Evaluating the associations between energy availability, eating attitudes, and bone strength in young endurance-trained individuals

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Abstract

Background: Endurance athletes are at a greater risk of low energy availability (EA) due to high-volume of training, elevated nutritional demands, and a higher prevalence of disordered eating. Low EA can lead to impaired bone health including an uncoupling in bone turnover and reductions in bone mineral density (BMD), bone microarchitecture, and bone strength. However, few studies have explored the associations between EA, eating attitudes, and bone health in endurance-trained individuals, with no studies describing sex differences.

Objective: The objective of this thesis is to evaluate the associations between EA, eating attitudes, and bone strength in young male and female endurance-trained individuals.

Methods: We recruited 43 healthy males and females aged 18-35 years with no known medical conditions/medication use affecting bone metabolism who participated on a competitive endurance sports team and/or in regular weight-bearing endurance exercise (≥180 minutes/week). Accelerometers were used to determine physical activity levels. Incremental treadmill testing to volitional exhaustion was performed to measure maximal aerobic capacity (VO_{2max}). Peripheral quantitative computed tomography (pQCT) was performed to measure total, trabecular, and cortical volumetric bone mineral density (vBMD) and area, and stress-strain index (SSI) at the distal (4% tibia length) and proximal tibia (38% and 66% tibia length). Dual-energy X-ray absorptiometry (DXA) was used to determine lean body mass (LBM) and areal BMD (aBMD) at the total hip, and femoral neck. EA was calculated over a 7-day period using dietary energy intake from 24-hour dietary recall and exercise energy expenditure using polar heart rate monitors and an individualized heart rate-VO_2 equation based on an incremental treadmill testing, adjusted for LBM from DXA. Drive for thinness, body dissatisfaction and bulimia subscales from the Eating Disorder Inventory-3 (EDI-3) were administered to assess DE

attitudes. Pearson/Spearman correlation coefficients and multivariable linear regression models adjusting for age, sex, and BMI were developed. Independent samples t-tests were used to compare outcomes between sexes.

Results: Forty-three participants participated in the study (72% male, age 25.54 ± 4.25 years, BMI 22.81 ± 2.88 kg/m^2, percent body fat 18.58 ± 6.24%, LBM 54.16 ± 10.67 kg, VO_{2max} 57.02 ml/min/kg, moderate-to-vigorous physical activity 667.3 (356 – 3349) minutes/week, EA 39.11 ± 14.02 kcal/kg LBM/d). No differences in EA and EDI-3 outcomes were observed between sexes. Male participants had higher total vBMD ($p = 0.020$), total area ($p = 0.011$), trabecular vBMD ($p = 0.005$), and trabecular area ($p < 0.001$) at 4% site; higher cortical area and SSI at 38% ($p < 0.001$) and 66% site ($p < 0.001$), and higher total area at 66% site ($p < 0.001$) compared to females. EA was negatively associated with trabecular area ($r = -0.333$, $p = 0.036$) and SSI at 38% site ($r = -0.339$, $p = 0.032$), and positively associated with cortical area at 66% site ($r_s = 0.459$, $p = 0.003$). Negative correlations were observed between drive for thinness and EA ($r_s = -0.380$, $p = 0.014$) and EDI-3 total score ($r_s = -0.316$, $p = 0.044$). No associations were observed between EDI-3 scores and pQCT bone outcomes. When adjusted for age, sex, and BMI, none of the associations between EA, EDI-3 outcomes, and pQCT outcomes remained significant.

Conclusion: The findings indicate that field-based EA measures may lack sensitivity in identifying associations between EA and bone strength among male and female endurance athletes. The observed associations agree with the existing evidence that DE attitudes may be robust surrogate markers of energy deficiency when screening athletes and exercising individuals who are at risk of low EA and bone fragility. Future prospective studies in a larger sample size

with longer-term assessments of EA are needed to confirm these associations between EA, eating attitudes, and pQCT measures of bone strength, and evaluate potential sex differences.

Résumé

Contexte: Les athlètes d'endurance courent un plus grand risque de faible disponibilité énergétique (EA) en raison du volume élevé d'entraînement, des exigences nutritionnelles élevées et d'une prévalence plus élevée de troubles de l'alimentation. Une faible disponibilité énergétique peut entraîner une détérioration de la santé osseuse, notamment un découplage du renouvellement osseux et une réduction de la solidité des os. Cependant, peu d'études ont exploré les associations entre la disponibilité énergétique, les attitudes alimentaires et la santé osseuse chez les personnes entraînées à l'endurance, et aucune étude n'a décrit les différences entre les sexes.

Objectif: L'objectif de cette thèse est d'évaluer les associations entre l'EA, les attitudes alimentaires et la solidité des os chez les jeunes hommes et femmes pratiquant l'endurance.

Méthodes: Nous avons recruté 43 hommes et femmes en bonne santé, âgés de 18 à 35 ans et faisant partie d'une équipe compétitive de sport d'endurance et/ou pratiquant régulièrement des exercices d'endurance avec mise en charge (≥180 minutes/semaine). Un test incrémental sur tapis roulant a été effectué pour mesurer la capacité aérobie maximale (VO_{2max}).Une tomographie quantitative périphérique (pQCT) a été réalisée pour mesurer la densité minérale osseuse volumétrique totale, trabéculaire et corticale (vBMD) et la surface, ainsi que l'indice de contrainte-déformation au niveau du tibia distal (4% de la longueur du tibia) et proximal (38% et 66% de la longueur du tibia). L'absorptiométrie à rayons X à double énergie (DXA) a été utilisée pour déterminer la masse corporelle maigre (LBM) et la DMO surfacique (aBMD) au niveau de la hanche totale et du col du fémur. L'EA a été calculée sur une période de 7 jours en utilisant l'apport énergétique alimentaire à partir d'un rappel alimentaire de 24 heures et la dépense énergétique liée à l'exercice à l'aide de moniteurs de fréquence cardiaque polaires et d'une

équation fréquence cardiaque-VO_2 individualisée basée. Les sous-échelles de la recherche de la minceur, de l'insatisfaction corporelle et de la boulimie de l'Inventaire des troubles de l'alimentation-3 (EDI-3) ont été administrées pour évaluer les attitudes de l'EDA. Des coefficients de corrélation de Pearson/Spearman et des modèles de régression linéaire multivariable ajustés en fonction de l'âge, du sexe et de l'IMC ont été élaborés. Des tests t d'échantillons indépendants ont été utilisés pour comparer les résultats entre les sexes.

Résultats: Quarante-trois participants ont pris part à l'étude (72 % d'hommes, âge 25,54 ± 4,25 ans, IMC 22,81 ± 2,88 kg/m2, pourcentage de graisse corporelle 18,58 ± 6,24 %, LBM 54,16 ± 10,67 kg, VO_{2max} 57,02 ml/min/kg, activité physique modérée à vigoureuse 667,3 (356 – 3349) minutes par semaine, EA 39,11 ± 14,02 kcal/kg LBM/j). Les participants masculins avaient une vBMD totale ($p = 0,020$), une surface totale ($p = 0,011$), une vBMD trabéculaire ($p = 0,005$) et une surface trabéculaire ($p < 0,001$) plus élevées au site de 4 % ; une surface corticale et un indice de contrainte-déformation plus élevés aux sites de 38 % ($p < 0,001$) et de 66 % ($p < 0,001$), et une surface totale plus élevée au site de 66 % ($p < 0,001$) par rapport aux femmes. L'EA était négativement associée à la surface trabéculaire ($r = -0,333$, $p = 0,036$) et à l'indice de contrainte-déformation sur le site de 38 % ($r = -0,339$, $p = 0,032$), et positivement associée à la surface corticale sur le site de 66 % ($r_s = 0,459$, $p = 0,003$). Des corrélations négatives ont été observées entre la recherche de lff minceur et l'EA ($r_s = -0,380$, $p = 0,014$) et le score total EDI-3 ($r_s = -0,316$, $p = 0,044$).

Conclusions: Les résultats indiquent que les mesures de l'EA sur le terrain peuvent manquer de sensibilité pour identifier les associations entre l'EA et la solidité des os chez les athlètes d'endurance masculins et féminins. De futures études prospectives sur un échantillon plus

important avec des évaluations à plus long terme de l'EA sont nécessaires pour confirmer nos résultats.

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Contribution of Authors

Ada Sevinc: Completed the data acquisition, analysis, and interpretation of data, and wrote the original draft

Jenna C. Gibbs: Supervision, conception, design, analysis, and interpretation of data, and revised work for important intellectual content

Jennifer Levee: Completed the data acquisition and analysis

Silar Gardy: Completed the data acquisition and analysis

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Tyler Churchward-Venne: Reviewed the protocol, and provided important intellectual content

Jennifer Reed: Reviewed the protocol, and provided important intellectual content

List of Tables and Figures

Abbreviation List

aBMD: Areal bone mineral density

vBMD: volumetric bone mineral density

BMD: Bone mineral density

BMI: Body mass index

BSI: Bone stress injuries

CTX: Carboxy-terminal collagen crosslinks

NTX: N-telopeptide

DE: Disordered eating

EA: Energy availability

ED: Eating disorder

EDE-Q: Eating Disorder Examination Questionnaire

EEE: Exercise energy expenditure

EE: Energy expenditure

EI: Energy Intake

FAST: Female Athlete Screening Tool

FFM: Fat-free mass

LBM: Lean body mass

FHA: Functional hypothalamic amenorrhea

GH: Growth hormone

HR-pQCT: High-resolution peripheral quantitative computed tomography

DXA: Dual-energy X-ray absorptiometry

IGF-1: Insulin-like growth factor

IOC: International Olympic Committee

LEAF-Q: Low Energy Availability in Females Questionnaire

LH: Luteinizing hormone

FSH: Follicle-stimulating hormone

P1NP: Procollagen 1 intact N-terminal peptide

RED-S: Relative Energy Deficiency-Syndrome

REE: Resting energy expenditure

TT_3: Tri-iodothyronine-3

HR-flex: Heart rate-flex

EDRC: Eating Disorder Risk Composite

SSI: Stress-strain index

VO_{2max}: Maximal aerobic capacity

Chapter 1: Introduction

1.1 Background

Endurance athletes are at a higher risk of low energy availability (EA) due to high training volume and elevated nutritional demands, with low EA affecting 31% and 25% of female and male long-distance runners, respectively (Heikura et al., 2018). The Female and Male Athlete Triad and Relative Energy Deficiency in Sports (RED-S) models are the two most widely adopted models describing the health and performance-related effects of low EA/energy deficiency in physically active individuals and athletes. Specifically, low EA is considered the key etiological factor underpinning the Female and Male Athlete Triad-related clinical sequelae, including an increased risk of low bone mineral density (BMD) and bone stress injury (BSI) (De Souza et al., 2014; Mountjoy et al., 2018; Nattiv et al., 2021). Several studies have investigated the effects of EA/energy deficiency and its surrogate indicators (e.g., presence of menstrual irregularity, history of disordered eating (DE)/eating disorders (ED)) on BMD, bone microarchitecture/strength, and bone turnover in physically active and sedentary individuals (Ackerman et al., 2011; Mitchell et al., 2015; Piasecki et al., 2018a; Southmayd et al., 2017; Melin et al., 2015). However, the associations between EA and indices of bone strength in male and female endurance athletes are not well-described.

EA refers to the amount of dietary energy remaining for physiological function after accounting for the energy demands of exercise (Loucks et al., 2011). Low EA triggers a cascade of hormonal changes that directly and indirectly impacts bone turnover (Wade et al., 1996; Wade & Schneider, 1992). Directly, low EA suppresses leptin, total triiodothyronine (TT_3), growth hormone (GH), and insulin-like growth factor (IGF-1), which have been shown to decrease bone formation (Elliott-Sale et al., 2018; Koehler et al., 2016; Papageorgiou et al., 2017). Indirectly,

low EA suppresses the hypothalamic-pituitary-gonadal axis by way of reductions in estrogen and testosterone concentrations, leading to an increase in bone resorption (Loucks et al., 1998). Together, these endocrine changes indicate an uncoupling of bone turnover which may lead to significant bone loss if sustained for a prolonged period (De Souza et al., 2008; Heikura et al., 2018; Papageorgiou et al., 2018). Low EA also causes menstrual disturbances (functional hypothalamic amenorrhea (FHA) being the most severe type), with the highest prevalence of menstrual disturbances observed in female athletes participating in leanness-focused sports (e.g., long-distance running, gymnastics). Female athletes with FHA have lower areal BMD (aBMD), trabecular volumetric (vBMD), number, and area, and estimated bone strength compared to their eumenorrheic counterparts (Lieberman et al., 2018), each of which may contribute to reduced bone quality and a higher risk of BSIs. While substantial literature exists in female athletes and exercising women, research comparing the associations between EA and indices of bone strength in male and female endurance athletes is lacking.

Low EA typically occurs through three main pathways: 1) inadvertently by failing to consume adequate energy intake (EI), 2) intentionally by modifying body composition/weight to achieve performance and appearance goals, and 3) compulsively by demonstrating DE behaviours and/or pathological weight control behavior. DE behaviors are commonly reported in athletes who engage in leanness-focused sports; however, not all DE leads to an energy deficit (Burke et al., 2018; Chatterton & Petrie, 2013; Gibbs et al., 2013; Sundgot-Borgen & Torstveit, 2004). High drive for thinness is characterized by a preoccupation with weight loss and dieting (Barrack et al., 2008; Gibbs et al., 2013, p. 201), and has been identified as a surrogate indicator of energy deficiency (i.e., lower resting energy expenditure (REE) and TT_3 concentrations) in exercising women (De Souza et al., 2007; Gibbs et al., 2011, 2013; Reed et al., 2011). Body

dissatisfaction, defined as a misalignment between the individual's ideal versus actual body image, is another psychological construct often associated with restrictive dietary behaviors to achieve a desired body mass and shape (Varnes et al., 2013). Bulimia nervosa is a mental disorder characterized by recurrent cycles of consuming large amount of food without control followed by compensatory behaviours to prevent weight gain such as vomiting, laxative use, and excessive exercise (Williams et al., 2012). Due to this cycle of overeating and fasting, individuals with bulimia nervosa often present with normal body weight (Sundgot-Borgen & Torstveit, 2010). High drive for thinness, body dissatisfaction, and bulimia scores have been linked to a greater prevalence of severe menstrual disturbances and lower EA in exercising women and athletes (Gibbs et al., 2011; Reed et al., 2013), yet less is known about whether these eating attitudes modify the relationship between EA and bone parameters in male and female endurance athletes. Since there are notable challenges with the accurate assessment of EA, measuring eating attitudes could be an alternative method to identify individuals at risk of low EA and associated health complications, including low BMD and BSIs (Heikura et al., 2018; Logue et al., 2018).

1.2. Knowledge Gaps and Objectives

Most studies investigating the effect of low EA/energy deficiency on bone strength have used surrogate measures of energy status, including menstrual status, REE, metabolic biomarkers (e.g., TT_3) and psychometric indicators of DE attitudes/behaviours (e.g., drive for thinness, body dissatisfaction) (Ackerman et al., 2011, 2013; Duckham et al., 2013; Mitchell et al., 2015; Piasecki et al., 2018; Southmayd et al., 2017). Although these outcomes may be indicative of an energy deficient state, proxy measures of low EA may neglect individuals with subclinical energy deficiency without overt changes in physiological/metabolic function and DE behaviours

(Reed et al., 2015; Sterringer & Larson-Meyer, 2022). Additionally, few studies have measured bone strength using advanced imaging measures, such as peripheral quantitative computed tomography (pQCT) (Gama et al., 2022; Southmayd et al., 2017). Male athletes are also at risk of the Triad/RED-S conditions. However, there are no studies which explore the link between EA and bone strength outcomes in both male and female endurance athletes. There is a need for studies which explore sex differences in these associations since male endurance-trained individuals have received little attention in previous studies in this field. Thus, the primary objective of this study is to examine the association between EA and total vBMD at the tibia in young endurance-trained individuals. The secondary objective is to explore the associations between EA, eating attitudes, and bone parameters. Through these objectives, we will test the following hypotheses: 1) EA will be positively associated with bone parameters; and 2) eating attitudes (drive for thinness, body dissatisfaction, bulimia) will be negatively associated with EA and bone parameters **(Table 1.1).**

Table 1.1. Primary and secondary objectives and methods of analysis.

