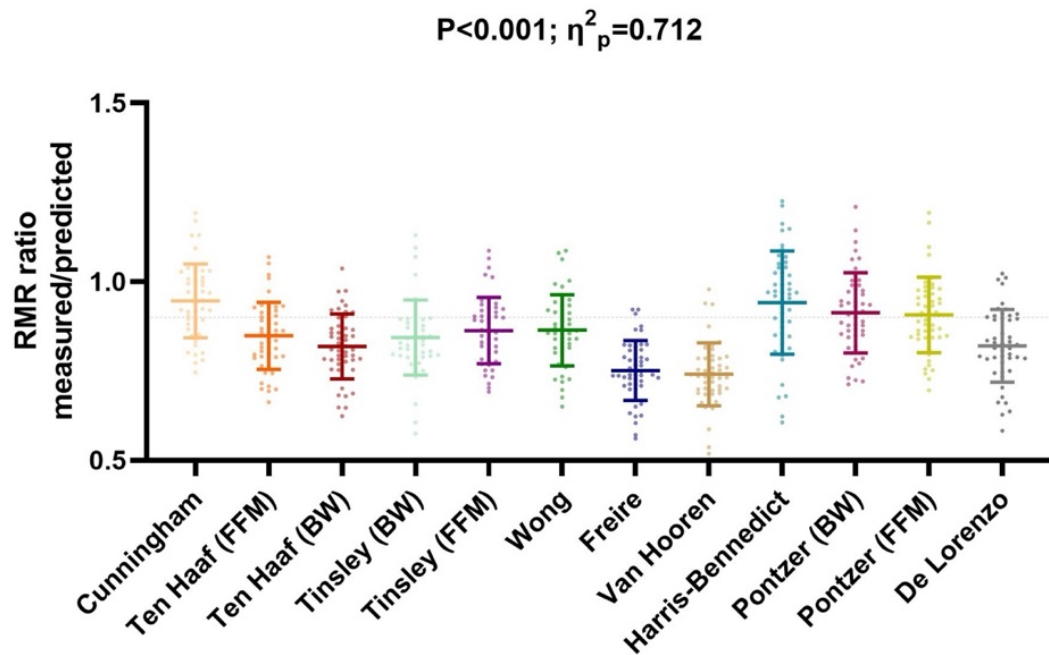


Figure 8. Differences in the RMR ratio measured/predicted depending on the predictive equation used to predict the RMR in all participants.

P values and partial eta squared (η^2_p) were obtained from repeated measures analysis of variance (ANOVA). Post-hoc Bonferroni correction is displayed in Table S1. The dashed grey line indicates the 0.9 threshold for considering a high or low RMR ratio. Abbreviations: BW, body weight; FFM, fat-free mass; RMR, resting metabolic rate.

Table 3. Differences in the prevalence of participants with a ratio lower or higher than 0.9. P value obtained from Chi-Square Test.

RMR ratio measured/predicted						P value
<0.9				>0.9		
		N	%	N	%	
Cunningham Ten Haaf (FFM) Ten Haaf (BW) Tinsley (BW) Tinsley (FFM) Wong Freire Van Hooren Harris- Benedict Pontzer (BW) Pontzer (FFM) De Lorenzo		17	34.7	32	65.3	<0.001
		34	69.4	15	30.6	
		39	79.6	10	20.4	
		40	81.6	9	18.4	
		32	65.3	17	34.7	
		32	65.3	17	34.7	
		46	93.9	3	6.1	
		46	93.9	3	6.1	
		18	36.7	31	63.3	
		24	49.0	25	51.0	
		24	49.0	25	51.0	
		37	75.5	12	54.5	

Abbreviations: BW, body weight; FFM, fat-free mass; RMR, resting metabolic rate.

The Ten Haaf equation based on FFM, which served as the reference model in this study, classified 69.4% of athletes as having suppressed RMR_{ratio}, reflecting a moderate sensitivity relative to other equations.

These analyses were similar when in the male subgroup (**Table S6**).

5.2.3. Relationship between RMR and RMR_{ratio} with energy availability

We found no significant association between RMR and EA ($r=-0.117$, $P=0.424$; **Figure 9A**), and when participants were classified in LEA (EA, <30 kcal/kg FFM/day) or adequate EA (EA, ≥ 30 kcal/kg FFM/day) no statistically significant differences were found in RMR ($P=0.520$; **Figure 9B**). Similarly, no relationship was observed between EA and RMR_{ratio} ($r=0.024$, $P=0.871$; **Figure 9C**), and no differences

were observed between participants with LEA or EA in the RMR_{ratio} ($P=0.819$; Figure 9D).

All these analyses were similar in the male subgroup (Table S7).

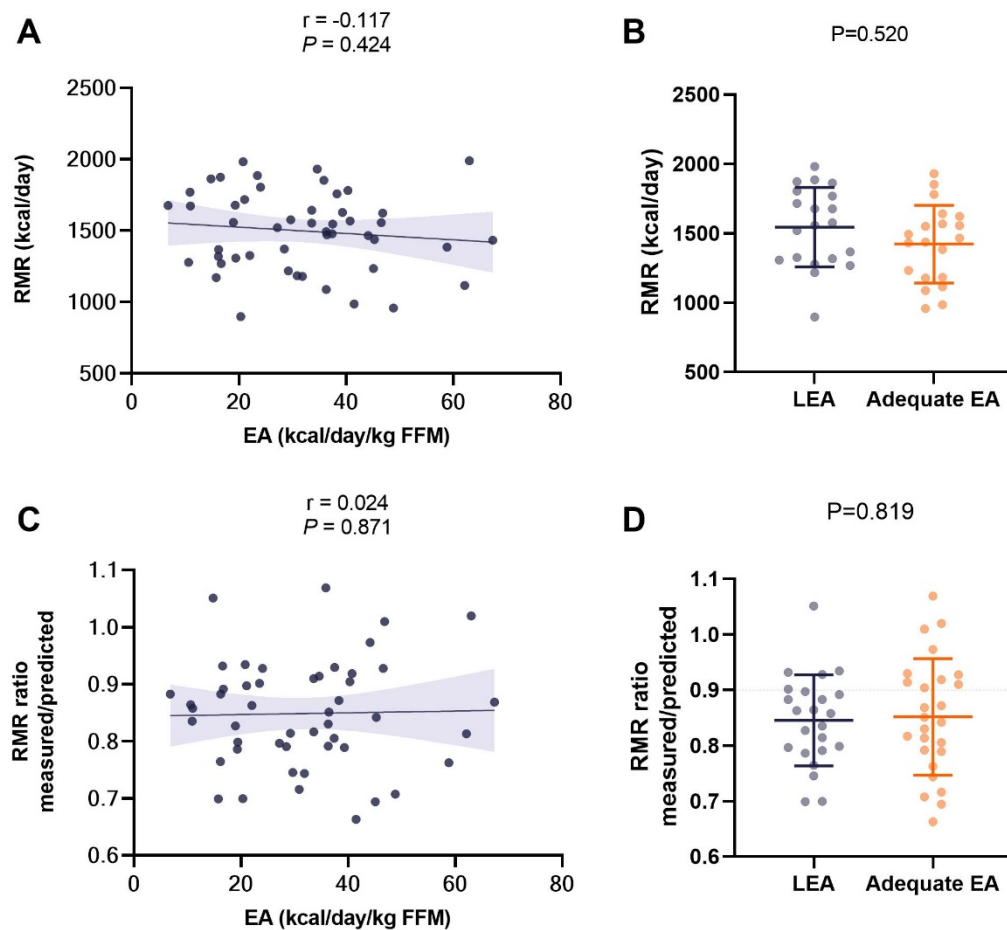


Figure 9. Relationship between RMR and RMR ratio measured/predicted with energy availability.

The RMR ratio measured/predicted was calculated using the ten Haaf (FFM) equation for the predicted RMR(186). Panels A and C display Pearson correlation analyses between RMR (A) and RMR ratio measured/predicted (C) with energy availability. Panels B and D represent differences in RMR (B) and RMR ratio measured/predicted (D) between participants with low energy availability and adequate energy availability. P values obtained from independent samples t-tests. In panel D, the dashed grey line indicates the 0.9 threshold for considering a high or low RMR ratio. Abbreviations: EA, energy availability; FFM, fat-free mass; LEA, low energy availability; RMR, resting metabolic rate.

5.2.4. Relationship between RMR_{ratio} and phenotypical characteristics

Given the potential predictive role of the RMR_{ratio} as a surrogate marker, we studied the association with different phenotypical parameters (**Figure 10A, B**, and **Figure 11A**), and participants were stratified into two groups according to their RMR_{ratio} based on the 0.9 threshold to compare the same parameters (**Figure 10C, D**, and **11B**).

Regarding body composition, the RMR_{ratio} was positively correlated with VAT mass ($r=0.317$, $P=0.026$; **Figure 10A** and **Table S4**). Similarly, individuals with RMR_{ratio} ≥ 0.9 presented significantly greater VAT mass ($P=0.003$), BMC ($P=0.042$), and BMD ($P=0.041$) compared to their counterparts with suppressed ratio (**Figure 10C** and **Table S5**).

We also observed a positive correlation between the RMR_{ratio} and moderate intensity physical activity ($r=0.34$; $P=0.029$; **Figure 10B** and **Table S4**). However, no statistically significant differences were observed between individuals with RMR_{ratio} higher or lower than 0.9 (all $P>0.05$; **Figure 10D** and **Table S5**).

In the sleep domain, we found that the RMR_{ratio} was negatively correlated with the time spent in sustained inactivity during waking hours of the night ($r=-0.332$, $P=0.034$; **Figure 10B** and **Table S4**) and for sustained inactivity bouts during night longer than 15 minutes ($r=-0.339$, $P=0.030$; **Figure 10B** and **Table S4**). Indeed, we also showed that individuals with RMR_{ratio} ≥ 0.9 presented lower time spent in sustained inactivity bouts during waking hours of the night ($P=0.002$) as well as the number of inactivity bouts ($P=0.009$) (**Figure 10D** and **Table S5**). Conversely, we revealed a positive correlation between the RMR_{ratio} and the sleep regularity index ($r=0.339$, $P=0.030$; **Figure 10B** and **Table S4**) and higher values of this index in individuals with RMR_{ratio} ≥ 0.9 than their counterparts ($P=0.022$; **Figure 10D** and **Table S5**).

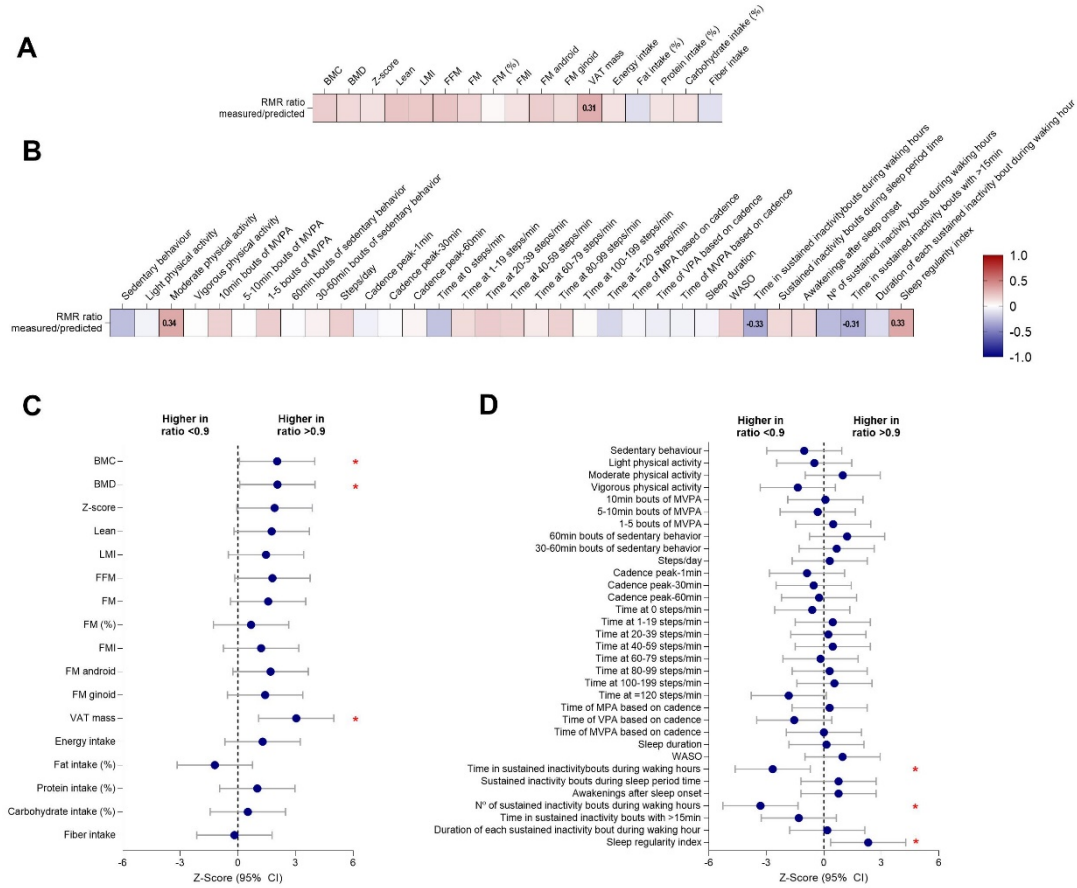


Figure 10. Relationship between RMR ratio measured/predicted and phenotypical traits (i.e., body composition, dietary outcomes, physical activity, sleep quantity and quality).

The RMR ratio measured/predicted was calculated using the ten Haaf (FFM) equation for the predicted RMR(186). Panels A and B: Heatmaps representing associations between energy availability and body composition (A), physical activity, sleep quantity and quality (B). The colors of the squares represent the Pearson correlation coefficients. Red colors represent positive Pearson coefficients, whereas blue depicts negative coefficients. Bold numbers inside the squares represent statistically significant Pearson correlation coefficients ($P < 0.05$). Panels C and D: Forest plots indicating the differences in body composition (C), and physical activity, sleep quantity and quality (D), between individuals with a ratio higher than 0.9 and individuals with a ratio lower than 0.9. Blue points indicate the z-score with the 95% confidence interval. Red asterisks indicate statistically significant differences between groups ($P < 0.05$). P values obtained from an independent samples *t*-test. Abbreviations: BMC, total body bone mineral content; BMD, total body bone mineral density; EA, energy availability; ECW, extracellular water; FFM, fat-free mass; FM, fat mass; FMI, fat mass index; LEA, low energy availability; LMI, lean mass index; MPA, moderate physical activity; MVPA, moderate to vigorous physical activity; VAT, visceral adipose tissue; VPA, vigorous physical activity; WASO, wake after sleep onset.

Regarding subjective and psychological variables (**Figure 11**), we showed a significant inverse correlation between the RMR_{ratio} and the ASPS score ($r=-0.384$, $P=0.006$), the ADAM-Q score ($r=-0.427$, $P=0.013$), the RESTQ-76 success ($r=-0.310$; $P=0.034$), and personal fulfillment ($r=-0.332$; $P=0.023$) scores (**Figure 11A** and **Table S4**). In addition, $RMR_{ratio} \geq 0.9$ reported lower general stress scores as measured by the RESTQ-76 ($P=0.024$; **Figure 11B** and **Table S5**).

Of note, no significant correlation or differences were observed in terms of dietary intake or macronutrient distribution (all $P>0.05$; **Figure 11** and **Tables 7-8**), and no significant relationship was observed between the RMR_{ratio} and phenotypical traits when accounting for multiple comparisons correction, adjusting for FDR (all $P>0.05$; **Tables S4-5**).

These analyses were similar when in the male subgroup (**Tables 7-8**).

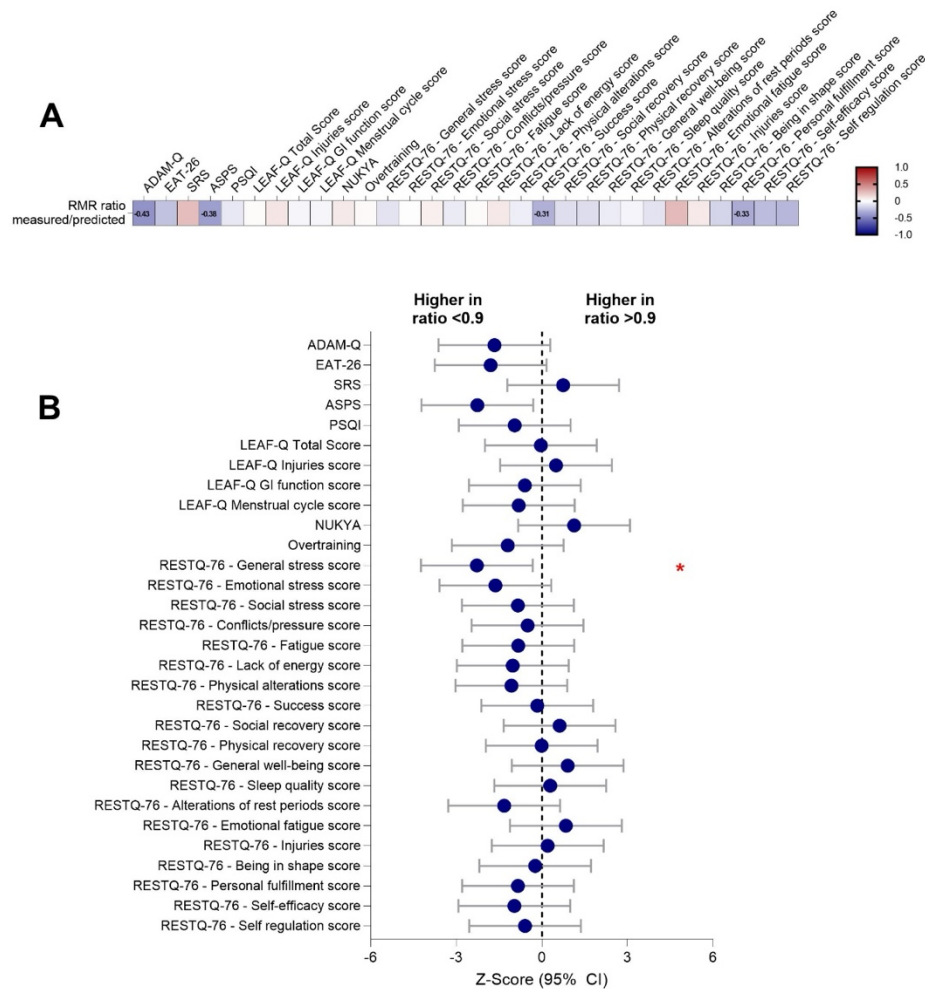


Figure 11. Relationship between RMR ratio measured/predicted and subjective well-being.

The RMR ratio measured/predicted was calculated using the ten Haaf (FFM) equation (186) for the predicted RMR. Panel A: Heatmap representing associations between energy availability and subjective well-being. The colors of the squares represent the Pearson correlation coefficients. Red colors represent positive Pearson coefficients, whereas blue depicts negative coefficients. Bold numbers inside the squares represent statistically significant Pearson correlation coefficients ($P < 0.05$). Panels B: Forest plot indicating the differences in subjective well-being between individuals with a ratio higher than 0.9 and individuals with a ratio lower than 0.9. Blue points indicate the z-score with the 95% confidence interval. Red asterisks indicate statistically significant differences between groups ($P < 0.05$). P values obtained from independent samples t-test. Abbreviations: ADAM-Q, Androgen Deficiency in the Aging Male Questionnaire; ASPS, Athlete's Subjective Performance Scale; EA, energy availability; EAT-26, Eating Attitudes Test-26; GI, gastrointestinal; LEA, low energy availability; LEAF-Q, Low Energy Availability in Females Questionnaire; NUKYA, Nutrition Knowledge for Young and Adult Athletes; PSQI, Pittsburgh Sleep Quality Index; RESTQ-76, Recovery-Stress Questionnaire for Athletes-76; SRS, Silhouette Rating Scale.

These analyses were similar when in the male subgroup (**Tables S9-10**).

