LOW ENERGY AVAILABILITY RISK IN FEMALE TRIATHLETES

Anna Brassell

A thesis submitted to the University of Sunderland in partial fulfilment of the requirements for the Degree of Doctor of Philosophy

May 2022

ABSTRACT

Background: The highly complex syndromes of the Female Athlete Triad (Triad) and Relative Energy Deficiency in Sport (RED-S) are underpinned by low energy availability (LEA). LEA has been defined as the imbalance between dietary energy intake (EI) and exercise energy expenditure (EEE), leading to inadequate energy available to optimally support physiological function and health. Chronic LEA, with or without disordered eating (DE) or eating disorder (ED), has been associated with direct and indirect links to the development of menstrual disturbances and impaired bone health. It has also been proposed that chronic LEA may result in impairments to several other health (i.e., cardiovascular, endocrine) and performance (i.e., muscle strength, cognitive function), factors, however limited evidence exists to support these findings. Endurance sports athletes have been reported to be at an increased risk of developing LEA and the subsequent health and performance impairments due to high daily EEE. High daily EEE may arise from high training volumes and/or an increased risk of DE/ED, and/or an increased risk of exercise dependence (EXD). Although it has been accepted endurance athletes may be at an increased risk of developing LEA, to date limited data exists in female athletes particularly from multi-sport endurance athletes.

Aim: The main purpose of this thesis was to investigate the prevalence of risk of LEA and associated risk factors (i.e., DE, ED, and EXD) in female triathletes.

Methods: To assess the prevalence of risk in Studies 1-3, a cross-sectional design using an anonymised online questionnaire was used. The questionnaire was constructed on established, validated, and reliable screening tools which included the LEAF-Q, FAST, and EDS-R. In Studies 1-3, participants were recruited using voluntary response sampling. Study 1 included 393 female triathletes (age: 36 (13) years; height: 1.66 (0.09) m; body mass: 64.0 (12.0) kg; BMI: 23.0 (4.3) $kg \cdot m^2$).

Study 2 included a total sample size of 393 female triathletes which were divided into three age groups: 18-29 years (n=101; height: 1.67 (0.08) m; body mass: 63.0 (9.5) kg; BMI: 22.6 (3.3) $kg \cdot m^2$), 30-39 years (n=159; height: 1.65 (0.08) m; body mass: 65.0 (12.0) kg; BMI: 23.3 (5.0) $kg \cdot m^2$), and 40-49 years (n=133; height: 1.67 (0.09) m; body mass: 23.2 (4.3) kg; BMI: 23.2 (4.3) $kg \cdot m^2$).

Study 3 included a total sample size of 383 female triathletes which were divided into two performance level groups: self-identified recreational age-groupers (n=293; age: 37 (12) years; height: 1.65 (0.08) m; body mass: 65.0 (12.0) kg; BMI: 23.5 (4.4) $kg \cdot m^2$, training time: 10.7 (5.5) h·week), and self-identified top-percentile age-groupers (n=90; age: 32 (13) years; height: 1.67 (0.09) m; body mass: 62.0 (11.0) kg; BMI: 21.6 (2.8) $kg \cdot m^2$, training time: 13.0 (7.0) h·week).

Study 4 was a longitudinal design with 10 female triathletes (age: 27.7 ± 8.6 years; height: 1.67 ± 0.04 m; body mass: 62.2 ± 3.2 kg; BMI: 22.6 ± 1.3 kg·m^2) were recruited using convenience sampling at local triathlon clubs. EA and eating attitudes were assessed every two-months throughout the season to assess changes. EA was assessed using both direct measures (i.e., lean body mass (LBM), EI, and EEE) and self-report screening tools (LEAF-Q). Eating attitudes was assessed using a self-report screening tool (FAST).

Results: In Study 1, it was shown that 42% of female triathletes, aged 18-54 years, were classified as at risk of LEA by the LEAF-Q. The FAST identified 25% with DE and 9% with ED symptoms, and 9% were at risk of EXD by the EDS-R. Eating attitudes and exercise behaviour were significant predictors of LEA and exercise behaviour was a significant predictor of eating attitudes. Participants with no ED had 3.375 times higher odds of being low risk of low EA than those with DE/ED. Similarly, participants not at risk of EXD had 2.489 times higher odds of being low risk of low EA than those at risk of EXD. Participants considered not at risk of EXD had 3.110 times higher odds of not having DE/ED than those at risk EXD.

In Study 2, it was shown that the prevalence of those considered at risk of LEA was significantly higher in younger participants aged 18-29 years compared to older participants aged 40-49 years ($p = .010$, $\varepsilon^2 = .023$). 49% of participants aged 18-29 years were considered at risk of LEA compared to 40% of those aged 30-39 years and 39% of those aged 40-49 years. No significant differences were observed between age groups for eating attitudes ($p = .070$). It was shown that prevalence of those considered at risk of DE and ED was 28% and 12%, respectively in those aged 18-29 years. In comparison, 24% and 10% of those aged 30-39 years were at risk of DE and ED, and 24% and 5% of those aged 40-49 years. It was shown that the prevalence of those considered at risk of EXD was significantly higher in younger participants aged 18-29 years compared to older participants aged 40-49 years ($p = <.001$, $\varepsilon^2 = .048$). 16% of participants aged 18-29 years were considered at risk of EXD in comparison to 6% of those aged 30-39 years and 40-49 years. Finally, younger participants were more likely to be categorised with maladaptive patterns of exercise than older participants ($p = .001$). Participants aged 18-29 years had 2.8 times higher odds of being symptomatic and 5.8 times higher odds of being at risk of EXD than their older counterparts.

In Study 3, no significant differences were observed between performance levels for LEA risk ($p = .083$) or DE/ED risk ($p = .990$). 47% of participants who identified as a top-percentile age-grouper were considered at risk of LEA compared to 39% of those who identified as a recreational age-grouper. It was shown that prevalence of those considered at risk of DE and ED was 30% and 9%, respectively in top-percentile age-groupers. In comparison, 24% and 9% of recreational age-groupers were at risk of DE and ED. Significant differences were observed between performance levels ($p = .023$) for EXD, with recreational age-groupers less likely to display maladaptive patterns of exercise than top-percentile

age-groupers (p = .017). Participants who were recreational age-gropers were 0.454 times less likely to be classified as symptomatic than top-percentile age-groupers and 0.489 times less likely to be classified as at-risk of EXD. Finally, 10% of top-percentile age-groupers were considered at risk of EXD compared to 8% of recreational age-groupers.

In Study 4, a single cohort of female triathletes were examined for changes in EA and eating attitudes across a full triathlon season. Overall prevalence rates (37%) of LEA were comparative to earlier findings in Study 1 (42%) and Study 6 (39%). No significant changes were observed across the season in measured EA (p = .591) or eating attitudes (p = .524). Statistically significant differences in LEA risk identified by the LEAF-Q were detected across the season (p = .011, $n^2_p = 0.274$). However, it was observed that for some, but not all, certain phases of the season may contribute to overall LEA risk.

Conclusion: Female triathletes are an athletic population at increased risk of developing LEA that may be underpinned by DE behaviour and/or EXD. Although younger athletes are considered at greater risk than their older counterparts, the risk still exists in significant proportions in older female triathletes. Similar findings existed between performance levels with a tendency for higher prevalence's as performance level improved. Despite no significant differences being observed in EA and eating attitudes across the season, the prevalence of subclinical LEA was high for the duration of the triathlon season. It demonstrated that risk factors for the development of LEA are individual to the athlete. Such findings have advanced our understanding and will facilitate identification and early detection and target educational resources to at-risk groups.

ACKNOWLEDGEMENTS

Firstly, this journey would not have been possible without my family (Steven, Maureen, Katie, Meg & Lottie) who have invested so much in supporting my education. I would like to thank the team of supervisors and mentors I have been fortunate enough to work with throughout my academic studies at the University of Sunderland; Dr Elisabeth (Lisa) Board, Dr David Archer and Dr Robert Hogg. I am grateful for your collective knowledge, individual expertise, guidance, and support over the last ten years. Thank you to the technical staff (Abbie, Shane & Jack) who's contribution to research/academia is too often overlooked. I especially want to extend my thanks and gratitude to Abbie Taylor. What started off as a professional working relationship became a friendship that I have relied on over the last three years. Thank you for your assistance with testing and data collection which was without a doubt the most time demanding and challenging part of this journey.

Thank you to all the participants who willingly gave their time and effort to take part in these studies. I am truly indebted to those participants who took part in the year-long study. Your support, encouragement, and belief in me throughout the most demanding part of this journey is truly appreciated.

Lastly, a note for my future self. This journey led you to the darkness, took away your peace, brought you unimaginable pain, and trapped you in a body that was trying to survive with a mind that simply was not. You struggled. You fought. You survived. You stayed. Academia is not worth your life. The carousel never stops turning. For now, it is time to let go, make the jump, dust off your hands and knees, and be free. Go find the sun and the place where your shine will not be stolen. But remember, you always had fire in your belly, your struggles, insecurity, and doubt were not weakness.

“Some nights, the wolf inside of me shrinks to nothing, she bares her teeth and runs away. The dragon in my chest rejects me, she is so tired of being slain. There are nights when the lioness cowers, says she can’t fight it another day…”

“What about the phoenix?”

“She sits with me in the darkness. She whispers *we will rise. Just you wait*.”

- SRWpoetry

TABLE OF CONTENTS

DECLARATION

I declare that the content presented in this thesis is original work, conducted and written by the author, and has not been submitted previously for another academic degree at any institution. This thesis is being submitted in partial fulfilment of the requirements for the Degree of Doctor of Philosophy.

LIST OF FIGURES

LIST OF TABLES

LIST OF ABBREVIATIONS

American College of Sports Medicine (ACSM)

Anorexia nervosa (AN)

Basal metabolic rate (BMR)

Binary logistic regression (BLR)

Binge eating disorder (BED)

Bone mineral density (BMD)

Bulimia nervosa (BN)

Carboxy-terminal propeptide of type 1 procollagen (P1CP)

Clinical eating disorder (ED)

C-telopeptide (CTx)

Diagnostic and Statistical Manual of Mental Disorders (DSM)

Dietary induced thermogenesis (DIT)

Disordered eating (DE)

Doubly labelled water (DLW)

Dual-energy X-ray absorptiometry (DXA)

Eating Attitudes Test (EAT)

Eating Disorder Examination (EDE)

Eating Disorder Examination questionnaire (EDE-Q)

Eating Disorder Inventory (EDI)

Eating disorder not otherwise specified (EDNOS)

Energy availability (EA)

Energy balance (EB)

Energy intake (EI)

Exercise Addiction Inventory (EAI)

Exercise Beliefs Questionnaire (EBQ)

Exercise dependence (EXD)

Exercise Dependence Questionnaire (EDQ)

Exercise Dependence Scale (EDS; revised: EDS-R)

Exercise energy expenditure (EEE)

Female Athlete Triad (Triad)

Female Athletes Screening Tool (FAST)

Follicle stimulating hormone (FSH)

Functional hypothalamic amenorrhea (FHA)

Gonadotropin-releasing hormone (GnRH)

Graded exercise test (GXT)

Heart rate (HR)

Hypothalamic-pituitary-gonadal axis (HPG axis)

Insulin-like growth factor-1 (IGF-1)

Interleukin-6 (IL-6)

International Olympic Committee (IOC)

International Triathlon Union (ITU)

Lactate threshold - onset of blood lactate accumulation (LT_{OBLA})

Lean body mass (LBM)

Low Energy Availability (LEA)

Low Energy Availability in Females Questionnaire (LEAF-Q)

Luteal phase defects (LPD)

Luteinising hormone (LH)

Maximal aerobic capacity ($\dot{V}O_{2max}$)

Menstrual dysfunction (MD)

Metabolic equivalent of tasks (METs)

Multinomial logistic regression (MLR)

Negative Addiction Scale (NAS)

Non-dependent-asymptomatic (AS)

Non-dependent-symptomatic (SY)

Non-exercise activity thermogenesis (NEAT)

N-terminal telopeptide (NTx)

Obligatory Exercise Questionnaire (OEQ)

Other specified feeding and eating disorders (OFSED)

Oestrogen (E_2)

Oxygen (O_2)

Oxygen consumption ($\dot{V}O_2$)

Peak bone mass (PBM)

Polycystic ovarian syndrome (PCOS)

Procollagen type 1 amino-terminal propeptide (P1NP)

RED-S clinical assessment tool (RED-S CAT)

Relative Energy Deficiency in Sport (RED-S)

Respiratory exchange ratio (RER)

Resting energy expenditure (RMR)

Resting metabolic rate (RMR)

Running Addiction Scale (RAS)

Thermic effect of food (TEF)

Three Factor Eating Questionnaire (TFEQ)

Thyroxine (T4)

Total energy expenditure (TEE)

Triiodothyronine (T3)

CHAPTER 1

GENERAL INTRODUCTION

The Olympic Movement Medical Code (2019) and the International Olympic Committee (IOC, Olympic Charter, 2019) have emphasised the importance of protecting the health of the athlete. When viewed as an integrated performance system, athlete health is multifaceted and forms part of an overarching system that impacts the function of other inter-related sub-systems, influencing outcomes of success and failure (Mooney, Charlton, Soltanzadeh & Drew, 2017). Across various sports, consensus exists regarding the importance of maintaining adequate energy and nutrient intake, as the foundation of optimal athletic health and performance (Otis, Drinkwater & Johnson, 1997; Nattiv, Loucks & Manore, et al., 2007; De Souza, Nattiv, Joy & Misra, et al., 2014; Mountjoy, Sundgot-Borgen, Burke & Carter, et al., 2014; 2018).

Currently findings from studies using single-sport endurance athletes (e.g., distance runners) are often generalised and applied to multi-sport endurance athletes (e.g., triathletes). This is despite the demands of training and competition being significantly different (Etxebarria, Mujika & Pyne, 2019). As a result, studies examining prevalence rates and the negative consequences of LEA in female athletes from multi-sport endurance events are limited. Additionally, findings from previous studies often have variable sample sizes (range 10 to 833; Schaal, et al., 2011a; Logue, et al., 2019). Based on a population size of the 10,000 registered female triathletes with British Triathlon (British Triathlon, 2021a), a sample size estimation of 370 was calculated with a confidence level of 95% and a 5% margin of error (Qualtrics, London, UK). However, calculations of sample size and power analyses should be interpreted with caution as they are estimates and variables may be manipulated (i.e., larger effect size; Prajapati, Dunne & Armstrong, 2010; Meyvis & Van Osselaer, 2018). Since leanness-sports are thought to be a major risk factor for the development of LEA, the implications are clear that female participation in triathlon may be associated with an increase in the prevalence of risk and/or incidence of LEA (Mountjoy, et al., 2014; 2018). LEA is the

aetiological factor underpinning the highly complex syndromes of Triad and RED-S. These syndromes are considered to be one of the most serious medical conditions in the female athlete (Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). LEA refers to the mismatch between EI and EEE, resulting in inadequate energy to support physiological function and maintain optimal health and performance (Nattiv, et al., 2007). Endurance sport athletes have been reported to be at increased risk of DE or ED (Nattiv, et al., 2007; Bratland-Sanda, et al., 2013; Sundgot-Borgen, et al., 2013; Joy, et al., 2016; Mountjoy, et al., 2018). They also may present with increased daily EEE from high training volumes. As a result, there may be an increased risk of developing LEA and associated health and performance consequences (Loucks, Kiens & Wright, 2011).

Short and long-term LEA plays both a direct and indirect role in the development of menstrual disturbances and impaired bone health, even without the presence of DE/ED (De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). The inter-related clinical conditions (i.e., osteoporosis and functional hypothalamic amenorrhea (FHA)) may have irreversible consequences (De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). Current evidence suggests LEA exists across all ages (Mountjoy, et al., 2018) and there may be critical phases (i.e., growth and development during adolescence/young adulthood or the menopause in adulthood) where the development of LEA may increase the severity of associated impairments (Thein-Nissenbaum, 2013; De Souza, et al., 2014). However, limited information exists regarding the prevalence of LEA risk or influence of age across different age groups, as LEA research has predominantly focused on adolescent, University students or young adults from various sports.

Similarly, LEA is not exclusive to elite athletic populations with current evidence suggesting non-elite athletic populations are at increased risk (Torstveit, et al., 2005; Slater, et al., 2016; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). However, the influence of performance level on the prevalence of LEA is not fully established as LEA research has

predominantly focused on Western elite athletic populations De Souza, et al., 2014; Mountjoy, et al., 2018; Logue, et al., 2018; 2020). It has been proposed that as performance level improves, training load increases thus increasing EEE which increases the risk of LEA development (Nattiv, et al., 2007; Mountjoy, et al., 2014; Wasserfurth, Palmowski, Hahn & Krüger, 2020). This is an area of concern if there is an increased risk in non-elite athletic populations as they may have limited access to nutritional, training, and sport-specific medical advice and support (Slater, et al., 2016; Black, et al., 2018; Logue, et al., 2019; Wasserfurth, et al., 2020). This has placed critical emphasis on prevention and early diagnosis of at-risk groups and individuals, to avoid the more serious clinical endpoints associated with the Triad or RED-S models (Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018).

To date there is no standardised or reference protocol for EA assessment. This has led to continuing variability in methods utilised and issues regarding the reliability and validity of such metrics (Mountjoy, et al., 2014; Burke, Lundy, Fahrenholtz & Melin, 2018). Measurement of EA components have included: EI assessment by retrospective or prospective methods, EEE assessment by activity logs or by quantitative data from heart rate (HR) or accelerometers, lean body mass (LBM) can be quantified by methods of bio-electrical impedance, surface anthropometry, or dual X-ray absorptiometry (DXA), and assessment of resting metabolic rate (RMR) via indirect calorimetry or prediction equations (Mountjoy, et al., 2014; Logue, Madigan, Melin & Delahunt, et al., (2020).

Recently, increased emphasis has been placed on the importance of early detection of at-risk groups through the use of self-report screening tools (i.e., questionnaires), followed by an individual clinical assessment (Mountjoy, et al., 2014; 2018). Screening tools provide an estimation of the prevalence of athletes at risk of LEA using self-report questionnaires that screen for the physiological symptoms associated with the Triad or RED-S models (De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). Examples include the LEA in Females

Questionnaire (LEAF-Q; Melin, Tornberg, Skouby & Faber, et al., 2014), the diagnostic criteria of the Triad (Joy, De Souza, Nattiv & Misra, et al., 2014), and the RED-S clinical assessment tool (RED-S CAT; Mountjoy, Sundgot-Borgen, Burke & Carter, et al., 2015a). Alongside the recommendation for screening physiological symptoms of LEA, it is recommended they are supplemented with screening for DE behaviour and clinical ED (De Souza, et al., 2014; Mountjoy, et al., 2014; 2018) and more recently exercise dependence (EXD; Torstveit, Fahrenholtz, Lichtenstein & Stenqvist, et al., 2019; Logue, et al., 2020). There is no consensus on which screening tools have the best efficacy (Mountjoy, et al., 2018). However, their implementation has furthered our understanding of the prevalence of LEA in various sports, highlighted potential risk factors and the role of LEA on long-term health and performance in female athletes.

A body of observational and cross-sectional literature using direct EA assessments or screening tools has identified female athletes from leanness sports at greater risk of LEA, with or without DE behaviour or clinical ED (Folscher, Grant, Fletcher & van Rensberg, 2015; Melin, Tornberg, Skouby & Møller, et al., 2016; Heikura, Uusitalo, Stellingwerff & Bergland, et al., 2018a). Current prevalence rates of risk have ranged from 18% to 80% across a variety of leanness sports (Muia, Wright, Onywera & Kuria, 2016; Jesus, Castela, Silva & Branco, et al., 2021). Narrative reviews and current consensus statements have supported the higher prevalence rates are increased risk of LEA, within-day energy deficiency, and associated health and performance impairments, in female athletes across a variety of sports and performance levels (De Souza, et al., 2014; Mountjoy, et al., 2014; 2018; Logue, Madigan, Delahunt & Heinen, et al., 2018; Logue, et al., 2020). While this has provided encouraging insights into the prevalence and effects of LEA, data from individual studies is not consistent. Limited studies have examined the prevalence and associations between LEA, DE behaviour and clinical ED, and/or EXD risk, in female multi-sport endurance athletes, despite regular exposure to high

training volumes (Hoch, Stavrakos & Schimke, 2007; Mongrain, Masson, Bégin & Lamarche, 2018). Additionally, there are few reports examining differences between age groups, performance levels, and changes in risk across a competitive season in multi-sport endurance athletes (Logue, et al., 2018; 2020).

Taken together, the true extent of the prevalence of and associations between LEA, DE behaviour or clinical ED, and EXD risk in female multi-sport endurance athletes is not entirely clear. Neither is it fully understood if differences exist in the level of risk across age groups, performance levels, or throughout the competitive season. Thus, further research has been recommended to further understand the scope of the problem across all athletic populations and help focus support to at-risk groups. Identification of at-risk groups will also help raise awareness and target educational resources to coaches and parents involved, who may be instrumental in the early detection of LEA (Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). Accordingly, this thesis aimed to further elucidate the prevalence of LEA, DE/ED, and EXD risk in female triathletes. Plus, explore the influence of age, performance level, and follow longitudinal changes through key phases of the competitive season.

CHAPTER 2

REVIEW OF LITERATURE

2.1 Chapter Overview

The following chapter will review the available literature surrounding topics relevant to this thesis and is divided into three main sections. The first section (2.2) provides an overview of the focus sport of this thesis, triathlon, and included the history and demands of the sport. Section 2.3 will then present and discuss literature related to LEA in athletes, including current conceptual models, assessment methods, alterations to health and performance, estimated prevalence and highlight areas that require further research. The final section (2.4) will discuss the DE behaviour and EXD literature in relation to their role as risk factors for the development of LEA.

2.2 Triathlon Overview

2.2.1 History

Triathlon has been described as a multidimensional endurance sport with three successive disciplines (swim, cycle, and run) and two transitions (swim-to-cycle and cycle-to-run; Bentley, Millet, Vleck & MacNaughton, 2002; Millet, Vleck & Bentley, 2011). The introduction of modern triathlon as an alternate workout to traditional track training was first founded in the early 1970s by the San Diego Track Club. It was initially characterised by a 10 km run, 8 km cycle, and 500 m swim known as the Mission Bay Triathlon (Hunt, 2019; Markus & Arimany, 2019). In 1978, the challenge to determine '*who was the toughest athlete*' led to the formation of the "Ironman" Triathlon where three of Hawaii's endurance events were combined: the Waikiki Rough Water Swim (2.4 miles), the Around-Oahu Bike race (112 miles), and the Honolulu Marathon (26.2 miles). In 1980, the global public learned about the Ironman World Championships as ABC's "Wide World of Sports" were permitted to film the 106 men and 2 women compete. It gained worldwide recognition in 1982 when collegiate athlete, Julie Moss, was shown collapsing and crawling to the finish line succumbing to a second-place finish by 29 seconds (Markus & Arimany, 2019).

Over the next decade, the exponential rise in participation and formation of National Governing Bodies led to the worldwide recognition of triathlon as a sport. In 1989, twenty-five nations came together to form the International Triathlon Union (ITU) and created the first official triathlon World Championships with the goal of gaining Olympic medal status for the sport. Triathlon was awarded Olympic status in 1994 and debuted at the Sydney 2000 Olympic Games (Hunt, 2019; Markus & Arimany, 2019). Today the sport continues to grow and in 2019 there was an estimated 150,000 committed active racing triathletes in the UK (British Triathlon, 2021a) and an estimated 400,000 USA Triathlon members (USA Triathlon, 2019). Both British Triathlon and USA Triathlon share a common goal to attract and retain more female participants with female triathletes currently representing 32% of memberships (British Triathlon, 2021b; USA Triathlon, 2019). These figures represent only those athletes who become members of British Triathlon or USA Triathlon, which is not a requirement to participate in a triathlon event. Various triathlon distances have been developed since the 1980s (Table 2.1) with the "Ironman" triathlon being the most recognisable distance, but the most popular being the international "standard" distance (Markus & Arimany, 2019).