Primary Research Question	**Independent Variable**	**Dependent Variable**	**Method of Analysis**
Is there an association between EA and total vBMD in young endurance-trained individuals?	EA	Total vBMD at the tibia (pQCT)	Pearson/Spearman correlations Multivariable linear regression
Secondary Research Questions	**Independent Variable**	**Dependent Variable**	**Method of Analysis**
Is there an association between EA and bone parameters in young endurance-trained individuals?	EA	Trabecular and cortical vBMD and area and SSI at the tibia (pQCT)	Pearson/Spearman correlations Multivariable linear regression
Is there an association between dietary EI and bone parameters in young endurance-trained individuals?	Dietary EI (24-hour dietary recall)	Total, trabecular and cortical vBMD and area and SSI at the tibia (pQCT)	Pearson/Spearman correlations Multivariable linear regression
Is there an association between EEE and bone parameters in young endurance-trained individuals?	EEE (Polar heart rate monitors)	Total, trabecular and cortical vBMD and area and SSI at the tibia (pQCT)	Pearson/Spearman correlations Multivariable linear regression
Is there an association between DE attitudes and EA in young endurance-trained individuals?	Drive for thinness, body dissatisfaction, and bulimia subscale scores (EDI-3)	EA, dietary EI (24-hour dietary recall), EEE (heart rate monitors and)	Pearson/Spearman correlations Multivariable linear regression
Is there an association between DE attitudes and bone parameters in young endurance-trained individuals?	Drive for thinness, body dissatisfaction, and bulimia subscale scores (EDI-3)	Total, trabecular and cortical vBMD and area and SSI at the tibia (pQCT)	Pearson/Spearman correlations Multivariable linear regression
Describe associations mentioned above stratified by sex	Sex (males versus females)	Bone parameters at the tibia (DXA and pQCT), EA variables (EA, dietary EI, EEE), eating attitudes (EDI-3)	Independent T-Tests

Footnotes: vBMD: volumetric bone mineral density; EA: energy availability; EI: energy intake; SSI: stress strain index; pQCT: peripheral quantitative computed tomography; EDI-3: Eating Disorder Inventory-3; DE: disordered eating; DXA: dual-energy X-ray absorptiometry.

Table 1.2. Outcome measures.

Energy Outcomes	
EA	Dietary EI by 24-hour dietary recall EEE by Polar heart rate monitors LBM by DXA
Bone Outcomes	
Bone strength	Total, trabecular, and cortical vBMD and area and SSI at the 4%, 38% and 66% tibia by pQCT
aBMD	aBMD at the lumbar spine and proximal femur by DXA
Eating Attitude Outcomes	
DE attitudes	Drive for thinness, body dissatisfaction, and bulimia subscale scores by EDI-3

Footnotes: EA: energy availability; EI: energy intake; LBM: lean body mass; vBMD: volumetric bone mineral density; SSI: stress strain index; pQCT: peripheral quantitative computed tomography; aBMD: areal bone mineral density; DXA: dual-energy x-ray absorptiometry; EDI-3: Eating Disorder Inventory-3; DE: disordered eating.

Chapter 2. Literature Review

2.1 Bone Fragility and BSIs in Athletes

2.1.1. Burden and Prevalence

There are well-documented health benefits associated with sports participation including enhanced aerobic fitness, lower risk of cardiovascular and metabolic diseases, and improved bone and muscle strength (Schwarz, 2004; Warburton & Bredin, 2017). However, certain physically active individuals and athletes who participate in high-volume training and/or have inadequate nutritional intake are at a higher risk of a BSI. BSIs are common overuse injuries associated with repetitive bone loading, which can lead to substantial morbidity, declines in athletic performance, and significant time lost from training and competition. While BSIs affect a wide range of physically active individuals, endurance athletes participating in strenuous weight-bearing activities, especially long-distance runners, are among the most impacted accounting for 69% of all stress fractures (Bennell & Brukner, 1997; Kaeding & Miller, 2013). BSIs occur in 3% to 21% of competitive endurance runners during a one-year period (Bennell et al., 1996; Duckham et al., 2015; Hutson et al., 2021; Kelsey et al., 2007; Tenforde et al., 2015). Given the high prevalence of BSIs in endurance athletes, safe and effective risk assessment and treatment strategies are essential for the competitive success of the athlete and for improvements in the management and prevention of BSIs and lifelong musculoskeletal impairments, including osteoporosis/low BMD and fractures (Engebretsen et al., 2014; Kelsey et al., 2007).

2.1.2 Pathophysiology and Risk Factors

BSIs occur due to cumulative, repetitive bone loading without adequate time for repair (Mayer et al., 2014), leading to an increase in bone remodeling and an accumulation of local microdamage (Engebretsen et al., 2014; Frost, 1991; Li et al., 1985). Microdamage formation is

determined by the number of loading cycles, strain magnitude, and strain rate. Microdamage formation is also threshold-dependent, which suggests that targeted remodeling may fail to sustain the equilibrium between bone damage formation and repair if the threshold is exceeded. Maladaptation to the new mechanical loading patterns may reduce bone mass and lead to further accumulation of microdamage, initiating the development of stress reactions and stress fractures (Warden et al., 2014). Unlike a fracture, BSIs do not require high applied force with a single load and are associated with localized pain and tenderness (Chen et al., 2013). BSIs exist along a continuum of severity, from a stress reaction to a stress fracture and ultimately, a complete fracture. Stress reactions present as periosteal and/or marrow edema. If left untreated, a stress fracture might develop which is detectable through bone imaging as a visible fracture line. If further bone uncoupling occurs, micro-fractures can lead to a fracture in the trabecular bone, and eventually a full cortical fracture (Nattiv & Armsey, 1997). Stress fractures often develop in response to sudden and/or significant increases in training frequency, intensity, or duration (Brunet et al., 1990; Jones et al., 1993; Korpelainen et al., 2001). Stress fractures of the navicular, tibia, and metatarsals are the most injured sites in track athletes, while tibia and fibula injuries are the most common injury sites among long-distance runners (Anderson & Greenspan, 1996; Arendt et al., 2003). Risk factors for BSIs can be classified into two categories: extrinsic and intrinsic. The latter refers to the risk factors which are internal, specifically within the individual's body. Both groups further divide into modifiable and non-modifiable risk factors (Warden et al., 2014). Importantly, risk factors for BSIs are often interrelated which adds another layer of complexity when determining the independent contribution of each factor.

Non-modifiable, intrinsic risk factors include demographics (e.g., sex, gender, race, age), fracture history, genetics, and lower-extremity alignment. Females are more susceptible to BSIs

than males due to anatomical, nutritional, biomechanical, and hormonal differences between sexes (Pepper et al., 2006). The risk of developing a BSI increases 5-fold after experiencing at least one BSI (Wright et al., 2015b). Additionally, individuals with the normal alignment of the knee, leg length discrepancy, and foot morphology are less likely to develop BSI (Korpelainen et al., 2001).

Modifiable, intrinsic risk factors include but are not limited to low aBMD, energy/caloric deficiency, calcium and vitamin D deficiency, low body mass index (BMI), and reduced muscle size and strength. Substantial evidence demonstrates that low aBMD is associated with a higher risk of stress fractures, particularly at bone sites with a higher proportion of trabecular bone (Bennell et al., 1996; Lauder et al., 2000; Marx et al., 2001; Mountjoy et al., 2018). Further, bone geometry and microarchitecture, including a lower moment of inertia, total and cortical area, trabecular thickness and number, and stress strain index (SSI), have been shown to correlate with prevalent stress fractures in military recruits and endurance athletes (Beck et al., 1996; Milgrom et al., 1989; Popp et al., 2009; Schanda et al., 2019). Nutritional factors, particularly calcium and vitamin D, can affect bone health and may influence bone turnover (Rizzoli, 2008). In a double-blind, randomized controlled study in 5201 female Navy recruits, Lappe et al. (2008) demonstrated that participants who received a vitamin D and calcium supplement had a lower incidence of stress fractures than the control group. However, evidence on the influence of vitamin D and calcium intake on stress fracture risk is conflicting and lacking in males. In a prospective study in 748 competitive high school runners, Tenforde et al. (2013) identified low BMI (<19 kg/m^2), late menarche, and prior participation in gymnastics and dance as risk factors for stress fractures in girls, whereas prior fracture and the number of competitive seasons were associated with an increased risk of stress fractures in boys. Biomechanical factors, such as an

abnormal alignment of the bone, increased force applied to the bone, and torsional loads, are associated with an increased risk of a stress fracture (Davis et al., 2004; Milner et al., 2006; Pohl et al., 2008). Muscle size and strength are also important modifiable factors in the etiology of BSIs and have a protective effect by acting as a shock absorber on the bone (Armstrong et al., 2004; Hoffman et al., 1999; Warden et al., 2014). Although these modifiable, intrinsic risk factors are outlined separately, some of these concepts should be considered as interrelated (Cobb et al., 2003a). Previous research has repeatedly shown the relationships between functional hypothalamic hypogonadism, low EA, and low aBMD, a syndrome referred to as the Female and Male Athlete Triad. Low EA, directly and indirectly, affects bone turnover and bone health through the suppression of reproductive hormones and alterations in metabolic hormones known to influence bone metabolism (De Souza et al., 2014).

Hormonal contraceptives, particularly oral contraceptive pills, are frequently used by female athletes to treat menstrual irregularity and reduce menstrual symptoms (Cheng et al., 2021). Evidence examining the association between hormonal contraceptive use and BSIs is conflicting, with some studies suggesting no associations while others suggest that they are protective against bone loss and BSI risk in female athletes (Barrow & Saha, 1988; Bennell et al., 1996; Cobb et al., 2007; Myburgh et al., 1990; Tenforde et al., 2013).

Training characteristics, equipment conditions, type of sport, and time of the season are external risk factors for a BSI. The first six weeks of training is the most common period to experience a stress fracture (Rizzone et al., 2017). Stress fracture incidence is greater at high training volumes. For example, a running volume of greater than 20 miles per week substantially increases the risk of developing a stress fracture in runners (Tenforde et al., 2013). A sharp increase in training frequency and/or intensity may also increase the risk of a stress fracture

(Pepper et al., 2006). Athletic equipment plays a key role in the development of a BSI, particularly the ground reaction forces and motion of the feet. Shoe properties may contribute to the development of a stress fracture such as shoe age and cushioning level of the shoe (Frey, 1997; Gardner et al., 1988; Ridge et al., 2013). However, the evidence for the role of footwear in stress fracture incidence among runners is unclear (Lieberman et al., 2018; Pepper et al., 2006; Warden et al., 2014, 2014). Cross-country running, gymnastics, and track and field are the sports with the highest percentage of individuals with stress fractures (Rizzone et al., 2017). Alternatively, past participation in ball sports (e.g., basketball, soccer, volleyball) may protect against stress fractures in runners due to the higher-impact, multi-directional loading patterns, which are known to positively influence BMD and bone strength (Fredericson et al., 2006; Milgrom et al., 1989; Tenforde et al., 2015; Tenforde & Fredericson, 2011). Collectively, the development of BSIs often involves an accumulation of several risk factors which modify either the ability of bone to resist load and/or the load applied to a bone.

2.2 EA: Definition and Scientific Basis

2.2.1 Operational Definition of EA

Low EA is defined as an inadequate dietary EI relative to the energy expended during exercise (Loucks et al., 2011). EA refers to the amount of dietary energy available for physiological functioning after exercise training (Mountjoy et al., 2018). Operationally, EA is calculated as dietary EI minus exercise energy expenditure (EEE) normalized for fat-free mass (FFM) or lean body mass (LBM) (EA = (EI (kJ) – EEE (kJ))/FFM or LBM (kg)). In the presence of low EA, redistribution of energy occurs in a hierarchical order to support the most vital (life-sustaining) metabolic functions and suppress non-essential physiological functions such as reproductive function and bone metabolism (Wade et al., 1996). Low EA is associated with

substantial morbidity, a higher risk of musculoskeletal declines, and reduced athletic performance in both male and female athletes. Previous research has established the negative impact of low EA on bone health and reproductive function in female athletes and exercising women (De Souza et al., 2014; Nattiv et al., 2007). Traditionally, energy balance calculations were used to manage the dietary needs of athletes. Energy balance is defined as EI minus total 24-hour EE. Conceptually, energy balance represents the amount of dietary EI added to or subtracted from bodily energy stores after physiological systems have expended their energy for the day. However, this concept has been deemed outdated in exercise physiology research as the calculation assumes optimally functioning physiological systems. This assumption does not hold true for an energy deficient state due to the suppression of physiological and metabolic processes (Areta et al., 2021; Mountjoy et al., 2018). The concept of EA recognizes that health (i.e., proper physiological function) depends on the amount of dietary energy that remains as an input to the various physiological systems after the individual has coped with a stressor (e.g., exercise training). Therefore, energy balance has been replaced in favor of the notion of EA in sports science research in recent years.

2.2.2 Methodological Considerations Related to EA

Although EA is a useful tool to assess health impairments and ensure optimal health and performance in athletes, calculating EA presents several methodological challenges which impacts the accuracy and reliability of the measurements (Burke, Close, et al., 2018; Heikura et al., 2021). While the associations between health impairments (i.e., reproductive dysfunction, low BMD) and low EA have been well-established in rigorous laboratory-based studies which manipulated EA by controlling EI and EEE, studies in free-living athletes have failed to report these associations, suggesting that caution should be taken when extrapolating the lab-based

findings to field settings (Fahrenholtz et al., 2018; Heikura et al., 2018, 2021; Koehler et al., 2013).

Currently, there is no single protocol to guide researchers and practitioners when assessing EA, leading to heterogeneity in the methods used to quantify EA (Burke, Close, et al., 2018). EA can be estimated either directly or indirectly. Direct assessment involves determining each component of the EA calculation including dietary EI, EEE, and FFM/LBM of the individual. Dietary EI is commonly measured using food records, 24-hour dietary recalls, or interviews where participants self-report their food and beverage consumption for a specified period, retrospectively or simultaneously. These methods can introduce error by way of under-reporting EI, failure to follow a habitual diet due to high intra-individual variability in daily intake, inaccuracy with estimating the portion sizes, recall errors, and low compliance (Burke et al., 2001; Burke, Lundy, et al., 2018; Capling et al., 2017; Magkos & Yannakoulia, 2003). A systematic review which compared two or more methods of dietary assessment in athletes demonstrated that self-reported EI was under-reported by 19% (−2793 ± 1134 kJ/day) compared to doubly labeled water, a method regarded as the gold standard to measure dietary EI (Capling et al., 2017). The 24-hour dietary recall method offers some advantages by reducing the burden to participants, therefore reducing issues related to participant response rates and missing data. Three to 7-day dietary recording is reasonable to capture longer-term EI and reduce day-to-day variability in EI, with longer durations of documentation providing an increased accuracy (Magkos & Yannakoulia, 2003).

Similarly, the assessment of EEE is also prone to error. Currently, a gold standard method does not exist to estimate EEE, resulting in high variability of protocols used to estimate EEE (Burke, Close, et al., 2018). In the literature, most assessments of EEE have relied on the

use of Global Positioning System (GPS) units, heart rate monitors, power meters, and accelerometers to generate an individualized assessment of EEE. A widely used, yet less accurate approach, involves estimating EEE using metabolic equivalency of the task based on training logs (Ainsworth et al., 2011; Heikura et al., 2021; Koehler et al., 2013; Reed et al., 2013). One of the most reliable methods involves using heart rate monitoring and indirect calorimetry to establish an individual-level calibration between heart rate and EE, which considers the heterogeneity in heart rate responses among individuals (Heikura et al., 2021; Leonard, 2003; Melin et al., 2015). Alongside the challenges with estimating EEE, it is difficult to distinguish EEE from the energy expended during activities of daily living such as commuting and physical labour-intensive employment (Burke, Close, et al., 2018). Since the operational definition of EA requires an estimation of EEE, the lack of standardized guidelines on the definition and terminology of EEE may influence the interpretation of EA.

Despite its high ecological validity, the direct, field-based estimation of EA is subject to significant errors of validity and reliability due to the complexity associated with defining and using accurate measurement tools for each of its components. Considering the difficulty implementing EA assessments in the field, many researchers have used indirect measurements to determine individuals who are at risk of low EA. Indirect measurement of EA refers to an assessment of possible symptoms of short to long-term low EA, including the use of blood biomarkers, laboratory-based/self-reported menstrual status, REE testing, questionnaires designed to screen for physiological symptoms of low EA, Low Energy Availability in Females Questionnaire, history of DE/ED, history of stress fractures or stress reactions, low BMI, and low aBMD for age (Z-score <-2). Specifically, blood biomarkers including leptin, IGF-1, and TT_3 were sensitive to detect low EA and provide an objective measure of energy conservation (De

Souza et al., 2019; Heikura et al., 2021; Mountjoy et al., 2015; Staal et al., 2018; Stenqvist et al., 2021). For REE, which is another accurate measurement associated with self-reported menstrual status, a cut-off value of 0.90 was determined to be indicative of low EA (De Souza et al., 2008; Strock et al., 2020). Drive for thinness score of ≥7 has been associated with signs of low EA such as lower TT_3, higher ghrelin, and lower REE (De Souza et al., 2007). A LEAF-Q score of ≥8 can identify females at high risk for Triad conditions (Melin et al., 2014). Another commonly used method involves determining menstrual status using self-reported logs or determining the levels of reproductive hormones (including estradiol), given that lower EA has been shown to be associated with greater menstrual disturbances in physically active females (Reed et al., 2015). Despite the wide use of surrogate markers to determine EA in the literature, these methods rely on self-reported symptoms or involve laboratory-based measurements which may not be a feasible method to determine the energy status of an athlete in a real-world context. Alternatively, estimating EA directly may increase the applicability of the results to field/practice and produce more ecologically valid studies.