5.3. MOVEMENT BEHAVIOURS, ENERGY AVAILABILITY AND PERCEIVED WELLNESS

5.3.1. Participants characteristics

Descriptive characteristics are shown in **Table 4**.

Table 4. Descriptive characteristics of the participants.

	All (n=49)	
	Mean	SD
Age (years)	25.0	5.9
Sex		
Male (n, %)	35	71.4
Female (n, %)	14	28.6
Weight (kg)	71.2	11.3
Height (cm)	173.8	8.2
BMI (kg/m ²)	23.5	2.7
Lean mass (kg)	53.6	9.5
Fat mass (%)	19.4	6.9
Training experience (years)	8.7	6.0
Competition level		
Regional (n,%)	23	46.9
National (n, %)	23	46.9

Abbreviations: BMI, body mass index.

5.3.2. Relationship between physical activity and energy availability

We observed a significant positive association between sedentary behaviour ($r = 0.38$, $P = 0.001$), time accumulated at 80–99 steps/min ($r = 0.36$, $P = 0.010$), and time accumulated at 100–199 steps/min ($r = 0.33$, $P = 0.019$) with EA (**Figure 12A** and **Table S9**). However, only sedentary behaviour was significantly associated with EA after correcting for FDR (**Figure 12A** and **Table S9**). No additional physical activity metrics demonstrated statistically significant associations with EA (**Figure 13A** and **Table S9**). Furthermore, comparative analysis between athletes classified with adequate EA ($\geq 30 \text{ kcal}\cdot\text{kg}^{-1} \text{ FFM}\cdot\text{day}^{-1}$) and those with LEA ($< 30 \text{ kcal}\cdot\text{kg}^{-1} \text{ FFM}\cdot\text{day}^{-1}$) revealed no significant between-group differences across physical activity variables (**Figure 12B** and **Table S10**), nor after adjusting for sex and sport classification (data not shown).

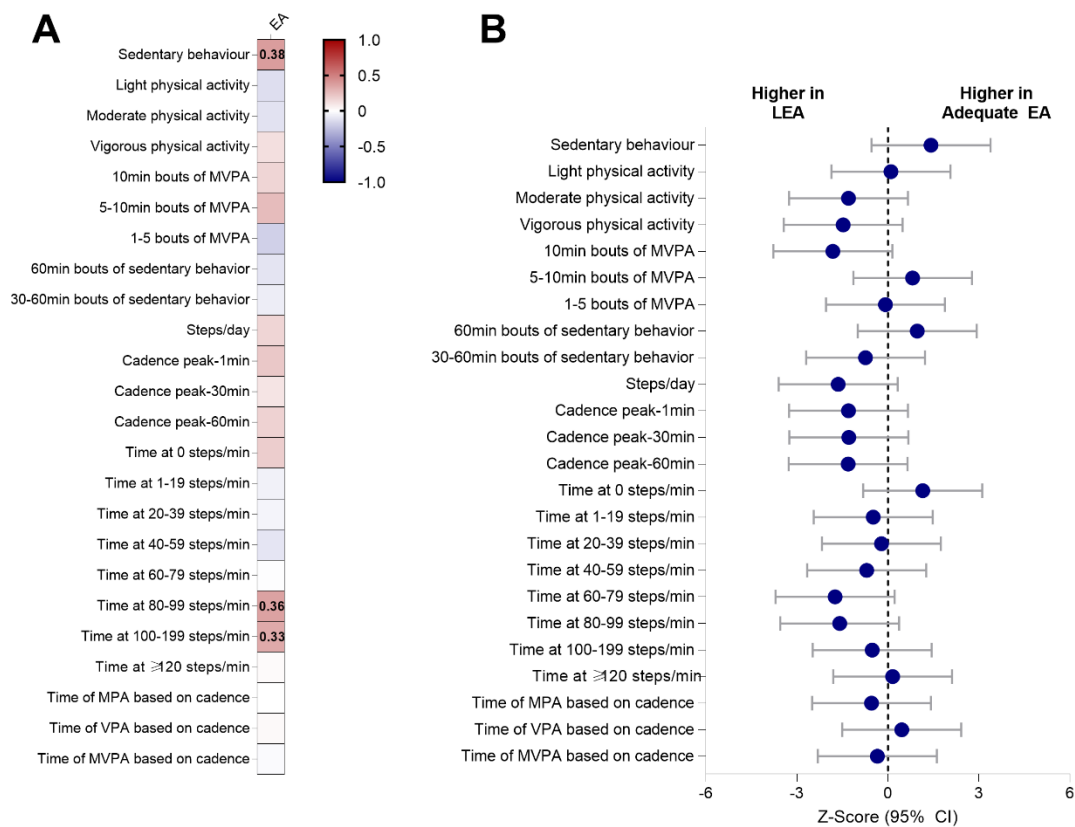


Figure 12. Relationship between physical activity and energy availability. Panel A: Heatmaps representing associations between physical activity and energy availability. The colors of the squares represent the Pearson correlation coefficients. Red colors represent positive Pearson coefficients, whereas blue depicts negative coefficients. Bold numbers inside the squares represent statistically significant Pearson correlation coefficients. Panel B: Forest plot indicating the differences in physical activity between participants with adequate energy availability (EA; ≥ 30 kcal/kg FFM/day) and participants with low energy availability (LEA; < 30 kcal/kg FFM/day). Blue points indicate the Z-scores with their corresponding 95% confidence intervals. Points placed to the left of the vertical axis indicate higher values in the adequate EA group, whereas points to the right indicate higher values in the LEA group. Red asterisks indicate statistically significant differences between groups. P values obtained from an independent samples t-test. Abbreviations: FDR, false discovery rate; MPA, moderate physical activity; MVPA, moderate-to-vigorous physical activity; VPA, vigorous physical activity.

The dose–response curves and the effect sizes relative to increasing MVPA, while proportionally reducing LPA, SB and sleep, did not show a significant

increase in EA ($P = 0.182$; **Figure 13A**). Similar non-significant results were found for increasing LPA, SB and sleep, while proportionally reducing the rest of movement behaviors respectively ($P > 0.242$; **Figures 13B-C**). The dose-response curves of the pairwise reallocation plots did not yield statistically significant associations with EA (**Figure S1**). These results remained non-significant after including individual covariates in the models (sex, age, sport classification), or all in pairwise combinations (data not shown).

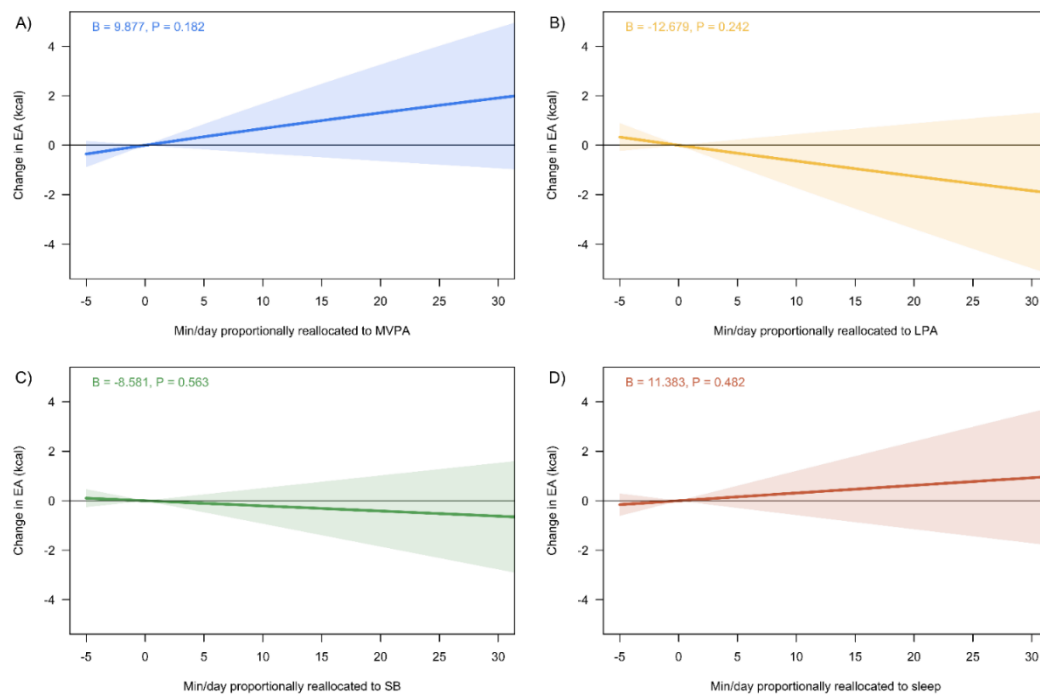


Figure 13. Predicted change in EA with time reallocation to specific movement behaviors.

(A) MVPA; (B) LPA; (C) SB; (D) Sleep. The lines represent the expected change in EA upon increasing the dominant behavior, while proportionally reducing the others. Compositional data analysis was used to model the impact of time-use shifts on EA (kcal/kg FFM/day). The lines represent the expected change in the EA upon increasing the dominant behavior, while proportionally reducing the others. Abbreviations: EA, Energy availability; MVPA, Moderate Vigorous Physical Activity; LPA, Light Physical Activity; SB, Sedentary Behaviour.

5.3.3. Relationship between physical activity and subjective well-being

Lastly, considering the role of physical activity patterns in recovery and health status, we explored their associations with subjective well-being outcomes. Sedentary behavior metrics showed positive associations with menstrual- and injury-related symptoms from the LEAF-Q (**Figure 14**). Specifically, total sedentary time ($r = 0.72$; $P = 0.004$), prolonged bouts of ≥ 60 minutes of sedentary behaviour ($r = 0.54$; $P = 0.046$), and time spent at 0 steps/min ($r = 0.55$; $P = 0.039$) correlated positively with the LEAF-Q menstrual cycle score (**Figure 14**), and 30–60 minutes of sedentary behaviour correlated positively with LEAF-Q injuries score ($r = 0.56$; $P = 0.039$). In addition, total sedentary time was positively related to the RESTQ-76 social recovery score ($r = 0.33$; $P = 0.027$), with a similar association observed for time at 0 steps/min ($r = 0.31$; $P = 0.039$) (**Figure 14**).

In contrast, LPA ($r = -0.63$; $P = 0.016$) and time accumulated at 40–59 steps/min ($r = -0.57$; $P = 0.034$) were inversely associated with LEAF-Q menstrual symptoms (**Figure 14**). Time spent at 60–79 steps/min correlated positively with self-rated performance on the ASPS ($r = 0.30$; $p = 0.037$) and negatively with LEAF-Q menstrual cycle score ($r = -0.54$; $P = 0.046$) (**Figure 14**).

No additional statistically significant associations were identified among the remaining physical activity parameters and well-being outcomes (**Figure 14**).

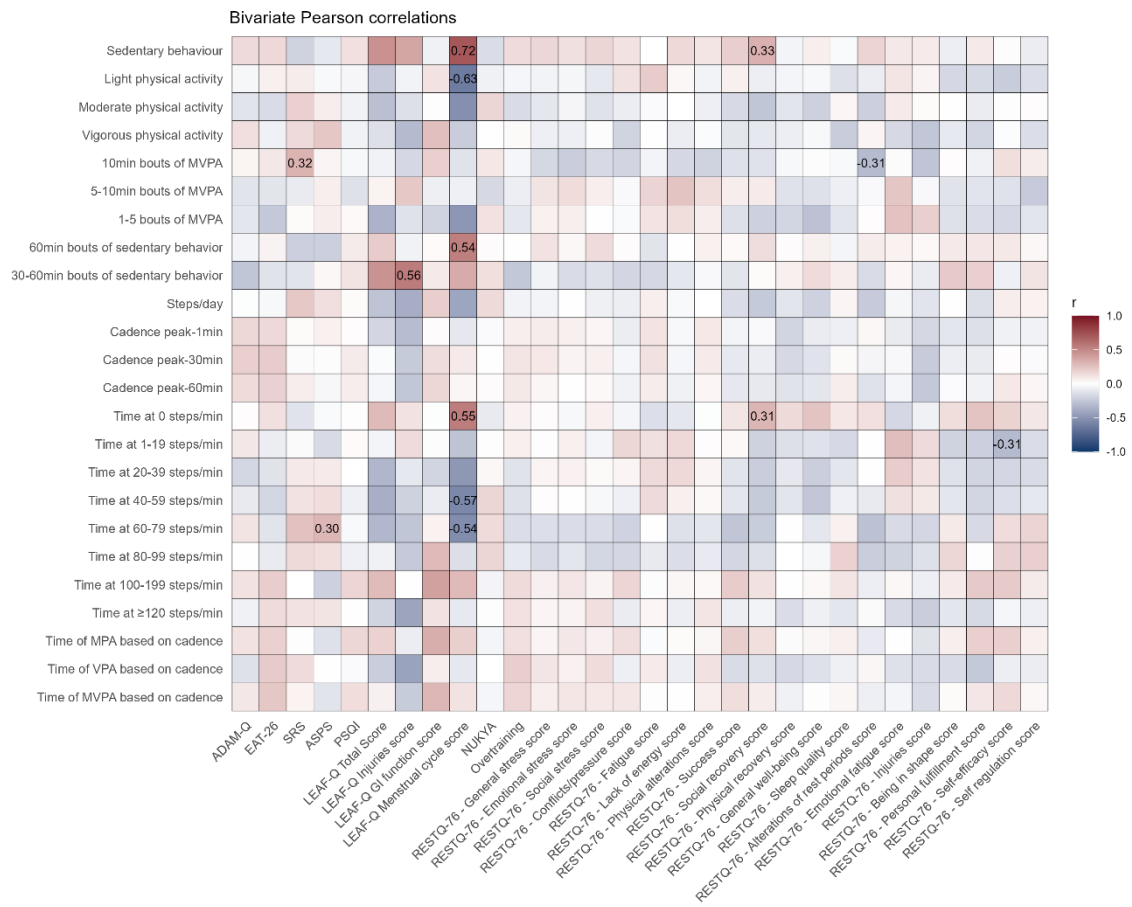


Figure 14. Relationship between physical activity and subjective well-being.

All the above-mentioned findings were consistent after adjusting for EA (**Figure S2**) or when the associations were performed separately in participants with adequate EA or with LEA (**Figure S3**).

VI – DISCUSSION

VI -DISCUSSION

6.1. GENERAL OVERVIEW OF THE MAIN FINDINGS

This Doctoral Thesis examined LEA using an integrative and multidimensional framework that combined physiological, metabolic, behavioural, and perceptual parameters in elite and pre-elite athletes. Across all analytical domains, the findings consistently challenge simplified approaches that rely on isolated markers to diagnostic LEA.

Overall, body composition, RMR/RMRratio, and daily movement behaviours showed limited or inconsistent associations with calculated EA. However, several of these variables were meaningfully related to symptom burden, perceived well-being, recovery, and health-related outcomes. Taken together, these findings indicate that LEA does not manifest as a discrete or uniform physiological state, but rather as a dynamic and individually modulated condition shaped by the interaction between training demands, nutritional adequacy, behavioural adaptations, and psychological context.

These results reinforce the conceptualization of LEA as a continuum rather than a binary condition defined by a fixed threshold, highlight the limitations of single-domain screening strategies. The following sections discuss each analytical domain in detail and situate the findings within the current literature.

6.2. BODY COMPOSITION AND LEA-RELATED SYMPTOMATOLOGY

In this study, we show that body composition parameters (i.e., adipose, lean, and bone) are not associated with the risk of low testosterone symptoms in male athletes and the risk of the Female Athlete Triad in female athletes. However, we observed that male athletes reporting symptoms of low testosterone had significantly greater lean mass compared to those without symptoms, and adiposity (i.e., fat mass percentage and VAT mass) was significantly positively correlated with LEAF-Q injury subscale score in female athletes. Our findings

provide valuable insights into how different components of body composition may be associated with hormonal function or performance outcomes in elite and pre-elite athletes.

6.2.1. Body Composition and Low Testosterone Symptoms in Male Athletes

We observed that most adipose indices were not significantly associated with symptoms of low testosterone scores. However, this finding differs from the results of Hagmar et al. (2013), who suggest that body fat levels and hormonal disturbances are correlated in athletes. Their study found that while total testosterone levels were similar across athletes with varying body fat percentages, leanest athletes showed lower levels of free testosterone, indicating a possible impact of low body fat on the bioavailable form of testosterone (209). Additionally, Hagmar et al. reported that serum levels of free testosterone were positively correlated with levels of IGF-I and the ratio of IGF-I to IGF binding protein-1, biomarkers associated with nutritional status. These biomarkers, along with sex hormone binding globulin, strongly positively correlated with body fat content, suggesting a link between adiposity and hormonal regulation through nutritional and metabolic pathways.

In addition to the findings on adipose indices, our study did not observe any significant relationships between bone health biomarkers (including BMC, BMD, and BMD Z-score) and symptoms of low testosterone. Although bone parameters did not differ significantly among participants with symptoms of low testosterone, it is plausible that the mechanical loading associated with high-impact or resistance-based training may have exerted a protective effect on bone mineral density (210). However, evidence also shows that prolonged LEA can compromise bone remodeling by impairing estrogen/testosterone and IGF-1 activity, ultimately leading to lower BMD, even in athletes (29,173).

Lean mass indices measured by both DXA and BIA, such as total lean mass, LMI, FFM, and muscle mass, showed significant positive associations with ADAM-Q scores. Furthermore, athletes reporting symptoms of low testosterone had significantly greater lean mass and higher thorax and thigh circumferences than those without symptoms. These findings imply that higher lean mass may be

linked to increased reporting of low testosterone symptoms among male athletes. A possible explanation is that athletes with greater lean mass may engage in more intense training regimens without sufficient recovery or nutritional support, potentially leading to a state of relative energy deficiency (211). This condition maintained over time can disrupt hormonal balance, including testosterone levels, despite high lean mass (211). Additionally, excessive training stress may suppress the hypothalamic-pituitary-gonadal axis, resulting in reduced testosterone production (30,212). Our results highlight the need for balanced training programs that consider recovery and nutritional strategies to maintain hormonal health in male athletes.

6.2.2. Body Composition and the Risk of the Female Athlete Triad in Female Athletes

In female athletes, most body composition variables, including adipose, lean, and bone parameters, were not significantly correlated with overall LEAF-Q scores, which assess the risk of the Female Athlete Triad. This lack of association suggests that factors beyond body composition, such as nutritional status or psychological stress, may play a role in the risk of developing the Female Athlete Triad (30). While body composition is often considered an essential factor in athlete health (213) our findings suggest that it may not be a direct predictor of low energy availability in this population.

However, significant positive associations were found between fat mass percentage, VAT mass, and the LEAF-Q injury score. This findings indicate that while adiposity may not be directly linked to the overall risk of the Female Athlete Triad, it may contribute to specific components such as injury susceptibility. Higher VAT mass is often associated with metabolic disturbances, low-grade systemic inflammation, and mechanical overloads which could increase injury risk (214). These results emphasize the importance of monitoring fat distribution and metabolic health in female athletes to prevent injuries.

6.2.3. Potential Implications for Athlete Health and Performance

The associations observed in this study underscore the complexity of the relationship between body composition and hormonal health in athletes. For male athletes, the positive correlation between lean mass and low testosterone symptoms highlights the potential impact of training load and recovery on hormonal balance. Coaches and sports health professionals should ensure that training programs are periodized appropriately and that athletes receive adequate nutritional support to prevent hormonal disruptions.

In female athletes, the link between higher fat mass percentage, VAT mass, and injury risk emphasizes the need to consider fat distribution in injury prevention strategies. While optimizing body composition is important for performance, it should not compromise health. Tailored nutritional interventions and training modifications may be necessary to address these risks without adversely affecting EA.

6.3. ENERGY AVAILABILITY AND METABOLIC FUNCTION

While the RMR_{ratio} has been proposed as a non-invasive marker of metabolic suppression and LEA, this thesis provides a broader perspective. We demonstrated that the choice of predictive equation substantially affects RMR_{ratio} estimation and, consequently, its potential diagnostic role. Moreover, we found no significant associations between RMR or RMR_{ratio} and EA, nor differences between athletes classified as having LEA or adequate EA. However, athletes with $RMR_{ratio} \geq 0.9$ exhibited greater bone mass, and exploratory analyses suggested potential links with certain behavioural and psychological traits, although these did not remain significant after correction for multiple comparisons. Taken together, these findings indicate that the RMR_{ratio} reflects multiple facets of athlete health and performance but has limited utility as a standalone marker, highlighting the need for its integration into multifactorial assessment models.