Table 2.1. Triathlon distances

	Sprint	**Standard**	**Half Ironman**	**Full Ironman**
Swim	750 m	1.5 km	1.9 km	3.9 km
Cycle	20 km	40 km	90 km	180.2 km
Run	5 km	10 km	21.1 km	42.4 km

2.2.1 Overview of demands

Elite participants and the millions of non-elite participants – known as Age-Groupers in triathlon – all compete within the same events following the same rules (Vleck, Millet & Alves, 2014). Defining an *athlete* often relies on imprecise and vague qualitative descriptors that fail to classify the type of sport performed (Solberg, Borisson, Sharma & Papadakis, et al., 2016; McKinney, Velghe, Fee & Isserow, et al., 2019). Traditionally, *elite* athletes are defined as those who exercise >10 hours·week and have achieved the highest level of competition (i.e., professional athletes or Olympians). *Competitive* athletes exercise >6 hours·week with a goal of improving performance and participating in official competitions. *Recreational* athletes exercise >4 hours·week for pleasure, fitness or to partake in unregulated competitions that typically do not require systematic training. Finally, an *exerciser* participates in >2.5 hours·week of physical activity to maintain health and fitness (Solberg, et al., 2016; McKinney, et al., 2019).

Regardless of athletic definition, triathlon has been classified as IIIC (high static, high dynamic) with a significant risk of bodily collision and an increased risk of a sudden syncopal event by the Mitchell Classification of sport shown in Figure 2.1 (Mitchell, Haskell, Snell & Van Camp, 2005). This is an established method for categorising sports related to the level of intensity (low, medium, high) of static and dynamic demand required to perform that sport. Sports are classified as IA (low static, low dynamic), IIB (moderate static, moderate dynamic), or IIC (high static, high dynamic). The classification also acknowledges the risk of bodily collision and syncope for each sport. Although the Mitchell Classification includes quantitative descriptors for each classification, it does not consider other contributors that athletes experience during competition (i.e., emotional stress, environmental factors) or account for the often-higher demands associated with training regimens (Mitchell, et al., 2005).

Triathlon poses a unique challenge to all triathletes. A common performance goal, irrespective of event distance, gender, or competitive level, is time minimisation in each individual sporting discipline and overall finish time to meet event cut-off times (Fröhlich, Balter, Pieter & Schwarz, et al., 2014). A variety of event formats exist (Table 2.1), ranging from the sprint with an average completion of sub-1 h, to the standard distance completed in sub-2 h, and the full Ironman distance taking 8-9 h, at the elite level (Millet, et al., 2011; Etxebarria, et al., 2019). Whilst each event format has its distinct demands for periodisation of training, endurance sports are typically characterised by high training volumes with various combinations of intensity. Triathlon also requires a large number of high-quality training sessions each week for three different disciplines (Millet, et al., 2011; Vescovi & VanHeest, 2016; Etxebarria, et al., 2019). Large volumes of sustained training, in addition to, the myriad of environmental, technical, nutritional, psychological, and social demands of triathlon could increase the risk of athletes developing LEA (intentional or inadvertent) and associated negative health consequences (Millet, et al., 2011; Vescovi & VanHeest, 2016).

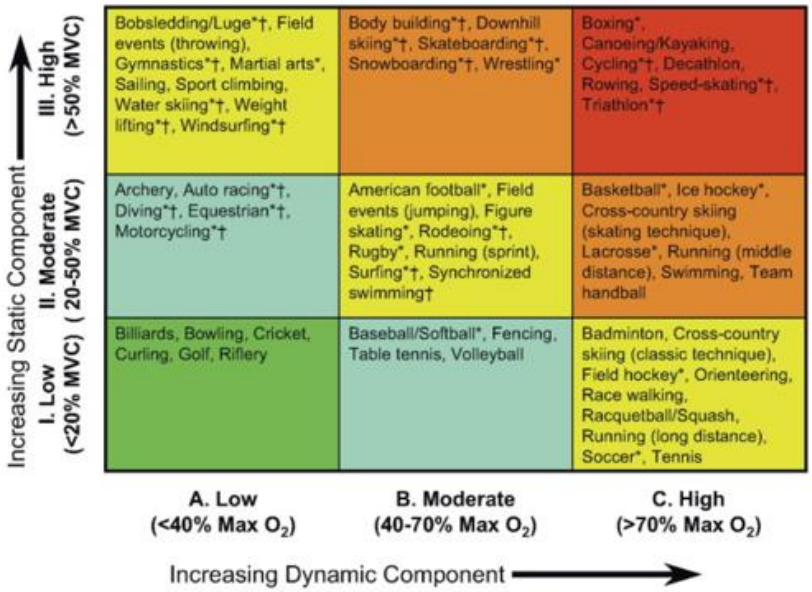


Figure 2.1. Mitchell Classification of Sport from Mitchell, et al., (2005, p. 1366). The lowest cardiovascular demands are shown in green and the highest in red. Blue, yellow, and orange shown low moderate, moderate, and high moderate total cardiovascular demands. Max

O_2, estimated maximal oxygen uptake; MVC, maximal voluntary contraction; * Danger of bodily collision; † Increased risk of syncope occurs.

2.3 Low energy availability in athletes

2.3.1 Energy balance & energy availability concepts

The aims of energy availability (EA) and energy balance (EB) are fundamentally different despite the similarities in both concepts relating energy intake to energy expenditure. Currently, EB is defined as dietary energy intake minus total energy expenditure (EB = EI – TEE; Westerterp & Saris, 1991; Westerterp, 2013). In the concept of EB (see figure 2.2), TEE is composed of three main components: basal metabolic rate (BMR), thermic effect of food (TEF), and activity thermogenesis (Levine, 2004). BMR represents 60-80% of energy expended during the post-absorptive state when an individual is at complete rest and resting energy expenditure (RMR) is considered to be within 10% of the BMR. TEF (also known as dietary induced thermogenesis (DIT)) refers to ~10% of energy expended during digestion, absorption, and conversion of food with both facultative and fixed components. Activity thermogenesis refers to ~15-30% of energy expended from exercise (EEE) and non-exercise activity thermogenesis (NEAT) such as, daily living, spontaneous muscle contraction or maintaining posture (Ravussin & Bogardus, 1989; Levine, 2004; Westerterp, 2013). EB is often referred to in the context of body mass and/or body composition changes related to diet and/or exercise interventions. When an imbalance occurs, weight gain results from a positive EB where the difference between EI and TEE is positive, and weight loss occurs from a negative EB (Areta, Taylor & Koehler, 2021).

Adaptive thermogenesis (or metabolic adaptation), although not unequivocally defined, relates to metabolic efficiency through alterations in TEE to conserve or dissipate energy (Müller & Bosy-Westphal, 2013). Comparable to most physiological systems, EB will return

to a state of equilibrium but this may not equate to a healthy metabolic balance been achieved. For instance, in the case of a negative EB weight loss may initially be evident before the initial energy deficit is decreased due to reductions in TEE (Hall & Kahan, 2018). This is often referred to as an *energy saving* mechanism where TEE is reduced as a result of decreased energy available to maintain the function of physiological systems. This has been quantified as a ~10-20% reduction in RMR plus a reduction in activity thermogenesis (Rion & Kawecki, 2007; Müller & Bosy-Westphal, 2013; Kosmiski, Schmiege, Mascolo & Gaudiani, et al., 2014; Koehler, Williams, Mallinson & Southmayard, et al., 2016). Consequently, these metabolic adaptations result in EB reaching a state of equilibrium, and therefore weight stability, at a lower threshold. This apparent state of homeostasis does not account for the downregulation of physiological systems (i.e., reproduction, growth, thermoregulation, immunity, and cellular maintenance) due to a lack of available energy for optimal functioning (Areta, et al., 2021). For that reason, it has emerged that EB may not be a useful measure within the athletic population. For measures solely of total or resting energy expenditure are considered unreliable in determining the energy available for the optimal functioning of physiological systems and will underestimate an athlete's energy requirements (Loucks, 2004; Loucks, et al., 2011).

In contrast to EB, the concept of EA only relates EI to EEE (figure 2.2). The most recent algebraic definition of EA defines it as dietary EI minus EEE, relative to each kilogram of LBM (EA = EI – EEE/ kg LBM). EA thereby represents the amount of residual energy available to sustain physiological systems after removing the energy cost of exercise training (Loucks, 2004; Loucks, et al., 2011; Loucks, 2020). In the context of adaptive thermogenesis, EA is viewed as an *input* into those physiological systems opposed to EB which is an *output* (Loucks, et al., 2011). The concept of EA provides a single numerical value that is not affected by adaptive thermogenesis as the focus relates EI solely to EEE which is independent of all the other components of TEE. The implication is that a threshold of energy availability is required

to support the optimal functioning of physiological systems. This concept allows the quantification of available energy independent of adaptive thermogenesis and body mass and/or body composition changes (Areta, et al., 2021). Importantly, the simplicity and minimalism of the current definition of EA is a limiting factor which in turn represents the main strength of the EB concept. In its simplification, the current concept of EA fails to account for energy expended from NEAT which may limit comparison of EA between studies or in the use of EA thresholds that may trigger physiological dysregulations. However, it is acknowledged that NEAT is highly variable between individuals, may be influenced by changes in EB and EA, is difficult to assess in free-living participants, and the relationship between EA and NEAT has yet to be established (Levine, 2004; Müller & Bosy-Westphal, 2013; Villablanca, Alegria, Mookadam & Holmes, et al., 2015).

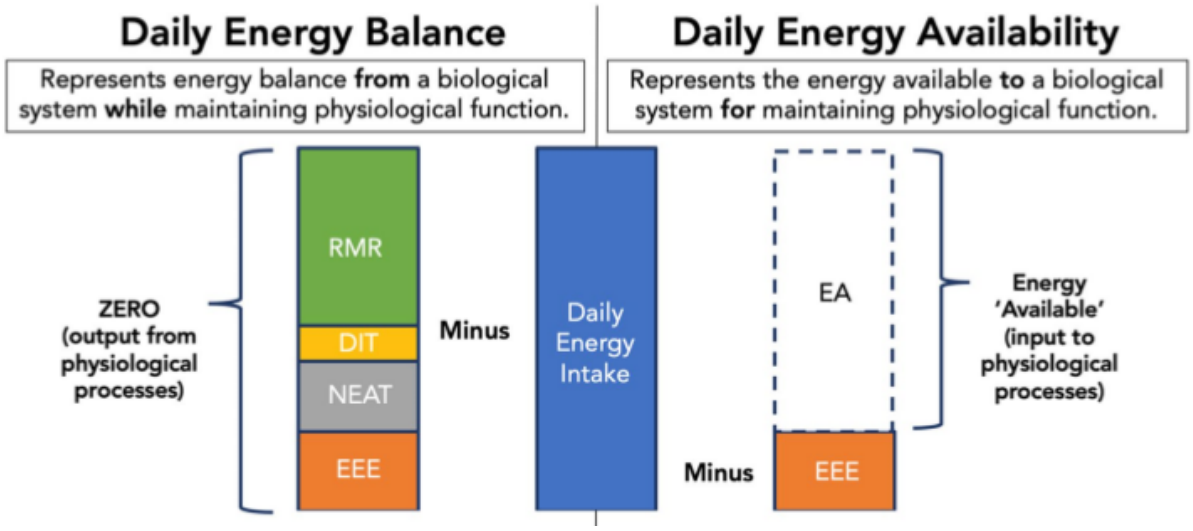


Figure 2.2. Illustration of the energy balance and energy availability concepts from Areta, et al., (2021, p. 10). RMR, resting metabolic rate; DIT, dietary induced thermogenesis; NEAT, non-exercise activity thermogenesis; EEE, exercise energy expenditure; EA, energy availability.

2.3.1.1 Evolution of the energy availability concept

To date, the evolution of the algebraic definition of EA in studies using humans has not been clear which may have contributed to a variety of definitions and calculations of EA being used. This has made comparisons of EA values across studies and its application to practice challenging. Although EA was not used as a quantifiable factor with algebraic formula, the original concept of EA was derived from mammalian experimental studies. These early studies assessed the role of nutrient availability and the energetic costs and gains associated with thermoregulatory and foraging efforts on reproductive success (Bronson, 1985; 1989). Schneider and Wade (1989; 1990) went on to determine changes in reproductive function in hamsters were related to changes in the general availability of metabolic fuels rather than changes in any specific fuel (i.e., fat or carbohydrates).

The concept of EA was first introduced in human trials by Loucks and Callister (1993) who examined the influence of exercise and EA treatments on thyroid metabolism in regularly menstruating sedentary women. This was the first study to use EA as a quantifiable parameter and the first algebraic definition of EA was introduced as dietary EI minus total EEE relative to body mass (EA = (EI – TEEE) / BM). Subsequently, Loucks and Heath (1994) aimed to characterise the functional relationship between thyroid metabolism and EA where the algebraic definition was refined to relate EA to LBM (EA = (EI – TEEE) / LBM). These previous algebraic definitions of EA used total EEE which includes energy expenditure from RMR and non-exercise waking activity. Loucks and Verdun (1998) recognised these components of energy expenditure should be subtracted from total EEE, thereby, the most current definition accounts only for net value of EEE (EA = (EI – EEE) /LBM).

2.3.1.2 Assessment of energy availability in free-living athletes

As LEA underpins both the Triad and RED-S models, it has been suggested that its presence and causes should be the focus of early detection and/or diagnosis (Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). Unfortunately, the direct measurement of EA in free-living athletes is not currently a practical or reliable option as several barriers exist. Most noteworthy is the lack of standardised or reference guidelines for undertaking an EA assessment in the field (Mountjoy, et al., 2014; 2018; Burke, et al., 2018). This relates to the period of assessment and methodologies used to assess the three core components of the most recent EA equation (EA = (EI/EEE) / LBM; Loucks & Verdun, 1998). Additionally, each of the three metrics have concerns related to their reliability, validity, and definitions of what should be assessed (Mountjoy, et al., 2014, 2018; Burke, et al., 2018). Table 2.2 summarises various assessment protocols used in studies estimating EA in various female athletic and exercising populations. A narrative review was conducted for the articles included in table 2.2 using targeted internet searches (i.e., Google Scholar and PubMed). Combinations of the following key search terms were included: recreational exerciser, athlete, endurance athlete, triathlon, triathlete, multi-sport endurance events, elite athlete, non-elite athlete, EA, LEA, Triad, RED-S, LEAF-Q, EI, and EB. Articles were considered if written in English, in full-text, and were conducted among free-living trained or exercising human subjects. Only studies that quantified the direct assessment of EA using quantified measures of EA (i.e., EI, EEE, and LBM) or screened for prevalence using the LEAF-Q within the text of the manuscript were included. No time limit on retrieval of articles was set and reference lists of articles retrieved were also reviewed. Animal studies were not included.

In comparison to other EA inputs, LBM measurement errors offer a comparatively small difference to estimates of EA. The main barrier for athletes and/or coaches gaining a reliable and accurate measurement of body composition is the cost and accessibility of the

equipment required (i.e., DXA scanning or bioelectrical impedance; Burke, et al., 2018; De Souza, Koltun, Strock & Williams, 2019). Although DXA scanning was the most commonly used method to assess body composition in studies included in table 2.2 (such as, Hoch, Pajewski, Moraski & Carrera, et al., 2009; Doyle-Lucas, Akers & Davy, 2010; Hoch, Papanek, Szabo & Widlansky, et al., 2011; Melin, Tornberg, Skouby & Møller, et al., 2015; 2016), estimates of LBM may still be influenced by the acute effects of hydration status, exercise or dietary EI. Thus, standardised DXA protocols should be employed to minimise the impact on measurement error (Nana, Slater, Hopkins & Halson, et al., 2016). Nevertheless, measurement error (~3% to 8%) is still common due to differences in techniques with the same machine or event different machines with the same technique (Burke, et al., 2018). A few studies also used skinfold measurements (surface anthropometry) to estimate LBM, such as, VanHeest, Rodgers, Mahoney & De Souza (2014), Muia, et al., (2016), Brown, Howatson, Quin & Redding, et al., (2017), Schaal, Tiollier, Le Meur & Casazza, et al., (2017), and Sygo, Coates, Sesbreno & Mountjoy, et al., (2018; Table 2.2). Despite having standardised and accredited protocols developed by the International Society for the Advancement of Kinanthropometry, this method is considered a doubly indirect estimate of LBM. Although cheaper and more accessible, its use is considered more applicable in monitoring differences in physical components such as subcutaneous fat or girths in athletes (Larsen-Myer, Woolf & Burke, 2018).

In contrast, dietary EI measurement errors contribute a significant difference to estimates of EA. This is related to dietary EI assessments in free-living athletes relying on self-reported sources to obtain a valid (how accurately the data measure actual EI) and reliable (how well the data reflect typical EI) record of either habitual or time specific dietary EI (Burke, Cox, Cummings & Desbrow, 2001; Mountjoy, et al., 2014; 2018; Burke, et al., 2018). Dietary EI can be assessed by either retrospective analysis or prospective recording. There is no gold standard for measuring dietary EI, however, most EA studies included in table 2.2 have relied

on prospective recording using diet records to assess EI (i.e., written record, electronic applications, and/or photo assessment; Table 2.2). Unfortunately, there is substantial evidence that self-reported diet records are prone to inaccurate reporting, particularly under-reporting in athletes, and fail to reflect a true representation of habitual or long-term EI (Burke, et al., 2001; Capling, Beck, Gifford & Slater, et al., 2017). These inaccuracies relate to several separate factors; it is known diet records often alter habitual dietary EI, quantification or description errors of food and drink recorded, and athletes may try to improve the perception of their dietary EI by inaccurate recording. The latter may be evidenced by omission of certain foods or drinks, under-reporting of portion sizes and foods and drinks deemed "unhealthy", and/or over-reporting of foods and drinks considered "healthy" (Burke, et al., 2001; Capling, et al., 2017; Burke, et al., 2018).

Significant and widespread dietary EI measurement errors evident in the athletic population exist even when alternative methods are employed to enhance both accuracy (e.g., duplication of assessments methods and/or weighted diet records) and reliability (e.g., repeated measures; Burke, et al., 2018). Burrows, et al., (2019) recently conducted a systematic review evaluating the validity of dietary assessment methods used to estimate EI of adults (≥18 years) against TEE measured by the reference method of doubly labelled water (DLW). It was reported that under-reporting of dietary EI ranged from 11% to 41% in studies using diet records, 1% to 47% in those using diet histories, and 5% to 42% in those studies using food frequency questionnaires. More recently, a similar study reported the use of weighed diet records in females underestimated daily EI by ~2286 kJ (546 kcal) and estimated diet records underestimated by ~1829kJ (437 kcal; McKenzie, Coyle, Santos & Burrows, et al., 2021). Measurement errors of self-reported dietary EI have not been as well studied in the athletic population compared to general population (Hill & Davies, 2001). However, a meta-analysis comparing self-report dietary EI to DLW in various athletic groups found a lower mean bias

of 19% under-reporting (range 0% to 36%) indicating a daily EI of ~2500kJ (598 kcal; Capling, et al., 2017).

The burden to both participants (i.e., time recording EI) and practitioners/researchers (i.e., time processing EI data) also need to be considered when assessing dietary EI as this may impact on participant compliance (Capling, et al., 2017; Burke, et al., 2018). Measurement error may too be derived from differences in the resources (i.e., nutrition software/databases) and protocols (i.e., coding of food and drink or food composition values) available to the practitioner/researcher to analyse diet records (Braakhuis, Meredith, Cox & Hopkins, et al., 2003; Larsen-Myer, et al., 2018). The validity of diet records can be compared to more rigorous assessments such as the DLW-method or using biomarkers (i.e., 24-h urea nitrogen excretion; Capling, et al., 2017), however, no studies in table 2.2 used either method and only a few (Schaal, van Loan & Casazza, 2011a; Woodruff & Meloche, 2013; Melin, et al., 2015; 2016) used more simple validity methods such as the Goldberg or Black cut-offs (Goldberg, Black, Jebb & Cole, et al., 1991; Black, 2000).

Similar to dietary EI, EEE measurement errors also contribute a significant difference to estimates of EA and cause significant participant and practitioner/researcher burden. Burke, et al., 2018) acknowledged the limited data available regarding the individualised energy expenditure of complex or field-based exercise (e.g., swimming or strength and conditioning) compared to simple exercise (e.g., running or cycling) when using HR monitors or GPS units. Varied approaches have been used in the EA literature to estimate EEE in various athletic populations with the most frequently used being training records with HR monitors in table 2.2. For instance, a few studies monitored body movements via accelerometers to estimate EEE (Woodruff & Meloche, 2013; Hoch et al, 2011; Brown, et al., 2017; Zabriskie, Currier, Harty & Stecker, et al., 2019) and others conducted laboratory testing to allow the relationship of HR

and $\dot{V}O_2$/respiratory exchange ratio (RER) to be compared to training records and HR monitors (Schaal, et al., 2011a; Melin, et al., 2015; 2016; Lagowska & Kapczuk, 2016).

Murakami, et al., (2016) conducted a validation study comparing 12 wearable devices for measuring TEE (e.g., Garmin Vivofit, Fitbit Flex, ActiGraph GT3X etc) against gold standard measurements for a standardised day (metabolic chamber) and free-living days (DLW method). In comparison to DLW estimates, all wearable devices underestimated TEE with the mean daily underestimation ranging from 400 to 2500 kJ (96 to 598 kcal). In a follow up study using the same wearable devices and methods, all except two devices significantly underestimated TEE in comparison to DLW estimates with the mean absolute percentage error ranging from 19% to 100% (Murakami, et al., 2019). The integration of both physiological data derived from indirect calorimetry and accelerometer data has been suggested as a method of improving precision and accuracy. Particularly for EEE estimates during vigorous exercise which are known to be underestimated when using accelerometers (Brage, Brage, Franks & Ekelund, et al., 2004; Strath, Brage & Ekelund, 2005; Brage, Westgate, Franks & Stegle, et al., 2015). Measurement error can also be minimised by ensuring the same device and methods for estimating EEE are consistent across all types of exercise recorded, especially in those athletes who cross-train (Burke, et al., 2018).

An alternative method used that is often cheaper and more accessible, albeit less precise, is the use of training records to calculate EEE from metabolic equivalent of tasks (METs; Ainsworth, Haskell, Herrmann & Meckes, et al., 2011) or equivalent (table 2.2). This method may also be used in combination with others when the use of wearable devices is not possible (i.e., swimming). There is also no universally accepted definition or terminology of exercise in free-living athletes with differences related to the difference between physical activity and purposeful training/competition, level of intensity, and the inclusion or exclusion of leisure activities and energy expended in transporting sport equipment (i.e., bikes; Burke, et

al., 2018 – table 2.2). Inconsistency in the different methods and definitions used in the same population can result in different calculations and interpretations of EA levels and makes comparisons of studies difficult (Guebels, Kam, Maddalozzo & Manore, 2014; Burke, et al., 2018).

As identified by Loucks and Verdun (1998), the current definition of EA accounts for non-exercise energy expenditure being subtracted from EEE during the exercise period to prevent the overestimation of EEE and underestimation of EA in athletes. Measurement error can therefore occur during measurements of RMR using indirect calorimetry or in the use of standard prediction equations (i.e., Cunningham, 1980). Although cheaper and more accessible, the use of such prediction equations in metabolically adapted athletes may overestimate RMR which in turn underestimates EEE and overestimates EA (Burke, et al., 2018). Limited studies have factored this into the estimation of EEE and EA calculation (Koehler, Achtzehn, Braun & Mester, et al., 2013; Melin, et al., 2015; 2016; Viner, Harris, Berning & Meyer, 2015; Heikura, Uusitalo, Stellingwerff & Bergland, et al., 2018a; Heikura, Burke, Bergland & Uusitalo, et al., 2018b).

There exist no reference guidelines for the period of assessment when undertaking an EA assessment (Mountjoy, et al., 2014; 2018; Burke, et al., 2018). Currently, no data exist across the athletic populations on the period of assessment required (i.e., number of days) to observe dietary EI and EEE to reflect true habitual practices (Braakhuis, et al., 2003; Mountjoy, et al., 2014; 2018; Burke, et al., 2018). Marr and Heady (1986) suggested the period of assessment required for this in sedentary populations ranged from 3 to 4 days. Table 2.2 summarises the typical period of assessment in EA studies in various athletic and exercising populations which ranges from 3 to 7 consecutive days. Assessment periods in the athletic population often reflect a specific phase of training, the social calendar or when recording compliance is maximised (Burke, et al., 2018). However, there may be dissociation between

the period of EA assessment and the period when mismatched eating and exercise behaviour caused reduced or LEA (Mountjoy, et al., 2014; 2018; Burke, et al., 2018). In summary, it is these barriers associated with the assessment of EA in free-living athletes that has prevented the universal acceptance and recommendation of it as a stand-alone diagnostic tool (De Souza, et al., 2014; Mountjoy, et al., 2014; 2018).