2.2.3 Overview of the Effects of Low EA on Reproductive Function

The detrimental effect of low EA on bone mass, strength, and microarchitecture, and menstrual function in physically active women and female athletes is well-established. Robust experimental studies which induced short-term low EA (<30 kcal/kg/FFM) in a laboratory setting have shown reductions in luteinizing hormone (LH) pulsatility, an uncoupling in bone turnover, and alterations in metabolic hormones known to influence bone metabolism in young, sedentary females (Ihle & Loucks, 2004; Loucks & Thuma, 2003). According to previous research, minimum cut-offs of EA equal to 45 kcal/kg FFM/day and 30 kcal/kg FFM/day were proposed as necessary to sustain optimal physiological functioning in sedentary, normally

menstruating women and physically active individuals, respectively. The basis of these cut-offs comes from a series of well-controlled experimental studies by Loucks and colleagues, wherein they investigated the effect of EA on LH pulsatility in sedentary, normally menstruating women. Using a repeated-measures, prospective cohort design, participants were exposed to two trials of dietary and exercise manipulation during the early follicular phase of the menstrual cycle with a 2-month wash-out period. In one of the trials, an EA of 45 kcal/kg FFM/day was induced to serve as a balanced EA. During the other trials, the participants were randomized to a restricted EA group of either 10, 20, or 30 kcal/kg FFM/day. All groups engaged in a supervised and standardized exercise session wherein they expended 15 kcal/kg of FFM per day. Although EEE was standardized across the groups, the calories consumed through standardized dietary products were modified for each group to match their targeted EA. The results of this work suggest that there is a threshold (30 kcal/kg FFM/day) below which there is a disruption in LH pulsatility.

Based on these findings, an EA less than 30 kcal/kg FFM/day is often used as a clinical cut-off for low EA in the management and prevention of the Female and Male Athlete Triad and RED-S. However, a consensus on a cut-off point for optimal EA in physically active individuals remains disputed, especially considering the studies by Loucks et al. were conducted on young sedentary women in a controlled laboratory setting. Recent studies have questioned the validity of a low EA threshold of 30 kcal/kg FFM/day and have shown that an impaired reproductive function was not associated with an EA below 30 kcal/kg FFM/day in some exercising women (De Souza et al., 2019; Lieberman et al., 2018; Williams et al., 2015). Therefore, it remains unclear whether the results from these laboratory studies can be extrapolated to a real-world environment.

Many exercising women develop exercise-associated menstrual disturbances due to hypoestrogenism secondary to low EA. These conditions range from subtle disorders including luteal phase defects and anovulation to more severe disorders like oligomenorrhea and amenorrhea. In a study in 35 regularly menstruating recreational young runners and sedentary controls, the 3-month incidence of luteal phase defects and anovulation were 33% and 46% in exercising women while there was no incidence of an inconsistent menstrual cycle in sedentary women (De Souza et al., 2010). Clinically, low EA (<30 kcal/kg FFM) may indicate a presence of FHA in females, which is a reproductive disorder characterized by an absence or cessation of menses. Primary amenorrhea is an absence of menses at the age of 16 and older whereas secondary amenorrhea refers to a failure to menstruate in the past 3 months alongside chronically suppressed estrogen and progesterone concentrations. Oligomenorrhea is when menses occurs at intervals of 36-90 days or an individual self-reports six or fewer menstrual cycles in the previous year. Eumenorrhea, on the contrary, describes a healthy, normal menstrual cycle occurring every 21-35 days. There is evidence for lower testosterone levels in males with low EA/energy deficiency, however, males do not present with as overt clinical signs of reproductive dysfunction as females, who display abnormalities in their menstrual cycle length and frequency.

To date, it is unclear whether a low EA can discriminate normal menstrual and ovarian function from severe menstrual disturbances (amenorrhea or oligomenorrhea) in exercising women (Williams et al., 2001). In a cross-sectional study in 91 young exercising women, Reed et al. (2015) demonstrated that EA was lower in exercising women with amenorrhea versus exercising women with eumenorrhea, which was corroborated by laboratory measures of lower REE, the ratio of actual REE to predicted REE (REE/pREE) and circulating TT_3 concentrations in those women with amenorrhea. These findings supported the use of current menstrual status as

a proxy indicator to distinguish between individuals with and without chronically low EA. Collectively, low EA is an important etiological factor for reproductive disturbances (i.e., reduced LH pulsatility, greater frequency of amenorrhea); yet data in support of a physiologically relevant threshold of low EA remain conflicting.

2.2.4 Overview of the Female and Male Athlete Triad

The Female Athlete Triad is a medical condition observed in physically active females that describes the interrelationships between low EA (with and without DE), menstrual dysfunction, and low aBMD. The Triad continuum exists from optimal EA, BMD, and menstrual function (i.e., eumenorrhea) to severe clinical conditions such as low EA, low aBMD/osteoporosis, and hypothalamic amenorrhea. The 2014 Female Athlete Triad consensus statement emphasizes the importance of preventative measures and early intervention to avoid the progression of the individual Triad components to their serious clinical endpoints. Further, the Triad consensus statement proposes a risk stratification point system to assist physicians and other healthcare providers by offering clinical guidelines for screening, diagnosis, and treatment.

Until the 2000s, research and clinical guidelines related to the Triad were focused primarily on exercising women and female athletes; however, male athletes have received increasing attention from the scientific and clinical communities. In response to the growing evidence of Triad-related conditions in male athletes, The Male Athlete Triad consensus statement was published in 2021, highlighting the effects of low EA/energy deficiency on reproductive and metabolic function and bone health in male athletes. In 2014, the International Olympic Committee (IOC) (updated in 2018) expanded the Triad syndrome to include males and the effects of energy deficiency on various bodily systems other than reproductive function and bone health, also referred to as RED-S (Mountjoy et al., 2014). However, more research is

needed to consider RED-S as an evidence-based condition, considering the causality between energy deficiency and many of the proposed health and physiological outcomes have not been established yet (Williams et al., 2019).

Few studies have reported the prevalence of both subclinical and clinical Triad conditions in physically active women. A systematic review by Gibbs et al. (2013) reported that 16-60% of exercising women presented with one of the Triad conditions, 3% to 27% had any two conditions, and 0% to 16% had all three conditions. Another study in 669 elite-level Norwegian female athletes showed that almost 75% of female athletes participating in lean sports presented with two of the Triad conditions in comparison to 39% of athletes participating in non-lean sports (Torstveit & Sundgot-Borgen, 2005). The same study showed that 70% of athletes participating in lean sports were considered at risk of the Triad versus 55% of those competing in non-lean sports, which contradicts findings from Beals and Hill (2006) who found no differences in Triad prevalence between these two athlete groups. A more recent study conducted by Melin et al. (2019) explored the prevalence of Triad conditions in a group of elite female endurance runners and reported 63% of athletes had low EA; 45% had low BMD, and 25% were diagnosed with DE/EDs. Moreover, half of the female athletes displayed at least one clinical Triad condition while 23% presented with all three subclinical or clinical conditions.

Although there is abundant evidence regarding the negative influence of reduced EA on bone health and reproductive function in female athletes, less research has explored the effect of low EA/energy deficiency in male athletes. Low EA has been acknowledged as one of the main factors associated with health and performance consequences in both female and male athletes. These negative health and performance outcomes in males have received growing attention following the introduction of the Male Athlete Triad and IOC RED-S consensus statements.

Thereafter, several studies have demonstrated the negative impact of reduced EA in male athletes (Tenforde et al., 2016; Viner et al., 2015). Energy deficiency downregulates reproductive function in males. Chronically exercising male athletes have been shown to have lower resting testosterone compared to their age-matched, non-athlete counterparts, due to the suppression of the hypothalamic-pituitary-testicular axis (Hackney, 2020; Lane & Hackney, 2014). Further, low EA may negatively affect metabolic and musculoskeletal health in physically active males, including reductions in REE; changes in leptin, insulin, and ghrelin; and declines in BMD and bone microarchitecture (Barrack et al., 2017; Koehler et al., 2016; McGuire et al., 2020; Papageorgiou et al., 2017; Torstveit et al., 2018). Many of these endocrine perturbations parallel those of female athletes. Nonetheless, current evidence does not support the presence of a critically low EA threshold below which disruptions occur in male athletes; however, more severe energy deficits may be needed for the induction of severe Triad outcomes in male athletes compared to female athletes (De Souza et al., 2019; Hackney, 2020; Koehler et al., 2016; Nattiv et al., 2021).

2.3 Associations between EA, Eating Attitudes, and Bone Health in Endurance Athletes

2.3.1 Pathways to low EA in Endurance Athletes

Athletes are often at an increased risk of developing low EA due to the energetically demanding nature of exercise/sport training and the challenges of matching energy input versus output (Mountjoy et al., 2014). Low EA can be achieved via alterations in dietary EI and/or EEE; however, nutrition plays a larger role in the etiology of negative health outcomes (Loucks et al., 2011; Papageorgiou et al., 2018). The prevalence of low EA in the athletic population ranges from 22% to 58% (Logue et al., 2020). Endurance athletes are more susceptible to low EA and/or chronic energy deficiency due to the emphasis on a lean physique and high-volume

training patterns inherent to endurance sports. Although energy deficiency is common in a wide range of sports, individuals who participate in leanness-focused sports are at higher risk of developing low EA compared to those who participate in non-leanness-focused sports (Gibbs et al., 2013; Sundgot-Borgen & Torstveit, 2004). Leanness sports are divided into three groups: gravitational sports, weight-class sports, and aesthetically judged sports. Endurance sports such as long-distance running, cross-country skiing, and road/mountain bike cycling are classified as gravitational sports because of the disadvantage of a high body mass countering gravity when performing these activities (Torstveit & Sundgot-Borgen, 2005). Thus, endurance athletes are typically at a higher risk of low EA than other athlete groups.

There are four proposed etiological pathways to low EA often observed among endurance athletes: 1) DE, 2) ED, 3) weight loss without DE/ED and 4) inadvertent undereating. To achieve an ideal body size, endurance athletes restrict their EI intending to reduce weight/fat mass and improve athletic performance (Martinsen et al., 2010). If low EA is induced through intentional weight loss attempts, they may present with or without ED/DE. DE is a term used to describe the full spectrum of abnormal eating-related problems from dieting to a clinically diagnosed ED. DE behaviours are often linked to disturbances in one's body image, body weight fluctuations, and affective disturbances (Mond et al., 2006; Otis et al., 1997). DE behaviours present as abnormal eating behaviours (e.g., purging, bingeing), food restriction, and other methods to lose or control weight (e.g., diuretics), which are less pathologically severe than those of clinical EDs. Clinical EDs include anorexia nervosa, bulimia nervosa, and EDs not otherwise specified. Anorexia nervosa is characterized by the refusal to eat resulting in significant weight loss. Bulimia nervosa is an ED involving binge-eating episodes followed by compensatory behaviours (e.g., vomiting, laxatives). Both anorexia and bulimia nervosa involve a serious disturbance in the way one

experiences their weight or body shape (Pereira & Alvarenga, 2007). The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) is a diagnostic tool and reference guide to diagnose and classify mental health disorders. The DSM-5 diagnostic criteria for EDs include binge eating disorder, anorexia nervosa, and bulimia nervosa while the rest of the disorders are classified as other specified eating and feeding disorders and unspecified eating and feeding disorders. The DE continuum ranges from short-term restrictive eating and dieting and extends to subclinical and clinical anorexia nervosa and bulimia nervosa. The prevalence of DE has historically increased in Western countries and is higher in athletes than non-athletes (Joy et al., 2016). These behaviors may contribute to macronutrient deficiencies including low carbohydrate, fat and protein intake and inadequate micronutrient intake (e.g., vitamin A, vitamin C, calcium, and zinc) (Dolan et al., 2011). The prevalence of EDs is higher among female endurance athletes than male endurance athletes, with almost one-quarter of female endurance athletes having a diagnosis of an ED (Sundgot-Borgen & Torstveit, 2004). Although research focused on DE behaviours among male athletes has not received as much attention as female athletes, 18% of male athletes in weight-class sports and 22-42% of male athletes in gravitational sports demonstrate DE behaviours (Sundgot-Borgen & Torstveit, 2010). Despite the lack of research, a substantial portion of male athletes suffer from DE/ED and related health consequences (Burke et al., 2018).

Currently, the Eating Disorder Examination (EDE) is the gold standard tool for diagnosing EDs which consists of a 62-item semi-structured interview based on the DSM-5 criteria (Thomas et al., 2014). Not all DE behaviours contribute to energy or nutrient deficiencies in athletes, thus previous literature has identified DE behaviours that are linked to low EA/energy deficiency. The most widely used assessments of DE and ED risk among athletes

include the EDE-Q, three subscales from the EDI, and the Cognitive Restraint Subscale from Three Factor Eating Questionnaire (TFEQ) (Koltun et al., 2019; Pope et al., 2015). EDE-Q is a self-report tool derived from the EDE with high reliability and convergent validity (Mond et al., 2006). EDE-Q consists of four subscales: dietary restraint, eating concern, weight concern, and shape concern. TFEQ includes 51-items that measure dietary cognitive restraint, disinhibition, and hunger. The cognitive restraint subscale assesses the extent to which an individual controls their food intake to maintain or lose body weight. Elevated cognitive restraint scores have been associated with lower energy intake and EA (De Souza et al., 2007; Gibbs et al., 2013; Vescovi et al., 2008). Lastly, EDI-3 is a self-reported questionnaire used by clinicians and researchers to identify individuals with DE. Previous studies showed that drive for thinness, bulimia, and body dissatisfaction subscales from the EDI were associated with indicators of low EA in athletes (Reed et al., 2013; Scheid et al., 2009).

Endurance athletes may also develop low EA inadvertently. Endurance athletes often engage in high-volume training which greatly reduces the availability of metabolic fuel. High-volume training creates a time constraint whereby the athlete may face barriers (e.g., busy schedules, significant time spent in training/competition) to consume adequate EI to match their excessive EE (De Souza et al., 2014). Despite higher nutrition knowledge in athletes versus non-athletes, there is a lack of education about the effects of low EA (De Souza et al., 2014). Aside from the time constraint and nutrition knowledge gaps, some individuals struggle with food access due to financial reasons and a lack of cooking skills (Heaney et al., 2008). An inadequate compensatory response in hunger levels to an exercise-induced energy deficit may also reduce EA. Athletes can have difficulty staying energy replete if they solely rely on appetite signals to consume food. Unlike food deprivation, increased EE due to strenuous training may not

correspond to an increase in hunger levels (Stubbs et al., 2004). Thus, it is of great importance for athletes to receive appropriate nutrition education/guidance and make mindful efforts to monitor their daily EI and EE.

2.3.2 Effect of low EA/Energy Deficiency on Bone Metabolism

The negative effect of low EA/energy deficiency on bone health has been well-established in the literature. In the presence of long-term energy deficiency, the repartitioning of oxidized fuel can disrupt bone turnover markers. Namely, in the presence of an energy deficiency, REE and important regulatory hormones such as TT_3, IGF-1, leptin, and insulin are typically suppressed while cortisol and GH are upregulated (Ihle & Loucks, 2004; Loucks & Thuma, 2003). Collectively, these metabolic hormones play a major role in modulating bone turnover; and subsequently, bone turnover rate, BMD, bone microarchitecture, and bone strength. Thyroid hormone is the main regulator of REE and is also crucial for normal skeletal growth. Hypothyroidism can reduce bone turnover by decreasing both bone formation and bone resorption via the hypothalamic-pituitary-thyroid axis, which is a crucial part of the development of peak bone mass (Gogakos et al., 2010). Another key hormone influencing bone turnover is GH, which is a peptide hormone involved in osteoblast proliferation and collagen production and in turn, positively contributes to BMD and bone turnover. The effect of GH is mediated by IGF-1, a hormone produced primarily in the liver. IGF-1 is responsible for systemic body growth and has anabolic effects on most body cells, including bone (Morel et al., 1993). Leptin and insulin are best known for their roles in energy homeostasis; however, they also maintain normal bone remodeling. Leptin is a cytokine-like hormone secreted by adipocytes known to regulate food intake but also regulates bone metabolism by acting on osteoblasts and osteocalcin (Driessler & Baldock, 2010; Ferron & Lacombe, 2014). Leptin acts on bone indirectly through its influence

on estrogen, cortisol, IGF-1, and parathyroid hormone (Khan et al., 2012; Upadhyay et al., 2015). Together, these metabolic hormones (particularly, TT_3, IGF-1, leptin, insulin) directly exert effects on bone remodeling and in the presence of an energy deficiency/low EA, their alterations can lead to an uncoupling in bone turnover and subsequently, reductions in BMD, bone microarchitecture, and bone strength.