6.3.1. Methodological sensitivity of RMR_{ratio} calculation to predictive equations

A key methodological finding of this investigation is the strong influence of the predictive equation used to estimate RMR on the resulting RMR_{ratio} and subsequent athlete classification. These results concur with previous studies showing that there is a different accuracy in the estimation of RMR depending on the equation used(215) which ultimately impacts the calculation of RMR_{ratio}. Consequently to this methodological issue, the proportion of athletes identified as metabolically suppressed ranged from 34.7% to 93.9% depending on the predictive equation used. Our results are in line with those from a previous study showing that the interpretation of the RMR_{ratio} is directly driven by the predictive equation chosen, with a prevalence of low RMR_{ratio} ranging from 1% to 68% (216). Consequently, Heikura et al. concluded that current generic equations are inadequate for REDs screening and advocated the development of sport- and phenotype-specific models with adaptable RMR_{ratio} thresholds.

This wide variability underscores the importance of standardizing predictive models when using RMR_{ratio} as a diagnostic or screening tool. Equations such as Freire or Van Hooren, which incorporate recent physiological assumptions or are derived from athletic populations(189,217), tended to produce lower predicted values, classifying a much higher proportion of athletes as suppressed. Thus, without a validated gold-standard prediction model specific to athletes, interpretation of the RMR_{ratio} remains vulnerable to methodological inconsistency.

6.3.2. RMR and RMR_{ratio} as inadequate as markers of EA

Contrary to previous studies that reported a reduction in RMR or RMR_{ratio} in athletes with LEA (140,218), our findings showed no significant differences in RMR or RMR_{ratio} between LEA and adequate EA groups. These discrepancies may be attributed to several factors. First, in our study EA was estimated using 24-hour dietary recalls and self-reported physical activity logs, which, despite being widely used, are subject to recall bias and underreporting, especially in athletic populations. Second, EA represents a dynamic and often fluctuating construct, and single-point measurements may not adequately capture chronic energetic status

(219,220). Lastly, individual variability in metabolic adaptation could result in some athletes maintaining relatively high RMR despite sustained periods of low EA, further complicating the interpretation of these markers (221).

LEA is a critical risk factor for developing the REDs (106). In this sense, a recent study found that 27.9% of athletes identified as moderate or high risk for REDs exhibited LEA based on $\text{RMR}_{\text{ratio}}$ (222). However, that study inferred energy deficiency from RMR-based metrics alone, without directly measuring EA or applying a validated clinical assessment for REDs. This highlights the ongoing uncertainty around the diagnostic validity of RMR and $\text{RMR}_{\text{ratio}}$ in the context of energy deficiency.

Our findings also contrast with those of Garay et al. (2025), who examined the relationship between predicted energy availability and $\text{RMR}_{\text{ratio}}$ in physically active college-aged females. Their results suggest that a low $\text{RMR}_{\text{ratio}}$ may be a more sensitive marker of energy deficiency than calculated EA, depending on the protocols used (223).

Although our study was not designed to diagnose or monitor REDs, the findings contribute to the ongoing debate on the role of RMR/ $\text{RMR}_{\text{ratio}}$ in this context. Recent consensus stresses the importance of integrating multiple objective indicators when assessing energy deficiency in athletes(175). In line with these recommendations, our results suggest that RMR and $\text{RMR}_{\text{ratio}}$ should not be used in isolation, as they may not reflect actual energy availability. Instead, they could serve as complementary tools when used in conjunction with standardized clinical assessments.

6.3.3. The RMR ratio as a physiological marker beyond EA

Although the $\text{RMR}_{\text{ratio}}$ was not associated with EA, it did demonstrate relationships with several indicators of physiological and psychological status. In this study, higher $\text{RMR}_{\text{ratio}}$ was associated with greater bone mineral content and density and more favorable patterns of sleep and activity, suggesting that this ratio may reflect broader systemic health. Notably, athletes with RMR ratios ≥ 0.9 also reported lower general stress and higher subjective performance. The observed associations between the $\text{RMR}_{\text{ratio}}$ and variables such as sleep behavior, sedentary

patterns, and psychological stress highlight the importance of integrating behavioural and psychosocial factors into the investigation of LEA and REDs. Additionally, the observed positive association between the RMR ratio and bone-related metrics (including BMD and BMC) aligns with previous research indicating that metabolic suppression may adversely affect bone remodeling and mineralization, particularly among physically active individuals (224).

These findings align with previous literature proposing that RMR suppression may reflect a broader metabolic adaptation to cumulative stressors—nutritional, physical, or psychological (3,225). Thus, the RMR_{ratio} may function less as a direct proxy for EA and more as an integrative marker of energy balance, behavioural regularity, and overall physiological adaptations. However, our results should be interpreted with caution since no significant relationships were observed when accounting for multiple comparisons.

6.4. MOVEMENT BEHAVIOURS, ENERGY AVAILABILITY, AND PERCEIVED WELLNESS

The present investigation set out to examine whether variations in daily movement behaviour are related to EA in free-living elite and pre-elite athletes. Contrary to our initial hypothesis, no significant association was found between time spent at any intensity of physical activity (LPA or MVPA) and EA. Compositional data analysis of 24 h behaviours further confirmed that reallocating time between activity categories (e.g., replacing SB with LPA, or MVPA) did not yield meaningful differences in EA. Collectively, these results suggest that EA is more strongly influenced by the adequacy of dietary intake relative to overall energy demands than by the specific distribution of time across daily physical activity behaviours.

This interpretation aligns with the constrained energy expenditure model (226) which proposes that increments in activity-related expenditure do not produce proportional increases in TDEE due to compensatory downregulation of other metabolic processes (227,228). Such adaptations have been observed not only in hunter-gatherer populations such as the Hadza, but also in athletes exposed to high training loads (229). In this context, increases in exercise-induced energy expenditure are often accompanied by downregulation of other physiological

processes, including reductions in basal metabolic rate, alterations in immune function, and suppression of reproductive hormones(106,230). Within this framework, our findings suggest that higher daily movement does not systematically reduce EA. As observed in other studies, the body compensates by adjusting energy allocated to reproduction, immunity, and cellular maintenance (228,231).

Interestingly, athletes who spent more time in SB showed slightly higher EA values. Although counterintuitive, this pattern is consistent with the well-described failure of appetite to increase *ad libitum* energy intake in compensation for exercise energy expenditure; thus, more active athletes may not spontaneously raise intake sufficiently, resulting in lower calculated EA unless intake is deliberately adjusted (232). Another possible explanation might be that EA is primarily determined by dietary intake, with physical activity playing a secondary role in shaping its variability(9,22,35,42). Nevertheless, these interpretations should be considered with caution due to the limitations of self-reported dietary intake assessment in athletes, particularly underreporting, which can obscure true associations (233).

Beyond EA *per se*, daily movement behaviours were related to subjective health outcomes. Female athletes engaging in greater amounts of LPA (particularly at 40–79 steps·min⁻¹) reported fewer menstrual-related symptoms, whereas those with higher sedentary time showed poorer LEAF-Q scores. These findings resonate with the literature on the anti-inflammatory effects of low-intensity physical activity and the enhanced modulation of prostaglandin activity in women (234–236).

Building on this perspective, it may be more appropriate to conceptualize EA as a multidimensional construct, determined not only by the magnitude of the energy deficit but also by its temporal duration. Severe but very brief restrictions may pass without overt symptoms(120), whereas more moderate but sustained deficits can progressively impair reproductive and skeletal health (22). Thus, it may not be only the absolute level of EA that matters, but also the length of the exposure. Moreover, substantial inter-individual variability exists: some athletes may display hormonal or performance-related disturbances at EA levels above the conventional 30 kcal/kg day threshold(88), while others remain asymptomatic even below this

cut-off. This spectrum-based view challenges the notion of a single static threshold and highlights the need for more nuanced, context-dependent evaluations of Relative Energy Deficiency in Sport (REDs) risk.

Overall, our findings suggest that the distribution of daily movement behaviours may have limited influence on EA under free-living conditions, whereas energy intake appears to be the more decisive factor. At the same time, movement patterns appear to modulate menstrual symptoms and subjective health outcomes, highlighting their complementary role in athlete care. Future research should further explore the complex relationship between NEAT and energy availability in athletes. Rather than relying on isolated markers or assuming linear dynamics between movement behavior and energetic status, future models should embrace more integrative, longitudinal monitoring approaches. Importantly, these findings suggest the need to move beyond the notion of a fixed EA threshold. It may be more appropriate to conceptualize LEA as existing along a functional–pathological spectrum, where the emergence of REDs symptoms depends on individual tolerance, metabolic compensation, and the duration of energy mismatch. Exploring this spectrum could support the development of more personalized diagnostic tools and intervention strategies, including the potential support of an “adaptable LEA” state, in which athletes exhibit subclinical energy deficits without overt physiological disruption (48).

VII – CONCLUSION

VII - CONCLUSION

7.1. GENERAL CONCLUSION

This Doctoral Thesis demonstrates that LEA is a complex and multifaceted phenomenon that cannot be adequately captured through isolated physiological, behavioural, or metabolic indicators. Across the three studies, the findings reveal that body composition, RMR, daily movement patterns, and subjective perceptions each provide only partial insight into the energetic status of competitive athletes. Taken together, the results emphasize that LEA should be conceptualized as a dynamic continuum of individual adaptations rather than a static state defined by a fixed numerical threshold. This work underscores the need for multidomain, personalized, and context-sensitive frameworks for evaluating and managing REDs in sport, offering evidence that can contribute to more precise identification and better prevention of its health and performance consequences.

7.2. SPECIFIC CONCLUSIONS

7.2.1. Conclusion I

No consistent associations were found between adipose or bone indices and symptoms of testosterone deficiency in males or Female Athlete Triad risk in females. Nonetheless, modest yet noteworthy patterns emerged: lean mass was positively associated with hypogonadal symptom scores in men, while higher levels of adiposity (particularly visceral fat) correlated with greater injury-related risk in women. These findings highlight the importance of interpreting body composition metrics within a multidimensional assessment framework.

7.2.2. Conclusion II

Our findings indicate that the predictive equation selected for estimating RMR significantly influences the RMR_{ratio} , thereby limiting its diagnostic consistency. In addition, although RMR and RMR_{ratio} were not associated with EA,

the RMR_{ratio} was related to several physiological, behavioral, and psychological traits in competitive athletes. In line with recent REDs CAT2 recommendations, our results reinforce that RMR and RMR_{ratio} should not be used in isolation but rather as complementary tools within multifactorial models for athlete monitoring. Future studies should explore the longitudinal predictive value of the RMR_{ratio} and its integration with other objective indicators to improve the assessment of energy deficiency risk.

7.2.3. Conclusion III

This thesis provides evidence that objective physical activity is not related to EA in free-living elite and pre-elite athletes. However, habitual activity patterns were related to subjective well-being, underscoring the dissociation between energy balance and health perception. We suggest that EA should be considered less as a binary state and more as a continuum of risk influenced by the magnitude, duration, and physiological context of energetic deficits. Further research is required to operationalize this continuum, providing practitioners with tools to anticipate dysfunction before overt REDs develop.

VIII – LIMITATIONS AND FUTURE RESEARCH



VIII - LIMITATIONS AND FUTURE RESEARCH

8.1. LIMITATIONS

Several limitations should be acknowledged when interpreting the findings of this Doctoral Thesis. The cross-sectional design employed across the three studies restricts the ability to establish causal relationships between EA, metabolic function, daily movement behaviors, and the physiological or psychological outcomes observed. LEA is inherently dynamic, and cross-sectional snapshots may not adequately reflect temporal fluctuations or cumulative effects of energy deficiency over extended periods. The estimation of EA under free-living conditions also remains constrained by methodological challenges. Self-reported dietary intake is prone to underestimation, while EE is sensitive to daily variability in training intensity and individual differences in movement patterns. Although validated procedures were followed, these sources of error likely introduced variability that may have attenuated associations with physiological and behavioural markers.

A further limitation relates to sample characteristics. Although both male and female athletes were included, the proportion of women was modest, and the multifactorial nature of menstrual disturbances may require larger, sex-specific samples to fully capture variability in hormonal outcomes.

The absence of biochemical markers (such as endocrine hormones) represents another limitation. While the thesis incorporated anthropometric, metabolic, and psychometric assessments, the inclusion of biochemical data would have enabled a more direct evaluation of physiological pathways affected by LEA.

8.2. FUTURE RESEARCH

Future research should prioritize longitudinal and multi-phase study designs to better capture the temporal dynamics of LEA and its cumulative effects on metabolic, endocrine, psychological, and performance-related outcomes. Longitudinal monitoring would help clarify individual trajectories of adaptation or maladaptation to energy deficiency and would enable the identification of early-

warning markers that precede clinically relevant dysfunction. Moreover, methodological advances in the assessment of EI and EEE, represent promising avenues for improving the accuracy and feasibility of EA assessment in free-living conditions.

Further studies should also explore the interactive roles of training periodization, recovery strategies, sleep, and psychological stress in shaping athletes' tolerance to low or fluctuating energy availability. Given the findings of this thesis on the relevance of sedentary behavior and low-intensity ambulation, future research would benefit from investigating how the organization of daily movement patterns modulates physiological resilience or vulnerability to LEA.

Expanding research across diverse sporting populations is also essential. Athletes in aesthetic sports, weight-category sports, ultra-endurance disciplines, and combat sports may exhibit distinct profiles and adaptive capacities. Similarly, female athletes require dedicated research, particularly regarding menstrual function, hormonal variability, and within-day energy balance. Integrating biochemical markers and psychophysiological measures would enable more multidimensional phenotyping and more precise identification of at-risk individuals. Ultimately, future investigations should aim to develop integrative diagnostic algorithms that combine metabolic, behavioural, hormonal, and perceptual data to improve the early detection, personalised management, and prevention of LEA and its associated health consequences.

IX - REFERENCES

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X – ANNEXES

X - ANNEXES

Annexe 1. Table S1: Spearman's correlation between body composition, and ADAM-Q and LEAF-Q scores.