Recently, emphasis has been placed on the use of screening tools for the early detection of those at risk of developing LEA as a means to prevent the long-term health and performance consequences (De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). Melin, et al., (2014) developed the LEAF-Q (see chapter 3.4) to examine LEA risk and associated physiological symptoms. Further validity testing is required across various athletic populations as it is currently only validated in female endurance athletes. Melin, et al., (2014) also recommend the LEAF-Q should be used in combination with a validated DE screening tool. Although yet to be validated, Mountjoy, et al., (2015a) developed the RED-S CAT to assist in screening for RED-S and return to play decisions. Although these subjective measures are more accessible to a larger population, they may not be accurate and are dependent on self-report answers, with inherent issues regarding false reporting and compliance. Plus, the efficacy of these measures has also been questioned and it is recommended they are supplemented with additional individualised measurement techniques to enable diagnosis. These have included serial measures of body mass and composition, metabolic status, eating behaviours, and other psychological risk factors (i.e., exercise dependence, perfectionism etc; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018; De Souza, et al., 2019). Thus, highlighting the complexity and multi-disciplinary approach required for the accurate and reliable assessment of EA in free-living athletes.

2.3.1.3 Low energy availability overview

Healthy adults are typically considered to be in a state of EB (0 kcal·day^{-1}) when EA is 45 kcal·kgLBM^{-1}·day^{-1} (Loucks & Heath, 1994; Loucks & Thuma, 2003). LEA is the current terminology used to describe the negative health and performance consequences observed in athletes when there is inadequate energy to support all physiological functions (Loucks, et al., 2011). LEA in athletes may be caused by three distinct origins, 1) ED, 2) intentional but mismanaged efforts to alter body composition that may include DE behaviour, and 3) inadvertent inability to increase dietary EI to match EEE (Nattiv, et al., 2007). Table 2.2 shows the estimated prevalence of LEA and EA measures in free-living, female athletes and/or exercisers in various sports groups.

2.3.2 Conceptual models

2.3.2.1 Female athlete triad model

The term 'female athlete triad' (Triad) was first recognised in 1992 by the ACSM with the first position stand on the Triad published in 1997 (Yeager, et al., 1993; Otis, et al., 1997). The Triad was primarily defined as a clinical syndrome of three distinct but inter-related conditions; disordered eating, amenorrhea, and osteoporosis which were frequently observed in adolescent and young adult female athletes and exercising females (Otis, et al., 1997). Subsequent studies found LEA could be intentional, inadvertent, or psychopathological and linked to disturbances in menstrual and bone health (Loucks, Verdun & Heath, 1998; Hilton & Loucks, 2000; Ihle & Loucks, 2004). It was also highlighted that the negative health consequences of Triad could occur at a subclinical level with reduced EA, subclinical menstrual disorders, and low bone mineral density (De Souza, Miller & Loucks, et al., 1998; Sowers, Randolph & Crutchfield, et al., 1998; Tomten, Falch & Birkeland, et al., 1998). An updated position stand published in 2007 acknowledged these findings and redefined the Triad model components to consist of LEA (with or without ED), FHA, and osteoporosis as the pathological clinical endpoints. The revised model (figure 2.3) views each of the three inter-

related conditions on a spectrum ranging from the optimal healthy endpoint to subclinical and clinical conditions (Nattiv, et al., 2007). However, there are currently no clear guidelines for the diagnosis of subclinical conditions (Nattiv, et al., 2007; De Souza, et al., 2014).

In the case of optimal health, it refers to adequate EA to support TEE and physiological function without compromise (i.e., bone mass is normal and maintenance of ovulatory menstrual cycles; Nattiv, et al., 2007). It recognised that females may present with one or more of the three Triad conditions and each individual condition may progress along each spectrum bi-directionally at different rates. For instance, changes in EA may take days to weeks, alterations to menstrual function may occur within several months but in some cases may take longer than a year, and changes to bone mineral density (BMD) are much slower and may take several years (Nattiv, et al., 2007). The most recent consensus statement (De Souza, et al., 2014) re-emphasises the importance of presenting the Triad on a spectrum to enable the early detection and intervention of females with subclinical conditions. Thus, preventing the potentially irreversible consequences of the clinical endpoints of the Triad. The 2014 statement established clinical guidelines on treatment, risk-management strategies, and return-to-play recommendations and re-iterated the need for continued research investigating these areas, along with, the prevalence and aetiology (De Souza, et al., 2014).

2.3.2.2 Relative energy deficiency in sport model

The term ‘relative energy deficiency in sport’ (RED-S) was first introduced in a consensus statement by the IOC in 2014 and later updated in 2018 (Mountjoy, et al., 2014; 2018). The aim of the consensus statement was to better current understanding and awareness of the Triad and provide a more inclusive term for the overall clinical syndromes originally referred to as Triad. The aetiological factor underpinning the model is relative energy deficiency defined as “an energy deficiency relative to the balance between EI and the energy

expenditure required to support homeostasis, health and the activities of daily living, growth, and sporting activities." (Mountjoy, et al., 2014). The RED-S model was proposed to be broader in scope and expand on the Triad by suggesting the overall clinical syndrome is not a triad of three components. Instead, it indicates ten health and ten performance-related consequences resulting *directly* from LEA that are not limited to disturbances in menstrual and bone health. These additional consequences are shown in Figure 2.4 and include metabolic rate, cardiovascular health, endocrine function, decreased endurance performance and aspects related to mood (Mountjoy, et al., 2014; 2018). In contrast to the Triad spectra, all of the suggested consequences have a uni-directional and direct relationship with LEA with the exception of psychological health. Psychological health is bi-directional as it is suggested it may precede or be the result of LEA. Additionally, the RED-S model further expands on the Triad by acknowledging male, non-Caucasian, and disabled athletes may too be affected (Mountjoy, et al., 2014; 2018).

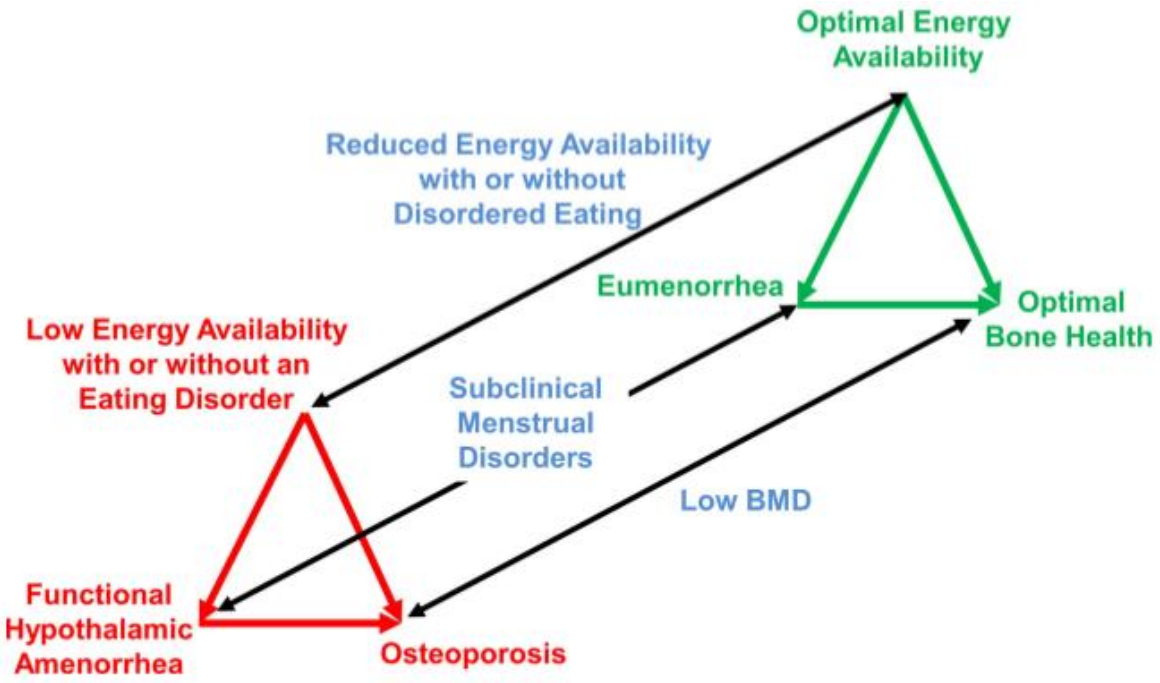


Figure 2.3. The current female athlete triad spectra by Nattiv, et al., (2007, p. 1868) illustrates the progression from optimal health to subclinical and clinical conditions. The black arrows represent the bidirectionality of each condition becoming worse or improving. The green and red arrows represent how the three Triad components are inter-related. BMD, bone mineral density.

Table 2.2. Estimated prevalence of LEA and EA measures in free-living, female athletes and/or exercisers in various sport groups.

Author	Population	Monitoring period	LBM measurement	DEI measurement	EEE measurement	DE/ED measurement	Additional measurements	Energy Availability	Prevalence of LEA
Black et al., (2018)	38 recreational exercisers (mean age: 23 years)	3-day period between two lab visits	Bioelectrical impedance	3-day weighed diet record	Training logs + METs	N/A	LEAF-Q + training background + self-reported physical characteristics + menstrual history + saliva samples for hormone profile	40.4 kcal·kgLBM^{-1}·day^{-1} (average) 36.3 / 47.5 kcal·kgLBM^{-1}·day^{-1} (at risk / not at risk by LEAF-Q)	63% at risk of LEA (LEAF-Q ≥ 8)
Brown et al., (2017)	25 pre-professional dancers (mean age: 21 years)	7-day period during normal training	Skinfold measurements (Durnin & Womersley, 1974)	7-day prospective weighed diet record + 24 h recall	Accelerometer + METs	Three-factor eating questionnaire (TFEQ-R18)	Menstrual cycle questionnaire	24.0 kcal·kgLBM^{-1}·day^{-1} (weekdays) 36.0 kcal·kgLBM^{-1}·day^{-1} (weekends)	N/A
Doyle-Lucas et al., (2010)	15 elite female ballet dancers (mean age: 24 years)	4-day period before laboratory visit	DXA scan	4-day weighed diet record	Estimated METs	TFEQ + EAT-26	Menstrual history questionnaire + RMR via indirect calorimetry + BMD	37.5 kcal·kgLBM^{-1}·day^{-1} 41.1 kcal·kgLBM^{-1}·day^{-1} (controls)	N/A

Folscher et al., (2015)	306 competitive ultra-endurance runners (mean age: 40 years)	1-day period at 2014 Comrades Marathon	N/A	N/A	N/A	FAST	LEAF-Q + training background + self-reported physical characteristics + Triad/RED-S knowledge	N/A	44% at risk of LEA during pre-season (LEAF-Q ≥ 8)
Heikura et al., (2018a)	13 AME+ 22 EU elite middle-and-long distance runners + race walkers (mean age: 24 and 27 years)	7-day period pre-competition training	DXA scan	7-day diet record (combined weighed and household measures)	Training logs + METs	N/A	BMD + LEAF-Q + blood markers + menstrual history + RED-S/Triad tool	32.0 kcal·kgLBM^{-1}·day^{-1} (AME) 35.0 kcal·kgLBM^{-1}·day^{-1} (EU)	31% had EA < 30 kcal·kgLBM^{-1}·day^{-1} AME mean LEAF-Q score = 12.8 EU = 8.3
Heikura et al., (2018b)	27 elite middle-and-long distance runners + race walkers (mean age: 26 years)	7-day period during altitude training camp – data recorded week 2	DXA scan	7-day weighed diet record	Training logs + METs + RPE	N/A	BMD + LEAF-Q + haemoglobin mass + blood markers	33.0 kcal·kgLBM^{-1}·day^{-1}	N/A

Hoch et al., (2009)	80 varsity athletes (various sports) (mean age: 17 years)	3-day period over a weekend	DXA scan	3-day prospective diet record	Questionnaire of training volume (weekly) + Compendium of Physical Activities (Ainsworth et al, 2011)	EAT-26	Menstrual history questionnaire + blood markers + BMD	N/A	6% had EA < 30 kcal·kgLBM^{-1}·day^{-1} 30% had EA > 30 and ≤ 45 kcal·kgLBM^{-1}·day^{-1}
Hoch et al., (2011)	22 professional ballet dancers (mean age: 23 years)	3-day period over a weekend	DXA scan	3-day prospective food diary	Accelerometer	EDE-Q	Menstrual history questionnaire with interview + hormonal profile + endothelial function + BMD	N/A *LEA defined as negative value	77% had LEA
Jesus et al., (2021)	83 elite cross-country runners (mean age: 22 years)	1-day period at the 2019 European Cross-Country Championships	N/A	N/A	N/A	N/A	LEAF-Q + training background + self-reported physical characteristics	N/A	80% at risk of LEA during pre-season (LEAF-Q ≥ 8)

Koehler et al., (2013)	185 young elite athletes (various sports) (mean age: 16 years)	7-day period during normal training	BIA	7-day diet record with standardised foods + interview	Training diary + Compendium of Physical Activities (Ainsworth et al, 2011)	N/A	Blood markers	29.4 kcal·kgLBM^{-1}·day^{-1} (average) 36.2 kcal·kgLBM^{-1}·day^{-1} (endurance sports)	51% had EA < 30 kcal·kgLBM^{-1}·day^{-1}
Lagowska & Kapczuk (2016)	31 athletes (various sports) + 27 ballet dancers (mean age: athletes 18 years + dancers 17 years)	3-day period during normal training	BIA	7-day diet record with photos	HR monitors + laboratory calculated $\dot{V}O_2$ + training questionnaire	N/A	Menstrual history questionnaire + gynaecological assessment + blood markers	28.3 kcal·kgLBM^{-1}·day^{-1} (athletes) 21.7 kcal·kgLBM^{-1}·day^{-1} (dancers)	N/A
Logue et al., (2019)	833 active females (various sports)	1-day period to complete online survey	N/A	N/A	N/A	N/A	LEAF-Q + participant demographics, training history, diet history and injury history	N/A	40% at risk of LEA (LEAF-Q ≥ 8)

Melin et al., (2015)	40 competitive and elite weight-bearing endurance athletes (mean age: 26 years)	7-day period during normal training	DXA scan	7-day prospective weighed diet record	HR monitors + training logs + laboratory calculated $\dot{V}O_2$	EDE-16 + EDI-3	Menstrual history questionnaire + gynaecological assessment + blood markers + aerobic capacity + BMD	39.6 kcal·kgLBM^{-1}·day^{-1} (average)	43% had EA > 30 and ≤ 45 kcal·kgLBM^{-1}·day^{-1} 20% had EA < 30 kcal·kgLBM^{-1}·day^{-1}
Melin et al., (2016)	25 competitive and elite weight-sensitive endurance athletes (mean age: 27 years)	7-day period during normal training	DXA scan	7-day prospective weighed diet record	HR monitors + training logs + laboratory calculated $\dot{V}O_2$	EDE-16 + EDI-3	Menstrual history questionnaire + gynaecological assessment + LEAF-Q + blood markers + BMD	42.5 kcal·kgLBM^{-1}·day^{-1} (average)	44% had EA > 30 and ≤ 45 kcal·kgLBM^{-1}·day^{-1} 12% had EA < 30 kcal·kgLBM^{-1}·day^{-1}
Meng et al., (2020)	52 elite and 114 recreational aesthetic sport athletes (mean age: 20 years)	1-day period during normal training	DXA scan	N/A	N/A	EDI-3	LEAF-Q + BMD + participant demographics, training history + blood markers	N/A	56% of elite and 35% at risk of LEA (LEAF-Q ≥ 8)

Moss et al., (2020)	13 professional soccer players (mean age: 24 years)	5-day period in-season	DXA scan	5-day weighed diet record	Training logs + METs	EDE-Q	BMD + LEAF-Q + blood markers + RMR via indirect calorimetry	35.0 kcal·kgLBM^{-1}·day^{-1} (all days) 29.0 kcal·kgLBM^{-1}·day^{-1} (heavy training or match days)	23% at risk of LEA (LEAF-Q ≥ 8) and had EA < 30 kcal·kgLBM^{-1}·day^{-1} 62% had EA > 30 and ≤ 45 kcal·kgLBM^{-1}·day^{-1}
Muia et al., (2016)	61 elite middle-and-long distance adolescent runners (median age: 16 years)	5-day period (3 training days and 2 rest days)	Skinfold measurement (Warner, Fornetti, Jallo, & Pivarnik, 2004)	5-day diet record (3 training days and 2 rest days)	Training logs with RPE for METs calculation	EDI-3 + TFEQ	Menstrual history questionnaire	36.5 kcal·kgLBM^{-1}·day^{-1} (average)	18% had EA < 30 kcal·kgLBM^{-1}·day^{-1}
Reed et al., (2011)	25 active females (various sports)	7-day period during normal training	DXA scan	2 sets of 3-day diet records recorded two weeks apart (2 weekdays and 1 weekend day)	HR monitors + training logs + METs for sessions without HR monitor	TFEQ + EDI	Aerobic capacity + RMR via indirect calorimetry + gynaecological assessment + blood markers + BMD	42.1 kcal·kgLBM^{-1}·day^{-1} (OVS) 28.8 kcal·kgLBM^{-1}·day^{-1} (EXMD)	N/A

Reed et al., (2013)	19 NCAA Division I soccer players (mean age: 19 years)	3-day period (pre, mid and post season)	DXA scan	3-day diet records (non-weighed + prospective)	HR monitors + training logs + METs for sessions without HR monitor	EDI-2	Aerobic capacity + blood markers + menstrual history questionnaire + BMD	43.5 kcal·kgLBM^{-1}·day^{-1} (pre) 35.2 kcal·kgLBM^{-1}·day^{-1} (mid) 44.5 kcal·kgLBM^{-1}·day^{-1} (post)	26% had < 30 kcal·kgLBM^{-1}·day^{-1} (pre) 33% had < 30 kcal·kgLBM^{-1}·day^{-1} (mid) 12% 26% had < 30 kcal·kgLBM^{-1}·day^{-1} (post)
Robbeson et al., (2015)	26 vocational student dancers (mean age: 19 years)	5-day period normal training	DXA scan	5-day weighed food diary	Estimated METs	EDI-3 + TFEQ	BMD	39.0 kcal·kgLBM^{-1}·day^{-1} 38.0 kcal·kgLBM^{-1}·day^{-1} (controls)	N/A
Schaal et al., (2011a)	10 competitive endurance trained athletes	7-day period (normal training)	DXA scan	7-day diet record	Training logs + HR and RPE monitoring compared to laboratory calculated $\dot{V}O_2$	EDE-Q	Aerobic capacity + blood markers + menstrual history questionnaire + BMD	29.0 kcal·kgLBM^{-1}·day^{-1} (EU) 18.0 kcal·kgLBM^{-1}·day^{-1} (AME)	N/A

Schaal et al., (2017)	9 national synchronised swimmers (mean age: 20 years)	4-day period (normal training + 2 and 4 weeks after intensified training)	Skinfold measurements (Jackson & Pollock, 1985)	4-day prospective photo record	HR monitors	N/A	Saliva samples for endocrine markers	25.0 kcal·kgLBM^{-1}·day^{-1} (normal training) 22.3 + 18.0 kcal·kgLBM^{-1}·day^{-1} (week 2 and 4 intensified training)	N/A
Slater et al., (2016)	109 recreational exercisers from team and individual sports (mean age: 24 years)	1-day period to complete online survey	N/A	N/A	N/A	N/A	LEAF-Q + training background + self-reported physical characteristics + menstrual history	N/A	45% at risk of LEA during pre-season (LEAF-Q ≥ 8) 70% + 35% at risk from individual sports + team sports

Silva et al., (2015)	67 rhythmic gymnasts	1 and 4 days before International competition	BIA	24 h record of dietary intake	Questionnaire of training volume (weekly) + Compendium of Physical Activities (Ainsworth et al, 2011)	N/A	Gynaecological history via questionnaire	31.5 kcal·kgLBM^{-1}·day^{-1} (average) 32.9 (16-18 years) 29.8 (19-26 years)	37% had EA < 45 kcal·kgLBM^{-1}·day^{-1} 45% had EA < 30 kcal·kgLBM^{-1}·day^{-1}
Sygo et al., (2018)	13 elite national level track and field sprinters and jumpers (mean age: 21 years)	Assessed once in the pre-and-post season	Skinfold measurements described by Stewart et al., 2011	N/A	N/A	N/A	LEAF-Q + BMD + RMR via indirect calorimetry + blood markers + menstrual history	N/A	23% at risk of LEA during pre-season (LEAF-Q ≥ 8) 39% at risk of LEA post-season
VanHeest et al., (2014)	10 junior national swimmers (mean age: 16 years) (CYC = 5) (OVS = 5)	Every 2-weeks during competitive training season	Skinfold measurements (Durnin & Womersley, 1974)	3-day prospective diet record + 24 h recall	Training logs + diaries	N/A	Menstrual cycle diary + maximum 400 m time trial + RMR via indirect calorimetry + blood markers	32.0 kcal·kgLBM^{-1}·day^{-1} (average: CYC) 11.0 kcal·kgLBM^{-1}·day^{-1} (average: OVS)	N/A

Viner et al., (2015)	4 competitive endurance cyclists (mean age: 38 years)	3-day period during the pre, mid and post season	DXA scan	3-day prospective weighed diet record	Training diary + Compendium of Physical Activities (Ainsworth et al, 2011)	TFEQ	BMD	26.2 kcal·kgLBM^{-1}·day^{-1} (pre) 25.5 (mid) 23.8 (post)	N/A
Woodruff & Meloche (2013)	10 Varsity volleyball players (mean age: 21 years)	7-day period during competitive season	Air-displacement plethysmography	7-day prospective diet record	Accelerometer + training diaries	N/A	Menstrual cycle history questionnaire	42.5 kcal·kgLBM^{-1}·day^{-1} (average)	60% had EA > 30 and ≤ 45 kcal·kgLBM^{-1}·day^{-1} 20% had EA < 30 kcal·kgLBM^{-1}·day^{-1}
Zabriskie et al., (2019)	20 National Division II Lacrosse athletes (mean age: 20 years)	4-day period at five time points across season	DXA scan	4-day diet record (2 weekdays + 2 weekend days)	Accelerometer	N/A	BMD + RMR + recovery assessment	27.4 kcal·kgLBM^{-1}·day^{-1} (average) 22.9 pre 28.8 mid 28.3 post	N/A

Zanders et al., (2021)	13 National Division II Basketball players (mean age: 20 years)	4-day period during 5 phases across the season, separated by ~1 month	DXA scan	4-day diet record	HR monitor + accelerometers	N/A	BMD + aerobic capacity + RMR + sleep and recovery	46.0 kcal·kgLBM^{-1}·day^{-1} (average)	N/A

AME, amenorrheic; BIA, bioelectrical impedance; BMD, bone mineral density; CYC, cyclic menstruation; DE, disordered eating; DEI, dietary energy intake; DXA, dual energy x-ray absorptiometry; EA, energy availability; EAT, eating attitudes test; ED, eating disorder; EDE, eating disorder examination; EDE-Q, eating disorder examination questionnaire; EDI, eating disorder inventory; EEE, exercise energy expenditure; EU, eumenorrheic; EXMD, exercise menstrual disturbances; FAST, female athlete screening tool; HR, heart rate; LBM, lean body mass; LEAF-Q, low energy availability in female's questionnaire, METs, metabolic equivalent of task; N/A, not available; OVS, ovarian suppressed; RED-S, relative energy deficiency in sport; RMR, resting metabolic rate; RPE, rating of perceived exertion; TFEQ, three factor eating questionnaire; Triad, female athlete triad; $\dot{V}O_2$, oxygen consumption.