Energy deficiency/EA can also impact bone indirectly through alterations in the hypothalamic-pituitary-gonadal axis. In an energy deficient state, the disruption of reproductive hormones is linked to the changes in the hypothalamic-pituitary-gonadal axis. Specifically, reductions in EA suppress gonadotrophin-releasing hormone and, in turn, decrease the secretion of gonadotrophins (i.e., LH and follicle-stimulating hormone (FSH)) from pituitary glands. Since LH and FSH are involved in the maturation and release of gonadal hormones from the gonads, the suppression of gonadotropins may prevent ovulation and reduce estradiol, progesterone, and testosterone concentrations (Iwasa et al., 2022). Gonadal steroids play a critical role in regulating bone metabolism. Estrogen is a major bone regulatory hormone that exerts effects on both osteoclast and osteoblast cells and is also critical for attenuating bone resorption by promoting osteoclast apoptosis (Siddiqui & Partridge, 2016). Thus, estrogen is essential for the maintenance of bone mass while both estrogen and progesterone are responsible for modulating skeletal growth (Khosla et al., 2012). Estrogen, along with testosterone, increases the area and density of cortical bone through periosteal apposition. Consequently, hypogonadism promotes bone resorption and suppresses bone formation (Clarke & Khosla, 2010).

Several experimental and observational studies have demonstrated the negative associations between low EA and metabolic biomarkers indicative of bone health in sedentary and exercising women and men. These studies have investigated the short-term (within-day to >6

days) or long-term/chronic (weeks to years) consequences of low EA on metabolic hormones, reproductive hormones, and bone turnover markers. In a series of controlled experiments, a short-term energy deficit was induced by manipulating EI and/or EEE. These studies investigated the impact of EA status on bone turnover in male runners, light-weight male rowers, recreationally active and sedentary men, and women (Grinspoon et al., 1995; Ihle & Loucks, 2004; Talbott & Shapses, 1998; Zanker & Swaine, 2000). In a cross-over, experimental study in 8 male distance runners, subjects underwent two 3-day trials in which their dietary EI was altered to achieve either energy restricted (50%) or energy balanced (100%) status while maintaining the same EEE through supervised treadmill running. All subjects in the energy restricted group experienced a significant decline in procollagen 1 intact N-terminal peptide (P1NP) and IGF-1 while a reduction in P1NP was strongly correlated with a decline in IGF-1. This was the first study to show a causal link between restricted EI and suppression in bone formation following daily endurance training (Zanker & Swaine, 2000). A randomized prospective experimental study was conducted by Ihle and Loucks, who determined the dose-response relationship between EA and bone turnover markers in 29 sedentary young women. Participants underwent two 5-day trials during the early follicular phase of the menstrual cycle, once in energy balance and once in one of the energy restricted treatments. They expended 15 kcal/kg LBM/day during supervised exercise training while consuming 60 kcal/kg LBM/day for the energy balanced treatment and consuming either 45, 35, or 25 kcal/kg LBM/day for the energy restricted treatments. P1NP and osteocalcin were suppressed in all energy restricted groups whereas N-telopeptide (NTX) showed an abrupt increase in the most severely restricted EA group only. Therefore, the uncoupling in bone turnover associated with severe energy restriction may lead to detrimental reductions in BMD in young women with chronically low EA. Despite the sedentary

nature of the participants that this study targeted, these findings were notable and paved the way for future research (Ihle & Loucks, 2004).

A more recent study by Papageorgiou et al. (2017) examined the effects of low EA on bone turnover markers in 22 young, physically active men and women (n=11 each). Participants underwent two 5-day trials, one energy restricted treatment and one energy balanced treatment separated by one menstrual cycle or at least 28 days. The target EA was achieved by manipulating dietary EI and maintaining the same EEE through supervised treadmill running. In women, the energy restricted group displayed a significant increase in carboxy-terminal collagen crosslinks (CTX) and a significant decrease in P1NP while men did not display any significant changes in response to energy restriction. The uncoupling in bone turnover in the energy restricted women favored bone resorption as indicated by a significant decrease in the ratio of P1NP (bone formation biomarker) to β-CTX (bone resorption marker). Interestingly, no sex differences in bone turnover markers were present between the energy restricted and balanced groups. Therefore, bone turnover markers may be more sensitive to reduced EA in women compared to men. However, longer-term studies in larger samples are still needed to confirm these findings.

2.3.4 Effects of low EA/Estrogen Deficiency on BMD and Bone Strength

Chronic energy deficiency is the key etiological factor associated with reproductive dysfunction and/or menstrual disorders in male and female athletes. Long-term hypogonadism can impair bone remodeling and lead to irreversible bone loss. Due to the challenges associated with measuring EA in free-living athletes, most studies measure self-reported menstrual status to indicate whether an individual is at risk for a chronic energy deficiency. To our knowledge, existing evidence on the effects of energy and estrogen deficiency on bone health is mostly based

on cross-sectional studies. In a cross-sectional study in 102 young weight-bearing endurance athletes and non-athlete controls, Ackerman et al. (2013) demonstrated that eumenorrheic individuals, regardless of their BMD, had greater bone strength measured by dual-energy X-ray absorptiometry (DXA)-based hip structural analysis (subperiosteal width, cross-sectional moment of inertia, section modulus) at the hip compared to non-athlete controls, while there were no differences in bone parameters between amenorrheic athletes and non-athlete controls. Amenorrheic athletes also displayed significantly lower cross-sectional area at the hip than eumenorrheic athletes. This finding indicates a loss of the exercise-related benefits on bone health in athletes with amenorrhea. Similarly, using high-resolution pQCT (HR-pQCT), Ackerman et al. (2011) found that amenorrheic athletes had lower trabecular number and separation at the tibia and lower trabecular vBMD at the radius compared to eumenorrheic athletes and non-athlete controls, supporting the association between hypoestrogenism and impaired bone microarchitecture. Alternatively, in a longitudinal observational study in 78 young female endurance runners and non-athlete controls, Singhal et al. (2019) examined changes in bone accrual over a 12-month period and found that there were no significant differences in changes in aBMD, aBMD Z-scores, and failure load between eumenorrheic athletes, oligomenorrheic athletes, and non-athlete control groups. At both baseline and follow-up, aBMD in oligo-amenorrheic did not differ compared to non-athletes. Similarly, oligo-amenorrheic athletes and non-athletes had similar failure load at the tibia, measured by micro-finite element analysis. Thus, oligomenorrheic athletes may display a lack of adaptive response to mechanical loading induced by weight-bearing exercise. In a cross-sectional study in 44 female endurance athletes and nonathletic controls aged 17-42, Piasecki et al. (2018) examined the influence of self-reported menstrual status on bone parameters using DXA and pQCT. Despite a lower aBMD

at the lumbar spine, trunk, and pelvis in amenorrheic women than eumenorrheic women, vBMD and estimates of bone strength were similar at the tibia and radius in both groups. As previously outlined, the influence of reproductive dysfunction on bone health has been mostly investigated in females, with limited evidence on possible sex-differences in these associations. In a cross-sectional study in 70 world-class female and male endurance runners and race walkers, Heikura et al. (2018) displayed lower aBMD in women with self-reported amenorrhea, while there was no difference in aBMD between men with low and normal testosterone levels. Moreover, women with amenorrhea and men with low testosterone had a 4.5-fold increased risk of BSI and up to 10-fold higher number of training days lost due to a BSI within the prior year. Collectively, these findings demonstrate that chronic energy deficiency may have detrimental consequences to BMD and bone strength/microarchitecture, which may place the athlete at an increased risk of BSIs and fractures.

Although the presence of long-term energy deficiency is often accompanied by hypoestrogenism and associated menstrual dysfunction, chronic energy deficiency has a direct, negative impact on bone health, independent from estrogen status. This is evident from studies which have determined the individual and combined effects of estrogen versus energy deficiency on bone turnover markers, BMD, and bone strength and microarchitecture (De Souza et al., 2008; Southmayd et al., 2017). In a cross-sectional study in 44 exercising women, De Souza et al. classified participants into four categories based on their reproductive and energy status: energy and estrogen replete, energy replete and estrogen deplete, energy deplete and estrogen replete, and energy and estrogen replete. De Souza et al. displayed that an energy deficiency is the primary factor driving alterations in bone remodeling, as bone turnover was not impaired regardless of estrogen status at a sufficient energy state. In addition, the greatest impairment in

bone turnover was observed in both estrogen and energy deficient groups, indicating that estrogen deficiency may have additive negative effects on bone health in the presence of an energy deficit. Exercising women who were energy and estrogen deficient had significantly lower serum PINP and greater CTX levels compared to other groups. Elevated bone resorption was likely attributed to both estrogen and energy status; and suppression in bone formation was likely related to energy status which is evident by the lower bone formation in the energy replete group. It is apparent that not only menstrual recovery, but also adequate EI, is crucial to prevent impairments in bone turnover and BMD in exercising women (De Souza et al., 2008). In another cross-sectional study by Southmayd et al. (2017), they determined vBMD, bone geometry and estimated bone strength in 60 premenopausal exercising women classified into the same categories as De Souza et al. The presence of both an estrogen and energy deficiency yielded the most adverse results for bone outcomes, with the highest trabecular and total bone area at the distal tibia observed in the estrogen and energy deficient group compared to the estrogen- or energy-replete groups. Notably, vBMD, bone geometry, and estimated bone strength were better associated with energy status at the tibia, while these same variables were better associated with estrogen status at the radius. These studies suggest the importance of treating both hypoestrogenism and energy deficiency in exercising women to prevent an uncoupling in bone turnover and declines in vBMD, bone microarchitecture, and bone strength.

2.3.5 Influence of DE Behaviors on Objective and Surrogate Measures of EA

DE behavior is one of the underlying pathways leading to low EA in physically active individuals and athletes. Several cross-sectional studies have examined the association between psychometric measures of DE and low EA. In a study in 112 female athletes, Sharps et al. (2021) observed that 16%, 44% and 53% of the participants displayed ED, DE, and low EA using the

Female Athlete Screening Tool (FAST) and LEAF-Q, respectively. Unlike majority of ED/DE questionnaires only validated in general populations, FAST is developed and validated to identify individuals with DE/ED particularly in female athletes, while LEAF-Q identifies individuals who are at risk of symptoms of low EA. A moderate positive correlation between was observed between FAST and LEAF-Q scores, demonstrating that individuals who were at higher risk of low EA were more likely to experience an eating pathology. Similarly, in a cross-sectional study in 202 young female endurance athletes, DE behaviour was observed more frequently in athletes who were at risk of low EA compared to the control group (Fahrenholtz et al., 2018). In a study in 642 female and 631 male athletes, Kuikman et al. (2021) used the Eating Disorder Examination Questionnaire (EDE-Q) and LEAF-Q to identify the prevalence of athletes at risk for DE and low EA and found that female athletes who were at risk of low EA displayed a higher risk of DE (i.e., higher global EDEQ score) when compared to non-athlete controls. Findings from these studies support the association between DE and low EA in female athletes.

DE and low EA are more common in certain athletic subgroups. Many of the studies in this field have excluded male athletes and female athletes typically display a higher prevalence of DE/ED. Gender-related stigma around DE may contribute to underdiagnosis and undertreatment of DE/ED in male athletes, because DE and physical appearance related problems are stereotyped as a women's health problem (Strother et al., 2012). Sonneville and Lipson (2018) showed that men with symptoms of EDs were less likely to receive diagnosis and treatment compared to women. Men were also less likely to recognize their need for treatment (Sonneville & Lipson, 2018). Athletes who engage in leanness-focused sports, especially long- and middle-distance running, are at a higher risk for DE and/or low EA, due to the belief that a

lower weight/leaner physique will result in improvements in athletic performance (Melin et al., 2019).

Although DE/EDs are key factors in the development of low EA and associated health consequences, not all DE behaviours result in an energy deficiency. When compared to other psychological constructs of eating behaviour and attitudes, drive for thinness has been shown to have a stronger association with low EA and reduced EI. Drive for thinness, a subscale of the Eating Disorder Inventory, is defined by a perceived discrepancy between actual and ideal body weight (Sands, 2001) Individuals with a high drive for thinness consciously limit their food intake due to the fear of gaining weight and have a preoccupation with a thin physique (Sundgot-Borgen & Torstveit, 2004). Drive for Thinness score has been shown to correlate negatively with REE and TT_3 levels and positively with ghrelin levels in 52 exercising women and sedentary controls, suggesting that drive for thinness may be a proxy indicator when screening for low EA/energy deficiency (De Souza et al., 2007). Further, in a cross-sectional study in 117 exercising women, Gibbs et al. (2011) demonstrated that women with a high drive for thinness may have a higher risk of an energy deficiency (expressed as a ratio between measured REE and predicted REE) and impaired reproductive function (expressed as a greater prevalence of amenorrhea and oligomenorrhea) compared to women with a normal drive for thinness. Previous studies have demonstrated a higher body dissatisfaction, bulimia, and drive for thinness in female athletes with severe menstrual disturbances (De Souza et al., 2007). Similarly, Reed et al. (2012) also found a negative association between body dissatisfaction and EA in 19 female collegiate athletes. Collectively, these results indicate that higher body dissatisfaction, drive for thinness, and bulimia subscale scores may be linked to EA through restrictions in EI and/or increased EEE.

2.4 Knowledge Gaps

Substantial evidence supports the positive relationship between energy status and bone parameters in physically active individuals and athletes (De Souza et al., 2014). Since no gold standard has been established for determining EA, there is considerable methodological heterogeneity in the literature which limits our understanding of its relationship with bone health in young endurance-trained individuals (Burke, Lundy, et al., 2018; Heikura et al., 2021). Due to the various challenges and potential errors in directly measuring EA, researchers have used indirect measures of EA through assessing symptoms of short/long term energy deficiency which includes but is not limited to blood parameters, menstrual status, measured versus predicted REE, and history of DE/ED (Heikura et al., 2018a; Reed et al., 2015; Staal et al., 2018). Although the current evidence suggests that blood parameters (leptin, IGF-1, bone formation and resorption markers, TT_3) may be the most reliable way to determine energy status, there are several challenges when extrapolating the observations from these laboratory-based outcomes to the field (Heikura et al., 2021). Thus, using a direct calculation of EA through assessments of EEE and dietary EI over a 7-day period may improve the ecological validity of these measurements. Prior research has mostly quantified aBMD using a two-dimensional measurement of aBMD (DXA), which does not provide information on bone structural and strength properties. Bone strength can be quantified more accurately using pQCT, which provides a better measure of bone geometry, structure, and strength (Hart et al., 2017; Stagi et al., 2016). To date, there are very few studies exploring the relationship between EA and pQCT-derived bone strength in endurance-trained individuals, a subset of the athletic population at a higher risk of bone loss and BSIs. Further, the research exploring Triad- or RED-S-related conditions in male endurance athletes has been lacking, and to our knowledge, there has not been

any comparisons of the associations between EA, eating attitudes, and bone strength between sexes. Therefore, this research will provide new knowledge on the interrelationship between EA, eating attitudes, and bone strength in young endurance-trained individuals, and describe potential sex-differences in these associations.

Chapter 3: Methods

3.1 Study Design

This study used a cross-sectional study design to evaluate the associations between EA, eating attitudes, and bone strength. Recruitment and data collection took place on a rolling basis, in which participants completed a virtual screening session and two in-person study visits, one at the Center of Innovative Medicine (CIM) (study visit #1), and the other at the Currie Gymnasium at McGill University (study visit #2). Participants were recruited using purposeful and snowball sampling techniques. Recruitment flyers were posted on departmental news outlets and social media platforms (Facebook and Instagram). The head of local running groups and collegiate endurance teams were contacted to circulate the study recruitment flyer to the members of their team/community. Individuals who contacted us via email or phone were sent information about the purpose of the study, description of the research procedures, and benefits and risks associated with participation. If still interested, a virtual screening visit was scheduled to determine their eligibility for the study. Study visits were scheduled for the participants who met the eligibility criteria.