	ADAM-Q score (n = 48)			LEAF-Q score (n = 23)			LEAF-Q injuries score (n = 23)			LEAF-Q gastrointestinal function score (n = 23)			LEAF-Q menstrual function and use of contraceptives score (n = 23)		
	ρ	95% CI	P	ρ	95% CI	P	ρ	95% CI	P	ρ	95% CI	P	ρ	95% CI	P
Weight (kg)	0.151	(-0.147; 0.424)	0.305	-0.089	(-0.494; 0.347)	0.685	0.119	(-0.320; 0.516)	0.590	-0.121	(-0.517; 0.318)	0.582	-0.371	(-0.686; 0.061)	0.081
Height (m)	0.048	(-0.247; 0.336)	0.745	-0.180	(-0.560; 0.283)	0.411	-0.093	(-0.496; 0.344)	0.674	-0.138	(-0.530; 0.303)	0.531	-0.238	(-0.600; 0.206)	0.275
BMI (kg/m ²)	0.210	(-0.087; 0.473)	0.152	0.085	(-0.350; 0.491)	0.698	0.317	(-0.123; 0.652)	0.141	-0.016	(-0.436; 0.410)	0.943	-0.292	(-0.636; 0.150)	0.177
Fat mass (kg) - DXA	0.021	(-0.272; 0.311)	0.886	0.044	(-0.386; 0.459)	0.841	0.317	(-0.122; 0.653)	0.140	-0.073	(-0.481; 0.361)	0.741	-0.323	(-0.656; 0.115)	0.132
Fat mass (kg) - BIA	-0.054	(-0.341; 0.241)	0.713	0.063	(-0.369; 0.474)	0.774	0.353	(-0.082; 0.675)	0.098	-0.101	(-0.503; 0.336)	0.646	-0.300	(-0.641; 0.141)	0.165
Fat mass (%) - DXA	-0.074	(-0.358; 0.223)	0.617	0.237	(-0.207; 0.600)	0.277	0.427	(0.005; 0.720)	0.042	0.061	(-0.371; 0.472)	0.781	-0.151	(-0.539; 0.291)	0.492
Fat mass (%) - BIA	-0.151	(-0.425; 0.147)	0.304	0.131	(-0.309; 0.525)	0.552	0.410	(-0.015; 0.710)	0.052	-0.021	(-0.440; 0.405)	0.923	-0.274	(-0.625; 0.168)	0.206
FMI (kg/m ²) - DXA	0.037	(-0.258; 0.325)	0.804	0.100	(-0.337; 0.502)	0.651	0.329	(-0.109; 0.660)	0.125	-0.008	(-0.430; 0.416)	0.971	-0.272	(-0.623; 0.171)	0.210
Android fat mass (kg)	0.112	(-0.186; 0.391)	0.448	0.137	(-0.303; 0.529)	0.533	0.385	(-0.046; 0.695)	0.070	0.001	(-0.422; 0.424)	0.996	-0.234	(-0.597; 0.210)	0.284
Gynoid fat mass (kg)	0.022	(-0.272; 0.312)	0.882	0.052	(-0.379; 0.465)	0.813	0.302	(-0.138; 0.643)	0.161	-0.049	(-0.462; 0.382)	0.826	-0.316	(-0.652; 0.123)	0.142
VAT mass (g)	0.256	(-0.039; 0.510)	0.079	0.186	(-0.257; 0.564)	0.396	0.438	(0.018; 0.726)	0.037	-0.081	(-0.488; 0.354)	0.712	-0.091	(-0.495; 0.345)	0.680
Adipose mass (kg)	0.000	(-0.292; 0.292)	0.998	0.036	(-0.393; 0.452)	0.870	0.202	(-0.241; 0.576)	0.354	-0.024	(-0.443; 0.403)	0.912	-0.270	(-0.622; 0.172)	0.212
Sum of six skinfolds (mm)	0.008	(-0.285; 0.299)	0.957	0.144	(-0.297; 0.535)	0.511	0.297	(-0.144; 0.640)	0.168	0.113	(-0.326; 0.511)	0.608	-0.221	(-0.589; 0.222)	0.310
Sum of eight skinfolds (mm)	0.020	(-0.273; 0.310)	0.891	0.162	(-0.280; 0.547)	0.460	0.322	(-0.117; 0.655)	0.135	0.113	(-0.326; 0.511)	0.609	-0.204	(-0.577; 0.240)	0.351
Adipose to muscle index	-0.169	(-0.440; 0.129)	0.250	0.098	(-0.339; 0.500)	0.658	0.189	(-0.254; 0.567)	0.388	0.003	(-0.420; 0.425)	0.989	-0.105	(-0.506; 0.332)	0.632
Lean mass (kg)	0.218	(-0.079; 0.479)	0.137	-0.109	(-0.508; 0.329)	0.621	-0.035	(-0.451; 0.393)	0.873	-0.093	(-0.496; 0.344)	0.674	-0.300	(-0.641; 0.141)	0.165
LMI (kg/m ²)	0.240	(-0.056; 0.497)	0.100	-0.108	(-0.508; 0.330)	0.623	-0.037	(-0.453; 0.392)	0.865	-0.099	(-0.501; 0.338)	0.652	-0.296	(-0.639; 0.145)	0.170
FFM (kg) - DXA	0.212	(-0.085; 0.475)	0.148	-0.098	(-0.500; 0.339)	0.655	-0.019	(-0.439; 0.407)	0.930	-0.086	(-0.491; 0.350)	0.698	-0.294	(-0.638; 0.147)	0.173
FFM (kg) - BIA	0.306	(0.015; 0.549)	0.035	-0.152	(-0.540; 0.290)	0.489	-0.039	(-0.454; 0.390)	0.860	-0.137	(-0.529; 0.303)	0.533	-0.318	(-0.653; 0.121)	0.140
Muscle mass (kg) - BIA	0.306	(0.015; 0.549)	0.035	-0.152	(-0.540; 0.290)	0.489	-0.039	(-0.454; 0.390)	0.860	-0.137	(-0.529; 0.303)	0.533	-0.318	(-0.653; 0.121)	0.140
Muscle mass (kg) - Anthropometry	0.188	(-0.110; 0.455)	0.200	-0.059	(-0.470; 0.373)	0.789	0.091	(-0.346; 0.495)	0.681	-0.056	(-0.468; 0.376)	0.801	-0.245	(-0.605; 0.199)	0.261
Corrected arm circumference (cm)	0.164	(-0.135; 0.435)	0.267	-0.125	(-0.520; 0.314)	0.569	-0.021	(-0.440; 0.406)	0.926	-0.093	(-0.496; 0.344)	0.674	-0.170	(-0.553; 0.272)	0.438
Corrected ~~taxa~~ circumference (cm)	0.247	(-0.048; 0.503)	0.090	-0.011	(-0.432; 0.414)	0.961	0.251	(-0.192; 0.609)	0.247	-0.018	(-0.438; 0.408)	0.934	-0.259	(-0.614; 0.184)	0.233
Corrected thigh circumference (cm)	0.239	(-0.057; 0.497)	0.102	-0.007	(-0.429; 0.417)	0.973	0.114	(-0.325; 0.512)	0.605	0.181	(-0.262; 0.561)	0.408	-0.436	(-0.725; -0.016)	0.037
Corrected calf circumference (cm)	0.130	(-0.168; 0.407)	0.378	-0.122	(-0.518; 0.317)	0.580	-0.066	(-0.476; 0.367)	0.763	-0.148	(-0.538; 0.293)	0.499	-0.088	(-0.493; 0.348)	0.689
Muscle to bone index	-0.014	(-0.305; 0.279)	0.924	-0.059	(-0.470; 0.373)	0.790	-0.018	(-0.437; 0.408)	0.936	0.037	(-0.392; 0.453)	0.865	-0.135	(-0.528; 0.305)	0.539
Residual mass (kg)	0.096	(-0.202; 0.378)	0.516	-0.294	(-0.638; 0.147)	0.173	-0.198	(-0.573; 0.246)	0.366	-0.407	(-0.708; 0.019)	0.054	-0.199	(-0.573; 0.245)	0.363
BMC (kg)	0.100	(-0.198; 0.381)	0.499	-0.121	(-0.517; 0.319)	0.584	-0.019	(-0.438; 0.407)	0.932	-0.022	(-0.441; 0.405)	0.921	-0.259	(-0.614; 0.184)	0.233
BMD (g/cm ³)	0.045	(-0.251; 0.332)	0.763	-0.095	(-0.498; 0.341)	0.665	-0.035	(-0.451; 0.393)	0.873	0.023	(-0.404; 0.441)	0.918	-0.100	(-0.502; 0.337)	0.650
BMD Z-score	0.045	(-0.250; 0.333)	0.759	-0.046	(-0.460; 0.384)	0.834	0.028	(-0.400; 0.445)	0.900	0.053	(-0.378; 0.466)	0.809	-0.077	(-0.484; 0.358)	0.728
Bone mass (kg)	0.237	(-0.059; 0.495)	0.105	-0.079	(-0.486; 0.355)	0.719	0.047	(-0.383; 0.461)	0.830	-0.159	(-0.546; 0.283)	0.467	-0.215	(-0.585; 0.228)	0.324
Total body water (kg)	0.223	(-0.074; 0.483)	0.128	0.082	(-0.353; 0.488)	0.709	0.262	(-0.181; 0.616)	0.228	0.006	(-0.418; 0.428)	0.978	-0.223	(-0.590; 0.221)	0.307
Total body water (%)	0.013	(-0.280; 0.304)	0.931	0.125	(-0.315; 0.520)	0.570	-0.144	(-0.534; 0.297)	0.512	0.178	(-0.265; 0.559)	0.417	0.384	(-0.046; 0.694)	0.070
Extracellular water (kg)	0.180	(-0.118; 0.449)	0.221	-0.019	(-0.438; 0.407)	0.932	0.153	(-0.288; 0.541)	0.485	-0.033	(-0.449; 0.396)	0.882	-0.302	(-0.643; 0.139)	0.161
Intracellular water (kg)	0.287	(-0.005; 0.534)	0.048	0.092	(-0.344; 0.496)	0.675	0.255	(-0.188; 0.612)	0.240	-0.075	(-0.483; 0.359)	0.732	-0.094	(-0.497; 0.342)	0.669
Phase angle (°)	-0.169	(-0.439; 0.130)	0.252	-0.142	(-0.533; 0.299)	0.518	-0.195	(-0.571; 0.249)	0.373	-0.039	(-0.455; 0.390)	0.858	0.102	(-0.335; 0.503)	0.642
Endomorphy	0.014	(-0.279; 0.305)	0.924	0.187	(-0.256; 0.565)	0.393	0.389	(-0.041; 0.697)	0.067	0.101	(-0.336; 0.503)	0.646	-0.213	(-0.584; 0.230)	0.328
Mesomorphy	0.229	(-0.067; 0.489)	0.117	0.315	(-0.125; 0.651)	0.144	0.466	(0.054; 0.743)	0.025	0.209	(-0.235; 0.580)	0.340	-0.043	(-0.458; 0.387)	0.844
Ectomorphy	-0.199	(-0.464; 0.098)	0.174	-0.086	(-0.491; 0.349)	0.695	-0.239	(-0.601; 0.205)	0.273	-0.003	(-0.425; 0.420)	0.989	0.157	(-0.285; 0.544)	0.475
Handgrip (kg)	0.121	(-0.177; 0.399)	0.411	-0.211	(-0.582; 0.233)	0.334	-0.183	(-0.562; 0.260)	0.404	-0.180	(-0.560; 0.263)	0.412	-0.203	(-0.576; 0.241)	0.353
HIP/lean mass index	-0.040	(-0.328; 0.255)	0.788	-0.054	(-0.466; 0.377)	0.806	-0.108	(-0.508; 0.330)	0.624	0.040	(-0.389; 0.456)	0.854	0.038	(-0.391; 0.454)	0.862

ρ = Spearman's correlation coefficient. P-values are from Spearman's correlation analyses between the questionnaire scores and body composition measures. Bold numbers indicate statistically significant associations. Significance was set at $P < 0.05$. Abbreviations: ADAM-Q, Androgen Deficiency in the Aging Male Questionnaire; BIA, bioelectrical impedance analysis; BMC, bone mineral content; BMD, bone mineral density; BMI, body mass index; DXA, dual-energy X-ray absorptiometry; FMI, fat mass index; LEAF-Q, Low Energy Availability in Females Questionnaire; LMI, lean mass index; VAT, visceral adipose tissue.

Annexe 2. Table S2: Differences in body composition between male athletes with no symptoms and symptoms of low testosterone levels, and female athletes at no risk and risk for the Female Athlete Triad.

	Male athletes (n = 48)				Female athletes (n = 23)			
	No symptoms (n = 31)	Symptoms (n = 17)	MD (95% CI)	P	No risk (n = 12)	Risk (n = 11)	MD (95% CI)	P
	Mean (SD)	Mean (SD)			Mean (SD)	Mean (SD)		
Weight (kg)	72.6 (9.6)	77.8 (8.8)	-5.2 (-10.8; 0.5)	0.075	61.8 (7.2)	66.3 (12.6)	-4.6 (-13.8; 4.6)	0.307
Height (m)	1.77 (0.07)	1.78 (0.06)	-0.02 (-0.06; 0.02)	0.414	1.66 (0.02)	1.65 (0.06)	0.01 (-0.04; 0.05)	0.702
BMI (kg/m ²)	23.2 (2.6)	24.5 (2.7)	-1.2 (-2.9; 0.4)	0.131	22.4 (2.2)	24.2 (3.6)	-1.8 (-4.3; 0.7)	0.157
Fat mass (kg) - DXA	12.3 (4.0)	12.1 (3.3)	0.1 (-2.1; 2.4)	0.906 ^a	15.7 (4.0)	19.3 (7.8)	-3.6 (-8.9; 1.7)	0.186
Fat mass (kg) - BIA	9.3 (4.3)	9.1 (4.1)	0.2 (-2.4; 2.8)	0.876	12.7 (3.8)	15.5 (7.2)	-2.9 (-7.8; 2.1)	0.262
Fat mass (%) - DXA	16.9 (3.9)	15.7 (3.1)	1.2 (-1.0; 3.4)	0.223 ^a	25.6 (4.4)	28.7 (7.2)	-3.1 (-8.2; 2.0)	0.221
Fat mass (%) - BIA	12.4 (4.4)	11.4 (4.2)	1.1 (-1.5; 3.7)	0.418	20.3 (4.1)	22.5 (7.0)	-2.3 (-7.2; 2.6)	0.413 ^a
FMI (kg/m ²) - DXA	3.9 (1.2)	3.8 (1.1)	0.1 (-0.6; 3.7)	0.889 ^a	5.7 (1.3)	7.0 (2.6)	-1.3 (-3.1; 2.6)	0.131
Android fat mass (kg)	0.7 (0.2)	0.8 (0.2)	0.0 (-0.2; 0.1)	0.419 ^a	0.8 (0.3)	1.1 (0.5)	-0.3 (-0.6; 0.1)	0.211 ^a
Gynoid fat mass (kg)	2.1 (0.7)	2.1 (0.7)	0.1 (-0.4; 0.5)	0.974 ^a	3.3 (0.9)	3.9 (1.6)	-0.6 (-1.7; 0.5)	0.315
VAT mass (g)	200.5 (43.8)	204.8 (39.9)	-4.3 (-30.1; 21.5)	0.739	117.3 (49.8)	171.8 (87.5)	-54.5 (-115.5; 6.5)	0.077
Adipose mass (kg)	17.2 (3.7)	17.1 (2.9)	0.1 (-2.0; 2.2)	0.941	20.4 (4.9)	22.2 (6.7)	-1.8 (-6.8; 3.2)	0.458
Sum of six skinfolds (mm)	55.3 (18.0)	55.6 (17.7)	-0.3 (-11.1; 10.6)	0.960	87.3 (32.4)	103.4 (36.5)	-16.1 (-46; 13.8)	0.275
Sum of eight skinfolds (mm)	70.4 (21.9)	71.0 (22.9)	-0.6 (-14.1; 13.0)	0.932	110.5 (42.9)	128.3 (45.9)	-17.8 (-56.3; 20.7)	0.347
Adipose to muscle index	0.51 (0.12)	0.45 (0.08)	0.05 (-0.01; 0.10)	0.106	0.85 (0.23)	0.91 (0.33)	-0.06 (-0.30; 0.20)	0.833 ^a
Lean mass (kg)	56.4 (6.3)	61.3 (6.4)	-5.0 (-8.8; -1.2)	0.012	42.7 (4.2)	43.6 (6.0)	-0.8 (-5.3; 3.6)	0.702
LMI (kg/m ²)	18.1 (1.8)	19.3 (1.9)	-1.2 (-2.3; -0.1)	0.027	15.5 (1.3)	15.9 (1.5)	-0.4 (-1.6; 0.8)	0.499
FFM (kg) - DXA	59.1 (6.8)	64.3 (6.7)	-5.2 (-9.3; -1.0)	0.015	45.0 (4.4)	45.9 (6.3)	-1.0 (-5.7; 3.7)	0.672
FFM (kg) - BIA	63.1 (6.5)	68.4 (5.7)	-5.3 (-9.1; -1.5)	0.007	48.8 (4.3)	50.5 (6.6)	-1.7 (-6.5; 3.1)	0.476
Muscle mass (kg) - BIA	60.0 (6.2)	65.0 (5.4)	-5.1 (-8.7; -1.5)	0.007	46.3 (4.1)	47.9 (6.3)	-1.6 (-6.1; 3.0)	0.478
Muscle mass (kg) - Anthropometry	34.6 (5.7)	38.0 (5.4)	-3.4 (-6.8; 0.0)	0.064 ^a	24.5 (3.8)	25.2 (4.6)	-0.7 (-4.3; 2.9)	0.701
Corrected arm circumference (cm)	28.0 (3.1)	29.4 (3.1)	-1.4 (-3.3; 0.5)	0.132	22.8 (2.6)	23.5 (2.4)	-0.7 (-2.9; 1.5)	0.494
Corrected torax circumference (cm)	91.7 (6.2)	95.6 (6.2)	-3.9 (-7.6; -0.1)	0.043	82.7 (3.6)	85.3 (5.0)	-2.6 (-6.3; 1.2)	0.169
Corrected thigh circumference (cm)	49.1 (3.5)	51.3 (3.3)	-2.2 (-4.3; -0.1)	0.044	43.3 (2.6)	44.3 (6.1)	-1.1 (-5.0; 2.9)	0.585
Corrected calf circumference (cm)	34.2 (2.3)	35.2 (1.8)	-1.0 (-2.3; 0.3)	0.072 ^a	30.5 (2.1)	30.4 (2.9)	0.1 (-2.1; 2.2)	0.949
Muscle to bone index	4.31 (0.85)	4.28 (0.53)	0.03 (-0.43; 0.49)	0.905	3.59 (0.76)	3.48 (0.61)	0.11 (-0.50; 0.71)	0.716
Residual mass (kg)	4.2 (0.4)	4.3 (0.3)	-0.1 (-0.3; 0.1)	0.377	3.5 (0.2)	3.4 (0.3)	0.1 (-0.1; 0.3)	0.347
BMC (kg)	2.74 (0.58)	2.91 (0.45)	-0.16 (-0.49; 0.17)	0.326	2.24 (0.29)	2.37 (0.42)	-0.14 (-0.45; 0.18)	0.378
BMD (g/cm ²)	1.23 (0.15)	1.26 (0.12)	-0.03 (-0.12; 0.05)	0.428	1.16 (0.09)	1.20 (0.12)	-0.04 (-0.13; 0.05)	0.394
BMD Z-score	0.3 (1.4)	0.6 (1.0)	-0.3 (-1.1; 0.5)	0.400	0.5 (1.0)	1.0 (1.3)	-0.5 (-1.5; 0.5)	0.327
Bone mass (kg)	8.2 (1.4)	8.9 (0.9)	-0.7 (-1.5; 0.1)	0.077	7.0 (1.0)	7.3 (1.3)	-0.4 (-1.4; 0.6)	0.449
Total body water (kg)	43.5 (3.8)	46.1 (3.6)	-2.6 (-4.8; -0.3)	0.030	32.3 (2.3)	33.8 (2.7)	-1.5 (-3.7; 0.7)	0.164
Total body water (%)	60.5 (3.3)	59.7 (2.5)	0.8 (-1.0; 2.6)	0.390	52.8 (2.7)	52.2 (6.6)	0.5 (-3.8; 4.8)	0.808
Extracellular water (kg)	17.1 (1.5)	17.7 (1.3)	-0.6 (-1.5; 0.3)	0.183	13.6 (1.1)	14.2 (1.8)	-0.6 (-1.9; 0.7)	0.322
Intracellular water (kg)	26.4 (2.6)	28.4 (2.8)	-2.0 (-3.6; -0.4)	0.018	18.7 (1.5)	19.6 (1.3)	-0.9 (-2.1; 0.4)	0.159
Phase angle (°)	-6.8 (0.6)	-7.1 (0.6)	0.3 (-0.1; 0.7)	0.133	-6.4 (0.7)	-6.6 (0.7)	0.2 (-0.4; 0.8)	0.444
<u>Endomorphy</u>	2.2 (0.6)	2.2 (0.9)	-0.1 (-0.5; 0.4)	0.743	3.5 (1.4)	4.2 (1.4)	-0.7 (-1.9; 0.5)	0.267
<u>Mesomorphy</u>	4.9 (1.2)	5.4 (1.3)	-0.5 (-1.3; 0.2)	0.140 ^a	3.9 (0.7)	4.5 (1.0)	-0.6 (-1.4; 0.1)	0.095
<u>Ectomorphy</u>	2.6 (1.2)	2.1 (1.2)	0.5 (-0.3; 1.2)	0.208	2.3 (0.9)	1.9 (1.1)	0.3 (-0.6; 1.2)	0.453

P-values are from independent-samples Student's t-test analyses comparing body composition between male athletes with no symptoms and those with symptoms of low testosterone levels, and comparing body composition between female athletes at no risk and at risk for the Female Athlete Triad. Bold numbers indicate statistically significant associations. Significance was set at $P < 0.05$. Abbreviations: 95% CI, 95% confidence intervals; ADAM-Q, Androgen Deficiency in the Aging Male Questionnaire; BIA, bioelectrical impedance analysis;

BMC, bone mineral content; BMD, bone mineral density; BMI, body mass index; DXA, dual-energy X-ray absorptiometry; FMI, fat mass index; LEAF-Q, Low Energy Availability in Females Questionnaire; LMI, lean mass index; MD, mean difference; VAT, visceral adipose tissue. ^a Symbol indicates P obtained from Mann-Whitney U tests.

Annexe 3. Table S3. Post-hoc pairwise comparisons of the differences in the RMR ratio measured/predicted depending on the equation used to calculate the RMR predicted.

	Mean	SD	P values from post-hoc Bonferroni pairwise comparisons											
			Cunningham	Ten Haaf (FFM)	Ten Haaf (BW)	Tinsley (BW)	Tinsley (FFM)	Wong	Freire	Van Hoozen	Harris-Benedict	Pontzer (BW)	Pontzer (FFM)	De Lorenzo
Cunningham	0.95	0.10	-	<0.001	<0.001	<0.001	0.004	0.005	<0.001	<0.001	1.000	1.000	1.000	<0.001
ten Haaf (FFM)	0.85	0.09	<0.001	-	1.000	1.000	1.000	1.000	<0.001	<0.001	0.001	0.115	0.288	0.002
ten Haaf (BW)	0.82	0.09	<0.001	1.000	-	1.000	1.000	1.000	0.070	0.011	<0.001	<0.001	0.002	1.000
Tinsley (BW)	0.84	0.10	<0.001	1.000	1.000	-	1.000	1.000	0.001	<0.001	<0.001	0.053	0.141	1.000
Tinsley (FFM)	0.86	0.09	0.004	1.000	1.000	1.000	-	1.000	<0.001	<0.001	0.010	0.920	1.000	<0.001
Wong	0.86	0.10	0.005	1.000	1.000	1.000	1.000	-	<0.001	<0.001	0.012	1.000	1.000	<0.001
Freire	0.75	0.08	<0.001	<0.001	0.070	0.001	<0.001	<0.001	-	1.000	<0.001	<0.001	<0.001	<0.001
Van Hoozen	0.74	0.09	<0.001	<0.001	0.011	<0.001	<0.001	<0.001	1.000	-	<0.001	<0.001	<0.001	<0.001
Harris-Benedict	0.94	0.14	1.000	0.001	<0.001	<0.001	0.010	0.012	<0.001	<0.001	-	1.000	1.000	<0.001
Pontzer (BW)	0.91	0.11	1.000	0.115	<0.001	0.053	0.920	1.000	<0.001	<0.001	1.000	-	1.000	<0.001
Pontzer (FFM)	0.91	0.11	1.000	0.288	0.002	0.141	1.000	1.000	<0.001	<0.001	1.000	1.000	-	<0.001
De Lorenzo	0.82	0.10	<0.001	0.002	1.000	1.000	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	-

P values represent post-hoc Bonferroni correction between equations obtained from a one-way analysis of variance (ANOVA). Abbreviations: BW, body weight; FFM, fat-free mass; RMR, resting metabolic rate.

Annexe 4: Table S4. Correlation between RMR ratio measured/predicted with body composition, dietary outcomes, physical activity, sleep quantity and quality, and subjective well-being.