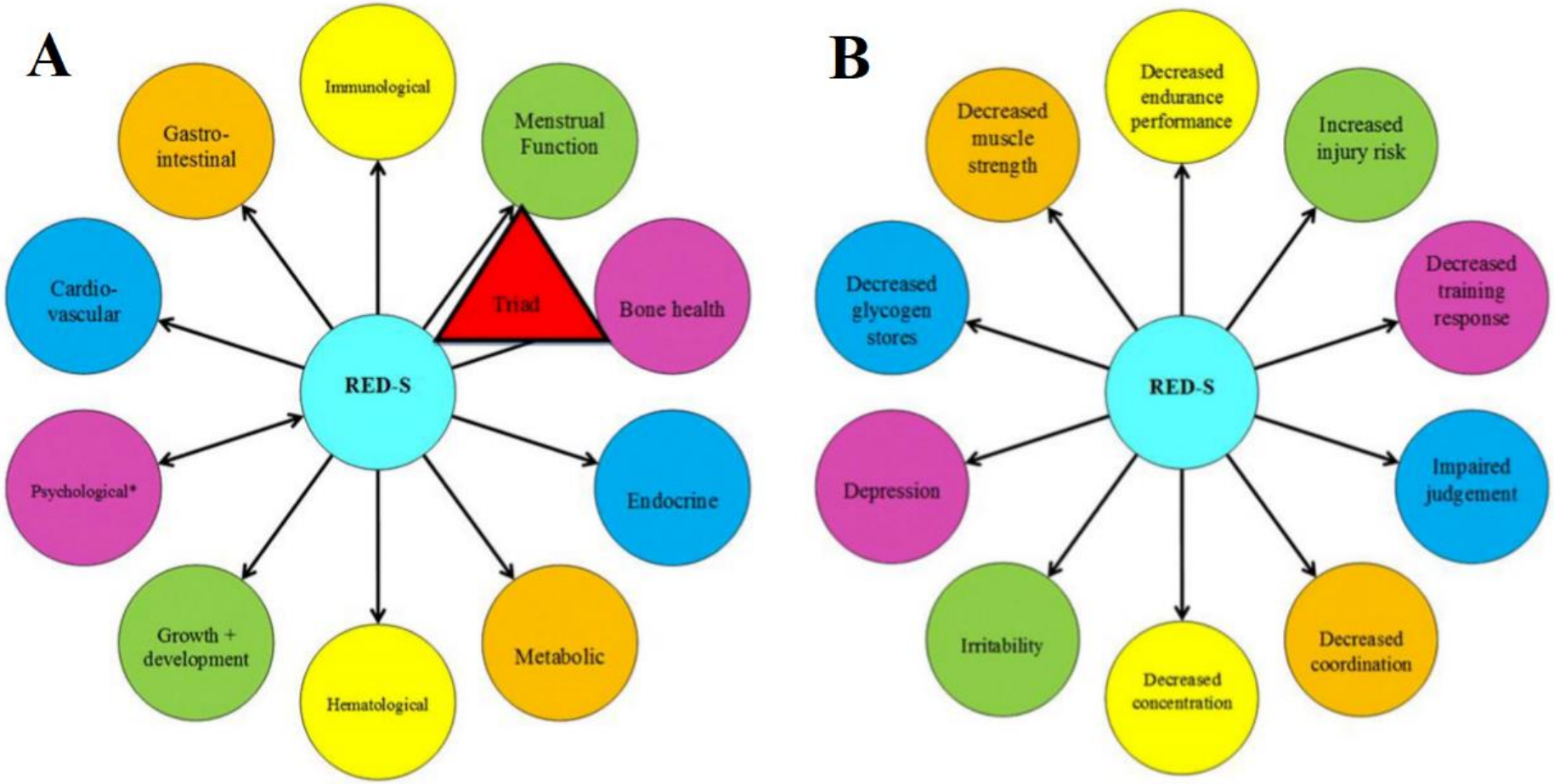


Figure 2.4. Health (A) and performance (B) consequences associated with the Relative Energy Deficiency in Sport model, from Mountjoy, et al., (2014, p. 493).

2.3.2.3 Critiques of the Triad and RED-S models

Both the Triad and RED-S models highlight the potential health and performance consequences of inadequate energy for sport, however, controversy and a lack of clarity around the models exist (De Souza, Williams & Nattiv, et al., 2014; Mountjoy, Sundgot-Borgen, Burke & Carter, et al., 2015b). The IOC authors have described the RED-S model as being broader in scope and more comprehensive than the Triad model and called for the new terminology of RED-S to replace the Triad (Mountjoy, et al., 2014). Scientific rigor (the strict compliance to all aspects of the scientific method) and reproducibility (different researchers obtaining consistent results based on the original studies methods) are vital in the development and interpretation of position stands and consensus statements. It reduces the risk of prematurely introducing unfounded hypotheses and builds evidence-based knowledge from high quality published studies, peer review and debate (Casadevall & Fang, 2016; 2018; Hofseth, 2018).

These factors have been evident in the advancements of the Triad model over the last three decades with research addressing the associated criticisms of the model. These have included the causal role of EA on menstrual function and bone health (De Souza & Williams, 2004; Loucks & Verdun, 1998; Loucks & Thuma, 2003; Metzger, Baek & Swift, et al., 2016; Williams, Helmreich, Parfitt & Caston-Balderrama, et al., 2001; Williams, Leidy & Hill, et al., 2015), sociological implications of the negative consequences associated with sport and exercise (DiPietro & Stachenfeld, 2006), the prevalence and interrelatedness of Triad components (De Souza, Toombs & Scheid, et al., 2010; Gibs, Williams & De Souza, 2013; Khan, Liu-Ambrose & Sran, et al., 2002; Sundgot-Borgen & Torstveit, 2004), and Triad recovery (Williams, Mallinson & De Souza, 2019). Current criticism, gaps in the literature or debates of the Triad relate to the long-term health consequences of Triad, the application of EA definitions and thresholds from laboratory to field settings, and the relationship between

psychological factors (i.e., stress) and menstrual function (Heikura, et al., 2018a; Loucks & Redman, 2004; Williams, et al., 2019).

In contrast, it has been argued that there is insufficient and inaccurate interpretation of supporting evidence and a lack of scientific rigor with regards to several facets of the RED-S model (De Souza, et al., 2014). For instance, the RED-S model refers to the ten health consequences of energy deficiency as '*impairments*' and considers each to be a threat to overall health and require treatment. The model does not offer evidence for physiological plasticity but rather implies all physiological impairments are independent and equal in their contribution to poor health. There is concern that this oversimplification may reduce the clinical relevance of the primary clinical conditions (DE/ED, menstrual function and/or bone health) associated with LEA (De Souza, et al., 2014). With reference to the health and performance consequences depicted in the RED-S model, these are not specifically defined and often refer to physiological systems. The amount of relative energy deficiency needed to cause impairment is also not defined. This has led to a lack of supporting evidence as the ability to reproduce findings is limited with a lack of quantifiable outcomes (Mountjoy, et al., 2014; 2018).

The IOC authors have argued that the Triad is not a true Triad (Mountjoy, et al., 2014; 2015b; 2018). The basis of this relates to the 2007 position stand and 2014 consensus paper stating individuals can present with one or more of the three clinical conditions and although the three clinical conditions are the most serious sequelae other clinical issues exist (Nattiv, et al., 2007; De Souza, et al., 2014). It is argued that the Triad model does not adequately illustrate the importance of LEA, the interrelatedness of all the factors, or the concern of subclinical levels of presentation (Mountjoy, et al., 2014; 2015b; 2018). Alternatively, it is viewed that the RED-S model misrepresents the physiological underpinnings of the Triad model and mis-identifies causality. A result of the uni-directional arrows implying a direct and equal effect of energy deficiency on health and performance consequences and insufficient supporting

evidence of the health and performance consequences. Unlike the Triad model, the RED-S model fails to depict or offer supporting evidence for the continuum from healthy to subclinical and clinical conditions for the health and performance consequences mentioned or the potential reversibility of these (De Souza, et al., 2014).

Another point of discussion centre on the unclear definition provided for energy deficiency which underpins the RED-S model (defined in Chapter 2.3.2.2). This definition more closely relates to the EB concept and is not in line with the definition of EA referenced throughout the RED-S consensus statement. EB and EA are not synonymous (see Chapter 2.3.1), but the IOC authors have continued to use the terms relative energy deficiency and LEA interchangeably (Mountjoy, et al., 2014; 2015b; 2018). The RED-S consensus statement provides no units of measure or guidelines on how to quantify relative energy deficiency and it is unclear how the concept of RED-S is used in the assessment of energy status (Mountjoy, et al., 2014). The RED-S concept is not experimentally derived and subsequent updates have used EA research originally used in the Triad model to support relative energy deficiency (De Souza, et al., 2014; Mountjoy, et al., 2014; 2015b; 2018). Thus, it is not clear if the aetiological factor of the RED-S model is relative energy deficiency or LEA.

A criticism of the Triad model, suggested by the IOC authors, has been its focus solely on the female athlete. They too suggest that the Triad model fails to recognise those who may be at risk but do not identify as an athlete (i.e., dancers or recreational exercisers) by using the term *athlete* in its title (Mountjoy, et al., 2014; 2015b). By being broader in scope the RED-S model has included male, non-Caucasian, and disabled athletes as groups potentially at risk of energy deficiency/LEA. Research is evolving within these focus groups (Chin, Hoggatt & McGregor, et al., 2016; James-Todd, Chiu & Zota, 2016; Tenforde, Barrack & Nattiv, et al., 2016), however, it is still in its infancy and it is thought their inclusion within the RED-S model is premature and unsubstantiated by case studies, observational studies, or gold-standard

randomised control trials. The RED-S model also does not account for the unique physiological differences found across these groups and their relation to energy deficiency/LEA (De Souza, et al., 2014). It is misleading to provide universal clinical guidelines for the prevention, detection, and treatment of energy deficiency/LEA across these groups that have predominantly been derived from females. There are concerns the inclusion of these groups under one model may detract the focus away from females when it is known they experience the most severe clinical consequences (De Souza, et al., 2014).

Although both the Triad and RED-S models' have a common goal, the lack of clarity, controversy, and inconsistencies in knowledge is confusing for the wider scientific and sporting community (Williams, Koltun, Strock & De Souza, 2019). After reviewing the two models, it is clear the RED-S model brings attention to impairments beyond menstrual and bone health whilst extending the model to acknowledge other potentially at-risk groups (i.e., males). However, unlike the Triad model, RED-S fails to define its components, the clinical relevance, the inter-relatedness of its components and the causal role of either LEA/relative energy deficiency. There is a distinct lack of supporting evidence and scientific rigour throughout the RED-S model. This results in increased confusion and misdiagnosis both in the research field and in clinical practice when managing at-risk individuals. After reviewing the two models, it is clear there is scope for the Triad research to extend in the direction of a male triad model, however, there appears to be an overgeneralised approach when including several focus groups under one umbrella for applying RED-S. For the purpose of this thesis, Triad is viewed as a diagnosable condition underpinned by supporting evidence and scientific rigour and RED-S is viewed as a concept.

2.3.3 Health effects of low energy availability

The Triad and RED-S models outline the negative health and performance consequences associated with LEA (Nattiv, et al., 2007; Mountjoy, et al., 2014). Although not limited to issues with menstrual function and bone health, to date these have been identified as the most serious clinical outcomes associated with LEA (De Souza, et al., 2014). As this thesis focuses on the prevalence of individuals at risk of LEA opposed to examining its effects on health and performance, this section will provide a brief overview of the most serious clinical outcomes for female athletes.

2.3.3.1 Energy availability and menstrual function

Definitions: Menstrual function exists upon a continuum of reproductive disturbances, fluctuating between eumenorrhea, to subclinical perturbations (i.e., luteal phase defects (LPD)), to clinical conditions (i.e., amenorrhea; De Souza, 2003; De Souza & Williams, 2004 – see figure 2.5). Within this range, eumenorrhea is defined as having a regular, ovulatory cycle, with a luteal phase >10 days and a cycle length ranging from 22-35 days (De Souza, 2003). LPD in athletes and physically active females has been defined as a luteal phase ≤10 days, with reduced progesterone concentrations. Though ovulation occurs, the reduction in progesterone concentrations reflect the inadequacy of the reproductive system to support implantation due to the poor quality of the endometrium (Jones, 1976; Balasch & Vanrell, 1987; De Souza, 2003). Anovulatory cycles refer to the absence of ovulation defined by low levels of luteinising hormone (LH) and follicle stimulating hormone (FSH) secretion and reduced oestrogen (E_2) levels (Hamilton-Fairley & Taylor, 2003). Due to the variation in cycle length with anovulation it has been associated with oligomenorrhoea (De Souza & Williams, 2004). Several methods exist to determine menstrual cycle phase which vary in both the accuracy of determining menstrual phase and precision of the measurement, as shown in table

2.3 (Alen, McRae-Clark, Carlson & Saladin, et al., 2016). However, it should be noted that recommendations made by Alen, et al., (2016) have not been directly compared to one another and rank ordering of methods based on precision, accuracy, cost, and participant burden was based on expert opinion opposed to scientific data.

Loucks and Horvath (1985) described oligomenorrhea as irregular and inconsistent menstrual cycles varying from 36-90 days in length. When methodological limitations for detection exist, an alternative definition used is ≤4 menstrual cycles per year (Cobb, Bachrach, Greendale & Marcus, et al., 2003). Primary amenorrhea (also referred as delayed menarche) is defined as menarche occurring after the age of 15 years in the presence of normal secondary sexual characteristics (American Society for Reproductive Medicine Practice Committee, 2004). Secondary amenorrhea has been conservatively defined as no menses for a minimum of three months (Loucks & Horvath, 1985). FHA is one of the most common causes of secondary amenorrhea and refers to recurring anovulation associated with weight-loss, stress, or exercise (Meczekalski, Katulski, Czyzyk & Podfigurna-Stopa, et al., 2014; Gordon, Ackerman, Berga & Kaplan, et al., 2017a). The current diagnostic approach for primary and secondary amenorrhea is presented in Figure 2.6 (ASRMPC, 2004; 2006; Klein, Paradise & Reeder, 2019).

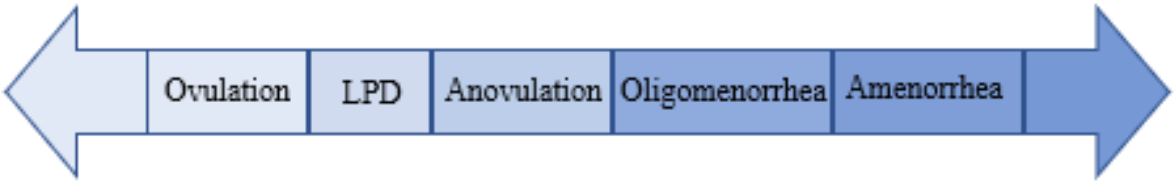


Figure 2.5. Continuum of menstrual disturbances in female athletes and physically active females. LPD, luteal phase defects.

Table 2.3. Precision and accuracy of methods used to determine menstrual cycle phase by Allen, et al., (2016, p. 22).

Method	Measurement precision	Accuracy of determining phase
Sonography	High	Very High
Blood sex hormone assessment	Medium	High
Salivary sex hormone assessment	Medium	High
Urine LH testing	Low	Medium
Basal-body temperature	Low	Medium
Self-report onset of menses	Low	Low

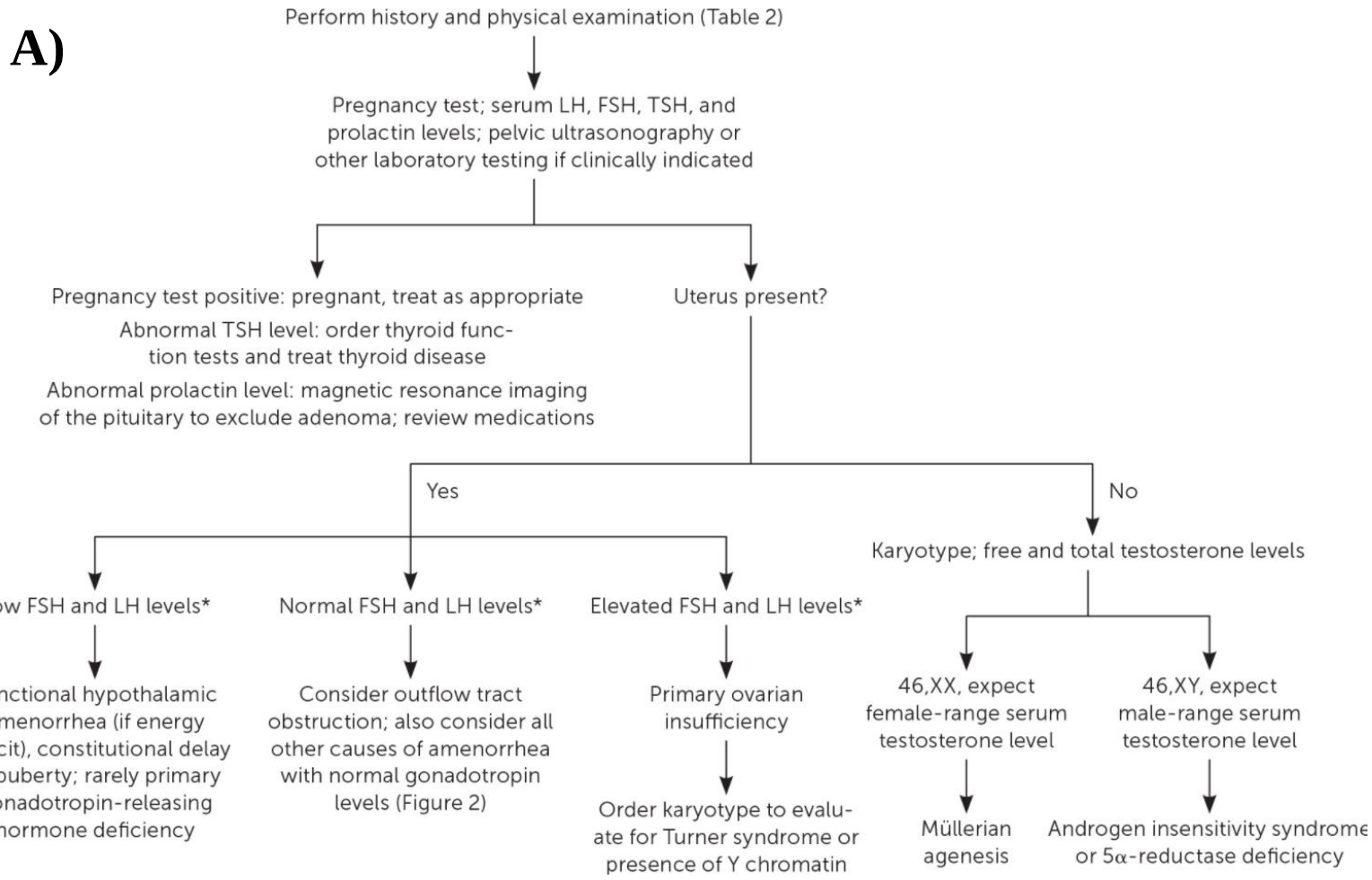


Figure 2.6. Diagnostic approach for the evaluation of A) primary amenorrhea and B) secondary amenorrhea, from Klein, et al., (2019, p. 41-2).

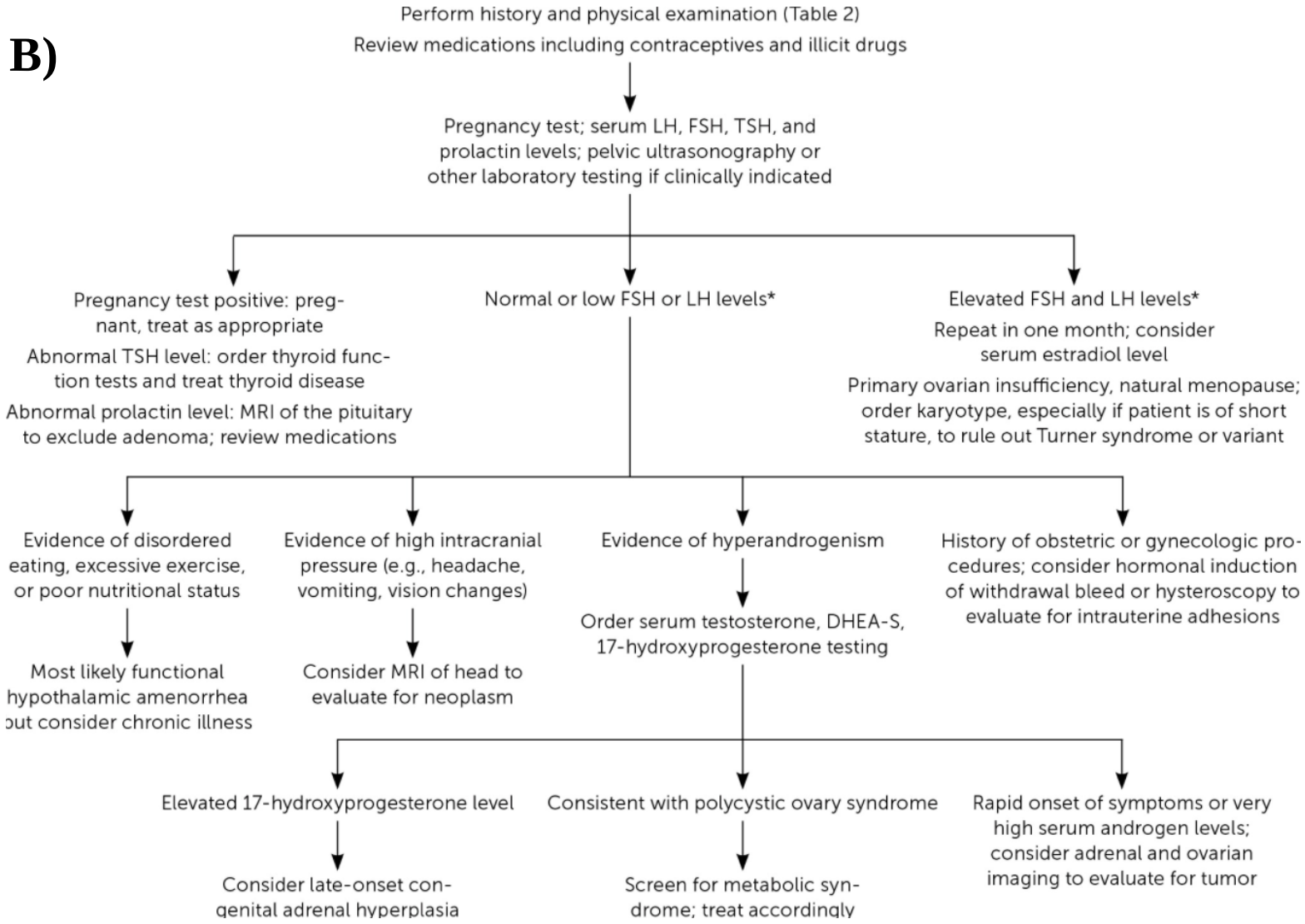


Figure 2.6. Diagnostic approach for the evaluation of A) primary amenorrhea and B) secondary amenorrhea, from Klein, et al., (2019, p. 41-2).

Prevalence: Limited data exist on the prevalence of LPD and anovulatory cycles in athletes and physically active females. A result of methodological difficulties in detecting these subtle menstrual disturbances when individuals present with consistent intermenstrual intervals of normal length (22-35 days). Specifically, with the sole use of menstrual history questionnaires assessing menstrual cycle length as an indicator of 'normal' menstrual function (De Souza, et al., 2010). Prevalence estimates of LPD in sedentary females range from 2% to 8% and 3% to 20% in females with infertility (McNeely & Soules, 1988; De Souza, Miller & Loucks, et al., 1998; Smith, Lenton, Landgren & Cooke, 2006). LPD are the most prevalent menstrual cycle disturbance associated with exercise with a greater incidence in active females than sedentary (McNeely & Soules, 1988; De Souza, Miller & Loucks, et al., 1998).

A 3-month prospective observational study in twenty-four, moderately active females (32 km/week of running) was conducted using urinary endocrine data for measures of total FSH, LH, pregnanediol-3-glucuronide, and estrone conjugates. (De Souza, Miller & Loucks, et al., 1998). It was observed that even with regular menstrual cycles of normal length, menstrual function was highly variable and frequently abnormal. A prevalence and sample incidence of LPD and anovulation of 42% and 16% respectively was reported (De Souza, et al., 1998). The importance of monitoring more than one menstrual cycle was highlighted with research often monitoring three consecutive menstrual cycles (De Souza, et al., 1998). As despite presenting with regular menstrual cycle lengths of 27 days, 42% of exercising females had intermittent presentations of ovulatory, LPD and anovulatory cycles across the 3-month monitoring period (McNeely & Soules, 1988; De Souza, Miller & Loucks, et al., 1998).