During study visit #1 at the CIM, participants underwent pQCT and DXA imaging scans. Following the imaging scans, participants filled out a series of questionnaires through the Research Electronic Data Capture (REDCap) web-based system, including a demographic and health history questionnaire, International Physical Activity Questionnaire (IPAQ), Bone-specific Physical Activity Questionnaire (BPAQ), the menstrual function and oral contraceptive use section of Low Energy Availability in Females Questionnaire (LEAF-Q) (female participants only), and the Drive for Thinness and Body Dissatisfaction subscales from the Eating Disorder Inventory-3 (EDI-3). During study visit #2 at the Currie Gymnasium, participants completed a

series of performance-based tests of upper- and lower-body muscle strength and maximal aerobic capacity. The participants were asked to arrive having abstained from caffeine, strenuous exercise, and alcohol for ≥12 hours and abstain from food 2 hours prior to the study visit. After study visit #2, participants were asked to: 1) wear a triaxial accelerometer on their waist for 7 consecutive days; 2) wear a polar heart monitor during every exercise training session in the 7-day period; and 3) complete a 24-hour dietary recall using the Automated Self-Administered 24-hour web-based dietary assessment tool over three days (two weekdays, one weekend day). However, twenty-six participants completed these measurements between the two study visits due to convenience. Each study visit lasted approximately 1.5 hours. For this thesis proposal, only relevant outcome measures are presented herein. This research was conducted according to the Tri-Council Policy Statement, second edition, and approval from the McGill University Health Centre Research Ethics Board was obtained.

3. 2 Participants

We recruited physically active men and women aged 18-35 years who engaged in high volume weight-bearing endurance training (i.e., running). Eligible participants were non-obese (BMI <30 kg/m^2), free of metabolic, neuromuscular, or intestinal disorders known to affect bone metabolism, and performed high-volume, weight-bearing endurance exercise >180 mins/week and/or 30 km/week in the past 6 months. We recruited only female participants with a naturally occurring menstrual cycle (i.e., between 21-35 days) or those using oral contraceptive pills at the time of study enrolment. To minimize confounding effects of reproductive hormones across the menstrual cycle, our intention was to test female participants during the early follicular phase of the menstrual cycle (days 1-5) or during the non-active pill phase if on oral contraceptives. However, due to scheduling constraints, some female participants were not tested entirely in the

early follicular phase. Additional exclusion criteria included medication known to affect bone metabolism (e.g., hormonal contraception use other than oral contraceptives within last 3 months prior to study participation, glucocorticoids, anti-hypertensive drugs, anti-epileptic drugs, osteoporosis therapy), orthopedic or musculoskeletal injury/disease that limits the capacity to exercise, pQCT scan impossible to perform, current diagnosis of an ED, current smokers, female participants with self-reported or diagnosed hypothalamic amenorrhea, polycystic ovarian syndrome, hyperprolactinemia, or primary ovarian insufficiency, and pregnant or breastfeeding. Written informed consent was obtained in all participants prior to the first study visit.

3.3 Outcome Measures (Table 1.2)

pQCT: pQCT is a three-dimensional, precise, and accurate imaging technique that provides a cross-sectional view of the bone tissue structure characteristics at peripheral sites (Stagi et al., 2016). pQCT is used to determine vBMD and area and estimate bone strength indices including SSI. Unlike DXA, pQCT can distinguish between cortical and trabecular bone compartments and measure vBMD independent of bone size, while also distinguishing bone from soft tissue. (Engelke et al., 2008; Erlandson et al., 2016; Frank et al., 2010).

A trained bone densitometry technologist performed pQCT scans. pQCT scans were performed at the tibia using the XCT 3000 scanner (Stratec Medizintechnik). pQCT acquisition parameters were 2.5 mm slice thickness, 0.5 x 0.5 mm in-plane pixel size and a tube voltage 60kV operated at 0.3 mA. Images were analyzed using the Stratec software (Orthometrix Inc., White Plains, NY) to derive the following variables at the 4%, 38%, and 66% sites of the tibia (measured from the distal end of the medial malleolus to the proximal end of the medial tibia plateau): total, trabecular, and cortical vBMD and area, and SSI (Giangregorio et al., 2013; Wong et al., 2015). Total and trabecular vBMD and area were analyzed from the 4% site using

the CALBD analysis – contour mode 1 with a threshold of 280 mg/cm^3. Total and cortical vBMD and area were analyzed at the 38% and 66% sites using the CORTBD analysis – contour mode 1 and a threshold of 710 mg/cm^3. SSI was analyzed at the 38% and 66% sites using a threshold of 480 mg/cm^3 with contour mode 1. Segmentation of muscle from subcutaneous fat used a threshold of 40 mg/cm^3 with contour mode 3 and peel mode 1. To determine muscle CSA, bone area was subtracted from total bone area + muscle area. We used muscle filter (F03F05) to enhance the detection of muscle area. Bone mass was subtracted from total bone + muscle mass to determine muscle mass. Muscle density was calculated by dividing total muscle mass by muscle CSA.

DXA: aBMD (lumbar spine, femoral neck, total hip) and body composition were measured using DXA (GE Lunar iDXA scanner). Each DXA scan involved lying on an open scanner for 1-7 minutes, depending on the scan, while two X-ray beams passed through the body aimed at the participants bones, fat mass, and LBM. Participants were asked to remove any metal or jewelry to avoid confounding the results of the scan. Whole-body and regional fat and LBM (kg) were determined using the enCORE software platform. Daily machine calibration using periodic phantom scans, daily and weekly quality assurance tests and longitudinal stability were monitored. According to the Triad risk assessment, aBMD Z-scores -1 and below were considered low risk, between -1 and -2 represented moderate risk, and -2 and below were considered high risk for Triad conditions (De Souza et al., 2014).

EA Calculations: EA was calculated as EI minus EEE divided by kilograms of LBM (Loucks & Thuma, 2003). The average values of EEE and dietary EI from the 7-day period were used to calculate the EA of each participant (see below for more details on these measures). Low EA

was characterized as having an EA below 30 kcal/kg LBM/day (Gibbs et al., 2013; Loucks & Thuma, 2003; Reed et al., 2015).

EEE

Participants completed a physical activity log wherein they recorded the type, intensity, rating of perceived exertion, and distance (if applicable) of their purposeful exercise for a 7-day period. In the same period, they were asked to wear a Polar heart rate sensor (Polar H10) and monitor (Polar Unite) during each of their purposeful exercise sessions. To compute EEE, the resting EEE expenditure (estimated using Cunningham equation) was subtracted from the total amount of calories expanded during the exercise session. The EEE data was calculated using the Heart Rate (HR)-flex method which translates heart rate data to EE estimates using individualized prediction equations (Cunningham, 1980; Spurr et al., 1988).The HR-flex method has been validated against doubly labeled water in athletes (Ekelund et al., 2002). Also, several studies in athletes have used this method to calculate EEE and EA (Łagowska et al., 2014; Łagowska & Kapczuk, 2016; Melin et al., 2015; Schaal et al., 2011). This bi-linear method considers the weak linear relationship between heart rate and EE at rest and low activity levels, and therefore estimates a heart rate cut-off point, typically calculated as the mean of the highest heart rate for the resting activities (supine, sitting, and standing) and the lowest heart rate of the exercise activities (Leonard, 2003). The HR-flex point and HR/VO_2 equation for each participant was calculated using the HR and VO_2 data from the laboratory-based progressive incremental test to volitional exhaustion which was performed to determine VO_{2max}. For this study, the HR-flex point was calculated as the mean of the heart rate at the end of a 5-minute standing period and the heart rate after the first stage of the laboratory-based incremental treadmill test to volitional exhaustion (0% incline, 8 km/hr). Below this point, EE was assumed to equal REE. At all

timepoints where heart rate was above the threshold, EE was estimated based on the heart rate/rate of oxygen consumption (HR/VO_2), assuming a caloric equivalency of 5 kcal per liter oxygen consumed. To determine the individual HR/VO_2 equation, the calibration procedure was performed by simultaneously measuring heart rate and VO_2 at the baseline (after standing for 5 minutes on the treadmill prior to the test) and after the completion of each stage of the laboratory-based incremental treadmill test to volitional exhaustion. This equation provided the basis for EEE calculations during the 7-day monitoring period. If 1) second-to-second heart rate from heart rate monitors, and/or 2) the rate of oxygen consumption and/or heart rate at baseline and during the incremental treadmill test were unavailable, EE for an exercise session was imputed using a physical activity compendium (Ainsworth et al., 2011). After multiplying the duration of the exercise session by the MET value which corresponds to the physical activity, REE was subtracted from this value to calculate EEE. Calories expended in each recorded exercise session during the 7-day monitoring period were summed and divided by 7 to calculate mean EEE (kcal/day).

Dietary EI

Dietary energy, macronutrient, and calcium intakes were measured by 24-hour dietary recall using the Automated Self-Administered 24-hour web-based tool over three days (two weekdays, one weekend day). Participants received the necessary information required (i.e., manual, user credentials) to fill out the dietary recalls via e-mail. A three-day dietary record was chosen over a 7-day record to reduce the subject burden and increase response rates (Magkos & Yannakoulia, 2003). The calories reported over three days were averaged to calculate dietary energy intake.

Drive for Thinness, Body Dissatisfaction, and Bulimia subscales from the Eating Disorder Inventory-3 (EDI-3): These subscales were three of the 12 subscales from the 91-item self-

report questionnaire designed for the assessment of psychological domains that have conceptual relevance in understanding and treating EDs (Garner, 2004). The drive for thinness subscale has 7 items and measures the presence of an excessive concern with dieting, preoccupation with weight, and an extreme pursuit of thinness. The score from each item was recoded and summed to generate the Drive for Thinness subscale score which ranged from 0 to 28. According to the Triad Coalition cumulative risk assessment tool, individuals with a drive for thinness score of 7 or higher were classified as being at high risk for Triad conditions. Scores between 3 and 6 were categorized as moderate risk, while scores below 3 were regarded as low risk (De Souza et al., 2014). As our study lacked statistical power to explore the associations between Triad risk categories for drive for thinness and primary outcomes of interest, we only reported the drive for thinness score categories to describe our sample. The Body Dissatisfaction subscale has 10 items and measures discontentment with the overall shape and with the size of those regions of the body of high concern to those with EDs (i.e., stomach, hips, thighs, buttocks). The score from each item was recoded and summed to generate the Body Dissatisfaction subscale score which ranged from 0 to 40. The Bulimia subscale has 8 items and assesses the tendency to think about and to engage in bouts of uncontrollable overeating (binge-eating). The score from each item was recoded and summed to generate the Bulimia subscale score which ranged from 0 to 32. EDI-3 total score was computed by summing the Drive for Thinness, Bulimia, and Body Dissatisfaction subscale scores. Eating Disorder Risk Composite (EDRC) score included the sum of T scores from the three subscales. Every summed T score had a corresponding EDRC score, found in the EDI-3 Professional Manual Appendix B, which provided a score that assigned equal weight to each of the three included subscales compared to the clinical diagnostic group. The appropriate diagnostic group for our sample was chosen as Anorexia Nervosa – Restricting type.

In our study, EDRC score was used to describe our sample and was not included in the correlation analysis. The range for EDI-3 total score and EDRC score were 0-100 and 26-82 respectively (Garner, 2004), with a higher score reflecting a higher ED risk. Reliability of EDRC ranges from .90 to .97 across the normative groups and clinical diagnostic groups. Reliability and validity of the EDI-3 has been established in individuals aged 13-53 years (Garner, 2004). Although EDI-3 was not developed for and validated in active population, drive for thinness, body dissatisfaction, and bulimia subscales are commonly used as a psychometric questionnaire assessing DE attitudes/behaviours (Sim & Burns, 2021).

Menstrual Function and Oral Contraceptive Use from Low Energy Availability in Females Questionnaire (LEAF-Q): The LEAF-Q was administered to female participants to gather data on menstrual status, history of menstrual irregularities, and past/current contraceptive use.

Demographics and Health History Questionnaire: Participants filled out a demographics and health history questionnaire which collected data on sociodemographic characteristics, endurance training history (type, frequency, volume) and competition level, current medication and supplement use, weight change patterns, alcohol and tobacco use, history of disease, illness, and musculoskeletal injury. Additional questions were added to evaluate the endurance training history (type, frequency, mileage) and competition level. For the competition level, participants were given three options: recreational, intermediate, and elite level. Elite competition level was defined as a participant who is a member of an elite-level competitive team including professional, national, provincial or varsity team. The intermediate competition level was defined as someone who participated in competitive endurance events in the past 4 years or who had previous competitive experience, and recreational level included little or no competitive experience.

International Physical Activity Questionnaire: The short version of the International Physical Activity Questionnaire was used to determine the self-reported time (number of sessions in the past 7 days, average duration per session) spent in moderate-to-vigorous physical activity, walking, and sedentary behaviour (sitting and lying down awake) (Craig et al., 2003). Additional questions assessed the self-reported time spent participating in resistance training (on own or as a part of a fitness class) and balance/flexibility exercise (e.g., yoga, pilates, Tai Chi). Reliability and validity of the IPAQ have been previously reported.

Bone-Specific Physical Activity Questionnaire: The Bone-Specific Physical Activity Questionnaire (BPAQ) was used to assess previous participation in bone-specific physical activity. Participants were asked to self-report the lifetime physical activity (types of physical activity, age, and years of participation) as well as types and frequency of physical activity participation in the past 12 months. BPAQ responses were analysed using algorithms (current and past BPAQ algorithms) and effective load ratings (assigned to common sports and activities from ground reaction force measures of fundamental actions observed in each sport/activity) to convert the raw data into a BPAQ score, which was shown to predict bone strength parameters (Weeks & Beck, 2008).

Tri-axial Accelerometer: Accelerometers were used to objectively determine physical activity levels. Triaxial accelerometer captures acceleration in three separate perpendicular axes, namely vertical, anteroposterior, and medio-lateral. Activity counts are generated by combining the acceleration values from these three axes into one composite vector magnitude (VM3). Participants wore a commercially available accelerometer (GT3X+ monitors, ActiGraph, FL, USA) over the hip for 7 consecutive days during waking hours. Data was used to compute the number of minutes spent in three intensity levels of activity (sedentary, light, moderate and

vigorous) based on standard counts/minute-based cut-points. Data was analyzed using Freedson Adult VM3 cut points to monitor time spent at each level of activity (Sasaki et al., 2011). The light activity was defined as 0-2690 counts per minute (cpm), and moderate-to-vigorous physical activity as ≥2691. Data was analyzed in 60 second epochs. Non-wear time was excluded if ≥60 minutes of consecutive zeros. Only 36 participants who wore the accelerometer for at least 4 days and 10 hours/day were analyzed (Colley et al., 2011; Troiano et al., 2008).

Cardiopulmonary Exercise Test: Maximal aerobic capacity ($VO_{2ma}x$ in mL·kg-1·min-1) was measured during a progressive treadmill test to volitional exhaustion using the modified Astrand protocol. Gas exchange was monitored continuously using a breath-by-breath indirect calorimetry system (SensorMedics Vmax metabolic cart, VIASYS Healthcare, CA, USA). The protocol involved treadmill running at a constant pace of 8 km/hr while incline went up by 2.5% at the end of each stage. Participants performed a 5-minute warm-up prior to testing followed by a cool-down period. Blood pressure measurements were taken before and after the testing. Heart rate and rating of perceived exertion were monitored throughout the testing. We considered VO2max to be achieved if three of the following four criteria were obtained: (1) attainment of age-predicted maximal heart rate; (2) respiratory exchange ratio ≥1.1; (3) plateau in oxygen consumption despite an increase in exercise workload; and (4) attainment of a rating of perceived exertion score ≥18.48

Anthropometric measures: Height was measured to nearest 0.1 cm using a calibrated well-mounted stadiometer. The participants heels, buttocks and head were flat on the wall, and eyes looking forward. After an exhalation, the head plate was brought down to the top of the participants head to determine their height. Weight was measured to the nearest 0.1 kg using a clinical, calibrated electronic weighing scale (Scale-Tronix, Welch Allyn, Skaneateles, NY). The

scale was zeroed, and the participant stepped onto the scale ensuring shoes, heavy jewelry, or any heavy clothing was removed. They stood straight, look forward, and stood still. The results were recorded. BMI was calculated as body weight (kg) divided by height squared (kg/m^2).

3.4 Statistical Analyses

Data analyses were conducted using the SPSS software package (version 24, Armonk, NY, USA). Participant characteristics and outcomes were summarized using descriptive measures: mean (standard deviation) for continuous variables and number (percentage) for categorical variables. Data was screened for statistical outliers and the assumptions for each statistical test were examined. Independent T-Test and Chi-Square analyses were used to compare continuous and categorical outcomes between sexes. Pearson/Spearman correlation coefficients were determined to assess the correlations between independent and dependent variables of interest. The correlation was considered negligible if correlation coefficient (r) is less than 0.2. The strength of the correlation was considered weak if r is between 0.2 and 0.4, moderate if r is between 0.4 and 0.7, strong if r is between 0.7 and 0.9, and very high if r is above 0.9 (Guilford, 1973). Multivariable linear regression models evaluated the associations between EA, eating attitudes and pQCT bone outcomes adjusting for age, sex, and BMI. A minimum of ten observations for each independent variable was used to avoid overfitting the models (Babyak, 2004). Regression coefficients were reported along with 95% confidence intervals and model fit was assessed using R^2 values. We performed Bonferroni correction (0.05/42) for multiple testing of independent variables and their associations with the dependent variables. Anticipating a moderate-to-strong association (effect size=0.33) between pQCT measures of calf muscle area and total vBMD at the tibia (power=0.80 and alpha=0.05) (the primary objective of the larger study), we determined that 50 adults at minimum were needed to detect a significant association.