Outcome	r	P value	P adjusted
<i>Body composition</i>			
BMC	0.195	0.179	0.672
BMD	0.147	0.312	0.739
Z-score	0.116	0.429	0.758
Lean	0.226	0.118	0.519
LMI	0.209	0.150	0.592
FFM	0.227	0.118	0.519
FM	0.164	0.259	0.688
FM (%)	0.024	0.871	0.977
FMI	0.112	0.445	0.758
FM android	0.190	0.191	0.684
FM ginoid	0.143	0.326	0.739
VAT mass	0.317	0.026	0.338
<i>Dietary outcomes</i>			
Energy intake	0.111	0.448	0.752
	-		
Fat intake (%)	0.133	0.362	0.739
Protein intake (%)	0.109	0.457	0.758
Carbohydrate intake (%)	0.115	0.433	0.758
	-		
Fiber intake	0.125	0.393	0.739
<i>Physical activity</i>			
	-		
Sedentary behaviour	0.257	0.101	0.498
	-		
Light physical activity	0.052	0.746	0.929
MPA	0.337	0.029	0.338
	-		
VPA	0.008	0.958	0.983
10min bouts of MVPA	0.174	0.270	0.688
5-10min bouts of MVPA	0.003	0.986	0.986
1-5 bouts of MVPA	0.191	0.226	0.688
	-		
60min bouts of sedentary behavior	0.013	0.933	0.982
30-60min bouts of sedentary behavior	0.053	0.740	0.929
Steps/day	0.188	0.233	0.688
	-		
Cadence peak-1min	0.060	0.706	0.929
	-		
Cadence peak-30min	0.017	0.913	0.977
Cadence peak-60min	0.041	0.795	0.937

Table S4 (continued). Correlation between RMR ratio measured/predicted with body composition, dietary outcomes, physical activity, sleep quantity and quality, and subjective well-being

	-		
Time at 0 steps/min	0.241	0.125	0.519
Time at 1-19 steps/min	0.140	0.377	0.739
Time at 20-39 steps/min	0.201	0.202	0.688
Time at 40-59 steps/min	0.183	0.247	0.688
Time at 60-79 steps/min	0.084	0.595	0.870
Time at 80-99 steps/min	0.176	0.264	0.688
Time at 100-199 steps/min	0.017	0.915	0.977
	-		
Time at ≥ 120 steps/min	0.160	0.311	0.739
	-		
Time of MPA based on cadence	0.042	0.793	0.937
	-		
Time of VPA based on cadence	0.072	0.651	0.907
	-		
Time of MVPA based on cadence	0.057	0.718	0.929
<i>Sleep quantity and quality</i>			
	-		
Sleep duration	0.047	0.768	0.934
WASO	0.197	0.216	0.688
	-		
Time in sustained inactivity bouts during waking hours	0.332	0.034	0.338
Sustained inactivity bouts during sleep period time	0.147	0.360	0.739
Awakenings after sleep onset	0.147	0.360	0.739
	-		
N° of sustained inactivity bouts during waking hours	0.270	0.088	0.478
	-		
Time in sustained inactivity bouts with >15min	0.316	0.044	0.387
	-		
Duration of each sustained inactivity bout during waking hour	0.138	0.390	0.739
Sleep regularity index	0.339	0.030	0.338
<i>Subjective well-being</i>			
	-		
ADAM-Q	0.427	0.013	0.338
	-		
EAT-26	0.247	0.091	0.478
SRS	0.256	0.076	0.459
	-		
ASPS	0.384	0.006	0.338
	-		
PSQI	0.091	0.541	0.864
LEAF-Q Total Score	0.017	0.949	0.983
LEAF-Q Injuries score	0.099	0.715	0.929
	-		
LEAF-Q GI function score	0.043	0.874	0.977
	-		
LEAF-Q Menstrual cycle score	0.042	0.878	0.977
NUKYA	0.086	0.558	0.864

Table S4 (continued). Correlation between RMR ratio measured/predicted with body composition, dietary outcomes, physical activity, sleep quantity and quality, and subjective well-being

Overtraining	0.021	0.889	0.977
	-		
RESTQ-76 - General stress score	0.110	0.460	0.758
RESTQ-76 - Emotional stress score	0.004	0.980	0.986
RESTQ-76 - Social stress score	0.060	0.691	0.929
	-		
RESTQ-76 - Conflicts/pressure score	0.082	0.585	0.870
RESTQ-76 - Fatigue score	0.019	0.897	0.977
RESTQ-76 - Lack of energy score	0.088	0.557	0.864
	-		
RESTQ-76 - Physical alterations score	0.067	0.655	0.907
	-		
RESTQ-76 - Success score	0.310	0.034	0.338
	-		
RESTQ-76 - Social recovery score	0.132	0.375	0.739
	-		
RESTQ-76 - Physical recovery score	0.136	0.362	0.739
	-		
RESTQ-76 - General well-being score	0.075	0.616	0.885
	-		
RESTQ-76 - Sleep quality score	0.047	0.752	0.929
	-		
RESTQ-76 - Alterations of rest periods score	0.112	0.452	0.758
RESTQ-76 - Emotional fatigue score	0.268	0.069	0.459
RESTQ-76 - Injuries score	0.084	0.574	0.870
	-		
RESTQ-76 - Being in shape score	0.171	0.250	0.688
	-		
RESTQ-76 - Personal fulfillment score	0.332	0.023	0.338
	-		
RESTQ-76 - Self-efficacy score	0.263	0.075	0.459
	-		
RESTQ-76 - Self regulation score	0.284	0.053	0.416

Bold P values indicate P values <0.05. r and P values obtained from Pearson correlation analyses. Adjusted P values obtained from multiple comparisons correction adjusting for FDR. The RMR ratio measured/predicted was calculated using the Ten Haaf (FFM) equation (237) for the predicted RMR. Abbreviations: ADAM-Q, Androgen Deficiency in the Aging Male Questionnaire; ASPS, Athlete's Subjective Performance Scale; BMC, bone mineral content; BMD, bone mineral density; EA, energy availability; EAT-26, Eating Attitudes Test-26; ECW, extracellular water; FDR, false discovery rate; FFM, fat-free mass; FM, fat mass; FMI, fat mass index; GI, gastrointestinal; LEA, low energy availability; LEAF-Q, Low Energy Availability in Females Questionnaire; LMI, lean mass index; MPA, moderate physical activity; MVPA, moderate to vigorous physical activity; NUKYA, Nutrition Knowledge for Young and Adult Athletes; PSQI, Pittsburgh Sleep Quality Index; RESTQ-76, Recovery-Stress Questionnaire for Athletes-76; SRS, Silhouette Rating Scale; VAT, visceral adipose tissue; VPA, vigorous physical activity; WASO, wake after sleep onset.

Annexe 5: Table S5. Differences in body composition, dietary outcomes, physical activity, sleep quantity, and quality, and subjective well-being between participants with an RMR ratio measured/predicted >0.9 vs. participants with a ratio <0.9.

Outcome	r	P value	P adjusted
<i>Body composition</i>			
BMC	0.195	0.179	0.672
BMD	0.147	0.312	0.739
Z-score	0.116	0.429	0.758
Lean	0.226	0.118	0.519
LMI	0.209	0.150	0.592
FFM	0.227	0.118	0.519
FM	0.164	0.259	0.688
FM (%)	0.024	0.871	0.977
FMI	0.112	0.445	0.758
FM android	0.190	0.191	0.684
FM ginoid	0.143	0.326	0.739
VAT mass	0.317	0.026	0.338
<i>Dietary outcomes</i>			
Energy intake	0.111	0.448	0.752
Fat intake (%)	-0.133	0.362	0.739
Protein intake (%)	0.109	0.457	0.758
Carbohydrate intake (%)	0.115	0.433	0.758
Fiber intake	-0.125	0.393	0.739
<i>Physical activity</i>			
Sedentary behaviour	-0.257	0.101	0.498
Light physical activity	-0.052	0.746	0.929
MPA	0.337	0.029	0.338
VPA	-0.008	0.958	0.983
10min bouts of MVPA	0.174	0.270	0.688
5-10min bouts of MVPA	0.003	0.986	0.986
1-5 bouts of MVPA	0.191	0.226	0.688
60min bouts of sedentary behavior	-0.013	0.933	0.982
30-60min bouts of sedentary behavior	0.053	0.740	0.929
Steps/day	0.188	0.233	0.688
Cadence peak-1min	-0.060	0.706	0.929
Cadence peak-30min	-0.017	0.913	0.977
Cadence peak-60min	0.041	0.795	0.937
Time at 0 steps/min	-0.241	0.125	0.519
Time at 1-19 steps/min	0.140	0.377	0.739
Time at 20-39 steps/min	0.201	0.202	0.688
Time at 40-59 steps/min	0.183	0.247	0.688
Time at 60-79 steps/min	0.084	0.595	0.870
Time at 80-99 steps/min	0.176	0.264	0.688
Time at 100-199 steps/min	0.017	0.915	0.977
Time at ≥120 steps/min	-0.160	0.311	0.739
Time of MPA based on cadence	-0.042	0.793	0.937

Table S5 (continued). Differences in body composition, dietary outcomes, physical activity, sleep quantity, and quality, and subjective well-being between participants with an RMR ratio measured/predicted >0.9 vs. participants with a ratio <0.9

<i>Sleep quantity and quality</i>			
Sleep duration	-0.047	0.768	0.934
WASO	0.197	0.216	0.688
Time in sustained inactivity bouts during waking hours	-0.332	0.034	0.338
Sustained inactivity bouts during sleep period time	0.147	0.360	0.739
Awakenings after sleep onset	0.147	0.360	0.739
N° of sustained inactivity bouts during waking hours	-0.270	0.088	0.478
Time in sustained inactivity bouts with >15min	-0.316	0.044	0.387
Duration of each sustained inactivity bout during waking hour	-0.138	0.390	0.739
Sleep regularity index	0.339	0.030	0.338
<i>Subjective well-being</i>			
ADAM-Q	-0.427	0.013	0.338
EAT-26	-0.247	0.091	0.478
SRS	0.256	0.076	0.459
ASPS	-0.384	0.006	0.338
PSQI	-0.091	0.541	0.864
LEAF-Q Total Score	0.017	0.949	0.983
LEAF-Q Injuries score	0.099	0.715	0.929
LEAF-Q GI function score	-0.043	0.874	0.977
LEAF-Q Menstrual cycle score	-0.042	0.878	0.977
NUKYA	0.086	0.558	0.864
Overtraining	0.021	0.889	0.977
RESTQ-76 - General stress score	-0.110	0.460	0.758
RESTQ-76 - Emotional stress score	0.004	0.980	0.986
RESTQ-76 - Social stress score	0.060	0.691	0.929
RESTQ-76 - Conflicts/pressure score	-0.082	0.585	0.870
RESTQ-76 - Fatigue score	0.019	0.897	0.977
RESTQ-76 - Lack of energy score	0.088	0.557	0.864
RESTQ-76 - Physical alterations score	-0.067	0.655	0.907
RESTQ-76 - Success score	-0.310	0.034	0.338
RESTQ-76 - Social recovery score	-0.132	0.375	0.739
RESTQ-76 - Physical recovery score	-0.136	0.362	0.739
RESTQ-76 - General well-being score	-0.075	0.616	0.885
RESTQ-76 - Sleep quality score	-0.047	0.752	0.929
RESTQ-76 - Alterations of rest periods score	-0.112	0.452	0.758
RESTQ-76 - Emotional fatigue score	0.268	0.069	0.459
RESTQ-76 - Injuries score	0.084	0.574	0.870
RESTQ-76 - Being in shape score	-0.171	0.250	0.688
RESTQ-76 - Personal fulfillment score	-0.332	0.023	0.338
RESTQ-76 - Self-efficacy score	-0.263	0.075	0.459
RESTQ-76 - Self regulation score	-0.284	0.053	0.416

Bold P values indicate P values <0.05. Mean differences, 95% confidence intervals (CI), and P values obtained from independent samples t-test. Adjusted P values obtained from multiple comparisons correction adjusting for FDR. The RMR ratio measured/predicted was calculated using the Ten Haaf (FFM) equation (237) for the predicted RMR.

Annexe 6: Table S6. Differences in the RMR ratio measured/predicted depending on the predictive equation used to predict the RMR in men participants with an RMR ratio measured/predicted >0.9 vs. participants with a ratio <0.9.

	N	%	P value
Cunningham	0.95	0.10	<0.001
Ten Haaf (FFM)	0.86	0.09	
Ten Haaf (BW)	0.82	0.08	
Tinsley (BW)	0.87	0.10	
Tinsley (FFM)	0.86	0.09	
Wong	0.88	0.09	
Freire	0.76	0.07	
Van Hooren	0.76	0.08	
Harris-Benedict	1.01	0.10	
Pontzer (BW)	0.91	0.10	
Pontzer (FFM)	0.92	0.10	

P values obtained from repeated measures analysis of variance (ANOVA).

Annexe 7. Table S7. Relationship between RMR and RMR ratio measured/predicted with energy availability in men

	Correlation analyses		Adequate EA (n=18)		LEA (n=15)		P value
	r	P value	Mean	SD	Mean	SD	
RMR	-0.158	0.381	1575.06	220.92	1647.28	206.27	0.343
RMR ratio	0.141	0.433	0.87	0.10	0.85	0.06	0.471

P values obtained from Pearson correlation analyses and independent samples t-tests.

Annexe. 8 Table S8. Relationship between RMR ratio measured/predicted and phenotypical traits (i.e., body composition, dietary outcomes, physical activity, sleep quantity and quality, and subjective well-being) in men

	r	P	Differences in the RMR ratio measured/predicted
			<0.9 or ≥0.9 P value
BMC	-0.022	0.902	0.301
BMD	-0.045	0.806	0.263
Z-score	-0.053	0.770	0.260
Lean	0.029	0.874	0.545
LMI	-0.012	0.946	0.922
FFM	0.025	0.891	0.518
FM	0.163	0.365	0.239
FM (%)	0.176	0.327	0.343
FMI	0.156	0.384	0.323
FM android	0.183	0.307	0.445
FM ginoid	0.195	0.277	0.219
VAT mass	0.261	0.143	0.349
MEAN_Energía kcal)	0.103	0.570	0.440
Fat intake (%)	-0.218	0.223	0.137
Protein intake (%)	0.124	0.490	0.388
Carbohydrate intake (%)	0.230	0.198	0.240
Fiber intake	-0.212	0.235	0.652
Sedentary behaviour	-0.259	0.159	0.669
Light physical activity	-0.084	0.654	0.313
Moderate physical activity	.406*	0.023	0.637
Vigorous physical activity	0.068	0.716	0.640
10min bouts of MVPA	0.353	0.052	0.649
5-10min bouts of MVPA	0.057	0.759	0.468
1-5 bouts of MVPA	0.174	0.349	0.803
60min bouts of sedentary behavior	0.088	0.637	0.066
30-60min bouts of sedentary behavior	0.161	0.386	0.217
Steps/day	.361	0.046	0.542
Cadence peak-1min	0.103	0.580	0.664

Table S8 (continued). Relationship between RMR ratio measured/predicted and phenotypical traits (i.e., body composition, dietary outcomes, physical activity, sleep quantity and quality, and subjective well-being) in men

Cadence peak-30min	0.214	0.248	0.392
Cadence peak-60min	0.301	0.100	0.277
Time at 0 steps/min	-0.250	0.175	0.994
Time at 1-19 steps/min	0.022	0.906	0.947
Time at 20-39 steps/min	0.182	0.327	0.575
Time at 40-59 steps/min	0.213	0.250	0.644
Time at 60-79 steps/min	0.198	0.286	0.799
Time at 80-99 steps/min	0.234	0.205	0.498
Time at 100-199 steps/min	0.177	0.341	0.108
Time at ≥120 steps/min	0.053	0.777	0.966
Time of MPA based on cadence	0.159	0.391	0.131
Time of VPA based on cadence	0.185	0.320	0.617
Time of MVPA based on cadence	0.186	0.317	0.115
Sleep duration	-0.084	0.658	0.742
WASO	0.186	0.325	0.224
Time in sustained inactivity bouts during waking hours	-0.354	0.055	0.120
Sustained inactivity bouts during sleep period time	0.035	0.854	0.443
Awakenings after sleep onset	0.035	0.854	0.443
N° of sustained inactivity bouts during waking hours	-0.331	0.074	0.052
Time in sustained inactivity bouts with >15min	-0.262	0.162	0.486
Duration of each sustained inactivity bout during waking hour	-0.101	0.594	0.763
Sleep regularity index	.365*	0.048	0.105
ADAM-Q	-.427*	0.013	0.149
EAT-26	-0.123	0.504	0.531
SRS	.364*	0.037	0.475

Table S8 (continued). Relationship between RMR ratio measured/predicted and phenotypical traits (i.e., body composition, dietary outcomes, physical activity, sleep quantity and quality, and subjective well-being) in men

ASPS	-0.299	0.090	0.024
PSQI	-0.122	0.500	0.522
LEAF-Q Total Score	NA		NA
LEAF-Q Injuries score	NA		NA
LEAF-Q GI function score	NA		NA
LEAF-Q Menstrual cycle score	NA		NA
NUKYA	0.216	0.227	0.275
Overtraining	-0.242	0.190	0.155
RESTQ-76 - General stress score	-0.144	0.433	0.210
RESTQ-76 - Emotional stress score	-0.102	0.578	0.331
RESTQ-76 - Social stress score	0.056	0.760	0.623
RESTQ-76 - Conflicts/pressure score	-0.155	0.396	0.771
RESTQ-76 - Fatigue score	-0.149	0.415	0.310
RESTQ-76 - Lack of energy score	0.125	0.496	0.892
RESTQ-76 - Physical alterations score	-0.022	0.907	0.803
RESTQ-76 - Success score	-0.280	0.121	0.828
RESTQ-76 - Social recovery score	-0.102	0.579	0.377
RESTQ-76 - Physical recovery score	-0.092	0.617	0.750
RESTQ-76 - General well-being score	-0.020	0.914	0.216
RESTQ-76 - Sleep quality score	0.009	0.962	0.619
RESTQ-76 - Alterations of rest periods score	-0.174	0.342	0.382
RESTQ-76 - Emotional fatigue score	0.245	0.176	0.297
RESTQ-76 - Injuries score	0.013	0.944	0.675

Table S8 (continued). Relationship between RMR ratio measured/predicted and phenotypical traits (i.e., body composition, dietary outcomes, physical activity, sleep quantity and quality, and subjective well-being) in men

RESTQ-76 - Being in shape score	-0.014	0.939	0.513
RESTQ-76 - Personal fulfillment score	-0.244	0.178	0.696
RESTQ-76 - Self-efficacy score	-0.168	0.357	0.735
RESTQ-76 - Self-regulation score	-0.089	0.630	0.557

P values obtained from Pearson correlation analyses and independent samples t-tests.

Annexe 9. Table S9. Correlation between sedentary behavior and physical activity levels with energy availability.

Outcome	r	P value	P adjusted
Sedentary behaviour	0.381	0.001	0.018
Light physical activity	-0.132	0.366	0.728
Moderate physical activity	-0.115	0.430	0.728
Vigorous physical activity	0.124	0.398	0.728
10min bouts of MVPA	0.164	0.260	0.624
5-10min bouts of MVPA	0.263	0.068	0.410
1-5 bouts of MVPA	-0.187	0.198	0.624
60min bouts of sedentary behavior	-0.110	0.453	0.728
30-60min bouts of sedentary behavior	-0.072	0.622	0.878
Steps/day	0.164	0.259	0.624
Cadence peak-1min	0.212	0.143	0.624
Cadence peak-30min	0.105	0.475	0.728
Cadence peak-60min	0.173	0.235	0.624
Time at 0 steps/min	0.193	0.185	0.624
Time at 1-19 steps/min	-0.057	0.695	0.927
Time at 20-39 steps/min	-0.047	0.750	0.947
Time at 40-59 steps/min	-0.102	0.485	0.728
Time at 60-79 steps/min	-0.009	0.952	0.993
Time at 80-99 steps/min	0.363	0.010	0.125
Time at 100-199 steps/min	0.333	0.019	0.156
Time at ≥120 steps/min	0.018	0.903	0.985
Time of MPA based on cadence	0.000	0.999	0.999
Time of VPA based on cadence	0.022	0.879	0.985
Time of MVPA based on cadence	-0.020	0.893	0.985

Bold P values indicate P values <0.05. r and P values obtained from Pearson correlation analyses. Adjusted P values obtained from multiple comparisons correction adjusting for FDR. Bold P adjusted values indicate P adjusted < 0.1. Abbreviations: FDR, false discovery rate; MPA, moderate physical activity; MVPA, moderate-to-vigorous physical activity; VPA, vigorous physical activity.