In a follow-up study by De Souza, et al., (2010), 52% (n = 25/48) of physically active females were categorised as having abnormal cycles despite presenting with regular menstrual cycles of normal length. This consisted of a prevalence and sample incidence of LPD and anovulation of 27% and 25% respectively (De Souza, et al., 2010). Both studies have

highlighted the significant differences in the prevalence of subtle menstrual disturbances between sedentary and exercising females. Both studies observed sedentary females as having consistent menstrual status of either ovulatory or LPD cycles. The prevalence of LPD in sedentary females in both studies was 9% and 5% respectively, with no reports of anovulatory cycles (De Souza, Miller & Loucks, et al., 1998; De Souza, et al., 2010). Further research is warranted on the prevalence of subclinical menstrual disorders across the sport and exercise spectrum. This will further define the scope of the problem and its associations with the Triad and RED-S models.

The characteristics of oligomenorrhea are inconsistent making it difficult to study. The estimated prevalence in the general population without polycystic ovarian syndrome (PCOS) is less than 18% and between 75% and 85% of females with PCOS (Harris, Babic, Webb & Nagle, et al., 2018). Within exercising and athletic females no definitive data exist on the prevalence of oligomenorrhea due to the methodological limitations (i.e., absence of daily measurements of hormones), variability in definitions used, and the frequent grouping of oligomenorrhea and amenorrhea presentation (Cobb, et al., 2003; De Souza, 2003; De Souza, et al., 2010). It has been accepted that exercising and/or athletic females frequently present with menstrual cycles of irregular length (Loucks & Horvath, 1985). In the De Souza, et al., (2010) study, 7% (3/43) of physically active females presented with oligomenorrhea compared to 0% of sedentary females. The low prevalence reported may be explained by the difficulty associated with collecting daily urine samples in females with long duration oligomenorrheic cycles and possibly linked to the exclusion of females with current or past PCOS. In contrast, studies using self-reported menstrual disturbances observed a prevalence of oligomenorrhea ranging from 10% to 40% in exercising females (Beals & Manore, 2002; Cobb, et al., 2003; Nichols, Rauh, Barrack & Barkai, et al., 2007).

Primary and secondary amenorrhea in female athletes and physically active females is considered to by hypothalamic in origin with a greater incidence of secondary amenorrhea (De Souza, 2003; De Souza & Williams, 2004; Gordon, et al., 2017a). It has been estimated that FHA accounts for 20% to 35% of secondary amenorrhea cases and 3% of primary amenorrhea (ASRMPC, 2006). Prevalence estimates of secondary amenorrhea in exercising women have been reported to range from 1% to 66%, exceeding estimates observed in sedentary females (2%-5%; Drew, 1961; Pettersson, Fires & Nillius, 1973; Feicht, Johnson, Martin & Sparkes, et al., 1978; Dale, Gerlack & Wilhite, 1979; Singh, 1981; Schwartz, Cumming, Riordan & Selye, et al., 1981; Sanborn, Martin & Wagner, 1982; Bachmann & Kemmann, 1982; Loucks & Horvath, 1985; De Souza, et al., 2010; Meczekalski, Katulski, Czyzyk & Podfigurna-Stopa, et al., 2014; Gordon, et al., 2017a). The majority of these studies used self-reported methods of detecting menstrual disturbances, varying definitions of amenorrhea and a range of athletic populations which may explain the variability observed.

In the De Souza, et al., (2010) study, 37% (16/43) of physically active females presented with secondary amenorrhea by assessment of ovarian steroids in daily urine samples. In a self-report study by Hoch, et al., (2007), it was observed that 40% of female club triathlon athletes had a history of primary or secondary amenorrhea. Using a sex hormone assessment (E_2, progesterone, LH, FSH, total testosterone, prolactin, sexual hormone binding globulin, dehydroepiandrosteron sulfate, and androstendion), 60% (24/40) of elite female endurance athletes presented with menstrual disturbances: 25% with oligomenorrhea, 17% with primary amenorrhea, and 58% with secondary amenorrhea (Melin, et al., 2015). Transvaginal ultrasound was used to diagnose and exclude females presenting with other menstrual disturbances (i.e., PCOS; Melin, et al., 2015). Menstrual disturbances exist across a range of sports. Endurance sports and sports emphasising low body mass or leanness have recorded the

highest incidence of the most severe menstrual disturbances, likely a result of LEA (De Souza & Williams, 2004).

Mechanisms: The causal role between LEA and menstrual dysfunction in athletes and physically active females has been well-established but the mechanisms underpinning its effects are not fully understood (Loucks & Thuma, 2003; Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018; Gordon, et al., 2017a). Regulation of the reproductive axis is centred on the gonadotropin-releasing hormone (GnRH; Tsutsumi & Webster, 2009). The release of GnRH from the hypothalamus includes both pulse and surge phases, which are regulated independently (Maeda, Ohkura, Uenoyama & Wakabayashi, et al., 2010). The anterior pituitary gonadotrope is the primary target of hypothalamic GnRH. Its pulsatile secretion determines the synthesis and secretion of the gonadotropins FSH and LH. FSH and LH regulate endocrine function and gonadal development (Conn & Crowley, 1994; Kaiser, et al., 1997). Current evidence has demonstrated alterations in the pulsatile release of GnRH are associated with LEA, which in turn results in alterations in FSH and LH pulsatility and decreased progesterone and E_2 levels. It is these LEA-associated disruptions to LH pulsatility, as a mechanism to conserve energy, that lead to disturbances in menstrual function in female athletes and/or exercisers (Curry, Logan, Ackerman & McInnis, et al., 2015; Gordon, et al., 2017a). A schematic representation of the hypothalamic-pituitary-gonadal (HPG) axis is presented in figure 2.7. Although there is a causal link between LEA and impairments to menstrual function, the duration and severity of reductions in EA required to cause such impairments are unclear.

Early work by Loucks and Heath (1994) examined dietary restriction on LH pulsatility in seven, sedentary, regularly menstruating females over two menstrual cycles. Five-day dietary EI was set at either 45 $kcal \cdot kgLBM^{-1} \cdot day^{-1}$ (balanced) or 10 $kcal \cdot kgLBM^{-1} \cdot day^{-1}$ (restricted) during the follicular phase of the menstrual cycle, whilst maintaining normal

sedentary habits. LH pulse frequency was significantly reduced (23%), especially during waking hours, and LH pulse amplitude significantly increased (40%), especially during sleep, in the restricted EA condition by the fifth day. The addition of an exercise treatment group (30 kcal·kgLBM^{-1}·day^{-1} of exercise) to both EA conditions in a follow-up study revealed similar LH pulse frequency and LH pulse amplitude findings in the restricted EA condition. New findings revealed no changes in LH pulsatility in the balanced EA condition which suggested EA was the cause of changes in LH pulsatility and not the stress of exercise (Loucks, Verdun & Heath, 1998).

The introduction of a specific EA threshold below which LH pulsatility is affected was established in subsequent work by Loucks and Thuma (2003). Twenty-nine, habitually sedentary, regularly menstruating females were examined for five days during the follicular phase. It was found LH pulsatility was not disrupted during EA conditions of 45 or 30 kcal·kgLBM^{-1}·day^{-1} while undertaking controlled exercise set at 15 kcal·kgLBM^{-1}·day^{-1}. However, during EA conditions of 10 and 20 kcal·kgLBM^{-1}·day^{-1} disruption in LH pulsatility were evident, suggesting a threshold <30 kcal·kgLBM^{-1}·day^{-1} at which significant impairments to menstrual function are observed. Important to note, only LH pulsatility was assessed as a marker of menstrual function and did not include assessment of ovarian hormonal characteristics or menstrual cycle length and the duration of assessment was short-term (5 days).

Although this concept of an absolute EA threshold of 30 kcal·kgLBM^{-1}·day^{-1} has been frequently cited in the Triad and RED-S supporting literature (Nattiv, et al., 2007; Mountjoy, et al., 2014; 2018) it has been met with contrasting findings (De Souza, et al., 2019). For instance, a cross-sectional study analysed EA in ninety-one exercising females who were categorised by menstrual status (amenorrheic, oligomenorrheic, ovulatory eumenorrheic, inconsistent subclinical menstrual dysfunction eumenorrheic, and anovulatory eumenorrheic;

Reed, De Souza, Mallinson & Scheid, et al., 2015). Across all groups mean EA was >30 kcal·kgLBM^{-1}·day^{-1} and EA could not differentiate ovulatory cycles from subclinical menstrual disturbances. However, EA was able to differentiate amenorrhea (31 kcal·kgLBM^{-1}·day^{-1}) from eumenorrhea (37 kcal·kgLBM^{-1}·day^{-1}; Reed, et al., 2015). Further work by Lieberman, et al., (2018) and Williams, et al., (2015) used a randomised control trial to assess EA over several menstrual cycles in untrained, previously eumenorrheic females by manipulating EI and EEE. These studies did not evidence an absolute EA threshold below which menstrual disturbances occur but did report linear increases in menstrual disturbances as EA decreased. It has been proposed that a dose response continuum exists between EA and menstrual function and more studies are needed to further elucidate this concept (De Souza, et al., 2019). It is also possible that there is individual variability when it comes to the use of EA thresholds and there is no 'one size fits all' (Loucks & Thuma, 2003; Loucks, 2007).

The exact signals and pathways of how LEA disrupts the HPG axis in female athletes and/or exercisers are complex and not fully understood. LEA causes a hypometabolic state which has been characterised by alterations in the secretion of insulin, cortisol, kisspeptin, insulin-like growth factor-1 (IGF-1), thyroid hormones, such as triiodothyronine (T3) and thyroxine (T4), and appetite-regulating hormones, such as ghrelin, leptin, and peptide YY (Gordon, et al., 2017a; Elliot-Sale, Tenforde, Parziale & Holtzman, et al., 2018 – see figure 2.8). Although currently unclear, it is believed such neuroendocrine factors likely signal nutritional status to the hypothalamus (Scheid & De Souza, 2010; Gordon, et al., 2017a). It is clear that short and long-term LEA impairs the HPG axis, reflected as disturbed LH pulsatility which leads to impaired menstrual function. Prolonged sub-clinical and clinical menstrual disturbances may have negative implications for the health and performance of female athletes and/or exercisers (De Souza, et al., 2014; Mountjoy, et al., 2014; 2018).

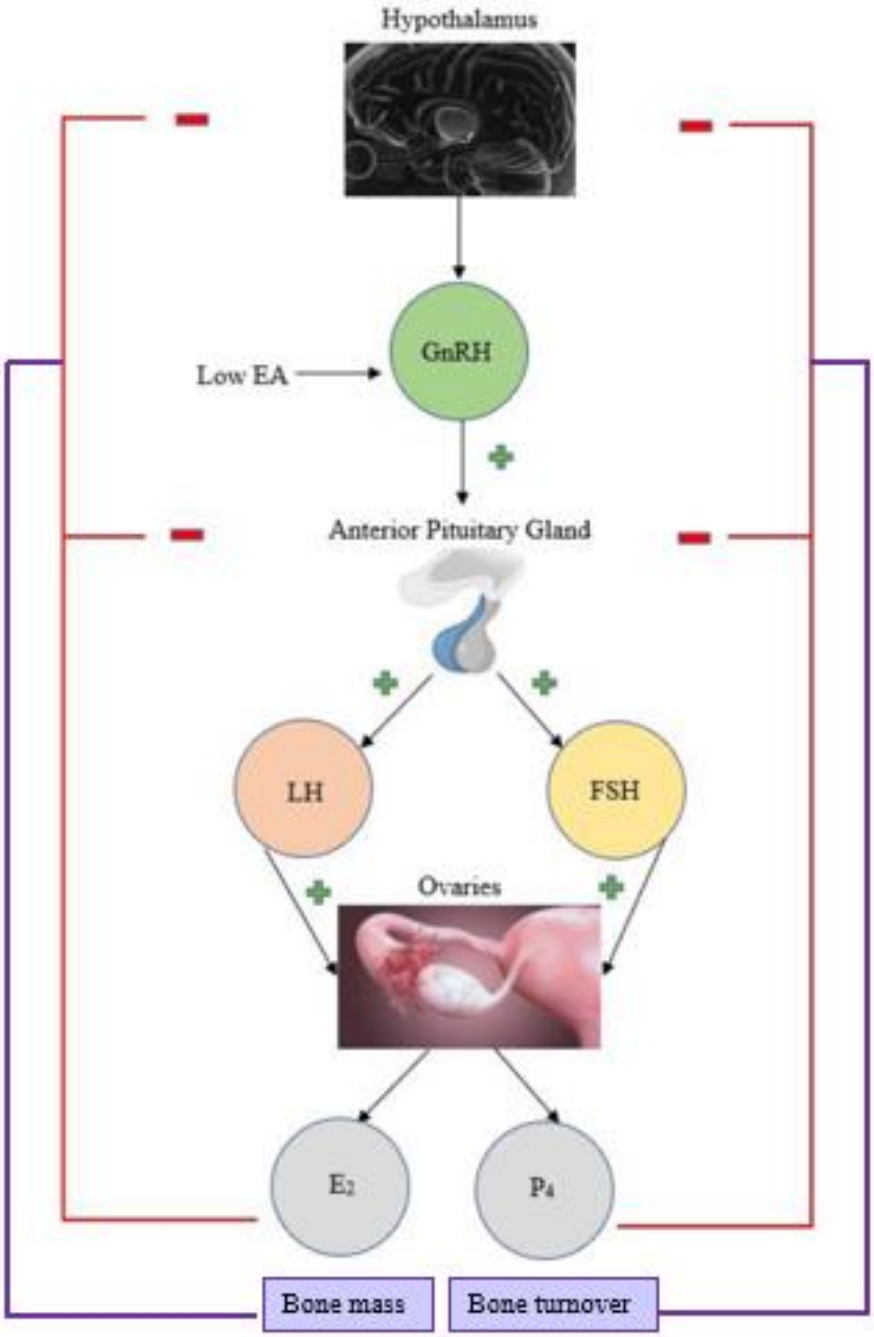


Figure 2.7. Schematic representation of the hypothalamic-pituitary-gonadal axis and low energy availability. + positive feedback; - negative feedback; EA, energy availability; E_2. Oestrogen, GnRH, gonadotropin-releasing hormone; LH, luteinising hormone; FSH, follicle stimulating hormone; P_4, progesterone. Alterations in any of the hormones within the HPG axis (with or without LEA) alters other bone-active hormones (i.e., FSH, E_2) in the whole feedback loop of the axis, leading to changes in bone mass and bone turnover.

2.3.3.2 Energy availability and bone

The beneficial effects of regular, weight-bearing exercise in improved bone health outcomes across the lifespan are well-established (Bailey & Brooke-Wavell, 2008; Boreham & McKay, 2011; Scofield & Hecht, 2012; Weaver, Gordon, Janz & Kalkwarf, et al., 2016). Peak bone mass (PBM) is defined as a combined measure of maximal bone size and mineral density present at the end of skeletal maturation (Matkovic, Jelic & Wardlaw, et al., 1994). It is during adolescence and young adulthood that these benefits of exercise to bone mass are maximised. In females, up to 90% of PBM is acquired by the age of 18-20 years and gains in BMD continuing, after the cessation of bone growth, into the third decade (Recker, Davies, Hinders & Heaney, et al., 1992; Bailey, McKay & Mirwald, et al., 1999; Bonjour & Rizzoli, 2001; Whiting, Vatanparast, Baxter-Jones & Faulkner, et al., 2004; Bailey & Brooke-Wavell, 2008). Bone mass accrual during adolescence and young adulthood and bone loss are the predominant factors in determining bone health (i.e., risk of fragility and osteoporosis) later in life. Accrual of a higher PBM is thought to provide protection when BMD inevitably declines due to aging, menopause, or other chronic disease-related causes in adulthood (Baxter-Jones, Faulkner & Forwood, et al., 2011).

PBM is largely pre-determined by genetics (60%-80%) but other factors such as the environment, hormones, nutrition, and mechanical loading also contribute (Havill, Mahaney, Binkley & Specker, 2007; Boudin & Van Hul, 2017; Gordon, Zemel, Wren & Leonard, et al., 2017b). Although exercise-induced gains to PBM are maximised during adolescence and young adulthood, this is considered a vulnerable time for the development of inadequate nutrition (including calcium deficiencies), DE/ED, menstrual dysfunction, and hypoestrogenism (Goolsby & Boniquit, 2017). Also, the positive effect of weight-bearing exercise on BMD is typically not replicated in sports such as swimming, cycling, distance running, ballet dancers and jockeys. Individuals from these sports typically present with lower

BMD than their counterparts in weight-bearing sports or controls (Schofield & Hecht, 2012; Dolan, McGoldrick, Davenport & Kelleher, et al., 2012; Wilson, Hill, Sale & Morton, et al., 2015; Amorim, Koutedakis, Nevill & Wyon, et al., 2017; Wewege & Ward, 2018). Such sports are often characterised as non-weight bearing, endurance based and/or have a higher risk of LEA (Sale & Elliot-Sale, 2019).

Early work in the 1980s and early 1990s first recognised the potential relationship between an athletes' menstrual status and their bone health. Drinkwater, et al., (1984) examined bone mineral content and density on four separate occasions, separated by seven days, in 14 amenorrheic and 14 eumenorrheic runners. It was found that lumbar BMD was significantly lower in amenorrheic athletes (mean, 1.12g/cm^2) compared to eumenorrheic athletes (mean, 1.30g/cm^2). In a follow-up study, BMD was examined over a 15.5-month period in athletes who regained menses from weight gain and reduced training, remained amenorrheic, and those with regular cycles. There were significant changes in lumbar BMD in amenorrheic athletes who resumed menses (+6.3%), but not for cyclic athletes (-0.3%), and a loss of -3.4% in BMD was observed in amenorrheic athletes (Drinkwater, Nilson, Ott & Chestnut, 1986).

Subsequent work recognised that an athletes' BMD reflects both current menstrual status and one's history of exercise-associated menstrual disturbances. It was also identified that resumption of menses may not fully restore BMD. Drinkwater, et al., (1990) found lumbar BMD was significantly related to menstrual patterns with BMD lower in athletes with a history of oligomenorrhea/amenorrhea (1.18g/cm^2) than those with a history of regular cycles (1.27g/cm^2). Athletes who had never had regular cycles reported the lowest lumbar BMD at 1.05g/cm^2. After two and eight years, it was observed athletes and dancers with a history of amenorrhea had significantly lower BMD compared to controls and their eumenorrheic counterparts (Jonnavithula, Warren, Fox & Lazaro, 1993; Keen & Drinkwater, 1997). Additionally, menstrual dysfunction has been related to higher prevalence's of stress reactions

and fracture independent of training volume (Warren, Brooks-Gunn, Hamilton & Warren, et al., 1986; Barrow & Saha, 1988; Myburgh, Hutchins, Fataar & Hough, et al., 1990; Bennell, Malcom, Thomas & Reid, et al., 1996; Duckham, Peirce, Meyer & Summers, et al., 2012; Ackerman, Cano Sokoloff & De Nardo Maffazioli, et al., 2015; Neidel, Wolfram, Hotfiel & Engelhardt, et al., 2019).

As such, both the Triad and RED-S models have acknowledged the interplay between EA and menstrual function on bone health in female athletes and/or exercisers (Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). The Triad model proposes that bone health exists upon a continuum from optimal bone health (i.e., BMD equal to or above average) to the clinical endpoint of osteoporosis (Nattiv, et al., 2007 – see figure 2.3). Along this continuum female athletes and/or exercisers may experience problems with achieving PBM, low areal and volumetric BMD, reduced bone strength, impaired bone geometry, and increased stress reactions and/or fractures (Drinkwater, et al., 1984; Ackerman, Nazem & Chapko, et al., 2011; Ackerman, Putman & Guereca, et al., 2012; Barrack, Gibbs & De Souza, et al., 2014; Mallinson, Williams & Gibbs, et al., 2016).

BMD in at risk females is typically assessed using DXA scanning, as recommended by the ACSM (Nattiv, et al., 2007). Low BMD is defined as Z scores of -1.0 to -2.0 together with a history of secondary clinical risk factors for fracture and osteoporosis is defined as Z scores ≤2.0 with secondary clinical risk factors for fracture (figure 2.8). Secondary clinical risk factors include stress fractures, hypoestrogenism, and nutritional deficiencies (ISCD, 2004; Khan, Bachrach & Brown, et al., 2004; Khan, Hanley & Bilezikian, et al., 2006; Nattiv, et al., 2007). It remains unclear if the use of DXA Z scores in estimating athletes bone health is appropriate. As Z scores use non-athletes of the same age and sex as a reference range and many athletes are considered smaller or larger than the average individual (Sale & Elliot-Sale, 2019). A review of the individual and combined components of Triad found the prevalence of low BMD

in exercising women ranged from 0% to 40% (Z score -1.0 to -2.0) and osteoporosis ranged from 0% to 15% (Z score ≤2.0; Gibbs, et al., 2013). Bone metabolic markers (i.e., procollagen type 1 amino-terminal propeptide (P1NP), N-terminal telopeptide (NTx), and C-telopeptide (CTx) can be used to examine the rate of bone formation and resorption (De Souza, Koltun, Etter & Southmayd, 2017). However, there is no gold standard bone metabolic marker and there has been a call for the adoption of international reference standards for bone formation and resorption markers to facilitate their use in clinical practice (Vasikaran, Cooper, Eastell & Griesmacher, et al., 2011). In practice this may not be easily achieved.

Ihle and Loucks (2004) were one of the first to directly examine the dose-response relationship of EA on bone metabolism in 29 regularly menstruating, habitually sedentary, young women. Using both dietary manipulation and exercise, bone metabolic markers were assessed for five days during the early follicular phase of two cycles separated by two months of a balanced EA (45 kcal·kgLBM^{-1}·day^{-1}) compared to three levels of LEA (10, 20 and 30 kcal·kgLBM^{-1}·day^{-1}). NTx concentrations (bone resorption) were only increased at 10 kcal·kgLBM^{-1}·day^{-1} and were inversely related to oestradiol. All levels of LEA reported a reduction of total osteocalcin and carboxy-terminal propeptide of type 1 procollagen (P1CP; bone formation) levels. A linear relationship was detected between changes in P1CP and insulin, and between total osteocalcin and T3 and IGF-1 concentrations. Importantly this study raised awareness of the potential relationship between LEA and bone metabolism. However, some of the bone metabolism markers (i.e., osteocalcin or P1CP) used are not considered optimal in their use today due to inadequate quality control, limited data for comparison and, limited understanding of their biological variability (Vasikaran, et al., 2011; Sale & Elliot-Sale, 2019). It is currently recommended that one marker for bone resorption (i.e., CTx) and bone formation (i.e., P1NP) be measured using standardised assays and used as reference markers (Vasikaran, et al., 2011).

The independent and combined effects of energy status (deficient or replete) and E_2 status (deficient or replete) on bone metabolism was assessed in 44 exercising women (De Souza, West, Jamal & Hawker, et al., 2008). The most severe metabolic impairments were evident in the energy and E_2-deificient group who had the lowest levels of P1NP and T3 and the highest levels of ghrelin and urinary CTx. Energy deficient groups presented with suppressed levels of osteocalcin and T3 and E_2-deficient groups had suppressant lumbar BMD and estrone glucuronides. Leptin was a significant predictor of bone formation but not resorption. The importance of maintaining balanced EA was noted as regardless of E_2 status impairments to bone metabolism were not evident in the energy replete group. In contrast, the group with both energy and E_2 deficiencies was associated with bone loss. More recently, the effects of reduced EA (15 $kcal \cdot kgLBM^{-1} \cdot day^{-1}$) on bone metabolism was assessed over a 5-day period. It was found that bone formation was significantly lower (P1NP), and bone resorption was significantly higher (β-CTX) and associated with decreased insulin and leptin levels, in women when in a state of reduced EA. Thus, highlighting the importance of EA for bone health (Papageorgiou, Elliot-Sale, Parsons & Tang, et al., 2017).