For the present study, the objectives were of a hypothesis-generating, exploratory nature and intended to inform sample size calculations for future studies.

Chapter 4: Results

4.1 Participant Characteristics

Thirty-one males and 12 females participated in the study (**Table 4.1**). Participants were 25.54 ± 4.25 years old, had a mean BMI of 22.81 ± 2.88 kg/m^2, percent body fat of 18.58 ± 6.24%, LBM of 54.16 ± 10.67 kg, and VO_{2max} of 57.02 ml/min/kg. There were significant differences in height (178.05 ± 5.45 cm vs 164.14 ± 6.34 cm, $p<0.001$), weight (73.35 ± 11.33 kg vs 58.16 ± 6.50 kg, $p<0.001$), LBM (58.92 ± 8.23 kg vs 41.86 ± 4.59 kg, $p<0.001$), FFM (62.01 ± 8.65 kg vs 44.18 ± 4.81 kg, $p<0.001$), and percent body fat (16.14 ± 5.505% vs 24.88 ± 2.54%, $p<0.001$) between males and females. Twenty-nine participants (67%) were White/Caucasian; 7 (16%) were Asian; 5 (12%) were either Black/African American, Middle Eastern/North African, or Hispanic/Latino; and 2 (5%) participants identified themselves as "other". Forty-two percent (n=18) of our sample engaged in full-time or part-time studies, while 51% (n=22) worked full-time. From 7% (n=3) of the participants who selected "other", one worked part-time, one worked and studied part-time, and one participant was self-employed.

When asked to report all current endurance sports participation (participants were able to select participation in multiple activities), ninety-one percent (n=39) of our sample engaged in long-distance running (more than 3000 metres) and 30% (n=13) in middle-distance running (between 800 to 3000 m). Fifty-five percent (n=24) also participated in either triathlon, swimming, or cycling. Twenty-one percent (9/43) of participants classified themselves as competing at the elite level, 51% (22/43) at the intermediate level, and 29% (12/43) at the recreational level. Based on the accelerometer data in 36 participants, the median moderate-to-vigorous physical activity was 667.33 (4417 – 7855.40) minutes/week and sedentary time was 7.73 (2.99 – 14.02) hours/day. Twenty-five percent (9/36) of participants engaged in more than

12 hours of moderate-to-vigorous physical activity per week, which is considered a high-volume of exercise training and risk factor for a BSI (Barrack et al., 2014). The mean IPAQ score was 407.73 ± 276.48 minutes/week. The current, past, and total BPAQ scores were 6.01 ± 11.65, 43.16 ± 36.12, and 24.59 ± 17.97 respectively. The mean past BPAQ score for our sample was lower than the average past BPAQ score of 57.7 reported by Weeks et al., (2023) in a sample of 532 healthy individuals aged 4-97.

Only 11.6% (5/43) of our sample had a history of stress fracture while thirty-nine percent (17/43) of participants reported a previous fracture. Two participants had experienced a stress fracture in the fibula, one in the knee, and two participants did not report the injury site. Eighty percent of those who reported a stress fracture (4/5) were competing at an elite or intermediate level and engaged in weight-bearing endurance training of more than 5 hours per week. Sixty percent (n=25) of our sample reported dieting in the past 5 years. Although most participants maintained a stable weight in the past 6 months, 5 (11.6%) individuals lost weight while 8 (18.6%) gained weight. Among those who had a weight change fluctuation, 15% (2/13) lost/gained more than 4.5 kg and 46.2% (6/13) reported that the change was intentional.

Among female participants, three participants currently used copper coil or oral contraceptive pills, and seven participants reported a history of oral contraceptive usage. Three females (25%) were 11 years old or younger when they had their first menses, six (50%) were between the age of 12 and 14, and three (25%) were 15 years old or older. Fifty-eight percent (7/11) reported that their menses has stopped for 3 consecutive months or longer in the past. Four females reported that they noticed a change in their menstruation with an increase in exercise intensity, frequency, or duration. None of the participants reported previous or current smoking.

Twenty-six percent (11/43) did not consume alcohol, 67% (29/43) consumed 1-7 alcoholic beverages per week, and 7% of participants (3/43) consumed 7-14 alcoholic beverages per week.

4.2 EA and Eating Attitude Parameters

EA was calculated for 42 participants; one participant had missing EI and EA data. Twenty-four percent (n=10) of our sample had low EA. No significant differences were observed in both EEE ($p = 0.096$) and EA ($p = 0.635$) between males and females (**Table 4.2**). Males had a significantly higher EI ($p = 0.019$) than females. None of the individual subscale scores from the EDI-3 were different between sexes. Mean scores for body dissatisfaction, drive for thinness, and bulimia subscales were 5.00 ± 6.04, 5.47 ± 6.47, 2.33 ± 2.83 for males; and 7.50 ± 5.90, 5.50 ± 6.54, 1.50 ± 1.45 for females. Forty-three percent (n=18) reported drive for thinness scores characterized as low Triad risk, 28.5% (n=12) had scores considered as moderate Triad risk and remaining 28.5% (n=12) had scores classified as high Triad risk. EDRC score ($p = 0.889$) and total score ($p = 0.410$) were also not significantly different between males and females.

4.3 DXA and pQCT Parameters

All participants completed pQCT scans at 4%, 38%, and 66% sites at the tibia, and DXA scans at the hip, lumbar spine, and total body. The aBMD Z-scores were not generated for four participants who were below the age of 19. Male participants had higher total vBMD ($p = 0.020$), total area ($p = 0.011$), trabecular vBMD ($p = 0.005$), and trabecular area ($p < 0.001$) at the 4% site compared to the females (**Table 4.3**). Cortical area and SSI at 38% ($p < 0.001$) and 66% sites ($p < 0.001$), as well as total area at 66% ($p < 0.001$) site were higher in males than females. No significant differences between sexes were observed in cortical vBMD at 38% and 66% site, and total vBMD at 66% site at the tibia. Males had higher aBMD at the total body ($p = 0.002$), total hip ($p = 0.002$), and femoral neck ($p = 0.036$). In our sample, participants were not classified as

high risk for Triad conditions based on their aBMD, except for one participant with a lumbar spine aBMD Z-score <-2. Five percent (n=2), 10% (n=4), and 18% (n=7) of the participants were at moderate risk based on their Z-scores at the hip, femoral neck, and lumbar spine, respectively. The remaining participants were classified as low risk.

4.4 Associations between EA and Bone Parameters

There were weak negative correlations between EA and trabecular area ($r = -0.333$, $p = 0.036$) and SSI at 38% site ($r = -0.339$, $p = 0.032$) while moderate positive correlations were observed between EA and cortical area at 66% site ($r_s = 0.459$, $p = 0.003$) (**Table 4.4**) **(Figure 1.A)**. EA was not significantly associated with total and trabecular vBMD at 4% site; cortical area at 38% site; cortical vBMD at 38% and 66% site; total area, total vBMD, and SSI at 66% site at the tibia. Weak-to-moderate positive correlations were found between EI and trabecular vBMD ($r = 0.315$, $p = 0.045$), total vBMD at 4% ($r_s = 0.331$, $p = 0.032$), cortical area and SSI at 38% ($r = 0.382$, $p = 0.014$; $r = 0.332$, $p = 0.034$) and 66% sites ($r_s = 0.375$, $p = 0.016$; $r_s = 0.373$, $p = 0.016$) **(Figure 1.B)**. EEE was positively correlated with cortical area ($r = 0.413$, $p = 0.007$; $r = 0.366$; $p = 0.019$) and SSI at 38% ($r = 0.366$, $p = 0.019$). EEE was positively correlated with total body aBMD ($r = 0.308$, $p = 0.045$). There were no significant associations between EA and aBMD variables. EI was positively correlated with aBMD at the total body ($r = 0.370$, $p = 0.015$), lumbar spine ($r = 0.350$, $p = 0.021$), and femoral neck ($r = 0.324$, $p = 0.034$). Total area at 4% and 66% sites, cortical and total vBMD at 66% site were not associated with any of the EA and eating attitude outcomes ($p>0.05$). When adjusted for age, sex, and BMI, none of the EA parameters were significantly associated with pQCT bone parameters, except for EEE and trabecular area ($R^2 = 0.080 – 0.668$, $F = 0.921 – 18.090$). (**Table 4.6 and 4.7**). The pQCT variables that did not exhibit any significant associations with EA, EEE, or EI (total area at 4%,

cortical vBMD at 38%, total area, cortical and total vBMD at 66% sites) were intentionally excluded from **Table 4.4 and 4.6**.

4.5 Associations between Eating Attitudes and EA

A weak negative correlation was observed between drive for thinness and EA (r_s= -0.380, p = 0.014) **(Figure 1.C)**. Body dissatisfaction was negatively correlated with EEE and EI (r_s = -0.337, p = 0.029; r_s = -0.443, p = 0.003) (**Table 4.5**). No significant correlations were found between bulimia and EA outcomes. EDI-3 total score was negatively correlated with EA (r_s = -0.316, p = 0.044) and EI (r_s = -0.365, p = 0.018). When adjusted for age, sex, and BMI, none of the EDI-3 subscale scores were significantly associated with EA parameters, except for EDI-3 total score and EI (R^2 = 0.160 – 0.251, F = 1.271 – 3.108 (**Table 4.8**).

4.6 Associations between Eating Attitudes and Bone Parameters

No significant correlations were observed between any of the EDI-3 subscales and pQCT bone outcomes (**Table 4.4**). Drive for thinness and EDI-3 total scores were positively correlated with total body aBMD (r_s = 0.444, p = 0.005; r_s = -0.466, p = 0.003). There was a weak positive correlation between EDI-3 total score and aBMD at the lumbar spine (r_s = 0.318, p = 0.004). When adjusted for age, sex, and BMI, none of the EDI-3 variables were significantly associated with pQCT bone outcomes (R^2 = 0.100 – 0.675, F = 0.975 – 18.143) **(Table 4.8).**

Table 4.1 Descriptive characteristics in study participants

	All (n = 43)	Male (n=31)	Female (n=12)	p-value
Age (years)	25.95 ± 4.57	25.68 ± 4.80	26.67 ± 4.01	0.531
Height (cm)	174.08 ± 2.88	178.05 ± 5.45	164.14 ± 6.34	**<0.001**
Weight (kg)	69.11±12.26	73.35 ± 11.33	58.16 ± 6.50	**<0.001**
BMI (kg/m^2)	22.81±2.88	23.29 ± 3.07	21.56 ± 1.91	0.077
LBM (kg)	54.16 ± 10.67	58.92 ± 8.23	41.86 ± 4.59	**<0.001**
FFM (kg)	57.04 ± 11.18	62.01 ± 8.65	44.18 ± 4.81	**<0.001**
Fat mass (kg)	12.30±4.84	11.69 ± 5.42	13.90 ± 2.37	0.070
Percent body fat (%)	18.57±6.24	16.14 ± 5.50	24.88 ± 2.54	**<0.001**
Race/ethnicity - N (%)				0.580
White/Caucasian	29 (67%)	21 (68%)	8 (67%)	
Black/African American	1 (2%)	1 (3%)	0 (0%)	
Hispanic/Latino	1 (2%)	0 (0%)	1 (8%)	
Asian	7 (16%)	5 (16%)	2 (17%)	
Middle Eastern or North African	3 (7%)	2 (7%)	1 (8%)	
Other	2 (5%)	2 (7%)	0 (0%)	
Employment Status				0.709
Full-time student	15 (35%)	11 (35%)	4 (33%)	
Part-time student	3 (7%)	2 (7%)	1 (8%)	
Full-time job	22 (51%)	15 (48%)	7 (58%)	
Other	3 (7%)	3 (10%)	0 (0%)	
Dieted in the past 5 years				0.299
Yes	25 (58%)	17 (55%)	8 (67%)	
No	17 (40%)	14 (45%)	3 (25%)	
Weight change in the last 6 months				0.196
Gained weight	8 (19%)	6 (19%)	2 (17%)	
Lost weight	5 (12%)	5 (16%)	0 (0%)	
Stable	29 (67%)	20 (65%)	9 (75%)	
Don't know	1 (2%)	0 (0%)	1 (8%)	
Current sport type (multiple answers allowed)				
Middle-distance running	13 (30%)	10 (32%)	3 (25%)	
Long-distance running	39 (90%)	27 (87%)	12 (100%)	
Triathlon	5 (12%)	3 (10%)	2 (17%)	
Swimming	7 (16%)	3 (10%)	4 (33%)	
Cycling	12 (28%)	9 (29%)	3 (25%)	
Other	8 (19%)	7 (23%)	1 (8%)	
Weight-bearing endurance exercise distance (km/week)				0.904
15-25	9 (21%)	7 (23%)	2 (17%)	
25-40	13 (30%)	9 (29%)	4 (33%)	

> 40	21 (49%)	15 (48%)	6 (50%)	
Fracture history	17 (40%)	11 (35%)	6 (50%)	0.629
Stress fracture history	5 (12%)	2 (6%)	3 (25%)	0.097
History of smoking	0 (0%)	0 (0%)	0 (0%)	
Alcohol use				0.107
None	11 (4%)	10 (32%)	1 (8%)	
1-7 drinks/week	29 (67%)	18 (42%)	11 (92%)	
8-14 drinks/week	3 (7%)	3 (7%)	0 (0%)	
Peak isometric knee extensor strength (Nm)	274.14 ± 86.79	306.1 ± 69.96	191.57 ± 71.38	**<0.001**
Maximal handgrip strength (kg)	44.36	48.37 ± 7.28	34.33 ± 4.94	**<0.001**
$VO2_{max}$ (ml/min/kg)	57.02 ± 38.49	58.76 ± 8.68	52.53 ± 6.25	**0.029**
Accelerometer sedentary time* (hours/day)	7.73 (2.99 – 14.02)	7.71 (2.99 – 9.54)	7.88 (6.04 – 14.02)	0.106
Accelerometer light PA* (min/week)	5520 (4417 – 7855.40)	5521.60 (4613 – 6202.0)	5515 (4417 – 7855.40)	0.942
Accelerometer MVPA* (min/week)	667.33 (355.83 – 3349)	667.33 (355.83 – 3349.0)	678 (372.40 – 1132.60)	0.525
IPAQ sedentary time (hours/day)	7.21 ± 3.01	7.15 ± 3.09	7.38 ± 2.95	0.825
IPAQ walking (min/week)	376.74 ± 290.46	418.71 ± 307.436	268.33 ± 215.78	0.129
IPAQ MVPA (min/week)	407.73 ± 276.48	447.98 ± 310.04	303.75 ± 115.54	**0.032**
Current BPAQ score	6.01 ± 11.65	4.47 ± 2.12	9.87 ± 21.71	0.568
Past BPAQ score	43.16 ± 36.12	42.0 ± 28.01	46.12 ± 52.74	0.742
Total BPAQ score	24.59 ± 17.97	23.23 ± 14.10	28.00 ± 25. 74	0.444

Footnotes: p-values < 0.05 are bolded. Mean and SDs are reported for continuous variables.
Number of participants and percentages are reported for categorical variables.
BMI: body mass index; LBM: lean body mass; FFM: fat-free mass $VO2_{max}$: Maximal aerobic capacity; MVPA: moderate-to-vigorous physical activity; IPAQ: International Physical Activity Questionnaire; BPAQ: Bone-specific Physical Activity Questionnaire.
***** = non-parametric t-test (median and range values were reported).

Table 4.2 EA and eating attitudes parameters in all participants and stratified by sex.

	All (n=43)	Male (n=31)	Female (n=12)	p-value
EA (kcal/kg LBM/day)	39.11 ± 14.02	38.44 ± 15.54	40.75 ± 15.54	0.635
EEE (kcal/day)	576.10 ± 288.60	621.71 ± 296.80	458.29 ± 238.22	0.096
EI (kcal/day)	2621.87 ± 862.38	2811.34 ± 904.27	2132.41± 496.96	**0.019**
Body dissatisfaction score*	4.50 (0 – 23)	3.5 (0 – 23)	6 (1 – 23)	0.066
Drive for thinness score*	3.0 (0 – 25)	3 (0 – 25)	2.5 (1 – 21)	0.877
Bulimia score*	1.0 (0 – 13)	1 (0 – 13)	1 (0 – 5)	0.538
EDI-3 total score	13.29 ± 11.98	12.8 ± 11.93	14.5 ± 12.56	0.410
EDI-3 EDRC score	33.64 ± 6.37	33.63 ± 6.41	33.67 ± 6.54	0.889

Footnotes: p-values < 0.05 are bolded. Data are expressed as mean and SD.
EA: energy availability; LBM: lean body mass EEE: exercise energy expenditure; EI: energy intake; EDI-3: Eating Disorder Inventory-3; EDRC: eating disorder risk composite.
*** =** non-parametric t-test (median and range values were reported).