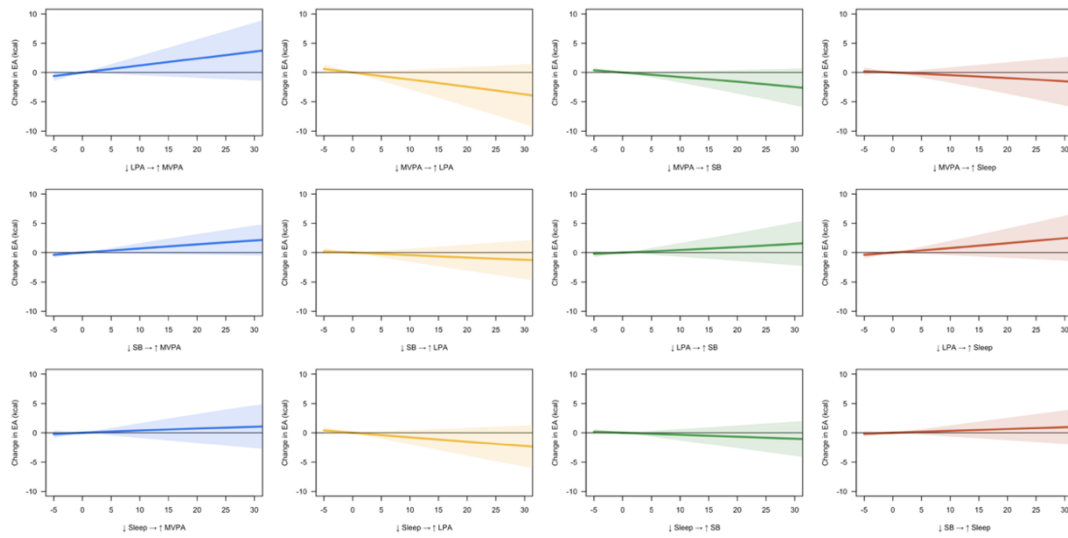
Annexe 10. Table S10. Differences in sedentary behavior and physical activity levels between participants with adequate energy availability (EA; >30kcal/kg FFM/day) and participants with low energy availability (LEA; <30kcal/kg FFM/day).

Outcome	Mean Difference	95%CI (Mean Difference)		P value	P adjusted
Sedentary behaviour	28.28	-67.30	10.74	0.151	0.459
Light physical activity	1.19	-24.60	22.22	0.919	0.933
Moderate physical activity	-16.27	-8.26	40.81	0.188	0.459
Vigorous physical activity	-8.40	-2.75	19.55	0.135	0.459
10min bouts of MVPA	-0.86	-0.07	1.78	0.069	0.459
5-10min bouts of MVPA	0.28	-0.96	0.40	0.406	0.749
1-5 bouts of MVPA	-0.27	-6.10	6.64	0.933	0.933
60min bouts of sedentary behavior	0.33	-0.99	0.34	0.326	0.652
30-60min bouts of sedentary behavior	-0.22	-0.36	0.80	0.453	0.763
Steps/day	-1764.83	-343.57	3873.23	0.099	0.459
Cadence peak-1min	-4.67	-2.37	11.71	0.187	0.459
Cadence peak-30min	-5.64	-2.94	14.22	0.191	0.459
Cadence peak-60min	-5.79	-2.87	14.44	0.184	0.459
Time at 0 steps/min	31.48	-85.37	22.41	0.245	0.534
Time at 1-19 steps/min	-7.22	-22.05	36.50	0.622	0.807
Time at 20-39 steps/min	-2.34	-19.01	23.70	0.826	0.933
Time at 40-59 steps/min	-4.45	-8.03	16.92	0.477	0.763
Time at 60-79 steps/min	-6.82	-0.84	14.49	0.080	0.459
Time at 80-99 steps/min	-8.73	-2.03	19.48	0.109	0.459
Time at 100-199 steps/min	-2.13	-5.89	10.16	0.595	0.807
Time at ≥120 steps/min	0.36	-4.79	4.08	0.872	0.933
Time of MPA based on cadence	-2.45	-6.45	11.35	0.582	0.807
Time of VPA based on cadence	0.68	-3.58	2.22	0.639	0.807
Time of MVPA based on cadence	-1.78	-8.22	11.78	0.722	0.867

Bold P values indicate P values <0.05. Mean differences, 95% confidence intervals (CI), and P values obtained from independent samples t-tests. Adjusted P values obtained from multiple comparisons correction adjusting for FDR. Bold P adjusted values indicate P adjusted < 0.1.

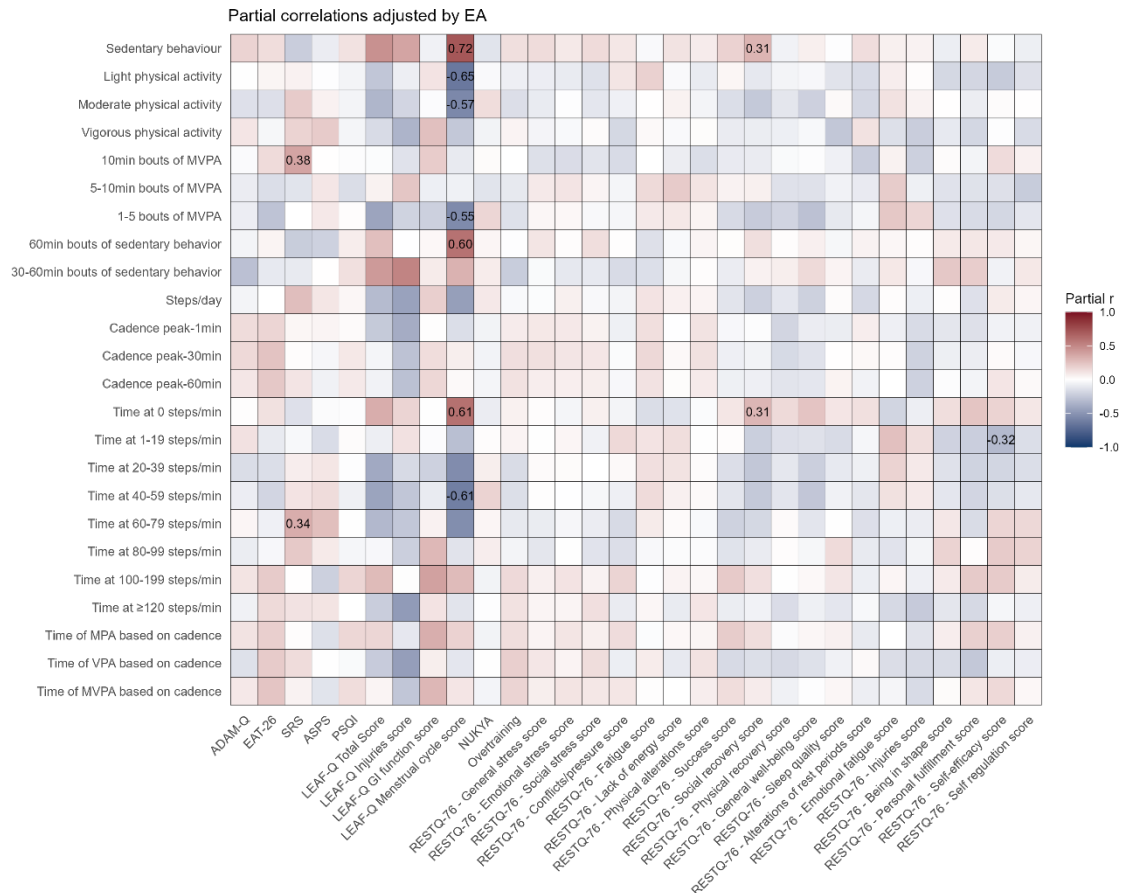
Abbreviations: FDR, false discovery rate; LEA, low energy availability; MPA, moderate physical activity; MVPA, moderate-to-vigorous physical activity; VPA, vigorous physical activity.

Annexe 11. Figure S1. Pairwise compositional substitution models showing the predicted change in EA with time reallocation to specific movement behaviors (per pair).

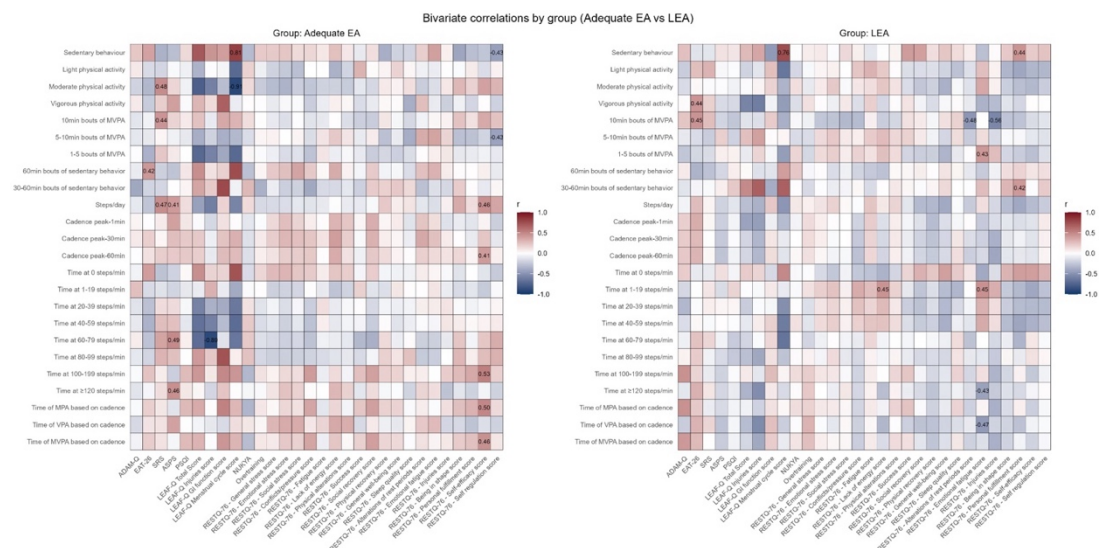


The lines represent the expected change in EA upon increasing the dominant behavior, relative to decreasing the other behavior. Compositional data analysis was used to model the impact of time-use shifts on EA (kcal/kg FFM/day). Abbreviations: EA, Energy availability; MVPA, Moderate Vigorous Physical Activity; LPA, Light Physical Activity; SB, Sedentary Behaviour.

Annexe 12. Figure S2. Relationship between physical activity and subjective well-being adjusted by EA.



Heatmap representing associations between energy availability and subjective well-being. The colours of the squares represent the partial Pearson correlation coefficients. Red colours represent positive Pearson coefficients, whereas blue depicts negative coefficients. Bold numbers inside the squares represent statistically significant partial Pearson correlation coefficients. Abbreviations: EA, Energy Availability; ADAM-Q, Androgen Deficiency in the Aging Male Questionnaire; ASPS, Athlete's Subjective Performance Scale; EAT-26, Eating Attitudes Test-26; GI, gastrointestinal; FDR, false discovery rate; LEAF-Q, Low Energy Availability in Females Questionnaire; MPA, moderate physical activity; MVPA, moderate-to-vigorous physical activity; NUKYA, Nutrition Knowledge for Young and Adult Athletes; PSQI, Pittsburgh Sleep Quality Index; RESTQ-76, Recovery-Stress Questionnaire for Athletes-76; SRS, Silhouette Rating Scale; VPA, vigorous physical activity.



Heatmap representing associations between energy availability and subjective well-being. The colours of the squares represent the Pearson correlation coefficients. Red colours represent positive Pearson coefficients, whereas blue depicts negative coefficients. Bold numbers inside the squares represent statistically significant Pearson correlation coefficients. Abbreviations: EA, Energy Availability; LEA, Low Energy Availability; ADAM-Q, Androgen Deficiency in the Aging Male Questionnaire; ASPS, Athlete's Subjective Performance Scale; EAT-26, Eating Attitudes Test-26; GI, gastrointestinal; FDR, false discovery rate; LEAF-Q, Low Energy Availability in Females Questionnaire; MPA, moderate physical activity; MVPA, moderate-to-vigorous physical activity; NUKYA, Nutrition Knowledge for Young and Adult Athletes; PSQI, Pittsburgh Sleep Quality Index; RESTQ-76, Recovery-Stress Questionnaire for Athletes-76; SRS, Silhouette Rating Scale; VPA, vigorous physical activity.

Annexe 14 . Ethics Committee Approval, Andalucia



DICTAMEN ÚNICO EN LA COMUNIDAD AUTÓNOMA DE ANDALUCÍA

D^a AURORA BUENO CAVANILLAS, EN CALIDAD DE PRESIDENTA DEL COMITÉ DE ÉTICA DE LA INVESTIGACIÓN BIOMÉDICA DE LA PROVINCIA DE GRANADA (CEIM/CEI GRANADA)

CERTIFICA:

Que este Comité ha evaluado la propuesta del promotor/investigador: , para realizar el estudio titulado:

TÍTULO DEL ESTUDIO: Prevalencia y consecuencias fisiológicas de una deficiencia energética relativa en el deporte (RED-S). Estudio PreCoRED.

Código protocolo: Estudio PreCoRED

Código Portal de Ética: 0909-N-22

Investigador Principal: LUCAS JURADO FASOLI

Centro: Universidad de Granada

Versión de los documentos:

Protocolo	Versión: 2 de fecha 01/06/2022
C.I.	Versión: 2 de fecha 01/06/2022
HIP	Versión: 2 de fecha 01/06/2022

Y considera que:

- Se cumplen los requisitos necesarios de idoneidad del protocolo en relación con los objetivos del estudio y se ajusta a los principios éticos aplicables a este tipo de estudios.
- La capacidad del investigador y los medios disponibles son apropiados para llevar a cabo el proyecto.
- El protocolo del estudio contempla de forma adecuada toda la legislación aplicable
- Que los aspectos económicos involucrados en el proyecto, no interfieren con respecto a los postulados éticos.

Por lo que este Comité ha acordado emitir **DICTAMEN FAVORABLE** para la realización del dicho estudio, para lo cual corresponde a la Dirección del Centro/os correspondiente/s determinar si la capacidad y los medios disponibles son apropiados para llevarlo a cabo.

En el caso de que su estudio requiera acceso y uso de la información sanitaria contenida en los sistemas de información del sistema sanitario público de Andalucía, tendrán que estar a lo establecido en la Resolución Conjunta 1/2021 de fecha 04/12/2021, de la Secretaría General de Investigación, Desarrollo e Innovación en Salud de la Consejería de Salud y Familias y de la Dirección Gerencia del SAS, por la que se aprueban las Instrucciones para la ordenación de acceso y uso de la información sanitaria contenida en los sistemas de información del Sistema Sanitario Público de Andalucía con fines de investigación e innovación por la entidades dependientes de la Consejería de Salud y Familias.

Lo que firmo en Granada

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Annexe 15 . Ethics Committee Approval, UCAM



COMITÉ DE ÉTICA DE LA UCAM

DATOS DEL PROYECTO

Título:	"Impacto del gasto total diario en la deficiencia energética relativa (RED-S) de atletas. Estudio PreCoRED"		
Investigador Principal	Nombre	Correo-e	
Dr.	Carlos Javier Contreras Fernández	ccontreras@ucam.edu	

INFORME DEL COMITÉ

Fecha	24/02/2023	Código	CE022301
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Tipo de Experimentación

Investigación experimental clínica con seres humanos	
Investigación experimental no clínica con seres humanos	X
Utilización de tejidos humanos procedentes de pacientes, personas sanas, tejidos embrionarios o fetales	
Utilización de tejidos humanos, tejidos embrionarios o fetales procedentes de bancos de muestras o tejidos	
Investigación observacional, psicológica o comportamental en humanos	X
Uso de datos personales, información genética, etc.	X
Experimentación animal	
Utilización de agentes biológicos de riesgo para la salud humana, animal o las plantas	
Uso de organismos modificados genéticamente (OMGs)	

Comentarios Respecto al Tipo de Experimentación

Nada Obsta

Comentarios Respecto a la Metodología de Experimentación

Nada Obsta



Universidad Católica San Antonio de Murcia. Vicerrectorado de Investigación. Salida nº REC- 27/02/2023 17:18:41



COMITÉ DE ÉTICA DE LA UCAM

Sugerencias al Investigador

A la vista de la solicitud de informe adjunto por el Investigador y de las recomendaciones anteriormente expuestas el dictamen del Comité es:

Emitir Informe Favorable	X
Emitir Informe Desfavorable	
Emitir Informe Favorable condicionado a Subsanación	

MOTIVACIÓN

Incrementará conocimientos en su área

Vº Bº El Presidente,

Fdo.: José Alberto Cánovas Sánchez



El Secretario,

Fdo.: José Alarcón Teruel

Annexe 16. Informed Consent form

CONSENTIMIENTO INFORMADO DEL PARTICIPANTE

(Los artículos se refieren a la Ley de Investigación Biomédica, BOE 4 de julio de 2007)

D. / Dña. _____ con D.N.I. _____, y

fecha de nacimiento _____

Declaro que:

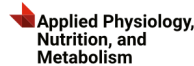
1. He leído (o me han leído) la hoja de información del proyecto: *Prevalencia y consecuencias fisiológicas de una deficiencia energética relativa en el deporte (RED-S). Estudio PreCoRED*, que me ha entregado el investigador responsable del proyecto: D. Lucas Jurado Fasoli con DNI 79034926A.

2. He comprendido la investigación que se va a realizar con mi participación y he tenido la oportunidad de resolver cualquier duda al respecto.

3. Así mismo, se me ha informado de que:

- Se hace constar que el participante manifiesta expresamente decir la verdad en sus respuestas para garantizar los datos reales sobre su estado físico o salud o los que se le solicitan (art. 23.1. L.I.B.).
- Tengo derecho a no otorgar mi consentimiento a participar y a revocarlo en cualquier momento del estudio (art. 4.3. L.I.B.).
- La falta de consentimiento a iniciar el estudio o su revocación una vez iniciado no me supondrá perjuicio alguno (en cualquier otro derecho) o discriminación (art. 4.4 y 6. L.I.B.).
- Seré informado, si así lo deseo, de los datos que se obtengan durante la investigación (art. 4.5 y 27.2. L.I.B.) y de la forma de obtener dicha información (art. 15.2. L.I.B.).
- Tengo derecho a decidir que no se me comuniquen los datos de la investigación (con las excepciones legales pertinentes) (art. 4.5. L.I.B.).
- Los datos que se obtengan con mi participación en la investigación son anónimos y si al publicarlos hay que mencionar mi nombre será precisa mi autorización (Ley Orgánica 3/2018, de 5 de diciembre, de Protección de Datos Personales y garantía de los derechos digitales).