The aetiology of impaired bone health in female athletes and/or exercisers relates to both E_2-dependent and E_2-independent mechanisms (figure 2.8). The E_2-dependent mechanism is secondary to hypoestrogenism (E_2 deficiency) associated with secondary amenorrhea (De Souza, et al., 2017). Bone mass and structure are maintained when the rate of bone resorption by osteoclasts and formation by osteoblasts are coupled (Delaisse, 2014). Chronic hypoestrogenism causes an imbalance in favour of osteoclasts which promotes reduced bone mass and impaired bone structure by stimulating the multi-complex process of osteoclastogenesis (Weitzman & Pacifici, 2006; Vasikaran, 2008; De Souza, et al., 2017). The E_2-independent mechanism is considered energy dependent as it relates to the hypometabolic state caused by LEA (Ihle & Loucks, 2004; De Souza, et al., 2008; Mallinson, Williams, Hill

& De Souza, 2013; Southmayard, Mallinson & Williams, et al., 2016). Hormones that regulate bone formation such as, T3 (stimulate osteoblast proliferation and differentiation), IGF-1 (stimulates osteoblastogenesis), and leptin (osteoblast proliferation and modulates hormones such as cortisol and IGF-1) are all suppressed in a state of LEA, particularly in amenorrheic athletes (Cornish, Callon, Bava & Lin, 2002; Combs, Nicholls, Duncan & Bassett, et al., 2011; Guntur & Rosen, 2013). Thus, bone metabolism may be impaired as a result of the hypometabolic state caused by LEA. Overall, it is the suppression of the HPG axis (figure 2.7) caused by either E_2-dependent (i.e., E_2 deficiency) or E_2-independent (i.e., suppression of T3, IGF-1, leptin) mechanisms, as a result of LEA, that may lead to negative perturbations to bone (Goolsby & Boniquit, 2017; De Souza, et al., 2017). To date, it remains unclear if it is the magnitude of LEA or the time-course of LEA that negatively influences bone health. The Triad and RED-S models have recognised LEA and menstrual function can independently or synergistically impair bone health, with the most significant impairments observed in females who are both E_2 and energy deficient (Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018).

2.3.4 LEA prevalence research

Both the Triad position stand and IOC consensus statement have highlighted the importance of identifying the prevalence of LEA and further elucidate the associated consequences on health and performance. This will enable further understanding of the scope of the problem across all athletic populations and help focus support to at-risk groups (Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). Table 2.2 summarises the prevalence of LEA across various sports in female athletes and/or exercisers using either direct measures of EA or self-report screening tools (i.e., LEAF-Q). Females participating in sports that emphasise leanness or low body mass (figure 2.9), particularly aesthetic and endurance sports, are more likely to be at risk of LEA and subsequently Triad or RED-S (Nattiv, et al.,

2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018; Logue, et al., 2018; 2020). However, the risk of LEA is not limited to females participating in leanness sports or those participating at the elite level.

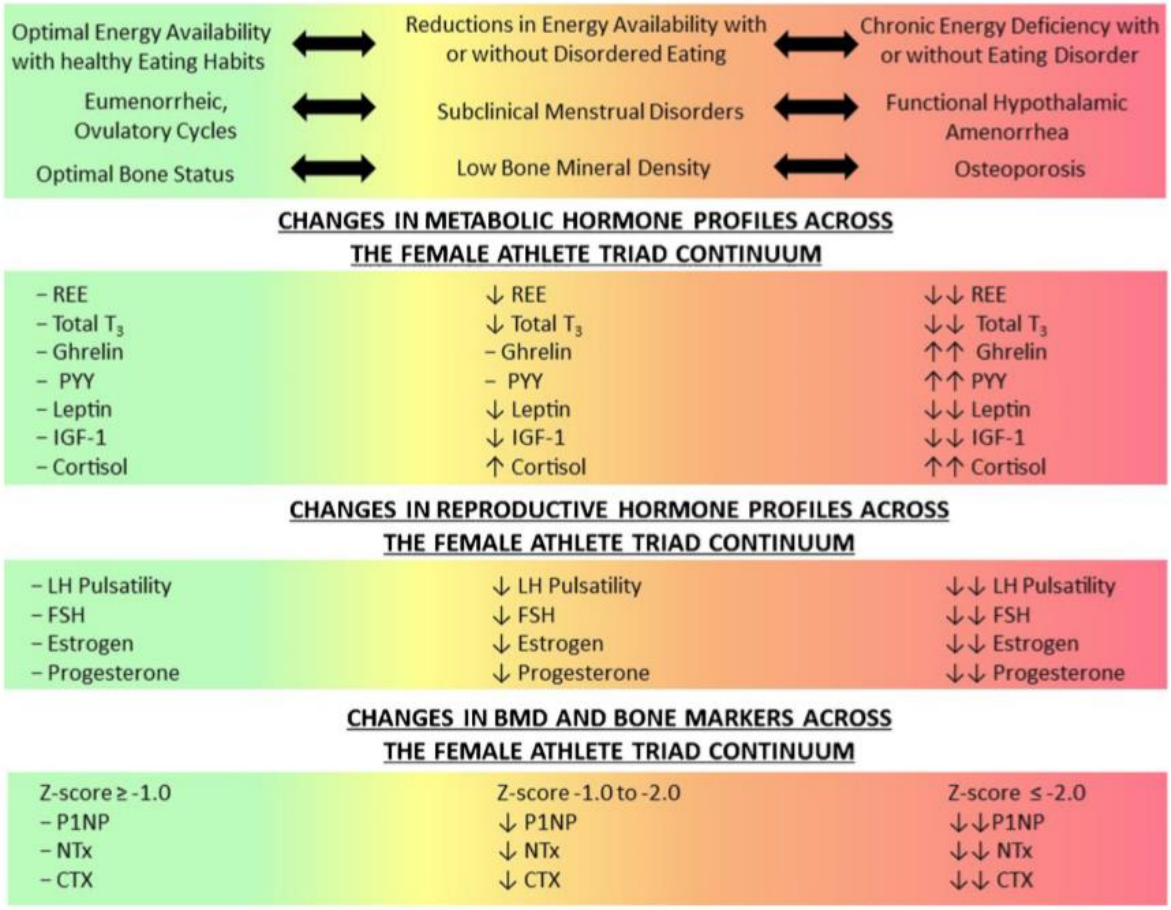


Figure 2.8. Changes in metabolism, reproductive hormones, and bone mineral density evident across the health continuum by De Souza, Koltun, Etter & Southmayd, (2017, p. 578). CTx, C-telopeptide; FSH, follicle stimulating hormone; IGF-1, insulin like-growth factor 1; LH, luteinising hormone; NTx, N-terminal telopeptide; P1NP, procollagen type 1 amino-terminal propeptide; PYY, peptide YY; REE, resting energy expenditure; T3, total triiodothyronine.

Recent studies have investigated the prevalence of LEA in females across an array of sports including dancers (Hoch, et al., 2011), rhythmic gymnasts (Silva, et al., 2015), soccer players (Reed, et al., 2013; Moss, et al., 2020), volleyball players (Woodruff & Meloche, 2013), sprinters and jumpers (Sygo, et al., 2018), endurance athletes (Folscher, et al., 2015; Melin, et al., 2015; 2016; Muia, et al., 2016; Heikura, et al., 2018a; Jesus, et al., 2021), and those combining various sports (Hoch, et al., 2009; Koehler, et al., 2013; Logue, et al., 2019). Such studies have also included groups of athletes from various performance levels including recreational exercisers (Slater, et al., 2016; Black, et al., 2017), active females (Logue, et al., 2019), collegiate athletes (Hoch, et al., 2009; Reed, et al., 2013; Woodruff & Meloche, 2013), competitive athletes (Folscher, et al., 2015), and elite, national, or professional athletes (Hoch, et al., 2011; Koehler, et al., 2013; Melin, et al., 2015; 2016; Sygo, et al., 2018). LEA prevalence ranges across all studies identified in table 2.2 range from 6% to 80%.

To date no studies have investigated the prevalence of LEA in females participating in multi-sport endurance events (e.g., triathlon or duathlon). This is despite leanness sports, particularly endurance sports, being identified as having a greater risk of LEA and prevalence rates ranging from 18% to 80% (table 2.2). The variability in EA methods used (see chapter 2.3.1.3), variability in the sports, performance level, and athletes examined, and the often-small sample sizes recruited (range: 4 to 833 – see table 2.2) have contributed to the consistent, low-quality evidence in available studies. For that reason, it has been advised that further work is required to better understand the prevalence of LEA across all sports, performance levels and athletes (i.e., age groups; Mountjoy, et al., 2014; 2018; Logue, et al., 2018; 2020).

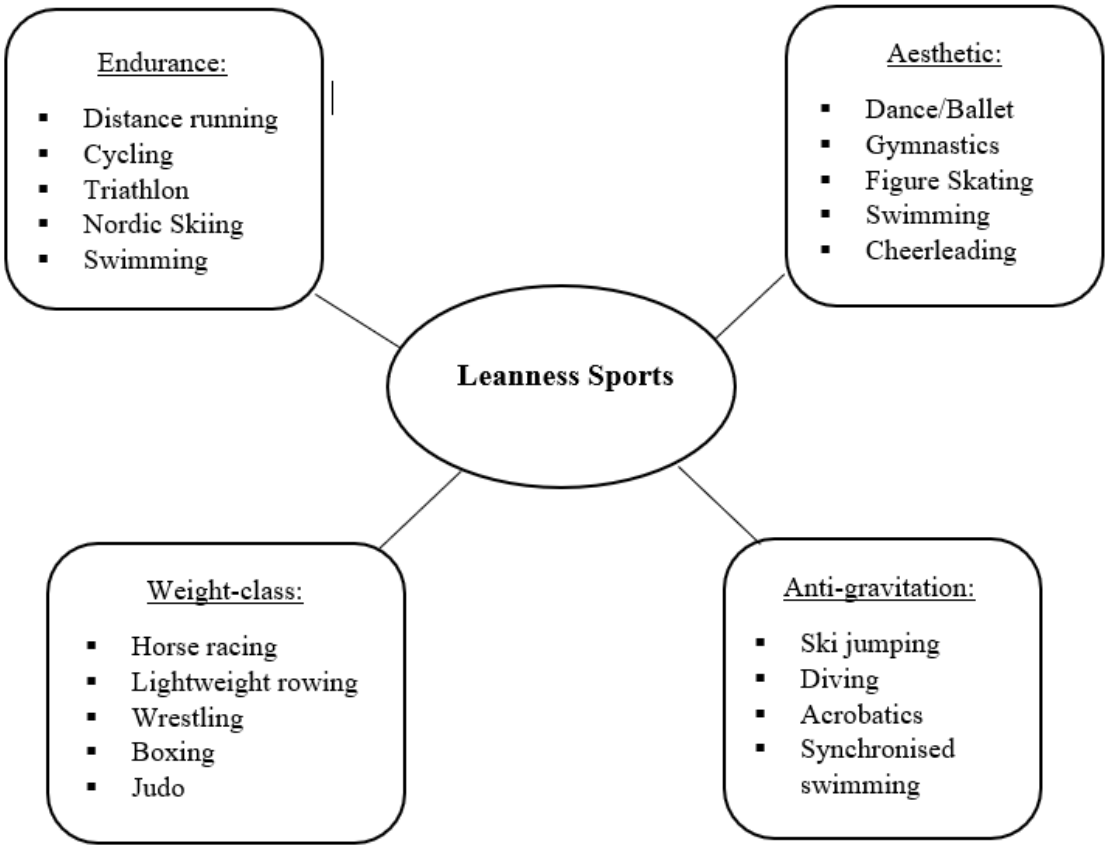


Figure 2.9. Examples of sports emphasising leanness or low body mass.

2.4 Disordered eating and exercise dependence

The Triad and RED-S models have outlined that DE or ED place athletes and/or exercisers at greater risk of developing LEA (Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). DE and ED in females have been associated with exercise dependence (EXD; also called exercise addiction and compulsive exercise behaviour; Bratland-Sanda, Martinsen & Rosenvinge, et al., 2011; Müller, Loeber, Söchtig, et al., 2015; Cook, Wonderlich & Mitchell, et al., 2016). More recently, it has been suggested that athletes with EXD, with or without DE/ED, may also be at greater risk of developing LEA if EI is not matched to such an extreme exercise commitment (Turton, Goodwin & Meyer, 2017). However, to date there is insufficient data available to substantiate this potential association across sports, performance levels or athletes. It is particularly unclear how prevalent EXD is in

female athletes and females participating in multi-sport endurance events like triathlon. As this thesis focuses on the prevalence of DE/ED and EXD in relation to LEA rather than as individual constructs, the following section will provide an overview of DE/ED and EXD pathophysiology, assessment methods and prevalence literature.

2.4.1 Disordered eating behaviour

2.4.1.1 Disordered eating spectrum

Eating behaviours exist upon a spectrum that can progress from optimised nutrition to DE behaviour and ending with overt clinical ED. Movement along the spectrum is considered bi-directional, however, recovery from clinical ED is complex with greater risk of relapse (American Psychiatric Association, 2013). Optimised nutrition has been characterised as individualised nutritional practices that balance health and performance and are considered to be safe, supported, and purposeful (American Psychiatric Association, 2013). Typically, the spectrum of eating behaviour starts with voluntary, healthy dieting evidenced by a reduction in EI and a gradual reduction in body mass. It then progresses to DE behaviour when an individual regularly engages in chronic dieting with the use of more extreme weight-control behaviours, increased pathological eating, body image issues, and frequent weight fluctuations (Sundgot-Borgen & Torstveit, 2010; Joy, Kussman & Nattiv, 2016; Wells, Jeacocke, Appaneal & Smith, et al., 2020). These more extreme weight-control behaviours may include restrictive diets (i.e., skipping meals, total energy and/or nutrient restriction), fasting, regurgitation and eat and spit, binge eating, active (e.g., exercise with sweat suits) and passive (e.g., sauna) dehydration, use of laxatives, diet pills, and diuretics, with or without compulsive exercise training (Nattiv, et al., 2007; Sundgot-Borgen, Meyer, & Lohman, et al., 2013; Wells, et al., 2020). Although an individual may engage with such behaviours, they do not occur with regularity and do not fully meet the criteria for clinical ED (American Psychiatric Association, 2013).

Clinical ED represents the end of the spectrum and are characterised by the frequent occurrence of extreme weight-control behaviours, distorted body image, pre-occupation, and obsession with food, eating, weight and body shape that prevent normal functioning, irrational fear of weight gain, variable athletic performance, and medical complications (American Psychiatric Association, 2013; Sundgot-Borgen, et al., 2013). Clinical ED are considered a clinical mental disorder as defined by the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013) and diagnostic classifications include anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), and ED not otherwise specified (EDNOS; also referred to as other specified feeding and ED (OFSED)).

The most common clinical ED amongst athletes is EDNOS/OFSED, with athletes more likely to present with DE than clinical ED (Sundgot-Borgen & Torstveit, 2004; Bonci, Bonci & Granger, et al., 2008; Martinsen & Sundgot-Borgen, 2013; Reardon, Hainline & Aron, et al., 2019; Wells, et al., 2020). Importantly, athletes who present with DE or ED can be underweight, normal weight or overweight (Torstveit & Sundgot-Borgen, 2011; Sundgot-Borgen, et al., 2013). Although the implications to health and performance are greatest in athletes with clinical ED, the risk exists regardless of placement on the spectrum and increases as eating behaviour deteriorates. As highlighted by the Triad and RED-S models, LEA can occur with or without DE and vice versa, therefore identification of one requires the examination of the other (Nattiv, et al., 2007; Sundgot-Borgen, et al., 2013; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018; Burke, et al., 2018; Wells, et al., 2020).

2.4.1.2 Aetiology for DE/ED

Studies examining the aetiology for DE behaviours and clinical ED in athletes from all performance levels (i.e., recreational to elite) are limited in number, focus, and methodological rigour. Specifically, there are no controlled prospective studies using the gold standard measure

of diagnostic interviews to assess DE or ED onset in athletes. Thus, it is not fully understood why some athletes progress from healthy dieting to chronic dieting with the use of extreme weight-loss methods and pathological eating to clinical ED (Petrie & Greenleaf, 2007; Stice, South & Shaw, 2012; Sundgot-Borgen, et al., 2013). While many theories have existed, currently there is general acceptance that the pathogenesis of DE behaviours and clinical ED is complex and multifactorial (Stice, et al., 2012; Sundgot-Borgen, et al., 2013). One of the main challenges associated with identifying the specific factors that may trigger or predispose an athlete to develop DE behaviour or clinical ED is the difficulty in distinguishing those same factors that are often a result of the impaired eating behaviour (Klein & Walsh, 2004).

To date, these risk factors have included biological, psychological, sociocultural, gender-based, sport-specific, and other factors outlined in figure 2.10 (Nattiv, et al., 2007; Bonci, et al., 2008; Stice, et al., 2012; Bratland-Sanda & Sundgot-Borgen, 2013; Sundgot-Borgen, et al., 2013; Jeacocke & Beals, 2015; Mountjoy, et al., 2018; Ackerman, Holtzman & Cooper, et al., 2019; Buckley, Hall & Lassemillante, et al., 2019; Wells, et al., 2020). Nattiv, et al., (2007) has acknowledged these factors can be considered predisposing (i.e., biological, psychological, and sociocultural), triggers (i.e., trauma or negative body comments), or DE and/or ED can be maintained by perpetuating factors (i.e., positive reinforcement by coach).

Specifically, the role of sport-specific factors in the development of DE behaviour and clinical ED in athletes often relay back to a desire to be leaner, thinner, or more muscular in the quest to enhance performance (Krentz & Warschburger, 2013). A high drive for these physical attributes may too be combined with psychological factors such as body dissatisfaction, distorted body image, low self-esteem, and neuroticism (i.e., anxiety, depression, emotional lability; Beals & Manore, 2000; De Souza, Hontscharuk & Olmsted, et al., 2007; Joy, et al., 2016). The risk of developing DE behaviour may increase, particularly in lean sports, when there is direct or perceived pressure from coaches, athletic peers, or the media

to alter body shape or composition to achieve an 'ideal' physique for their sport (Beals & Manore, 2000; Torstveit & Sundgot-Borgen, 2005; Shanmugam, Jowett, & Meyer, 2014). Additionally, injury and illness, transition periods (i.e., training phase, retirement, non-selection, or de-selection, progressing in performance level), performance pressure, increases in training volume and intensity, weight cycling, group weigh-ins or public display of results, and modelling DE behaviours from their peers may also increase the risk of DE progressing along the spectrum (Sundgot-Borgen, 1994; Krentz & Warschburger, 2013; Arthur-Cameselle, Sossin & Quatromoni, 2017). The role of personality traits such as perfectionism, particularly in female athletes, competitiveness, pain tolerance, or a need for order and symmetry have also been identified as potential risk factors (Bardone-Cone, Wonderlich, Frost & Bulik, et al., 2007; Stirling & Kerr, 2012).

As an individual progress along the DE spectrum the psychological, behavioural, physiological, and social disturbances observed become more persistent and detrimental to overall health and recovery (Klein & Walsh, 2004; Joy, et al., 2016). Nevertheless, further validation work is required to determine a causal relationship between the onset of DE behaviour in athletes and the proposed risk factors discussed (Arthur-Cameselle, et al., 2017; Mountjoy, et al., 2018). It also reiterates the requirement for enhanced screening for DE/ED risk in the athletic population.

2.4.1.3 Health and performance consequences

If DE behaviours or clinical ED are a contributing factor in the development of chronic LEA in athletes, this may lead to the health and performance consequences associated with Triad or RED-S (Loucks, et al., 2011; Bratland-Sanda, et al., 2013; Mountjoy, et al., 2014; 2018). These have previously been discussed in chapter 2.3.3 and depicted in figure 2.4 and 2.7 of this thesis. Current guidance for sport participation in cases of athletes with known ED

are to cease all training and competition and in cases of athletes with known DE are to be cleared for sport participation only with supervised participation and a medical treatment plan (Mountjoy, et al., 2014; 2018).

Specific health consequences that may arise from DE behaviours include cardiovascular (i.e., hypotension, bradycardia), endocrine (i.e., menstrual dysfunction, hypoglycaemia), renal (i.e., dehydration, electrolyte imbalances, oedema), gastrointestinal (i.e., constipation, postprandial distress, swollen parotid glands), dermatological and dental (i.e., dental and gum problems, hair loss, lanugo hair), psychological and behavioural (i.e., insomnia, mood swings, poor coping skills), and other problems (i.e., nutritional deficiencies, anaemia, weight fluctuations; Sundgot-Borgen, et al., 2013; Joy, et al., 2016).

The most severe health consequence associated with clinical ED is death caused by suicide (~20% in AN and ~23% in BN) or cardiac arrhythmia likely caused by electrolyte imbalances (Crow, Peterson & Swanson, et al., 2009; Arcelus, Mitchell & Wales, et al., 2011; American Psychiatric Association, 2013). Smith, et al., (2013) observed a strong association between suicidal behaviour and compulsive exercise (usually observed in athletes) as the DE behaviour in individuals with clinical ED. Performance consequences that may arise from DE behaviours often relate to reductions in training quality and consistency, a greater risk of injury and illness, and an overall reduction in sports performance (Mountjoy, et al., 2014; 2018). There is also evidence to suggest that athletes who weight cycle may also experience impaired performance resulting from nausea, headaches, hot flushes, dizziness, and nosebleeds (Sundgot-Borgen & Garthe, 2011; Sundgot-Borgen, et al., 2013). Athletes presenting with severe cases of clinical ED (AN or BN) should not be involved in sport participation (De Souza, et al., 2014).

Impairments to health and performance are dependent on several factors including the duration of the ED, age, amount and rate of weight loss, frequency of weight fluctuation and health and body composition before weight loss (Joy, et al., 2016). However, the more severe clinical ED and impairments are often observed in individuals presenting with more than one pathogenic DE behaviour (Joy, et al., 2016). Further work is required across athletic groups as to date no studies have examined the health and performance consequences (acute and chronic) in athletes with DE behaviour or clinical ED.

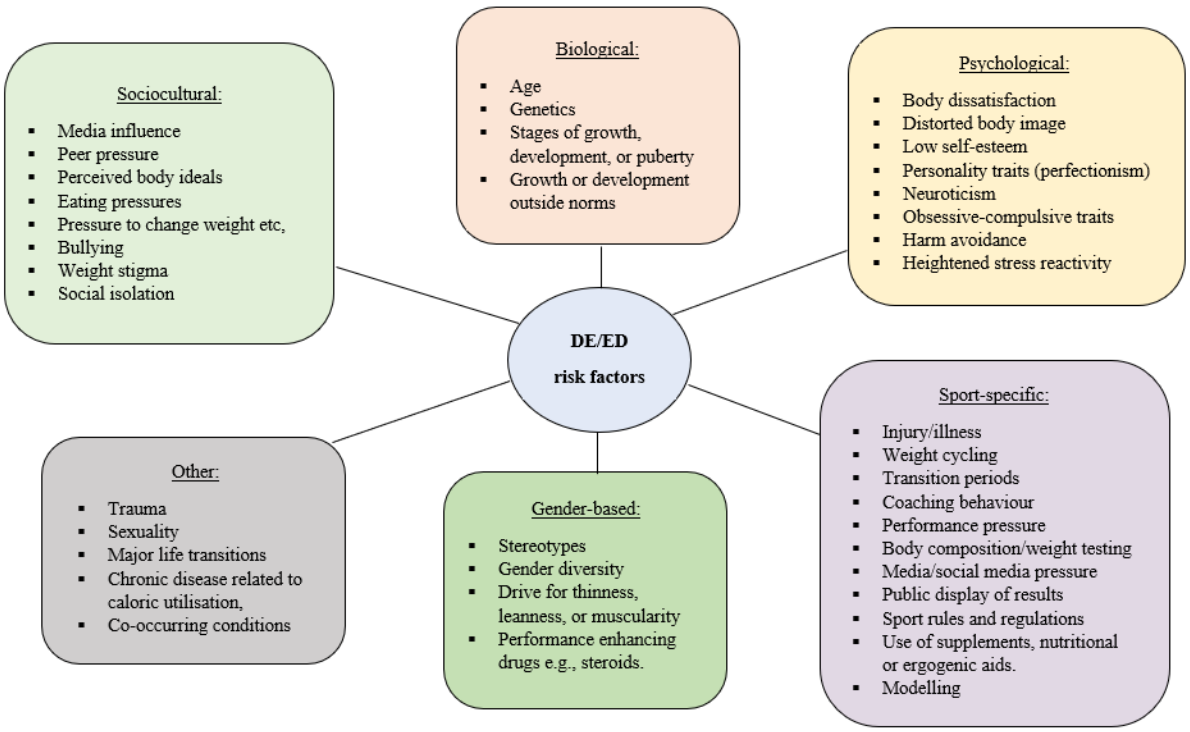


Figure 2.10. Suggested risk factors of disordered eating (DE) and eating disorder (ED) in athletes, adapted from Bratland-Sanda & Sundgot-Borgen, (2013, p. 503) and Wells, et al., (2020, p. 1251

Table 2.4. Estimated prevalence of disordered eating in female athletes in various sport groups and controls.