Table 4.3 Bone outcomes from pQCT and DXA in all participants and stratified by sex.

	All (n=43)	Male (n=31)	Female (n=12)	p-value
4% Tibia				
Total vBMD* (mg/cm^3)	344.35 (262.1 – 1146)	355.65 (262.10 – 1146.0)	321.20 (270.50 – 362.70)	**0.02**
Trabecular vBMD (mg/cm^3)	267.90 ± 42.05	276.31 ± 44.65	244.96 ± 22.32	**0.005**
Total area (mm^2)	1095.55 ± 216.37	1148.31 ± 221.16	963.65 ± 137.75	**0.011**
Trabecular area (mm^2)	502.39 ± 86.30	531.28 ± 77.78	423.59 ± 53.98	**<0.001**
38% Tibia				
Cortical vBMD (mg/cm^3)	1154.91 ± 25.95	1153.01 ± 21.47	1160.09 ± 36.30	0.553
Cortical area (mm^2)	348.31 ± 58.44	366.52 ± 49.19	298.64 ± 54.17	**<0.001**
SSI (mm^3)	2074.44 ± 500.15	2255.24 ± 407.30	1581.33 ± 392.41	**<0.001**
66% Tibia				
Total vBMD* (mg/cm^3)	569.1 (462.10 – 1109)	569.1 (462.10 – 1109)	572.7 (490.6 – 729.9)	0.636
Cortical vBMD* (mg/cm^3)	1098 (1037.10 – 1172.20)	1094.25 (1037.10 – 1129.40)	1113.10 (1068.70 – 1172.20)	0.070
Total area (mm^2)	770.92 ± 170.97	825. 22 ± 150.39	635.18 ± 145.51	**0.001**
Cortical area (mm^2)	334.79 ± 59.25	357.68 ± 48.06	272.36 ± 38.56	**<0.001**
SSI (mm^3)	3356.10 ± 910.92	3718.65 ± 719.58	2367.29 ± 589.65	**<0.001**
DXA				
Total body aBMD (g/cm^2)	1.27 ± 0.13	1.31 ± 0.13	1.17 ± 0.08	**0.002**
Total hip aBMD* (g/cm^2)	0.3 (-1.50 – 4.10)	1.13 (0.80 – 1.60)	1.02 (0.948 – 1.173)	**0.011**
Total hip Z-score	0.29 ± 0.82	0.30 ± 0.92	0.29 ± 0.57	0.967
Femoral neck aBMD (g/cm^2)	1.11 ± 0.16	1.14 ± 0.17	1.03 ± 0.09	**0.036**
Femoral neck Z-score*	0.30 (-1.50 – 4.10)	0.3 (-1.5 – 4.10)	0 (-1.2 – -.90)	0.315
L1-L4 aBMD (g/cm^2)	1.20 ± 0.14	1.22 ± 0.16	1.16 ± 0.10	0.261
L1-L4 Z-score	0.03 ± 1.11	0.10 ± 1.23	-0.13 ± 0.80	0.565

Footnotes: Data are expressed as mean and SD.
DXA: dual-energy X-ray absorptiometry; pQCT: peripheral quantitative computed tomography; vBMD: volumetric bone mineral density; aBMD: areal bone mineral density; SSI: stress-strain index.
* = non-parametric t-test (median and range values were reported).

Table 4.4 Associations between EA, eating attitudes, and bone outcomes from pQCT in all participants.

	pQCT @ 4% site						**pQCT @ 38% site**						**pQCT @ 66% site**					
	Total vBMD (mg/cm^3)		**Trabecular vBMD (mg/cm^3)**		**Trabecular area (cm^2)**		**Cortical area (cm^2)**		**SSI (mm^3)**		**Cortical vBMD (mg/cm^3)**		**Total area (cm^2)**		**Cortical area (cm^2)**		**SSI (mm^3)**	
	r	**p**	**r**	**p**	**r**	**p**	**r**	**p**	**r**	**p**	**r**	**p**	**r**	**p**	**r**	**p**	**r**	**p**
EA (kcal//kg LBM/day)	0.119	0.457	0.103	0.527	**-0.333**	0.036	-0.267	0.095	**-0.339**	0.032	0.136	0.403	0.285	0.067	**0.459***	0.003	0.299	0.057
EEE (kcal/day)	0.101*	0.523	0.145	0.366	0.294	0.062	**0.413**	0.007	**0.366**	0.019	-0.09	0.577	0.285	0.067	-0.214*	0.185	0.299	0.057
EI (kcal/day)	**0.331***	0.032	**0.315**	0.045	0.21	0.187	**0.382**	0.014	**0.332**	0.034	-0.178	0.265	0.248	0.114	**0.375***	0.016	**0.373**	0.016
EDI-3 Drive for thinness score	0.06*	0.678	0.091*	0.576	0.119*	0.465	0.239*	0.137	0.211*	0.191	-0.227*	0.159	0.122*	0.448	0.181*	0.265	0.205*	0.204
EDI-3 Bulimia score	0.061*	0.704	0.306*	0.055	0.238*	0.139	0.244*	0.129	0.285*	0.074	-0.198*	0.221	0.161*	0.313	0.232*	0.15	0.194*	0.231
EDI-3 Body dissatisfaction score	0.061*	0.706	0.06*	0.712	-0.271*	0.091	-0.077*	0.635	-0.15*	0.356	-0.081*	0.618	-0.181*	0.256	-0.099*	0.542	-0.087*	0.595
EDI-3 total score	0.08*	0.621	0.138*	0.396	-0.016*	0.921	0.171*	0.292	0.146*	0.37	-0.183*	0.257	0.057*	0.724	0.156*	0.336	0.128*	0.431

Footnotes: p-values < 0.05 are bolded.

pQCT: peripheral quantitative computed tomography; vBMD: volumetric bone mineral density; EA: energy availability; LBM: lean body mass; EEE: exercise energy expenditure; EI: energy intake; EDI-3: Eating Disorder Inventory – 3; SSI: stress-strain index.

* = Spearman's correlations.

Table 4.5 Associations between eating attitudes and EA in all participants.

	EA (kcal/kg LBM/day)		EEE (kcal/day)		EI (kcal/day)	
	r	**p**	**r**	**p**	**r**	**p**
EDI-3 Drive for thinness score	**-0.380***	0.014	-0.121*	0.445	-0.299*	0.055
EDI-3 Bulimia score	0.018*	0.91	0.004	0.982	-0.004*	0.981
EDI-3 Body dissatisfaction score	-0.218*	0.171	**-0.337***	0.029	**-0.443***	0.003
EDI-3 total score	**-0.316***	0.044	-0.216*	0.169	**-0.365***	0.018

Footnotes: p-values < 0.05 are bolded.
EDI-3: Eating Disorder Inventory-3; EA: energy availability; LBM: lean body mass; EEE: exercise energy expenditure; EI: energy intake.
* = Spearman's correlations.

Table 4.6 Multiple regression analyses of unstandardized coefficients and 95% confidence intervals for associations between pQCT bone outcomes at 4% site of the tibia and eating attitudes adjusted for age, sex, and BMI.

	Total vBMD @ 4% site (mg/cm^3)		**Trabecular vBMD @ 4% site (mg/cm^3)**		**Trabecular area @ 4% site (cm^2)**	
	Unadjusted	**Adjusted**	**Unadjusted**	**Adjusted**	**Unadjusted**	**Adjusted**
	B (95% CI)	**B (95% CI)**	**B (95% CI)**	**B (95% CI)**	**B (95% CI)**	**B (95% CI)**
EA (kcal/kg LBM/day)	-0.607 (-3.647, 2.432)	-1.487 (-4.626, 1.652)	0.312 (-0.677, 1.301)	0.351 (-0.602, 1.303)	**-2.095 (-4.043, 0.148)**	-1.244 (-2.974, 0.486)
EEE (kcal/day)	0.031 (-0.114, 0.176)	0.025 (-0.129, 0.179)	0.023 (-0.28, 0.74)	0.016 (-0.36, 0.068)	0.096 (-0.005, 0.197)	0.03 (-0.63, 0.122)
EI (kcal/day)	0.005 (-0.043, 0.054)	-0.003 (-0.055, 0.049)	**0.015 (0.000, 0.030)**	0.012 (-0.003, -0.183)	0.021 (-0.11, 0.053)	-0.001 (-0.29, 0.027)
EDI-3 Drive for thinness score	-0.490 (-7.159, 6.178)	1.150 (-5.883, 8.184)	0.730 (-1.338, 2.848)	0.743 (-1.363, 2.848)	0.028 (-4.333, 4.389)	-1.455 (0.432, -5.170)
EDI-3 Bulimia score	-6.066 (-29.092, 16.960)	-10.621 (-35.008, 13.767)	5.044 (-0.107, 10.195)	2.829 (-2.559, 8.217)	8.027 (-2.726, 18.779)	6.240 (-3.194, 15.674)
EDI-3 Body dissatisfaction score	-1.820 (-8.945, 5.304)	0.351 (-7.132, 7.834)	0.163 (-2.134, 2.461)	0.554 (-1.689, 2.797)	-2.280 (-6.922, 2.362)	-2.237 (-6.140, 1.666)
EDI-3 total score	0.652 (-5.187, 6.491)	-1.172 (-6.445, 4.101)	0.480 (-0.658, 1.618)	0.477 (-0.649, 1.603)	-0.200 (-2.549, 2.150)	-0.720 (-2.716, 1.275)

Footnotes: p-values < 0.05 are bolded.

B: unstandardized regression coefficient; CI: confidence intervals; pQCT: peripheral quantitative computed tomography; BMI: body mass index; vBMD: volumetric bone mineral density; EA: energy availability, LBM: lean body mass; EEE: exercise energy expenditure; EI: energy intake; EDI-3: Eating Disorder Inventory – 3.

* = p-values < 0.001

Table 4.7 Multiple regression analyses of unstandardized coefficients and 95% confidence intervals for associations between pQCT bone outcomes at 38% and 66% site at the tibia and eating attitudes adjusted for age, sex, and BMI.

	Cortical area @ 38% site (cm^2)		**SSI @ 38% site (mm^3)**		**Cortical area @ 66% site (cm^2)**		**SSI @ 66% site (mm^3)**	
	Unadjusted	**Adjusted**	**Unadjusted**	**Adjusted**	**Unadjusted**	**Adjusted**	**Unadjusted**	**Adjusted**
	B (95% CI)	**B (95% CI)**	**B (95% CI)**	**B (95% CI)**	**B (95% CI)**	**B (95% CI)**	**B (95% CI)**	**B (95% CI)**
EA (kcal/kg LBM/day)	-1.094 (-2.389, 0.201)	-0.298 (-1.337, 0.742)	**-11.945 (-22.823, 1.066)**	-5.628 (-14.014, 2.757)	-0.942 (-2.278, 0.394)	-0.186 (-1.180, 0.808)	-17.446 (-37.857, 2.966)	-3.648 (-17.307, 10.010)
EEE (kcal/day)	**0.092 (0.026, 0.157)**	**0.054 (0.001, 0.107)**	**0.694 (0.122, 1.265)**	0.242 (-0.217, 0.701)	**0.097 (0.031, 0.162)**	0.046 (-0.006, 0.099)	1.033 (-0.034, 2.100)	0.085 (-0.655, 0.824)
EI (kcal/day)	**0.026 (0.006, 0.046)**	0.011 (-0.005, 0.028)	**0.193 (0.015-0.370)**	0.051 (-0.088, 0.191)	**0.029 (0.009, 0.050)**	0.013 (-0.003, 0.029)	**0.395 (-1.107-0.285)**	0.114 (-0.106-0.334)
EDI-3 Drive for thinness score	2.151 (-0.725, 5.027)	0.584 (0.601, -1.661)	22.377 (-1.879, 46.633)	12.816 (-5.611, 31.242)	2.038 (-0.890, 4.966)	0.967 (-1.229, 3.162)	35.480 (-8. 818, 80.498)	15.630 (-13.986, 45.246)
EDI-3 Bulimia score	5.582 (-1.709, 12.872)	3.553 (-2.134, 9.239)	55.917 (-5.748, 117.581)	51.960 (6.433, 97.487)	8.058 (0.907, 15.209)	6.967 (1.758, 12.175)	69.215 (-45.801, 184.230)	49.807 (-25.973, 125.586)
EDI-3 Body dissatisfaction score	0.219 (-2.973, 3.411)	-0.318 (-2.707, 2.070)	-0.597 (-27.922, 26.729)	-1.810 (-21. 904, 18.283)	0.536 (-2.698, 3.769),	0.661 (-1.685, 3.006)	-5.398 (-55.160, 44.363)	-7.984 (-39.807, 23.838)
EDI-3 total score	0.931 (-0.635, 2.497)	0.240 (-0.966, 1.446)	8.870 (-4.475, 22.214)	5.468 (-4.518, 15.454)	1.089 (-0.490, 2.668)	0.748 (-0.415, 1.911)	12.179 (-12.386, 36.745)	4.607 (-11.458, 20.673)

Footnotes: p-values < 0.05 are bolded.

B: unstandardized regression coefficient; CI: confidence intervals; BMI: body mass index; pQCT: peripheral quantitative computed tomography; EA: energy availability, LBM: lean body mass; EEE: exercise energy expenditure; EI: energy intake; EDI-3: Eating Disorder Inventory – 3; SSI: stress strain index.

* = p-values < 0.001

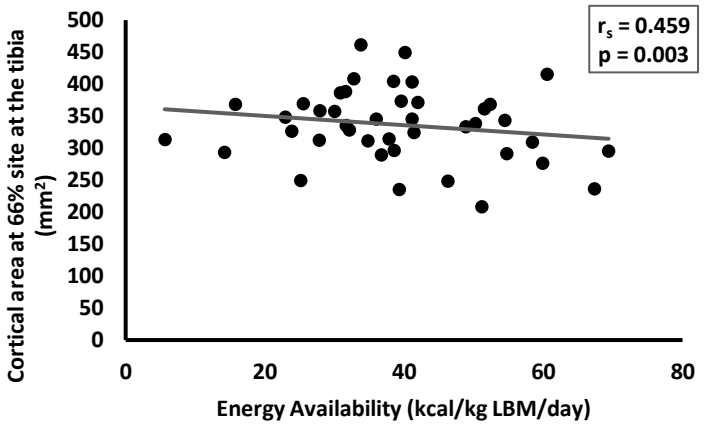
Table 4.8. Multiple regression analyses of coefficients and 95% confidence intervals for associations between EA and eating attitudes adjusted for age, sex, and BMI.

	EA (kcal/kg LBM/day)		**EEE (kcal/day)**		**EI (kcal/day)**	
	Unadjusted	**Adjusted**	**Unadjusted**	**Adjusted**	**Unadjusted**	**Adjusted**
	B (95% CI)	**B (95% CI)**	**B (95% CI)**	**B (95% CI)**	**B (95% CI)**	**B (95% CI)**
EDI-3 Drive for thinness score	-0.639 (-1.310, 0.032)	-0.411 (-1.107, 0.285)	-6.491 (-20.876, 7.894)	-10.105 (-24.843, 4.634)	-30.055 (-71.984, 11.874)	-38.437 (-80.250, 3.376)
EDI-3 Bulimia score	-0.476 (-2.240, 1.288)	-0.581 (-2.410, 1.248)	5.694 (-31.140, 42.528)	8.384 (-30.711, 47.479)	4.199 (-104.941, 113.340)	-9.654 (-122.977, 103.669)
EDI-3 Body dissatisfaction score	-0.484 (-1.208, 0.241)	-0.289 (-1.038, 0.460)	-10.366 (-25.436, 4.703)	-11.126 (-26.606, 4.355)	**-48.869 (-91.782, -5.956)**	-46.492 (-89.851, -3.133)
EDI-3 total score	-0.326 (-0.684, 0.33)	-0.216 (-0.588, 0.157)	-4.236 (-11.892, 3.420)	-5.376 (-13.240, 2.487)	-20.821 (-42.849, 1.206)	**-23.281 (-45.281, -1.280)**

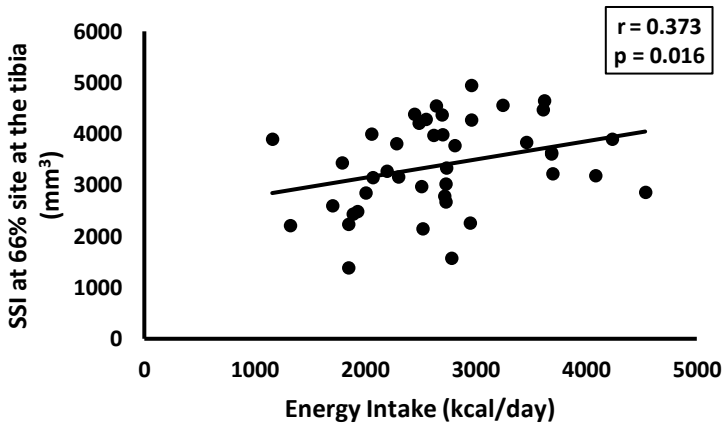
Footnotes: p-values < 0.05 are bolded.