Annexe 17. Published Article: “RMR and RMR Ratio Are Not Related to Energy Availability in Elite and Pre-Elite Athletes”



Research Article

RMR and RMR ratio are not related to energy availability in elite and pre-elite athletes

Sergio Espinar[✉], Juan J. Martín-Olmedo[✉], Marcos Rueda-Córdoba[✉], Olalla Prado-Nóvoa[✉], Carlos Contreras[✉], José Miguel Martínez-Sanz[✉], and Lucas Jurado-Fasoli[✉]

^{*}Faculty of Health Sciences, Catholic University of Murcia, Murcia, Spain; [†]Department of Physiology, Faculty of Medicine, Sport and Health University Research Institute (iMUDS), University of Granada, Granada, Spain; [‡]Department of Physical Education and Sports, Faculty of Sports Science, Sport and Health University Research Institute (iMUDS), University of Granada, Granada, Spain;

[§]Department of Education and Teacher Training, Faculty of Law, Education, and Humanities, Universidad Europea de Madrid, Madrid, Spain; ^{||}Innova, Health and Sport Institute, Murcia, Spain; [¶]Department of Nursing, Applied Dietetics, Nutrition and Body Composition Research Group (DANuC), Faculty of Health Sciences, University of Alicante, Alicante, Spain; ^{|||}Centro de Investigación Biomédica en Red Fisiopatología de la Obesidad y Nutrición (CIBERObn), Instituto de Salud Carlos III, 28029 Madrid, Spain; ^{||||}Instituto de Investigación Biosanitaria ibs.GRANADA, Granada, Spain

Corresponding author: Lucas Jurado-Fasoli (email: juradofasoli@ugr.es)

Abstract

Low energy availability (LEA), defined as an imbalance between dietary intake and exercise energy expenditure, has been associated with physiological and psychological dysfunction in athletes. The resting metabolic rate ratio (RMR_{ratio}), calculated as measured RMR divided by predicted RMR, is increasingly used as a surrogate marker for chronic energy deficiency when values fall below 0.9. Therefore, this study aimed to (1) compare the RMR_{ratio} across different predictive equations, (2) assess the association between energy availability (EA) and both RMR and RMR_{ratio}, and (3) explore the relationship between the RMR_{ratio} and phenotypical characteristics, including body composition, dietary intake, physical activity, sleep, and subjective well-being in Spanish competitive athletes. A total of 49 competitive athletes (33 males; 25.2 ± 5.6 years) from various sports were evaluated in this cross-sectional study. RMR was measured using indirect calorimetry and predicted using eleven validated equations. EA was estimated through dietary recalls and training logs. The prevalence of athletes classified as having metabolic suppression (RMR_{ratio} < 0.9) ranged from 34.7% to 93.9%, depending on the predictive equation used. No significant associations were observed between EA and either RMR or RMR_{ratio}. However, athletes with RMR_{ratio} ≥ 0.9 had significantly higher visceral adipose tissue mass, bone mineral content, and density. They also showed more favorable behavioral and psychological profiles, including greater sleep regularity, fewer sustained inactivity bouts, higher self-perceived performance, and lower general stress scores. In conclusion, while no relationship was observed between RMR or RMR_{ratio} and EA, the RMR_{ratio} was associated with different physiological, behavioral, and psychological parameters.

Key words: resting metabolic rate, energy availability, metabolic suppression, bone health, physical activity, athlete monitoring

1. Introduction

Energy availability (EA) is an essential determinant in understanding the physiological requirements of athletes and their impact on performance and health. EA is defined as the energy remaining for essential physiological functions after subtracting exercise energy expenditure (EEE) and is expressed in terms of fat-free mass (FFM) (Mountjoy et al. 2014). This metric provides insights into the balance between energy intake (EI) and the energy requirements of high exercise levels. An adequate EA is essential for sustaining normal physiological functioning, encompassing hormone regulation, bone integrity, and immune response (Areta et al. 2021; Mountjoy et al. 2023). When EA drops below a crit-

ical threshold (typically defined as <30 kcal/kg FFM/day), it leads to low energy availability (LEA), a condition associated with various detrimental effects, including those related to the Female Athlete Triad (Triad) and/or relative energy deficiency in sport (REDs) (Dipla et al. 2021; Maya and Misra 2022).

The causes of LEA involve a complex interplay between high training loads, calorie restriction, and sociocultural pressures, driven by widespread misconceptions such as the fear of carbohydrates or the belief that eliminating them enhances performance or aids weight management (Martinsen et al. 2010; Sundgot-Borgen and Torstveit 2010). These restrictive practices create a precarious state of energy imbal-



ance, leaving athletes at heightened risk for the physiological and performance-related consequences of LEA (Melin et al. 2024). Chronic LEA can result in serious health consequences, including hormonal disruptions such as functional hypothalamic amenorrhea in females and reduced testosterone levels in males, impaired bone health, increased injury risk, and psychological stress (Gordon et al. 2017; Nattiv et al. 2021; Gallant et al. 2025). These physiological disturbances not only compromise long-term health but also have immediate detrimental effects on athletic performance (Melin et al. 2019).

One key mechanism underlying these effects is the impact of LEA on resting metabolic rate (RMR). RMR, the largest component of total daily energy expenditure, refers to the energy required to sustain basic physiological functions at rest (Maclean et al. 2011; Fernández et al. 2024). Studies suggest that lower RMR could be used to determine energy status under conditions of LEA (De Souza et al. 2008; Siedler et al. 2023). This metabolic suppression may serve as a protective mechanism to conserve energy during periods of caloric restriction or inadequate EI (Trexler et al. 2014; Martin et al. 2022). However, in practical terms, a reduced RMR further exacerbates the mismatch between EI and expenditure, creating a feedback loop that perpetuates the state of energy imbalance and amplifies the physiological and performance-related consequences of LEA (Melin et al. 2019). Findings from previous research are inconsistent, with some studies reporting a direct link between LEA and suppressed RMR, while others report no significant association (Lee et al. 2020; Kuikman et al. 2024).

Beyond RMR, the RMR ratio (RMR_{ratio}), defined as the measured RMR divided by the predicted RMR, has emerged as a potential indicator of metabolic adaptation in athletes (Sterringer and Larson-Meyer 2022). An RMR_{ratio} below 0.9 is a frequently established threshold for identifying energy conservation mechanisms linked to LEA (Staal et al. 2018). However, recent consensus, such as the REDs CAT2 (Stellingwerff et al. 2023) highlights that the use of RMR as a standalone marker of LEA has notable limitations, and recommends its interpretation in conjunction with other physiological and clinical indicators. Given that a reduced RMR_{ratio} may reflect subclinical metabolic adaptations, its potential relationship with phenotypic traits could provide complementary insights into athletes' health status and risk of LEA beyond what is captured by traditional EA metrics.

Given the inconsistencies in the literature, further research is needed to clarify the utility of RMR/RMR_{ratio} in assessing energy status. In particular, the relationship between RMR and RMR_{ratio} with calculated EA in athletes remains unclear due to physiological adaptations and methodological variability. This study aimed to contribute to this knowledge gap by (1) comparing the RMR_{ratio} across different predictive equations, (2) examining the relationship between RMR and RMR_{ratio} with EA, and (3) exploring the association between RMR_{ratio} and phenotypic traits (i.e., body composition, dietary outcomes, physical activity, sleep quantity and quality, and subjective well-being) in competitive athletes.

2. Methods

2.1. Study design

This cross-sectional investigation recruited 49 competitive athletes (16 women) from diverse sports disciplines at regional, national, or international competitive tiers. Athletes' competitive level was determined using validated classification criteria as outlined by McKay et al. (2022). Participants were required to complete two scheduled visits to the Sport and Health University Research Center (iMUDS). The initial session comprised a detailed briefing on study objectives, protocol disclosure, and acquisition of written informed consent. A series of physiological and psychosocial assessments were conducted, including body composition analysis and RMR, along with a set of validated questionnaires designed to evaluate potential risk factors such as low testosterone levels in male athletes, the female athlete triad in female athletes, stress levels, sleep disturbances, eating disorders, and body image perception. Ethical approval for this study was granted by the Human Research Ethics Committee of the "Junta de Andalucía" (approval code: 0909-N-22) and followed the last revised Declaration of Helsinki. All participants received comprehensive information regarding the study's aims, procedures, and any associated risks and provided written informed consent prior to participation.

2.2. Participants

Participants were eligible if they met the following criteria: (1) aged between 18 and 35 years, (2) actively competing as nationally registered athletes with at least two consecutive years of experience, (3) for women, not pregnant or breastfeeding during the study period, (4) absence of any diagnosed cardiovascular conditions or chronic illnesses, and (5) not using tobacco, recreational drugs, alcohol excessively, or any medications with potential to interfere with study outcomes.

2.3. Resting metabolic rate

RMR was assessed using indirect calorimetry under standardized conditions to ensure accuracy and reproducibility. Participants arrived at the laboratory in a fasted state (at least 8 h) and refrained from strenuous physical activity, caffeine, and alcohol consumption for at least 24 h prior to the measurement. Upon arrival, they rested in a supine position on a bed in a thermoneutral environment for a minimum of 20 min to acclimate before data collection (Fullmer et al. 2015).

Gas exchange measurements were obtained using a ventilated canopy hood system connected to the Quark RMR metabolic cart (COSMED, Rome, Italy). The measurement period lasted 30 min, during which oxygen consumption (VO_2) and carbon dioxide production (VCO_2) were continuously recorded at a sampling frequency of 5 s. To ensure data quality, the first 5 min of recorded values were discarded, and the mean VO_2 and VCO_2 from the remaining 25 min were used for analysis. Resting energy expenditure (REE) was then calculated using Weir's abbreviated equation, assuming negligible nitrogen excretion (Weir 1949).

2.4. RMR_{ratio} calculation

To determine the RMR_{ratio}, we first obtained the measured RMR via indirect calorimetry. Predicted RMR values were then estimated using a suite of published equations, including Cunningham (Cunningham 1980), Ten Haaf (both FFM and body weight-based variants), Tinsley (body weight and fat-free mass versions) (Ten Haaf and Weijs 2014), Wong (Wong et al. 2012), Freire (Freire et al. 2022), Van Hooren (Van Hooren et al. 2023), Harris-Benedict, Pontzer (body weight and FFM versions) (Garrel et al. 1996; Pontzer et al. 2021), and De Lorenzo (De Lorenzo et al. 1999).

Among these, Ten Haaf et al.'s equation was selected to examine the relationship between RMR_{ratio} and phenotypic traits, based on recent work by O'Neill et al. (2023) indicating its superior accuracy (especially in athletic cohorts) relative to other commonly utilized predictive models (O'Neill et al. 2023). In addition, the Ten Haaf equation was specifically validated in physically active populations and incorporates FFM as a key determinant (Ten Haaf and Weijs 2014), making it more suitable for sports performance and athlete monitoring contexts.

The RMR_{ratio} was determined by dividing the measured RMR by the predicted RMR. A threshold of 0.90 was used as the cut-off point to indicate metabolic suppression, as commonly referenced in the literature for detecting metabolic adaptations associated with LEA (Sterringer and Larson-Meyer 2022). This approach allows for a more precise assessment of potential metabolic adaptations that may arise in competitive athletes due to energy deficiency, training load, or other physiological stressors.

2.5. Energy availability assessment

EA was estimated following the method described by Loucks et al. (1998), which integrates dietary EI and FFM to provide a physiologically relevant index. The formula applied was: $EA = (EI \text{ (kcal)} - EEE \text{ (kcal)})/FFM \text{ (kg)}$. FFM was derived from a whole-body Dual-Energy X-ray Absorptiometry (DXA) scan.

Dietary intake was assessed through three non-consecutive 24 h dietary recalls. Participants were systematically guided through the 24 h dietary recall, strategically distributed to include two weekdays and one weekend or holiday. All interviews were conducted in person or via video call by qualified dietitians trained in standardized data collection techniques. To enhance the accuracy of portion size estimation, standardized photographic aids depicting commonly consumed food portions were employed, allowing for more precise and consistent reporting of both food and beverage intake. Nutritional analysis, including total EI and macronutrient distribution, was performed using EvalFINUT[®] software, which incorporates compositional data from both the United States Department of Agriculture and the Spanish Food Composition Database (BEDCA).

Participants maintained a daily log documenting the duration (in hours) and frequency of various physical activities. Energy expenditure for each activity was calculated using the following equation: $\text{Energy Expenditure (kcal)} = \text{MET (kcal/kg/h)} \times \text{Body Weight (kg)} \times \text{Duration (hours)}$ (Herrmann

et al. 2024). These daily values were then averaged over seven consecutive days to derive a weekly mean, integrating both resting metabolic demands and activity-specific energy costs to provide a comprehensive estimate of total energy expenditure.

Importantly, the three non-consecutive 24-h dietary recalls were conducted within the same 7-day period in which physical activity logs were recorded for the estimation of EEE. This ensured temporal alignment between EI and EEE measures. While the recalls did not cover all seven days, they were distributed strategically (two weekdays and one weekend/holiday) to reflect habitual intake and to overlap with the week of activity monitoring. Thus, both dietary and training data represent the same period, providing a coherent basis for the calculation of EA.

2.6. Body composition and anthropometrics

Body weight and height were measured to the nearest 0.1 kg and 0.1 cm, respectively, using a digital scale and stadiometer (Seca model 799, Electronic Column Scale, Hamburg, Germany). Participants wore light clothing and no shoes. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m²). A whole-body DXA scan was then performed with a Discovery Wi device (Hologic, Bedford, Massachusetts, USA) employing APEX software (version 4.0.2), providing values for lean mass, lean mass index (LMI), fat-free mass (FFM), fat mass (FM), fat mass index (FMI), android FM, gynoid FM, visceral adipose tissue (VAT) mass, total body bone mineral density (BMD), total body bone mineral content (BMC), and the total body BMD Z-score. Lean and fat mass indices were expressed in kg/m², while fat mass was also noted as a percentage of total body weight. During the DXA procedure, participants remained motionless as the scanning arm collected detailed images. All measurements were obtained under standardized conditions, adhering to the manufacturer's guidelines for calibration and quality control.

2.7. Assessment of phenotypical traits

A broad set of phenotypical traits relevant to LEA was evaluated. Dietary outcomes (energy, macronutrient, and fiber intakes) were evaluated through 24-h recalls as aforementioned. We assessed physical activity and sleep quality through accelerometry as detailed elsewhere (Rueda-Cordoba et al. 2025). Subjective health and wellness were assessed through validated questionnaires. Symptoms of androgen deficiency by the Androgen Deficiency in Aging Males Questionnaire (ADAM-Q) (Mohamed et al. 2010), eating behaviors and attitudes linked to eating disorders by the Eating Attitudes Test-26 (EAT-26) (Constain et al. 2014), body image perceptions by the Silhouette Rating Scale (SRS) (Lombardo et al. 2022), athlete's self-perceived performance by the Athlete's Subjective Performance Scale (ASPS) (Lee et al. 2023), subjective quality and quantity of sleep by the Pittsburgh Sleep Quality Index (PSQI) (Royuela 1996), indicators of LEA in female athletes by the Low Energy Availability in Females Questionnaire (LEAF-Q) (Melin et al. 2014), athletes' nutritional knowledge by the Nutrition Knowledge Questionnaire for Young Athletes (NUKYA) (Vázquez-Espino et al. 2020), the

Table 1. Descriptive parameters.

	All (n = 49)		RMR ratio measured/ predicted < 0.9 (n = 34)		RMR ratio measured/ predicted ≥ 0.9 (n = 15)	
	Mean	SD	Mean	SD	Mean	SD
Age (years)	25.2	5.6	26.1	6.1	23.3	3.7
Sex (n, %)						
Male (n, %)	33	67.3	22	64.7	11	73.3
Female (n, %)	16	32.7	12	35.3	4	26.7
Weight (kg)	71.4	11.7	68.7	11.3	77.6	10.5
Height (cm)	173.6	8.2	172.4	8.2	176.4	7.8
BMI (kg/m ²)	23.6	3.0	23.0	2.8	24.9	3.2
FFM (kg)	56.0	10.1	54.4	10.4	59.7	8.7
FM (%)	20.0	7.0	19.5	6.2	21.3	8.6
Training experience (years)	9.0	6.4	9.8	7.0	7.4	4.7
Competition level						
Regional (n, %)	28	57.1	21	61.8	7	46.7
National (n, %)	18	36.7	10	29.4	8	53.3
International (n, %)	3	6.1	3	8.8	0	0

Note: Data presented as mean and standard deviation (SD), unless otherwise indicated. The RMR_{ratio} measured/predicted was calculated using the ten Haaf (FFM) equation for the predicted RMR. Abbreviations: BMI, body mass index; FFM, fat-free mass; FM, fat mass; RMR, resting metabolic rate.

risk of overtraining by the Overtraining Score (Brun 2003a), and the balance between stress and recovery by the Recovery-Stress Questionnaire for Athletes (RESTQ-76) (Brun 2003b).

2.8. Statistical analysis

Unless otherwise indicated, descriptive data are expressed as mean and standard deviation (SD). Data normality was checked using histograms, Q-Q plots, box plots, and the Shapiro-Wilk test. Differences in the RMR_{ratio} depending on the equation used to obtain the RMR predicted were analyzed using repeated measures Analysis of variance (ANOVA) with post hoc Bonferroni pairwise comparisons. Sphericity was determined by Mauchly's test, and the Greenhouse-Geisser correction was applied when required. Differences in the prevalence of participants with a ratio lower or higher than 0.9 depending on the predictive equation used were analyzed by the χ^2 test.

Then, we conducted Pearson correlation analyses to examine the relationship between RMR and RMR_{ratio} with EA. Differences in RMR and RMR_{ratio} between participants with LEA and adequate EA were assessed using independent samples *t* tests.

We also used Pearson correlation analyses to study the relationship between the RMR_{ratio} and phenotypical traits (i.e., body composition, dietary outcomes, physical activity, sleep quantity and quality, and subjective well-being). Lastly, we conducted independent samples *t* tests to compare body composition, dietary outcomes, sleep quality and quantity, and subjective well-being between individuals with an RMR_{ratio} higher or lower than 0.9. To represent differences between individuals with an RMR_{ratio} higher or lower than 0.9, we further calculated the Z-scores and 95% confidence interval of the mean differences between groups. To account for multiple testing in the Pearson correlation analyses and independent samples *t* test analyses, we used the two-stage step-

up method of the false discovery rate (FDR) (Benjamini et al. 2001).

The level of statistical significance was set at $P < 0.05$. Analyses were performed in Statistical Package for the Social Sciences v.27.0 (IBM Corporation, Pittsburgh, PA, USA) and R software version 4.1.2 (<https://cran.r-project.org/>). The R Project for Statistical Computing, Vienna, Austria). Plots were built using GraphPad Prism software v.8 (GraphPad Software, San Diego, CA, USA) and R software version 4.1.2 (<https://cran.r-project.org/>). The R Project for Statistical Computing, Vienna, Austria).

3. Results

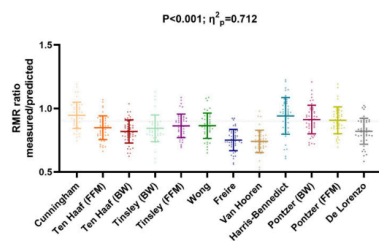
3.1. Participant characteristics

A total of 49 Spanish competitive athletes (33 males and 16 females), with a mean age of 25.2 ± 5.6 years, participated in the study (Table 1). The cohort included athletes competing at regional (57.1%), national (36.7%), and international (6.1%) levels. The average BMI was 23.6 ± 3.0 kg/m², and the mean training experience was 9.0 ± 6.4 years. No significant sex-based differences were observed in RMR_{ratio} categories or EA status (both $P > 0.05$; Table S1); consequently, the analyses are presented together for both sexes.

3.2. Differences in the RMR_{ratio} based on predictive equations

Statistical differences in the RMR_{ratio} values were identified depending on the predictive equation used for RMR estimation ($P < 0.001$; Fig. 1 and post hoc comparisons shown in Table S2). The mean RMR ratio ranged from 0.74 ± 0.09 using the Van Hooren equation to 0.95 ± 0.10 with the Cunningham equation ($P < 0.001$; Table S2). In line with these findings, the proportion of participants classified as having a suppressed RMR ratio (<0.9) also varied significantly across pre-

Fig. 1. Differences in the RMR ratio measured/predicted depending on the predictive equation used to predict the RMR in all participants ($n = 49$). P values and partial eta squared (η^2_p) were obtained from repeated measures ANOVA. Post hoc Bonferroni correction is displayed in Table S2. The dashed grey line indicates the 0.9 threshold for considering a high or low RMR ratio. Abbreviations: BW, body weight; FFM, fat-free mass; RMR, resting metabolic rate.



dictive equations ($P < 0.001$; Table 2). Notably, the Freire and Van Hooren equations identified the highest proportion of participants as metabolically suppressed, with 93.9% falling below and only 6.1% upper the 0.9 threshold (Table 2). Meanwhile, other models, such as both versions of the Pontzer equation, resulted in more balanced classifications, with over half of the athletes exhibiting $\text{RMR}_{\text{ratio}} \geq 0.9$ (Table 2).

The Ten Haaf equation based on FFM, which served as the reference model in this study, classified 69.4% of athletes as having suppressed $\text{RMR}_{\text{ratio}}$, reflecting a moderate sensitivity relative to other equations.

These analyses were similar when in the male subgroup (Table S5).