Author	Population	Age (years)	DE/ED measurement	Prevalence
Beals et al, (2006)	112 female collegiate athletes (various sports)	Mean: 19.5 ± 1.2	EDE-Q + EDI symptoms checklist	*DE:* 25%
Byrne et al, (2002)	155 elite females + 108 elite male athletes (various sports) + 263 controls	Range: 15 – 36	Composite International Diagnostic Interview	*ED:* Female athletes: 22% Female controls: 6% Male athletes: 4% Male controls: 0%
Folscher et al, (2015)	306 female competitive ultra-endurance runners	Mean: 39.5 ± 8.0	Female Athlete Screening Tool	*Subclinical DE:* 27% *Clinical ED:* 5%
Greenleaf et al, (2009)	204 female NCAA Division I college athletes (various sports)	Mean: 20.2 ± 1.3	Questionnaire for ED Diagnosis + 36-item Bulimia Test-Revised	ED: 2% (EDNOS) Symptomatic: 26%

Hauck et al, (2020)	1022 amateur endurance athletes (450 male + 572 female)	Range: 18 – 78 Mean: 36.4 ± 19	Eating Disorder Diagnostic Scale	*At risk of ED:* Total: 7%
Hoch et al, (2009)	80 female varsity athletes (various sports) + 80 non-athlete controls	Athletes mean: 16.5 ± 1.0	EAT-26	*At risk of DE:* Athletes: 4% / Non-athletes: 6%
Hulley et al, (2001)	181 elite female distance runners	Mean: 28.5 ± 0.5	EDE-Q	*ED:* 16% AN: 13%, BN: 4% & EDNOS: 36%
Johnson et al, (1999)	562 females + 883 male collegiate athletes (various sports)	Mean: 19.9	EDI-2 body dissatisfaction, drive for thinness & bulimia	*Clinical ED (AN/BN):* Females: 0%/1% Males: 0%/0% *Subclinical ED (AN/BN):* Females: 3%/9% Males: 0%/0% *At risk ED (AN/BN):* Females: 35%/38% & Males: 10%/38%

Kong et al, (2015)	320 female athletes of varying levels and sports	Range: 17 – 30 Mean: 21.7 ± 3.5	EAT-26	*At risk of DE/ED:* Total: 23% Leanness sports: 35% Non-leanness sports: 9%
Martinsen et al, (2010)	606 elite high school athletes (various sports - F: 217 & M: 389) and 355 age-matched controls (F: 158 & M: 197)	Range: 15 – 16	EDI-2 drive for thinness & body dissatisfaction, BMI, current and/or previous attempts (≥3) to lose weight, use of pathogenic weight control methods & MD (self-report)	*Symptoms of ED:* Athletes (F: 45% & M: 13%) Controls (F: 71% & M: 31%)
Melin et al, (2015)	40 female competitive and elite weight-bearing endurance athletes	Mean: 26.3 ± 5.7	EDE-16 + EDI-3	*Clinical ED:* 25%

Meng et al, (2020)	52 elite and 114 recreational female aesthetic sport athletes	Mean: 20	EDI-3	*ED risk:* Elite: 52% Recreational: 60%
Mongrain et al, (2018)	162 non-elite multisport endurance athletes (female: 48 / male: 114)	Mean: 38.0 ± 11.7	EAT-26	*At risk of ED:* Total: 6% Winter Triathlon: 2% Ironman 70.3: 9% Ironman: 0%
Muia et al, (2016)	61 elite middle-and-long distance adolescent runners + 49 non-athletes	Range: 16 – 17	EDI-3 + Three Factor Eating Questionnaire	*Subclinical DE:* Athletes: 75% Non-athletes: 71% *Clinical DE:* Athletes: 5% Non-athletes: 10%
Muros et al, (2020)	401 female and 3636 male cyclists and triathletes	Mean: 36.1 ± 9.3	SCOFF questionnaire (sick, control, one stone, fat and food)	*At risk of ED:* Male: 1% / Female: 23% / Cyclists: 20% / Triathletes: 16%

Nichols et al, (2006)	170 female high school athletes (various sports)	Range: 13 – 18 Mean: 15.7 ± 1.3	EDE-Q	*DE:* 18%
Nichols et al, (2007)	423 female high school athletes from leanness (146) and non-leanness sports (277)	Range: 13 – 18 Mean: 15.7 ± 1.7	EDE-Q	*DE:* Total: 20% Leanness sports: 19% Non-leanness sports: 21%
Pernick et al, (2006)	453 female high school athletes (various sports)	Range: 13 – 18 Mean: 15.7 ± 1.2	EDE-Q	*DE:*20% African Americans: 19% Caucasians: 18% Latinas: 23%
Pettersen et al, (2016)	225 female adolescent cross-country skiers and biathletes	Competitive age groups: 17, 18 and 19+ years	EDI-2 drive for thinness and body dissatisfaction	*At risk of DE:* Total: 19% Cross-country skiing: 15% Biathlon: 22% Both sports: 24%

Quah et al, (2009)	67 elite female athletes (various sports)	Range: 13 – 30	EDI-2 body dissatisfaction, drive for thinness, bulimia & perfectionism subscales	*At risk of ED:* Leanness sports (n=30): 89% Non-leanness sports (n=37): 89%
Rosendahl et al, (2009)	576 high school elite athletes (various sports – F: 210 & M: 366) and 291 non-athlete controls (F: 169 & M: 122)	Range: 14 – 18	EAT-26	*Females at risk of DE:* Athletes: 26.7% Controls: 36.1% *Males at risk of DE:* Athletes: 10.4% Controls: 12.3%
Rousselet et al, (2017)	340 high level athletes (leanness and non-leanness sports) Male: 213 / Female: 127	Mean: 16.8 ± 3.5	DSM-IV criteria + EDI	*DE detected:* 33% of all athletes – 47% of those athletes were female and 50% from leanness sports. *No DE detected:* 67% of all athletes – 33% were female and 31% from leanness sports

Schaal et al, (2011b)	2067 adolescent and adult elite athletes (various sports) F: 728 / M: 1339	Range: 12 – 35 Mean: 18.5 ± 4.9	Psychological consultation using DSM-IV for AN, BN & EDNOS	*Current ED (< 6 months):* At-least 1 ED – F: 7%, M: 4%, all: 5% AN & BN – <1% across all groups EDNOS – F: 6%, M: 4%, all: 4% *Lifetime ED:* At-least 1 ED – F: 11%, M: 6%, all: 8% AN: 2% & BN: 3% all groups EDNOS – F: 9%, M: 5%, all: 6%
Schtyscherbyna et al, (2009)	78 adolescent elite swimmers	Range: 11 – 19 Mean: 14.6 ± 2.0	EAT-26 + Bulimic Investigatory Test Edinburgh (BITE) + Body Shape Questionnaire (BSQ)	*At risk of DE by one of the tests:* 45% *EAT-26*: 7.7% *BITE*: 22% *BSQ*: 37%
Sundgot-Borgen et al, (1993)	133 elite female athletes (various sports) + 60 controls	Range: 12 – 35	Diagnostic survey for ED based on DSM-III – structured clinical interview	*ED:* Athletes: 18% Controls: 5%

Sundgot-Borgen et al, (2004)	572 elite females + 687 elite male athletes (various sports) + 574 females + 629 male controls	Range 15 – 39 Athletes & Controls (F) mean: 21.4 ± 4.6 & 24.7 ± 6.5. Athletes & Controls (M) mean: 23.2 ± 4.9 & 25.2 ± 6.2	EDE structured clinical interview	*ED:* Female athletes: 20% Female endurance athletes (n=102): 24% Female controls: 9% Male athletes: 8% Male endurance athletes (n=149): 9% Male controls: 1%
Thein-Nissenbaum et al, (2011)	311 female high school athletes (various sports) Aesthetic sports: n=41 Endurance: n=89 Team/Anaerobic: n=181	Mean: 15.4 ± 1.2	EDE-Q (self-report)	*DE:* Total: 35.4% Aesthetic Sports: 41.5% Endurance Sports: 37.1% Team/Anaerobic Sports: 33.1%
Toro et al, (2005)	283 elite female athletes (various sports)	Mean: 15.3 ± 3.1	EAT + Eating Disorders Evaluation Questionnaire	*EAT:* 11% ED Evaluation Questionnaire AN: 3% ED Evaluation Questionnaire BN: 20%

Torstveit et al, (2008)	186 elite female athletes (various sports) + 145 controls	Range: 13 – 39 Mean athletes: 22.2 ± 5.8 Mean controls: 29.6 ± 7.9	Eating Disorder Examination (EDE) structured interview for clinical ED + EDI-2 drive for thinness & body dissatisfaction for DE	*Total clinical ED:* Athletes: 33% Controls: 21% *1+ of the five indicators of DE:* Athletes: 46% Controls: 52% Leanness sports: 49% Non-leanness sports: 44%
Vardar et al, (2007)	240 competitive female athletes (various sports)	Range: 15 – 25 Mean: 19 ± 2	EAT-40	*DE:* 17%

AN, anorexia nervosa; BMI, body mass index; BN, bulimia nervosa; DE, disordered eating; DSM-III, diagnostic and statistical manual of mental disorder, third edition; DSM-IV, diagnostic and statistical manual of mental disorder, fourth edition; ED, eating disorder; EDE-Q, eating disorder examination questionnaire; EDI, eating disorder inventory; EDNOS, eating disorder not otherwise specified; MD, menstrual dysfunction; F, female; M, male.

2.4.1.4 Assessment of disordered eating behaviour

The importance of prevention, early detection and treatment of DE behaviour and clinical ED in athletes have been widely acknowledged in published position stands by several sports medicine organisations (including IOC, ACSM, Australian Institute of Sport, and the National Athletic Trainer Association; Sherman & Thompson, 2006; Nattiv, et al., 2007; Bonci, et al., 2008; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018; Wells, et al., 2020). This is irrespective of the presence of Triad or RED-S as clinical ED have one of the highest mortality rates (crude mortality ~5% per decade) among all mental health conditions (Harris & Barraclough, 1998; American Psychiatric Association, 2013; Chesney, Goodwin & Fazel, 2014; Mehler & Brown, 2015). There is universal agreement that an individual's prognosis and recovery improve with the early detection and treatment of DE behaviour (Nattiv, et al., 2007; Bonci, et al., 2008; Ozier & Henry, 2011; Bratland-Sanda, et al., 2013; Chesney, et al., 2014; Mountjoy, et al., 2018; Wells, et al., 2020).

Although predominantly targeted at elite athletes, the current recommendation for the assessment of DE behaviour in athletes comprises of a self-report screening tool and subsequent clinical interview for diagnostic purposes using sport-specific resources where available (Bonci, et al., 2008; Reardon, et al., 2019). A range of standardised, self-report screening tools and questionnaires designed and validated to assess DE behaviour in the general population exist. These resources are frequently utilised in the assessment of DE in athletes as shown in table 2.4. The current gold standard measure for diagnosing overt, clinical ED in the general population is the Eating Disorder Examination 17.0 (EDE 17.0). This includes the 36-item EDE questionnaire (EDE-Q) and a semi-structured, clinical interview conducted by a specialised clinician (Fairburn, Cooper & O'Connor, 2014). More accessible screening tools validated in the general population often used include the Eating Disorder Inventory (EDI; developed to characterise the psychological characteristics of anorexia and bulimia; Garner,

Olmsted, & Polivy, 1983; Garner, 1991; Garner, 2004), the Three Factor Eating Questionnaire (TFEQ; measuring dietary restraint, disinhibition, and hunger; Stunkard & Messick, 1985), the Eating Attitudes Test (EAT; Garner, Olmsted, Bohr & Garfinkel, 1982), and the SCOFF Questionnaire (Morgan, Reid & Lacey, 2000). Importantly, these screening tools and questionnaires have demonstrated suboptimal psychometric properties in athletes despite their widespread use and may lead to inaccurate diagnosis (Bonci, et al., 2008; Pope, Gao, Bolter & Pritchard, 2015).

Validated screening tools and questionnaires to assess DE behaviour in athletes are limited in number and quality (Knapp, Aerni & Anderson, 2014; Wagner, Erickson & Tierney, 2016; Wells, et al., 2020). The four most common include the Athletic Milieu Direct Questionnaire (Nagel, Black, Leverenze & Coster, 2000), the Physiological Screening Test (Black, Larkin, Coster & Leverenze et al, 2003), the Brief ED in Athletes Questionnaire (Martinsen, Holme, Pensgaard & Torstveit et al, 2014), and the Female Athletes Screening Tool (FAST; McNulty, Adams, Anderson & Affenito, 2001). Despite all being able to discriminate between athletes with and without DE behaviour they should be used with caution. It is currently unknown if these tools are valid for athletes across all sports, ages, and performance levels as they have only been validated in female collegiate athletes and female adolescent athletes. It is also unclear if these screening tools can be generalised to use with male athletes (Knapp, et al., 2014). Further work is required to increase the validity of existing screening tools and questionnaires designed for athletes by assessing the strength of their psychometric properties (i.e., content and convergent validity, test-retest, and internal consistency reliability) across athletic populations (including males and females, elite and non-elite). It is recommended that athletes identified with DE behaviour subsequently undergo a nutritional assessment for LEA which was discussed in Chapter 2.3.1.2 of this thesis.

Additionally, assessment should not focus on assessment of body mass as athletes can be weight stable and present with DE, ED, or LEA (Wells, et al., 2020).

Despite the importance of early detection and treatment of DE behaviour and clinical ED, several barriers exist. As previously highlighted in chapter 2.3.1.2, subjective measures are inherently inaccurate due to false reporting and compliance. This may be exacerbated in the athletic population when screening for DE behaviour and clinical ED due to the stigma, shame and discrimination often associated with disclosing mental health concerns (Bonci, et al., 2008; Ozier, et al., 2011; Walker & Lloyd, 2011; Reardon, et al., 2019). Rather than seeking direct help for DE behaviour, athletes often seek help indirectly for a health consequence associated with DE or clinical ED (Joy, et al., 2016). Alternatively, from a coach or parental perspective limited knowledge related to signs and symptoms of DE behaviours or how to approach an athlete may also prevent early detection (Reardon, et al., 2019). From a clinical perspective, poor understanding of an individual's sports environment may prevent the distinction between acceptable and problematic behaviours. Thus, signs and symptoms of DE behaviour may be masked (Chapa, Hagan & Forbush, et al., 2018; Ströhle, 2019; Wells, et al., 2020).

2.4.1.5 DE/ED prevalence research

As previously stated, the screening or identification of either LEA or DE behaviours in athletes necessitates the examination of the other (Nattiv, et al., 2007; Sundgot-Borgen, et al., 2013; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018; Burke, et al., 2018; Wells, et al., 2020). This is due to the absence of a singular screening tool or questionnaire that can identify individuals at risk of LEA without the presence of DE behaviours or clinical ED (Joy, et al., 2016). Table 2.4 provides an overview of studies reporting the prevalence of DE and ED in predominantly female athletes from various sports or between athletes and controls. A narrative

review was conducted for the articles included in table 2.4 using targeted internet searches (i.e., Google Scholar and PubMed). Combinations of the following key search terms were included: athlete, endurance athlete, recreational exercise, elite athlete, non-elite athlete, DE, ED, eating attitudes, eating behaviour, EAT-26, FAST, EDI, and EDE. Articles were considered if written in English, in full-text, and were conducted among free-living trained or exercising human subjects. Only studies that quantified the assessment of DE/ED and screened for the prevalence within the text of the manuscript were included. No time limit on retrieval of articles was set and reference lists of articles retrieved were also reviewed.

Overall, the athletic population are more likely to have a greater prevalence of DE and/or ED than the general population (Sundgot-Borgen & Torstveit, 2004; Bratland-Sanda, et al., 2013; Sundgot-Borgen, et al., 2013). Plus, athletes from sports that emphasise leanness or low body mass as advantageous for performance (figure 2.9), are more likely to be identified at risk for DE and/or ED (Nattiv, et al., 2007; Bratland-Sanda, et al., 2013; Sundgot-Borgen, et al., 2013; Joy, et al., 2016; Mountjoy, et al., 2018). Despite a high prevalence of DE and/or ED observed in elite athletes, DE and ED can occur in any athlete, irrespective of gender, sport, age, culture, performance level, socioeconomic background, time, or body composition/weight (Sundgot-Borgen, et al., 2013; Joy, et al., 2016; Wells, et al., 2020).

Many of the studies examining the prevalence of DE and/or ED have focused on elite female athletes (Sundgot-Borgen, et al., 1993; 2004; Hulley, et al., 2001; Torstveit, et al., 2008; Schaal, et al., 2011b; Muia, et al., 2016), high school athletes (Nichols, et al., 2006; 2007; Pernick, et al., 2006; Rosendahl, et al., 2009; Martinsen, et al., 2010; Thein-Nissenbaum, et al., 2011), and collegiate athletes (Johnson, et al., 1999; Beals, et al., 2006; Greenleaf, et al., 2009; Hoch, et al., 2010). Much of these studies have combined athletes from various sports (Sundgot-Borgen, et al., 1993; 2004; Vardar, et al., 2007; Rosendahl, et al., 2009; Quah, et al., 2009; Martinsen, et al., 2010; Kong, et al., 2015), or focused on single-sport endurance events

(Hulley, et al., 2001; Schtyscherbyna, et al., 2009; Folscher, et al., 2015; Melin, et al., 2015; Muia, et al., 2016; Petterson, et al., 2016; Hauck, et al., 2020). Many of the studies presented in table 2.4 include adolescent or young adult Caucasian athletes, from North American or Scandinavian cohorts, who are able-bodied. Prevalence ranges for athletes at risk of DE (including ED) across all studies identified in table 2.4 range from 4% to 89%.

Importantly, estimates of DE and/or ED prevalence remain unclear across athletic populations and are likely to be significantly underestimated due to the stigma and discrimination associated with mental health conditions (Joy, et al., 2016). A reflection of the often-unreliable results reported resulting from differing criteria and definitions used, variability in the screening tools and questionnaires used that are frequently self-report and unvalidated in athletes, lack of or unsuitable control groups, small sample sizes, and heterogeneous samples examined (Byrne & Mclean, 2001; Nattiv, et al., 2007). Currently, there are a limited number of studies examining DE and/or ED in athletes using the gold standard assessment method to obtain unbiased and reliable estimates of prevalence. Sundgot-Borgen, et al., (2004) examined 572 elite females and 687 elite males from various sports compared to controls using the EDE. In females, it was found that 20% of all females and 24% of female endurance athletes (n=102) had clinical ED compared to 9% of the general population. Despite leanness sports being at greater risk for the development of DE and ED, limited studies exist examining the prevalence in females participating in multi-sport endurance events (e.g., triathlon). Recently, Mongrain, et al., (2018) examined 162 non-elite male and female multi-sport endurance athletes using the EAT-26 and found 6% at risk of ED. Muros, et al., (2020) examined 401 female and 3636 male cyclists and triathletes using the SCOFF and found 23% of females and 16% of triathletes at risk of ED.

It should also be acknowledged that prevalence estimates identified in table 2.4 are largely based on early versions of the DSM which may influence previous diagnoses in athletes

(American Psychiatric Association, 2013). Although self-report measures only provide an estimate their use remains valuable in identifying at-risk groups to enable early intervention and target awareness (Bratland-Sanda, et al., 2013). Future work is advised to continue examining the prevalence of DE and ED across a range of athletic populations (i.e., sports, gender, cultures, performance level, age; Mountjoy, et al., 2014; 2018; Joy, et al., 2016; Wells, et al., 2020).

2.4.2 Exercise dependence

2.4.2.1 Definitions and classification

Pathological exercise was first identified in the 1970s and despite growing support its existence continues to spark debate in the literature (Baekeland, 1970; Morgan, 1979; Hailey & Bailey, 1982). It is recognised individuals can develop a negative relationship with exercise, however, several barriers in the literature exist. Currently, there is no universally accepted definition or classification of pathological exercise (Hausenblas & Downs, 2002a; Landolfi, 2013). This has led to a variety of terms being used including compulsive exercise (Dalle Grave, Calugi & Marchesini, 2008; Holland & Tiggemann, 2017; Dittmer, Jacobi & Voderholzer, 2018), exercise addiction (Adams & Kirkby, 2002; Aidman & Woollard, 2003; Oberle, Watkins & Burkot, 2018), exercise dependence (Hausenblas & Downs, 2002b; Bamber, Cockerill, Rodgers & Carroll, 2003; Adams, 2009; MacIntyre, Heron, Howard & Symons Downs, 2020), excessive exercise (Long, Smith, Midgley & Cassidy, 1993; Shroff, Reba, Thornton & Tozzi, et al., 2006), obligatory (Thompson & Pasman, 1991; Brehm & Steffen, 1998; Serier, Smith, Lash & Gianini, 2018), and overcommitted (Yates, Shisslak, Crago & Allender, 1994). Current literature often does not provide clear definitions making it difficult to understand if the variety of terms used all denote the same concept (Landolfi, 2013). This variety in terminology has led to challenges in establishing a consensus due to

misunderstanding, ambiguity, irreproducible results, and interpretation errors (Cook & Hausenblas, 2008; Landolfi, 2013; Szabo, Griffiths, Marcos & Mervó, 2015; Bratland-Sanda, Mathisen, Sundgot-Borgen & Rosenvinge, 2019).

Debate in the literature also exists in relation to the proposal that pathological exercise can be considered as either a primary disorder or a secondary symptom of another pathological disorder. This proposal emphasises the core motivation behind the exercise behaviour (Veale, 1987; Szabo, 2010). Classification as a primary disorder refers to the absence of a clinical ED (AN or BN), whereby, the motivation for exercise is the reduction of negative affect for exercise in itself and not solely for weight loss. In contrast, a secondary symptom refers to the co-existence with another pathological disorder, most often clinical ED, where the motivation for exercise is weight loss (Veale, 1987; Bamber, Cockerill & Carroll, 2000; Blaydon, Lindner & Kerr, 2002; Szabo, 2010; Scharmer, Gorrell, Schaumberg & Anderson, 2020). However, this theory remains critically challenged as little is currently known about the aetiology of pathological exercise, irrespective of primary or secondary classification (Szabo, 2010; Berczik, Szabo, Griffiths & Kurimay, et al., 2012; Cunningham, Pearman & Brewerton, 2016; Colledge, Cody, Buchner & Schmidt, et al., 2020). Moreover, this classification within the literature has often characterised primary pathological exercise as addictive in nature and secondary as compulsive (Cunningham, et al., 2016). Yet, research by Cook, et al., (2014) has suggested secondary pathological exercise, specifically with clinical ED, exhibits greater levels of addictive and compulsive qualities. Thus, highlighting the importance of continued work in this area to further elucidate these findings.

Despite the variability in terminology, it has been proposed by some authors that exercise addiction may be the most applicable term due to the inclusion of both compulsion and dependence (Goodman, 1990; Szabo, 2010; Berczik, et al., 2012). In a recent analytical review by Szabo, et al., (2015) it was reported that the most frequently used term in the

literature was exercise dependence. For the that reason, this thesis will use the term exercise dependence (EXD) to facilitate comparison between studies, however, it is acknowledged that several terms exist that aim to conceptualise pathological exercise. No definitive definition of EXD exists (Hausenblas & Downs, 2002a; 2002b; Marques, Peralta, Sarmento & Loureiro, et al., 2019). Hausenblas and Downs (2002b) recommended, based on the DSM-IV definition of substance dependence (American Psychiatric Association, 2000) and the early work of Veale (1987), that EXD is conceptualised as a "*multidimensional maladaptive pattern of exercise leading to clinically significant impairment or distress"*, including physiological, cognitive, and behavioural symptoms. EXD is defined by presentation of three or more of these symptoms: tolerance, withdrawal, intention effects, lack of control, time, reduction in other activities, and continuance (outlined in chapter 3.4.3; Hausenblas & Downs, 2002b; Bamber, et al., 2003; Allegre, Souville, Therme & Griffiths, 2006).

Although likened to other behavioural addictions (e.g., gambling disorder), EXD or any variation of pathological exercise has not been positioned as a mental disorder in the DSM-V. A result of insufficient peer-reviewed evidence and methodological rigour (American Psychiatric Association, 2013). More recently, Bratland-Sanda, et al., (2019) has proposed the concept of pathological exercise should instead be aligned more closely to the DSM-V criteria for obsessive-compulsive disorder. It is suggested this may enable further understanding and evaluation of the obsessions and compulsions underpinning pathological exercise rather than the frequency and motives.