B: unstandardized regression coefficient; CI: confidence intervals; BMI: body mass index; EA: energy availability, EEE: exercise energy expenditure; EI: energy intake; EDI-3: Eating Disorder Inventory – 3; LBM: lean body mass.

A



B



C

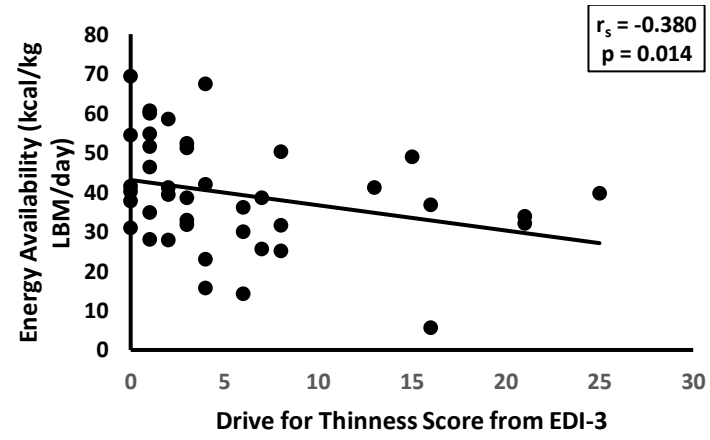


Figure 1. Scatterplots displaying the correlation between A) energy availability (kcal/kg LBM/day) and cortical area at 66% site at the tibia (mm^2), B) energy intake (kcal/day) and SSI at 66% site at the tibia (mm^3) and C) drive for thinness score from EDI-3 and EA (kcal/kg LBM/day). SSI: stress-strain index; EDI-3: Eating Disorder Inventory – 3; LBM: lean body mass.

Chapter 5: Discussion

5.1 Summary of the Findings

This exploratory, observational study evaluated the associations between EA, eating attitudes and bone strength in young endurance-trained individuals. Our findings demonstrated no major or statistically significant associations between EA and pQCT-derived bone outcomes at the tibia in this subset of athletes often at a higher risk of bone fragility due to their high training volume and elevated nutritional demands. While we observed weak positive correlations between EA and cortical area at the proximal tibia, EA was negatively correlated with trabecular area at the distal tibia and SSI at the mid-tibial shaft. These results are not entirely surprising considering the relatively low percentage of individuals with markers of chronic energy deficiency (e.g., low BMI, FHA, history of DE/ED) in our sample as well as the methodological challenges of assessing EA using field-based measures of EI and EEE, wherein substantial inter-day variability may be observed. Our study did not measure certain surrogate markers of low EA (i.e., metabolic/reproductive hormone levels and REE/pREE), which may have better correlated with pQCT estimates of bone. Apart from acute and rigorous laboratory-based experimental studies wherein low EA/energy deficiency is induced, there is no conclusive evidence to support that reduced EA leads to deteriorations in clinically relevant bone health endpoints in athletes (Burke et al., 2018). Experimental studies with a longer duration assessment of EA in athletes with evidence of chronic energy deficiency are needed to determine the direction and strength of the association between EA and pQCT measures of bone strength. As expected, higher drive for thinness and EDI-3 total scores were associated with lower EA. However, no associations were observed between other DE attitudes (body dissatisfaction, bulimia, drive for thinness) and pQCT measures of bone strength (Cobb et al., 2003). Notably, most of these associations

between EA, eating attitudes, and bone parameters were no longer significant after adjusting for age, sex, and BMI, suggesting the potential value in examining these links in a larger sample stratified by these variables. Overall, our study offers a unique perspective on the link between field-based EA measures and advanced, three-dimensional estimates of bone strength, which have a stronger influence on BSI risk.

5.2 Associations between EA and Bone Health

The detrimental effects of energy restriction and/or high training volume on bone health are well established. Prior research in female military recruits and long-distance runners has shown that cortical bone variables measured using pQCT at the tibia were able to significantly predict BSI (Koltun et al., 2020; Popp et al., 2009, 2020). In our study wherein we assessed EA in free-living, endurance-trained individuals, we found modest-to-no associations between EA and pQCT variables except cortical area at the proximal tibia, which displayed positive correlations with EA. Our findings differ from a previous cross-sectional study which compared pQCT bone outcomes in 60 young exercising women grouped as energy deficient and energy replete (Southmayd et al., 2017). Individuals who had an mREE/pREE ratio of $\geq$0.9 were classified as energy replete and a ratio of < 0.9 were classified as energy deficient. Southmayd et al. (2017) demonstrated that energy deficient women had lower trabecular vBMD at the proximal tibia, and lower total vBMD and cortical area at the distal and proximal tibia compared to the energy replete group. Ackerman et al. (2011) compared bone outcomes measured by HR-pQCT in 50 female athletes and non-athletes classified by current self-reported menstrual disturbances (often an indicator of chronic energy deficiency). Unlike our study, Ackerman et al. (2011) reported lower cortical area and thickness, and trabecular number at the distal tibia in amenorrheic athletes compared to eumenorrheic athletes. Alternatively, Heikura et al. (2021)

demonstrated no differences in metabolic hormones (IGF-1, TT_3, insulin), BSI prevalence, total body aBMD, and lumbar spine, right and left femur aBMD Z-scores between male and female distance runners with low EA versus moderate EA using a similar field-based EA measures as the current study. Interestingly, when their sample was stratified by self-reported menstrual status, females with amenorrhea exhibited lower total body aBMD than their eumenorrheic counterparts. Further, both amenorrheic females and males with low testosterone exhibited lower TT_3 and a 4.5 times higher risk of BSIs. Similar to our findings, Melin et al. (2015) found no differences in EA between individuals with normal and impaired bone health, where impaired bone health was defined as having an aBMD Z-score of -1 or lower at one or more of the measured sites (whole body, lumbar spine, and hip). Previous evidence displays considerable heterogeneity in the methods used to measure EA/energy status. Our findings suggest that studies which used surrogate markers of EA including menstrual status, metabolic hormones, and REE/pREE may be better able to identify individuals with bone fragility (including low aBMD, BSI risk) compared to using field-based EA measures. Additionally, our study did not specifically target individuals with chronic energy deficiency, which was evident from the very few individuals who exhibited Triad risk factors such as low BMI, history of BSIs, and hypogonadism. Future prospective research exploring EA and bone strength should target these higher-risk individuals to better understand energy deficiency-related bone loss and BSI incidence.

It is also worth mentioning the methodological challenges in estimating EA in the field. The main components of EA (both EI and EEE) are highly variable from day to day, making it difficult to accurately identify "true" energy status. Our study measured EA over a 7-day period, which may not be representative of their long-term energy status. We measured EI using 24-hour

dietary recall for three days, which is prone to measurement error due to the lack of knowledge of portion sizes, recall bias, and tendency to under-report foods perceived as unhealthy. Underreporting is one of the most prevalent measurement errors involved in dietary recalls (Black et al., 1997). A previous meta-analysis of 18 studies which included more than one assessment of dietary intake and a measure of EE in athletes has shown an average of 19% underreporting of self-reported EI (0.4%-36%), showing the extent of error that may be involved in these measurements (Capling et al., 2017). While we did not identify implausible reporters in our study, there are several methods to assess the plausibility of self-reported EI. The Goldberg method is a commonly used equation which compares the ratio between self-reported EI and resting metabolic rate to the physical activity level of the participant (Goldberg et al., 1991). Similarly, the estimation of EEE is prone to significant error. In our study, we determined the relationship between heart rate and VO_2 for each individual to convert their second-to-second HR during the exercise sessions to EE estimates, a method that is thought to be superior than measuring EE using accelerometers, METs, or fitness watches (Ceesay et al., 1989; Leonard, 2003; Spurr et al., 1988). However, EEE also fluctuates daily, making it difficult to accurately identify longer-term EEE from a seven-day calculation. Future experimental studies should investigate the associations between EA and bone outcomes using a longer-term assessment of energy status in endurance-trained individuals.

5.3 Associations between Eating Attitudes and EA

DE is one of the pathways that contributes to the development of inadequate EA and has been widely used as a surrogate marker to indirectly determine energy status and other Triad-related outcomes, including menstrual disturbances, low aBMD, and risk of BSI (De Souza et al., 2007, 2014). Specifically, drive for thinness score from the EDI-3 has been studied as a key

correlate of energy deficiency and subsequent Triad/RED-S risk (Gibbs et al., 2011) (De Souza et al., 2007; Garner, 2004; Sundgot-Borgen, 1994). In our study, drive for thinness and total EDI-3 scores were negatively associated with EA, while these associations did not remain significant when adjusted for age, sex, and BMI. This is consistent with the large body of literature displaying a higher prevalence of DE defined as elevated drive for thinness and body dissatisfaction scores in females with menstrual disturbances, and negative correlations between each of drive for thinness and body dissatisfaction and low EA/energy deficiency (Cobb et al., 2003; Gibbs et al., 2011; Reed et al., 2013). In a cross-sectional study in 117 young exercising women, Gibbs et al. (2013) displayed that individuals with higher drive for thinness had a greater frequency of severe menstrual disturbances (oligoamenorrhea, FHA) corroborated by urinary assays of estrogen and progesterone metabolites. High drive for thinness was also associated with a lower mREE/pREE (a widely used surrogate indicator of energy deficiency). Similarly, Cobb et al. (2003) found a positive association between EDI total score and frequency of self-reported menstrual disturbances in young female distance runners. Collectively, these findings suggest that DE attitudes identified from psychometric questionnaires (i.e., EDI-3) may serve as a useful screening tool when identifying athletes and physically active individuals at risk of low EA. The negative associations between each of drive for thinness and total EDI-3 score and EA also aligns with findings from a study in 19 young female soccer players investigating eating attitudes and EA using field-based measurements, which demonstrated that lower EA was moderately associated with higher drive for thinness and body dissatisfaction during the mid-season (Reed et al., 2015). Therefore, drive for thinness and body dissatisfaction scores from EDI-3 and total EDI-3 score may represent surrogate markers of energy deficiency/low EA in active individuals at higher risk of the Triad/RED-S outcomes, including low aBMD and BSIs. It

is important to clarify that the current study is of a hypothesis-generating nature focused on secondary objectives. Future prospective studies with a larger sample size are needed to examine the theoretical basis of the association between DE attitudes and EA in endurance-trained individuals, and determine whether DE predicts low aBMD/BSIs independent of EA.

5.4 Associations between Eating Attitudes and Bone Health

DE attitudes were not correlated with pQCT measures of bone strength. In a previous study in 91 female distance runners, Cobb et al. (2003) found a lower aBMD at the lumbar spine in individuals with higher total EDI scores. Prior research in young exercising females also suggests that individuals with elevated shape concern from the EDE-Q was associated with a 4.2-fold higher musculoskeletal injury risk. Although lacking significance, elevated dietary restraint score or global score from the EDE-Q (≥4) were associated with a 7-fold increased risk for musculoskeletal injuries than those with normal EDE-Q scores (Holtzman et al., 2022; Rauh et al., 2010). Additionally, in a 5-year prospective study in 211 male and female runners, Nattiv et al. (2013) showed that athletes with a history of a bone injury at skeletal sites that are predominantly made of trabecular bone (femoral neck, sacrum, pubic bone) were more likely to report a history of DE (classified through a multidisciplinary assessment using the DSM-4 criteria (Bell, 1994). Similarly, a cross-sectional study by Holtzman et al. (2022) in 127 female athletes showed higher rates of self-reported DE/ED, assessed by Eating Disorder in Athletes Questionnaire or the Eating Disorder Screen for Primary Care, among individuals who had BSI to high risk (sacrum, pelvis, femoral neck, hip) bones versus low-risk (other) bones. Further, in a cross-sectional study in 51 young female runners, Gehman et al. (2022) examined whether recurring BSI history is associated with Triad risk factors including bone strength at the tibia measured by pQCT and DE attitudes using EDE-Q and history of restrictive eating periods.

Female runners with multiple BSIs displayed higher total EDEQ score and shape and weight concern scores, and greater tendency for restrictive eating than the control group with one or no BSIs, while there was no difference in pQCT outcomes between the multiple BSI and control groups. Notably different from our study, Gehman et al. (2022) did not evaluate the independent associations between DE and pQCT outcomes. The lack of associations between DE attitudes by EDI-3 and pQCT measures of bone strength in the current study may be attributed to the lower number of participants with elevated scores on EDI-3 subscales. As previously mentioned, athletes who may be at higher risk of bone fragility were likely excluded from this study which led to a healthier cohort, as evidenced by the low prevalence of individuals (11.6%) with a history of BSIs in our sample. To our knowledge, few studies have examined the independent associations between eating attitudes and bone variables using pQCT measures of bone strength in young endurance athletes, suggesting the unique insight our study may bring to prevent these deteriorations in bone health and the occurrence of BSIs (Popp et al., 2009). Further investigation in a larger sample is needed to fully understand the influence of ED on pQCT bone outcomes in endurance athletes at higher risk of chronic energy deficiency and related bone fragility.

5.5 Strengths and Limitations

The current study is not free from limitations. This study focused on secondary objectives from a larger observational project that primarily is investigating the relationship between muscle and bone strength in endurance-trained individuals. The sample size calculations were conducted to observe associations between muscle and bone outcomes, rather than specifically testing the relationship between EA and pQCT measures of bone strength. Although the recruitment for the present study is ongoing, the results reported herein are preliminary with an

inadequate sample size for identifying the independent associations between EA, eating attitudes and bone strength in this group. Additionally, the unequal number of male (n=31) and female (n=12) participants may compromise the strength and reliability of statistical analyses when describing comparisons between the sexes. Further, there were logistical challenges with scheduling female participants during the early follicular phase, resulting in variations in menstrual phase among female participants on the testing day. By increasing the number of females in our sample for the final dataset, we will improve our ability to examine the influence of menstrual history within the multivariable regression models. In addition to the limitations of field-based EA calculations mentioned in Section **4.2**, it is worth mentioning that our EA calculations do not account for EE from normal daily activities that are outside the planned training sessions (also known as non-exercise activity thermogenesis (NEAT)). The four components of total EE include REE, diet-induced thermogenesis, NEAT, and EEE. Ideally, all types of physical activity (i.e., the sum of EEE and NEAT) should be considered to yield true energy status (Chung et al., 2018).

There were several strengths of our study. This study recruited both male and female endurance-trained individuals. Unlike the historical underrepresentation of females in the broad field of sports science, most of the research studies investigating EA and Triad/RED-S conditions have predominantly focused on female athletes (Loucks et al., 1998). The inclusion of males in this study contribute to our understanding of the unique relationships between EA, eating attitudes and bone strength in male athletes. Our study used pQCT to estimate bone strength which is a measure of "true" bone density versus the measure of aBMD obtained from DXA. Lastly, our use of field-based calculations of EI and EEE serve as more ecologically valid

methods of measuring EA in a free-living individual, which is thought to better reflect the complexity and dynamics of the real-world contexts.

5.6 Conclusion

In conclusion, our results demonstrated no major or statistically significant associations between field-based EA calculations and pQCT measures of bone strength in young male and female endurance-trained individuals. The lack of associations may be partly explained by the methodological challenges associated with EA calculations and low percentage of participants who exhibit chronic energy deficiency and bone fragility (low aBMD, BSI) in our sample. Future experimental studies with longer-term assessments of EA may provide a better understanding of these associations between EA and bone strength, given the high inter-day variability in the EA components. Unlike surrogate measures of EA/energy deficiency, the field-based EA assessment may not serve as a sensitive approach to detect Triad-related decrements in bone strength. However, considering the sample size and hypothesis-generating nature of this study, we are unable to make definitive inferences based on these findings. Future studies should target individuals at higher risk of chronic energy deficiency and bone fragility to advance our knowledge regarding the energetic/nutritional mechanisms underlying bone loss and BSIs in endurance athletes. While drive for thinness and EDI-3 total scores demonstrated negative associations with EA, no statistically significant associations were observed between any of the other DE outcomes from the EDI-3 (body dissatisfaction, bulimia) and bone parameters. Almost none of the associations between EA, eating attitudes, and bone strength remained significant after adjusting for age, sex, and BMI, suggesting future stratified analysis in a larger sample may be valuable. Overall, this preliminary analysis from the present study provides new knowledge on the physiological and behavioural determinants of bone strength at the tibia which may serve

as pilot data for future exercise and nutrition interventions to improve bone strength and BSI risk in endurance athletes.

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