3.3. Relationship between RMR and $\text{RMR}_{\text{ratio}}$ with energy availability

We found no significant association between RMR and EA ($r = -0.117$, $P = 0.424$; Fig. 2A), and when participants were classified in LEA (EA, <30 kcal/kg FFM/day) or adequate EA (EA, ≥ 30 kcal/kg FFM/day) no statistically significant differences were found in RMR ($P = 0.520$; Fig. 2B). Similarly, no relationship was observed between EA and $\text{RMR}_{\text{ratio}}$ ($r = 0.024$, $P = 0.871$; Fig. 2C), and no differences were observed between participants with LEA or EA in the $\text{RMR}_{\text{ratio}}$ ($P = 0.819$; Fig. 2D).

All these analyses were similar in the male subgroup (Table S6).

3.4. Relationship between $\text{RMR}_{\text{ratio}}$ and phenotypical characteristics

Given the potential predictive role of the $\text{RMR}_{\text{ratio}}$ as a surrogate marker, we studied the association with different phenotypical parameters (Figs. 3A, 3B, and 4A), and participants were stratified into two groups according to their $\text{RMR}_{\text{ratio}}$

based on the 0.9 threshold to compare the same parameters (Figs. 3C, 3D, and 4B).

Regarding body composition, the $\text{RMR}_{\text{ratio}}$ was positively correlated with VAT mass ($r = 0.317$, $P = 0.026$; Fig. 3A and Table S3). Similarly, individuals with $\text{RMR}_{\text{ratio}} \geq 0.9$ presented significantly greater VAT mass ($P = 0.003$), BMC ($P = 0.042$), and BMD ($P = 0.041$) compared to their counterparts with suppressed ratio (Fig. 3C and Table S4).

We also observed a positive correlation between the $\text{RMR}_{\text{ratio}}$ and moderate intensity physical activity ($r = 0.34$; $P = 0.029$; Fig. 3B and Table S3). However, no statistically significant differences were observed between individuals with $\text{RMR}_{\text{ratio}}$ higher or lower than 0.9 (all $P > 0.05$; Fig. 3D and Table S4).

In the sleep domain, we found that the $\text{RMR}_{\text{ratio}}$ was negatively correlated with the time spent in sustained inactivity during waking hours of the night ($r = -0.332$, $P = 0.034$; Fig. 3B and Table S3) and for sustained inactivity bouts during night longer than 15 min ($r = -0.339$, $P = 0.030$; Fig. 3B and Table S3). Indeed, we also showed that individuals with $\text{RMR}_{\text{ratio}} \geq 0.9$ presented lower time spent in sustained inactivity bouts during waking hours of the night ($P = 0.002$) as well as the number of inactivity bouts ($P = 0.009$) (Fig. 3D and Table S4). Conversely, we revealed a positive correlation between the $\text{RMR}_{\text{ratio}}$ and the sleep regularity index ($r = 0.339$, $P = 0.030$; Fig. 3B and Table S3) and higher values of this index in individuals with $\text{RMR}_{\text{ratio}} \geq 0.9$ than their counterparts ($P = 0.022$; Fig. 3D and Table S4).

Regarding subjective and psychological variables (Fig. 4), we showed a significant inverse correlation between the $\text{RMR}_{\text{ratio}}$ and the ASPS score ($r = -0.384$, $P = 0.006$), the ADAM-Q score ($r = -0.427$, $P = 0.013$), the RESTQ-76 success ($r = -0.310$; $P = 0.034$), and personal fulfillment ($r = -0.332$; $P = 0.023$) scores (Fig. 4A and Table S3). In addition, $\text{RMR}_{\text{ratio}} \geq 0.9$ reported lower general stress scores as measured by the RESTQ-76 ($P = 0.024$; Fig. 4B and Table S4).

Of note, no significant correlation or differences were observed in terms of dietary intake or macronutrient distribution (all $P > 0.05$; Fig. 3 and Tables S3-4), and no significant relationship was observed between the $\text{RMR}_{\text{ratio}}$ and phenotypical traits when accounting for multiple comparisons correction, adjusting for FDR (all $P > 0.05$; Tables S3-4).

These analyses were similar when in the male subgroup (Tables S6-7).

4. Discussion

While the $\text{RMR}_{\text{ratio}}$ has been proposed as a non-invasive marker of metabolic suppression and LEA, our study provides a broader perspective. We demonstrated that the choice of predictive equation substantially affects $\text{RMR}_{\text{ratio}}$ estimation and, consequently, its potential diagnostic role. Moreover, we found no significant associations between RMR or $\text{RMR}_{\text{ratio}}$ and EA, nor differences between athletes classified as having LEA or adequate EA. However, athletes with $\text{RMR}_{\text{ratio}} \geq 0.9$ exhibited greater bone mass, and exploratory analyses suggested potential links with certain behavioral and psychological traits, although these did not remain significant after correction for multiple comparisons. Taken together, these

Table 2. Differences in the prevalence of participants with a ratio lower or higher than 0.9.

	RMR ratio measured/predicted				P value
	<0.9		>0.9		
	N	%	N	%	
Cunningham	17	34.7	32	65.3	<0.001
Ten Haaf (FFM)	34	69.4	15	30.6	
Ten Haaf (BW)	39	79.6	10	20.4	
Tinsley (BW)	40	81.6	9	18.4	
Tinsley (FFM)	32	65.3	17	34.7	
Wong	32	65.3	17	34.7	
Freire	46	93.9	3	6.1	
Van Hooren	46	93.9	3	6.1	
Harris-Benedict	18	36.7	31	63.3	
Pontzer (BW)	24	49.0	25	51.0	
Pontzer (FFM)	24	49.0	25	51.0	
De Lorenzo	37	75.5	12	24.5	

Note: P value obtained from χ^2 test. Abbreviations: BW, body weight; FFM, fat-free mass; RMR, resting metabolic rate.

Fig. 2. Relationship between RMR and RMR ratio measured/predicted with energy availability. The RMR ratio measured/predicted was calculated using the ten Haaf (FFM) equation for the predicted RMR. Panels A and C display Pearson correlation analyses between RMR (A) and RMR ratio measured/predicted (C) with energy availability. Panels B and D represent differences in RMR (B) and RMR ratio measured/predicted (D) between participants with low energy availability and adequate energy availability. P values obtained from independent samples t tests. In panel D, the dashed grey line indicates the 0.9 threshold for considering a high or low RMR ratio. Abbreviations: EA, energy availability; FFM, fat-free mass; LEA, low energy availability; RMR, resting metabolic rate.

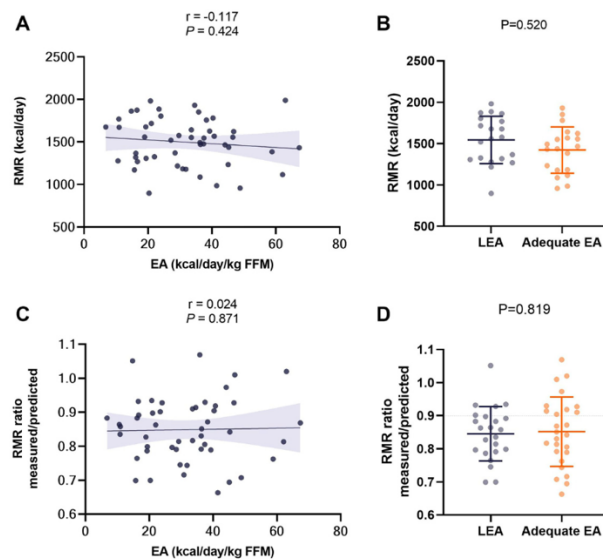




Fig. 3. Relationship between resting metabolic rate (RMR) ratio measured/predicted and phenotypical traits (i.e., body composition, dietary outcomes, physical activity, sleep quantity and quality). The RMR ratio measured/predicted was calculated using the ten Haaf (FFM) equation for the predicted RMR. Panels A and B: Heatmaps representing associations between energy availability and body composition (A), physical activity, sleep quantity and quality (B). The colors of the squares represent the Pearson correlation coefficients. Red colors represent positive Pearson coefficients, whereas blue depicts negative coefficients. Bold numbers inside the squares represent statistically significant Pearson correlation coefficients ($P < 0.05$). Panels C and D: Forest plots indicating the differences in body composition (C), and physical activity, sleep quantity and quality (D), between individuals with a ratio higher than 0.9 and individuals with a ratio lower than 0.9. Blue points indicate the z-score with the 95% confidence interval. Red asterisks indicate statistically significant differences between groups ($P < 0.05$). P values obtained from an independent samples t test. Abbreviations: BMC, total body bone mineral content; BMD, total body bone mineral density; EA, energy availability; ECW, extracellular water; FFM, fat-free mass; FM, fat mass; FMI, fat mass index; LEA, low energy availability; LMI, lean mass index; MPA, moderate physical activity; MVPA, moderate to vigorous physical activity; VAT, visceral adipose tissue; VPA, vigorous physical activity; WASO, wake after sleep onset.

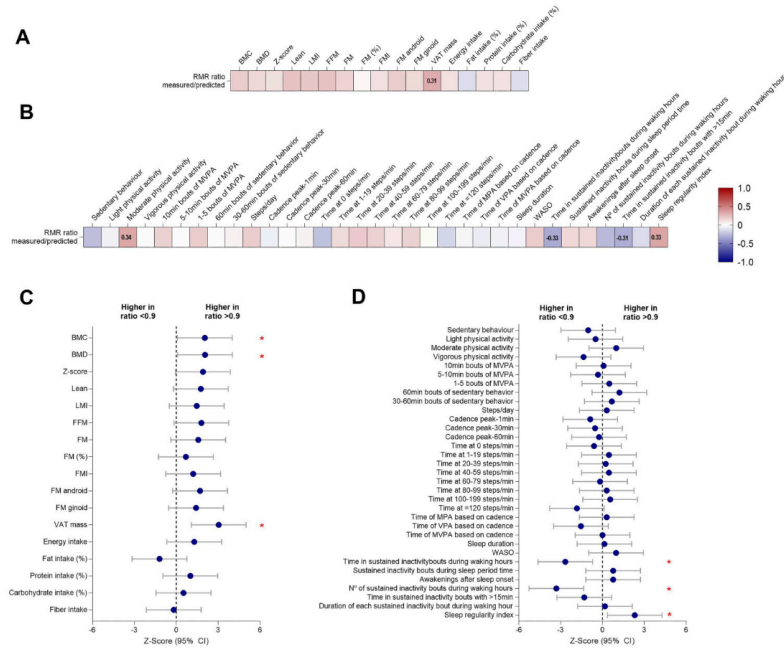
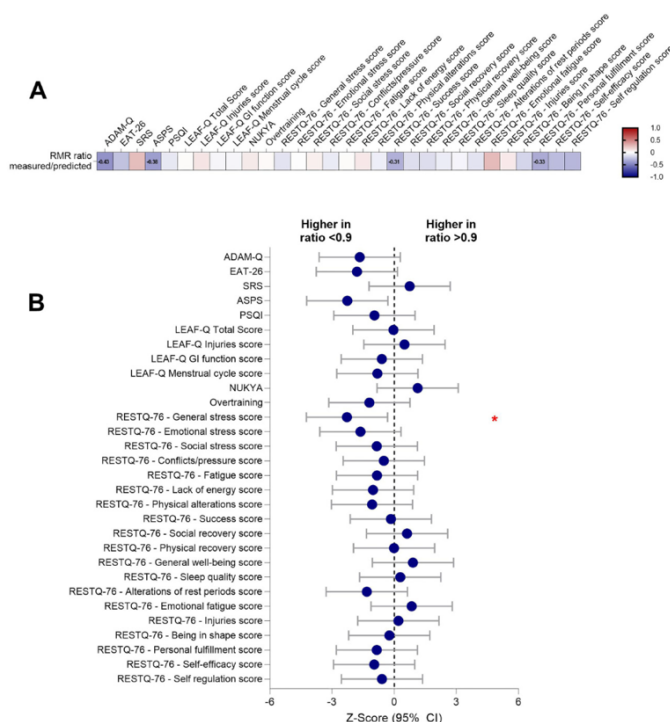


Fig. 4. Relationship between resting metabolic rate (RMR) ratio measured/predicted and subjective well-being. The RMR ratio measured/predicted was calculated using the ten Haaf (FFM) equation for the predicted RMR. Panel A: Heatmap representing associations between energy availability and subjective well-being. The colors of the squares represent the Pearson correlation coefficients. Red colors represent positive Pearson coefficients, whereas blue depicts negative coefficients. Bold numbers inside the squares represent statistically significant Pearson correlation coefficients ($P < 0.05$). Panels B: Forest plot indicating the differences in subjective well-being between individuals with a ratio higher than 0.9 and individuals with a ratio lower than 0.9. Blue points indicate the z-score with the 95% confidence interval. Red asterisks indicate statistically significant differences between groups ($P < 0.05$). P values obtained from independent samples t test. Abbreviations: ADAM-Q, Androgen Deficiency in the Aging Male Questionnaire; ASPS, Athlete's Subjective Performance Scale; EA, energy availability; EAT-26, Eating Attitudes Test-26; GI, gastrointestinal; LEA, low energy availability; LEAF-Q, Low Energy Availability in Females Questionnaire; NUKYA, Nutrition Knowledge for Young and Adult Athletes; PSQI, Pittsburgh Sleep Quality Index; RESTQ-76, Recovery-Stress Questionnaire for Athletes-76; SRS, Silhouette Rating Scale.



interpretation of the RMR_{ratio} is directly driven by the predictive equation chosen, with a prevalence of low RMR_{ratio} ranging from 1% to 68% (Heikura et al. 2025). Consequently, Heikura et al. concluded that current generic equations are inadequate for REDs screening and advocated the develop-

ment of sport- and phenotype-specific models with adaptable RMR_{ratio} thresholds.

This wide variability underscores the importance of standardizing predictive models when using RMR_{ratio} as a diagnostic or screening tool. Equations such as Freire or Van

Hooren, which incorporate recent physiological assumptions or are derived from athletic populations (Alcantara et al. 2024), tended to produce lower predicted values, classifying a much higher proportion of athletes as suppressed. Thus, without a validated gold-standard prediction model specific to athletes, interpretation of the RMR_{ratio} remains vulnerable to methodological inconsistency.

4.2. RMR and RMR_{ratio} as inadequate as markers of energy availability

Contrary to previous studies that reported a reduction in RMR or RMR_{ratio} in athletes with LEA (De Souza et al. 2008; Staal et al. 2018), our findings showed no significant differences in RMR or RMR_{ratio} between LEA and adequate EA groups. These discrepancies may be attributed to several factors. First, in our study EA was estimated using 24-h dietary recalls and self-reported physical activity logs, which, despite being widely used, are subject to recall bias and underreporting—especially in athletic populations (Johansson et al. 2001; Lopes et al. 2016). Second, EA represents a dynamic and often fluctuating construct, and single-point measurements may not adequately capture chronic energetic status (Reed et al. 2013; Jesus et al. 2022). Lastly, individual variability in metabolic adaptation could result in some athletes maintaining relatively high RMR despite sustained periods of low EA, further complicating the interpretation of these markers (Kinoshita et al. 2021).

LEA is a critical risk factor for developing the REDs (Gallant et al. 2025). In this sense, a recent study found that 27.9% of athletes identified as moderate or high risk for REDs exhibited LEA based on RMR_{ratio} (Marzuki et al. 2024). However, that study inferred energy deficiency from RMR-based metrics alone, without directly measuring EA or applying a validated clinical assessment for REDs. This highlights the ongoing uncertainty around the diagnostic validity of RMR and RMR_{ratio} in the context of energy deficiency.

Our findings also contrast with those of Garay et al. (2025), who examined the relationship between predicted EA and RMR_{ratio} in physically active college-aged females. Their results suggest that a low RMR_{ratio} may be a more sensitive marker of energy deficiency than calculated EA, depending on the protocols used (Garay et al. 2025).

Although our study was not designed to diagnose or monitor REDs, the findings contribute to the ongoing debate on the role of RMR/RMR_{ratio} in this context. Recent consensus stresses the importance of integrating multiple objective indicators when assessing energy deficiency in athletes (Stellingwerff et al. 2023). In line with these recommendations, our results suggest that RMR and RMR_{ratio} should not be used in isolation, as they may not reflect actual EA. Instead, they could serve as complementary tools when used in conjunction with standardized clinical assessments.

4.3. The RMR ratio as a physiological marker beyond energy availability

Although the RMR_{ratio} was not associated with EA, it did demonstrate relationships with several indicators of physiological and psychological status. In this study, higher RMR_{ratio}

was associated with greater bone mineral content and density and more favorable patterns of sleep and activity, suggesting that this ratio may reflect broader systemic health. Notably, athletes with RMR_{ratio} ≥ 0.9 also reported lower general stress and higher subjective performance. The observed associations between the RMR_{ratio} and variables such as sleep behavior, sedentary patterns, and psychological stress highlight the importance of integrating behavioral and psychosocial factors into the investigation of LEA and REDs. Additionally, the observed positive association between the RMR ratio and bone-related metrics—including bone mineral density and content—aligns with previous research indicating that metabolic suppression may adversely affect bone remodeling and mineralization, particularly among physically active individuals (Hilkens et al. 2023).

These findings align with previous literature proposing that RMR suppression may reflect a broader metabolic adaptation to cumulative stressors—nutritional, physical, or psychological (Trexler et al. 2014; Martin et al. 2022). Thus, the RMR_{ratio} may function less as a direct proxy for EA and more as an integrative marker of energy balance, behavioral regularity, and overall physiological adaptations. However, our results should be interpreted with caution since no significant relationships were observed when accounting for multiple comparisons.

4.4. Strengths and limitations

This study's strengths include the use of indirect calorimetry for RMR measurement, DXA for body composition, and accelerometry for sleep and physical activity monitoring. In addition, the inclusion of validated psychological scales adds further depth to the characterization of phenotypic traits. Another notable contribution is the inclusion of a wide range of predictive equations, including some that had not been previously applied in athletic populations, contributing valuable comparative data to the literature on their potential applicability in sports science. However, limitations include its cross-sectional design, which precludes causal inference, and reliance on self-reported food and training logs to estimate EA. Additionally, the unbalanced sex distribution of our population could affect the results obtained in this study. For this reason, future studies with larger and more sex-balanced cohorts are warranted to explore potential sex differences in this relationship.

5. Conclusion

Our findings indicate that the predictive equation selected for estimating RMR significantly influences the RMR_{ratio} , thereby limiting its diagnostic consistency. In addition, although RMR and RMR_{ratio} were not associated with EA, the RMR_{ratio} was related to several physiological, behavioral, and psychological traits in competitive athletes. In line with recent REDs CAT2 recommendations, our results reinforce that RMR and RMR_{ratio} should not be used in isolation but rather as complementary tools within multifactorial models for athlete monitoring. Future studies should explore the longitudinal predictive value of the RMR_{ratio} and its integration with



other objective indicators to improve the assessment of energy deficiency risk.

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Data availability

The data supporting this study's findings are available from the corresponding author, LJ-F, upon reasonable request.

Author information

Author ORCIDs

Sergio Espinar <https://orcid.org/0000-0002-8928-6054>

Juan J. Martin-Olmedo <https://orcid.org/0009-0005-8072-0116>

Marcos Rueda-Córdoba <https://orcid.org/0009-0000-4486-6998>

Lucas Jurado-Fasoli <https://orcid.org/0000-0002-5254-1816>

Author notes

Sergio Espinar and Juan J. Martin-Olmedo contributed equally to this work

Author contributions

Conceptualization: SE, JJM, MR, LJ

Data curation: JJM

Formal analysis: JJM

Investigation: SE, LJ

Methodology: SE, JJM, MR, OP, CC, JMM

Project administration: LJ

Software: SE, JJM, MR, OP, LJ

Supervision: CC, JMM, LJ

Writing – original draft: SE, JJM

Writing – review & editing: SE, JJM, MR, OP, CC, JMM, LJ

Competing interests

The authors state that there are no conflicts of interest.

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Supplementary material

Supplementary data are available with the article at <https://doi.org/10.1139/apnm-2025-0200>.

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