2.4.2.2 Theoretical models, risk factors, and consequences

Theoretical research is the current focus in the literature as little is currently understood about the aetiology of EXD. To date several theoretical models have been proposed (Egorov & Szabo, 2013; Landolfi, 2013; Chen, 2016). A physiological model, the Sympathetic Arousal

Hypothesis, proposed by Thompson and Blanton (1987) suggests EXD relates to an individual's adaptation to habitual exercise in a cycle of sympathetic activity. Briefly, it suggests lower levels of sympathetic arousal may occur at rest when an individual engages in regular exercise. This may lead to physical feelings of lethargy and tiredness, and psychological feelings of being low or negative, resulting in the desire to increase arousal. When exercise is used as a means to increase arousal, the increased arousal levels are temporary, leading to increased levels (i.e., frequency and volume) of exercise being required to reach optimal arousal. This refers to the tolerance component of the EXD concept. The main limitation of this model relates to sympathetic adaption to exercise is universal, yet EXD is only experienced by a small percentage of exercisers (~3%; Sussman, Lisha & Griffiths, 2011).

Szabo (1995) proposed the Cognitive Appraisal Hypothesis which accounts for individuals (although it is unknown who) using exercise as a coping mechanism for life-stress. The individual rationalises the use of exercise as a healthy coping mechanism, however, in order to function the individual becomes dependent on it. Withdrawal symptoms may become evident when life-obligations and daily activities force a reduction in exercise levels. Thus, in the absence of exercise (coping mechanism) they become more vulnerable to stress and in order to abate the negative feelings they resume previous patterns of exercise, at the expense of other obligations. The main limitation of this model is the lack of explanation for the onset of exercise dependence as it only depicts the maintenance of dependence (Egorov & Szabo, 2013).

Hamer and Karageorghis (2007) proposed a psychobiological model examining the role of interleukin-6 (IL-6) on EXD. It suggests that exercise may act as a trigger (although unknown) that results in elevated levels of IL-6 that result in cytokine-induced sickness behaviours, causing negative affect. Individuals may use exercise to increase arousal which creates the exercise-increased IL-6 loop. The main limitation of this model is that it does not

account for why an individual chooses exercise over substance abuse for example (Egorov & Szabo, 2013).

Freimuth, et al., (2011) proposed a Four Phased Model of EXD with each phase focused on the components of motivation, consequences, and frequency/control. Phase 1 represents recreational exercise and is considered to be pleasurable activity with minor negative consequences (i.e., muscle soreness or minor strains). Phase 2 represents at-risk exercise which is characterised by the adoption of exercise as a coping mechanism to improve mood. Phase 3 represents problematic exercise where an individual is highly engaged with rigid organisation of exercise, and negative consequences are greater due to exercise being the sole coping mechanism. The final phase represents an individual meeting the symptoms to be considered as EXD. Although the model suggests the onset of EXD occurs in phase 2, it does not provide detail regarding the distress that causes the onset of EXD (i.e., gradual, or sudden), the conditions resulting in the adoption of exercise as a coping mechanism, or who specifically in the exercising population is at risk of using exercise as a coping mechanism and why (Egorov & Szabo, 2013).

McNamara and McCabe (2012) proposed the Biopsychosocial model which has focused on elite athletes. The model has proposed that EXD is triggered by biological factors (i.e., BMI) that interact with psychological (i.e., self-esteem) and social factors (i.e., coaches) that determine the onset of EXD. However, this model has been criticised in relation to its focus on elite athletes. It has been advised that the intense training and ambitious strivings for success evident in elite athletes does not compare to the proposed symptoms of EXD. The model suggests biological factors are the onset of EXD, but it has been argued that addictions or dependence originate from psychological factors and biological factors affect psychology (Freimuth, Moniz & Kim, 2011; Egorov & Szabo, 2013). Nevertheless, it is important to acknowledge that these counterarguments do not suggest that elite athletes are not at risk of

developing poor coping mechanisms or pathological exercise. Rather, it is the perspective from which EXD is examined that is contested.

The final model proposed by Egorov and Szabo (2013) is in line with the Pragmatics, Attraction, Communication and Expectation model for general addictions and is called the Interactional Model. It is proposed the primary motivation for exercise is determined by the interaction between environmental and personal factors. These exercise motivations may be described as therapeutic-orientation or mastery-orientation. The model acknowledges that an individual may experience a gradual or sudden intolerable life-stressor that causes the individual to seek a means to cope. It is the subconscious and conscious interaction between these motivations for exercise and previous exercise behaviours that may determine if an individual uses exercise as a coping mechanism. It is at this phase that mastery-orientated exercisers may change to therapeutic-orientation. The greater the perceived benefit of exercise for coping, the more likely an individual is to continue using it as a coping mechanism (Egorov & Szabo, 2013). Overall, the inconsistencies evident between these models clearly evidence a lack of aetiologic consensus across the field.

To date, several risk factors for the development of EXD have been proposed (Back, Josefsson, Ivarsson & Gustafsson, 2019). Previous research has suggested EXD is significantly associated with anxiety and obsessive-compulsive disorders in both general and exercise populations (Ogden, Veale & Summers, 1997; Gulker, Laskis & Kuba, 2001; Spano, 2001; Grandi, Clementi, Guidi & Benassi, et al., 2011; Costa, Hausenblas, Oliva & Cuzzocrea, et al., 2013; Landolfi, 2013; Young, Rhodes, Touyz & Hay, 2013; Paradis, Cookie, Martin & Hall, 2013; Schreiber & Hausenblas, 2015). Hausenblas & Giacobbi (2004) also suggested personality traits such as impulsiveness and extroversion may also have a higher risk for EXD. Several studies have also reported significant associations between obsessive passion (intra- and/or interpersonal pressure to participate in exercise and feelings of guilt or anxiety when

unable to; Vallerand, Blanchard, Mageau & Koestner, et al., 2003; Parastatidou, Doganis, Theodorakis & Vlachopoulos, et al., 2012; Kovacsik, Griffiths, Pontes & Soós, et al., 2018; Back, et al., 2019). Finally, distorted body image, body dissatisfaction and physical appearance-orientated individuals have also been significantly associated with the development of EXD when exercise is used to achieve body ideals (Hausenblas & Giacobbi, 2004; Hausenblas & Fallon, 2006; Landolfi, 2013; Schreiber & Hausenblas, 2015).

Although the consequences of EXD are not fully elucidated and require further work, several co-occurring dependencies related to EXD have been proposed to negatively influence health. These have included dependence on work, illegal drugs, nicotine, buying, alcohol, sex, and the internet (Freimuth, et al., 2011; Landolfi, 2013; Lichtenstein, Hinze & Emborg, et al., 2017). Sussman, et al., (2011) estimated a third of individuals with EXD will also have co-occurring dependencies. In addition to co-dependencies, individuals with EXD may also experience negative consequences related to pain, injury, impaired social life, reduced sleep quality, depression, and anxiety related to overtraining (Landolfi, 2013; Lichtenstein, et al., 2017; Marques, et al., 2020). Importantly, current evidence does not suggest the level of psychological morbidity is sufficient to indicate psychological distress as defined by the DSM-V (American Psychiatric Association, 2013). Although complex and poorly understood, there is significant literature focusing on the significant associations between DE behaviour, clinical ED, and EXD (Bratland-Sanda, et al., 2011; Freimuth, et al., 2011; Sussman, et al., 2011; Muller, Loeber, Sochtig & Te Wildt, et al., 2015; Cook, et al., 2016; Scharmer, et al., 2020). The negative health consequences of EXD in the context of DE behaviour and clinical ED is outlined in chapter 2.4.1.3. Recently, it has been proposed that the link between EXD and DE behaviour may also be associated with a greater risk of Triad or RED-S (Turton, et al., 2017). A study by Torstveit, et al., (2019) found significant associations between higher EXD scores, ED symptoms, biomarkers of RED-S in healthy male endurance athletes. However, studies

investigating these potential associations are in their infancy and further work is required across all athletic groups (i.e., gender, performance level and sports).

2.4.2.3 Assessment methods

Unfortunately, there is no consensus for the assessment of EXD, however, it has been acknowledged that the volume and intensity of exercise is not an indicator of EXD (Lichtenstein, et al., 2017). Several assessment instruments have been developed for the assessment of pathological exercise (Berczik, et al., 2012; Lichtenstein, et al., 2017). These include the Negative Addiction Scale (NAS; Hailey & Bailey, 1982), the Obligatory Exercise Questionnaire (OEQ; Thompson & Pasman, 1991), the Running Addiction Scale (RAS; Chapman & De Castro, 1990), the Exercise Dependence Questionnaire (EDQ; Ogden, et al., 1997), and the Exercise Beliefs Questionnaire (EBQ; Loumidis & Wells, 1998). The two most frequently adopted and well-validated instruments are the Exercise Addiction Inventory (EAI; Terry, Szabo & Griffiths, 2004) and the Exercise Dependence Scale (EDS; Hausenblas & Downs, 2002b).

Terry, et al., (2004) developed the 6-item EAI in the context of the theoretical concepts of behavioural addiction proposed by Brown (1997). Each item is indicative of one of the addictive behaviour components and rated on a 5-point Likert scale with a maximum score of 30. Cut-off points of ≥24 indicates risk of addiction, a score of 12-23 indicates symptomatic, and <12 indicates asymptomatic individuals. Hausenblas and Downs (2002b) developed the 29-item EDS in the context of DSM-IV diagnostic criteria for substance abuse (e.g., tolerance, time – see chapter 2.4.2.1). Downs, et al., (2004) revised the EDS (EDS-R) to 21-items which is outlined in detail in chapter 3.4.3. The EDS-R produces a mean score and categorises individuals as at-risk of EXD, non-dependent symptomatic, or non-dependent asymptomatic (Hausenblas & Downs, 2002b).

Although good psychometric properties have been reported in the abovementioned assessment instruments, the heterogeneity of these instruments has led to considerable variance in reported prevalence rates (Lichtenstein, et al., 2017). For instance, the NAS and RAS only focus on a specific sport but the EDS and EAI focus on general physical exercise. The EAI is developed based on addiction theory but the EDS on diagnostic DSM-IV criteria. The EDS and EBQ do not have cut-off points but the EAI and EDQ do (Lichtenstein, et al., 2017). Importantly, while the EAI and EDS are frequently used, self-report instruments cannot be used as diagnostic tools for EXD. This is due to the inherent issues previously discussed of using self-report measures (i.e., honesty and compliance), the lack of empirical research, and inaccurate interpretation of data (Szabo, et al., 2015). It has been recommended that self-report assessment instruments be supplemented with clinical interview. This will allow confirmation of true positives, false positives, true negatives, and false negatives (Müller, Cook, Zander & Herberg, et al., 2014; Szabo, et al., 2015; Lichtenstein, et al., 2017).

2.4.2.4 Prevalence research

To date, the prevalence of EXD remains unclear due to the issues highlighted regarding definitions, theoretical models, risk factors, and assessment instruments (Berczik, et al., 2012; Landolfi, 2013; Lichtenstein, et al., 2017; Di Lodovico, Poulnais & Gorwood, 2019; Marques, et al., 2019). Additionally, current research examining the prevalence of EXD using self-report assessment instruments are in fact measuring the prevalence of individuals 'at-risk' of EXD, rather than 'diagnosed' EXD (Szabo, et al., 2015). Mónok, et al., (2012) reported the prevalence rate of EXD in the general population ranged from 0.3% to 0.5% and a study by Sussman, et al., (2011) reported up to 3% in general population. A recent review by Marques, et al., (2019) highlighted the prevalence in regular exercisers ranged between 2% and 10% in 14 identified studies using either the EAI or EDS. In university students, Marques, et al., (2019) identified a prevalence ranging from 3% to 21% in 8 identified studies and in the athlete

population a prevalence ranging from 1% to 17% in 11 identified studies using either the EAI or EDS. However, some studies have reported higher prevalence rates ranging from 3% to 52% in various populations (Blaydon & Lindner, 2002; Lejoyeux, Avril, Richoux & Embouazza, et al., 2008; Villella, Martinotti & Nicola, et al., 2011; Lejoyeux, Guillot, Chalvin & Lequen, 2012; McNamara & McCabe, 2012; Szabo, Vega Rde, Ruiz-BarquIn & Rivera, 2013; Lichtenstein & Jensen, 2016).

Currently, there is limited data available examining the risk of developing EXD in specific athletic groups (i.e., sports, age, gender, performance level, culture). A recent review by Di Lodovico, et al., (2019) examined which sports are more likely to be at-risk of EXD using the EAI and/or EDS. The review identified 48 cross-sectional, observational studies from various sports (EAI: n=20, EDS: n=26, EAI & EDS: n=2). In studies using the EAI, endurance sports reported the highest prevalence at 14% and mixed disciplines, health and fitness, power sports, and general population were reported at 10%, 8%. 6% and 3%, respectively. In studies using the EDS, mixed disciplines reported the highest prevalence at 15% and endurance sports, health and fitness, power sports, and general population were reported at 4%, 6%, 11% and 2%, respectively (Di Lodovico, et al., 2019).

Both Marques, et al., (2019) and Di Lodovico, et al., (2019) found limited studies differentiating between primary and secondary risk for EXD or between the categorisations found within the EDS of ‘at-risk’, ‘non-dependent symptomatic’, and ‘non-dependent asymptomatic’. In addition to the potential reasons for the apparent inconsistencies in prevalence rates, comparisons and generalisability of studies is difficult due to the lack of descriptions provided of the populations studied (Mónok, et al., 2012; Landolfi, 2013). Further work is advised considering the lack of studies examining prevalence in specific populations and the potential of negative consequences from the development of EXD.

2.5 Overall summary and research questions

LEA describes the mismatch between EI and EEE, resulting in inadequate energy to support physiological function and maintain optimal health and performance. LEA may be caused by clinical ED, intentional but mismanaged efforts to alter body composition that may include DE, and/or an inadvertent inability to increase EI to match EEE (Nattiv, et al., 2007). Additionally, it has been recognised that DE/ED in females has been associated with EXD with recent work suggesting that EXD, with or without DE/ED, may increase the risk of developing LEA (Turton, et al., 2017). Current position stands have stressed the importance of prevention and the early detection of at-risk groups, to avoid the more serious clinical disturbances associated with LEA (i.e., impaired menstrual and bone health; Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018).

Current work has identified that individuals from endurance sports and/or leanness sports are at greater risk of LEA due to increased risk of DE, ED, EXD and increased daily EEE from high training volumes (Loucks, et al., 2011). However, previous studies have focused on single-sport endurance athletes (i.e., runners) and studies examining LEA in multi-sport endurance athletes (i.e., triathletes) are limited. Triathlon requires large volumes of sustained training across three sports in addition to the nutritional and psychological demands (Vescovi & VanHeest, 2016). Participation in triathlon may be associated with an increased risk of developing LEA and warrants further investigation into its prevalence and potential origins. LEA is associated with sub-clinical and clinical disturbances to menstrual and bone health that may have irreversible consequences (De Souza, et al., 2014). There may be critical phases of throughout the lifespan (i.e., puberty and/or menopause) where the development of LEA may increase and/or increase the severity of associated impairments. However, whilst current evidence suggests LEA exists across all ages, current work has focused on young adults and limited information exists on LEA across age groups or the influence of age. Similarly,

previous work has focused on elite athletic populations, but recent evidence suggests non-elite athletic groups are also at increased risk but may have less access to advice and support. It has been proposed that performance level development may increase the risk of know risk factors for LEA (i.e., increased daily EEE from training, DE, ED) but more work is needed to elucidate the influence of performance level on LEA. It is also unclear how seasonal changes in respect of training and competition impact on LEA and associated risk factors.

In order to address some of the discrepancies and gaps identified with the present literature review, the following research questions for this thesis were generated:

1. What is the prevalence of risk for LEA, DE/ED and EXD in competitive female triathletes?
2. What are the associations between LEA, DE/ED and EXD in competitive female triathletes?
3. Does age influence the prevalence of and associations between LEA, DE/ED and EXD in competitive female triathletes?
4. Does performance level influence the prevalence of and associations between LEA, DE/ED and EXD in competitive female triathletes?
5. Does EA and eating attitudes change across the triathlon season in female triathletes and does the prevalence change?

CHAPTER 3

GENERAL METHODOLOGY

3.1 Overview

This chapter will outline the general methodology of the research studies within this thesis, relating to the ethical approval, recruitment procedures, and questionnaires used in Studies 1-4. Variations in data collection methods or specific information where relevant will be detailed within the respective studies.

3.2 Ethical approval

All studies were conducted having gained ethical approval (Appendix 1) from the University of Sunderland Research Ethics Group (studies 1-4). The key ethical issues related to studies 1-7 included repeated measures of body composition, EI, EEE, and eating attitudes over a prolonged data collection period, measurement of maximal aerobic capacity ($\dot{V}O_{2max}$), potential data breach due to the online nature of the questionnaires, and screening and subsequent exclusion of participants if they had a previous or current diagnosis of LEA, FHA, and/or DE/ED. All studies were subsequently conducted in accordance with the Declaration of Helsinki (World Medical Association, 2013).

3.3 Research design

To assess the prevalence of risk in studies 1-3, a cross-sectional design using an anonymised online questionnaire was used. The questionnaire was titled the 'Female Health Questionnaire' and took 15-20 minutes to complete on average. It comprised of demographic questions selected after reviewing the literature which included age, nationality, self-identified performance level, training hours, height, and weight. BMI was subsequently calculated from the self-reported height and weight. The 'Female Health Questionnaire' included the questions from the original LEAF-Q (Melin, et al., 2014), FAST (McNulty, et al., 2001), and EDS-R (Downs, et al., 2004) questionnaires to assess LEA risk, eating attitudes and EXD. The LEAF-Q, FAST, and EDS-R are established, validated and reliable screening tools previously used in

EA literature (i.e., Melin, et al., 2014, Folscher, et al., 2015, Slater, et al., 2016 and Logue, et al., 2019).

The 'Female Health Questionnaire' was primarily used to investigate if female triathletes were an athletic population considered to be at risk of developing LEA, DE/ED, and/or EXD – forming study 1 of this thesis. Findings from study 1 influenced the formation of studies 2-3 of this thesis, whereby subsequent analysis investigated the influence of age (study 2) and performance level (study 3) on the prevalence of risk and/or associations with the key components (i.e., LEA, DE/ED, EXD). Study 4 was a longitudinal design following a cohort of female triathletes across a full triathlon season. EA and eating attitudes were assessed every two months throughout the season to assess prevalence and seasonal changes using both direct EA measures and screening tools for LEA and eating attitudes. Study 4 was the first study to commence and informed the decision to design studies 1-3, as after reviewing the literature there was limited prevalence studies within multi-sport endurance athletes to enable comparison. Figure 3.1 illustrates the overall research design of this thesis.

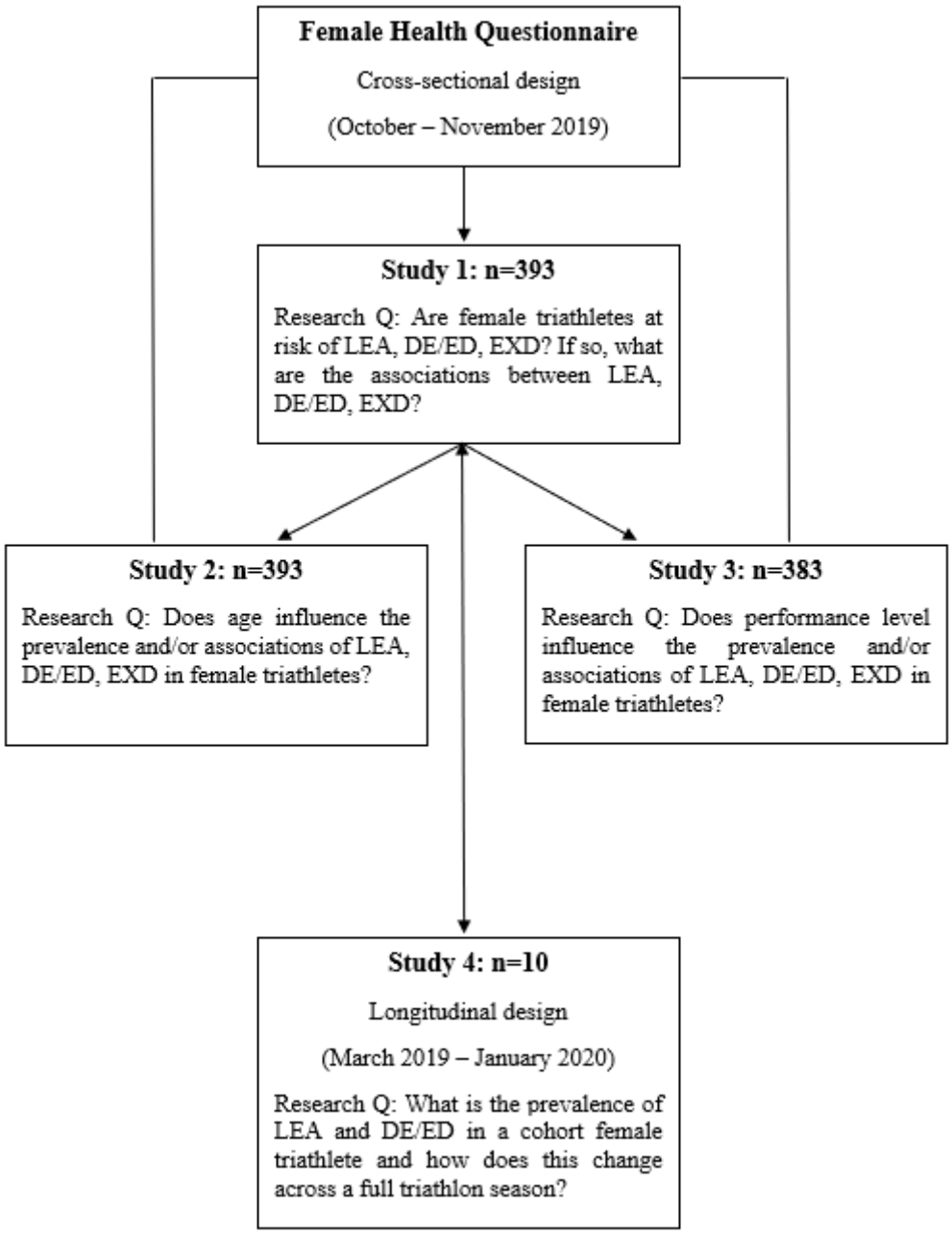


Figure 3.1. Schematic of thesis research design.

3.4 Participant recruitment and sample size

All participants took part in the studies voluntarily and were provided with information specifying the experimental procedures, including the requirements of their involvement in the studies via a participant information sheet (Appendix 2). Subsequently, participants were made aware that they could ask any questions for clarification before providing informed, written consent to take part in the study 4 (Appendix 3). A statement of implied consent was used in the study presented in studies 1-3 (Appendix 3). In study 4, all participants completed an Institutional Review Board-approved pre-participation health screening medical form, prior to any data collection (Appendix 4). Inclusion and exclusion criteria for studies 1-4 are presented in table 3.1.

For studies 1-3, participants were recruited using voluntary response sampling using recruitment posters advertised via social media platforms (Facebook, Instagram, and Twitter), supported by flyers and word-of-mouth approaches when relevant. An alternate form of participant recruitment was used in study 4 by using convenience sampling at local triathlon clubs. A recruitment poster for female triathletes was emailed to registered British Triathlon clubs (including University triathlon clubs) in the North-East of England. Word-of-mouth approaches were also used when relevant.

Table 3.1. Inclusion and exclusion criteria for studies 1-4.

Studies 1-3	
Inclusion	*Exclusion*
• Female • Aged ≥ 18 years • Pre-menopausal • Currently participating in triathlon (any performance level)	• Male • Aged < 18 years • Menopausal or post-menopausal • Non-triathlete or not currently participating in triathlon • LEAF-Q exclusion criteria by Melin, et al., 2014; pregnant, breastfeeding, chronic illness, use of forms of contraceptive other than oral (i.e., hormonal coil)

	• Incomplete questionnaire • Unable to understand study requirements • Unable to provide implied consent
Study 4	
Inclusion	*Exclusion*
• Female • Aged ≥ 18 years • Pre-menopausal • Currently participating in triathlon (non-elite) • Non-smoker • Apparently healthy and no previous diagnosis of LEA, FHA, DE, or ED • A minimum of 12 months experience of participating in training and competition for triathlon	• Male • Aged < 18 years • Menopausal or post-menopausal • Non-triathlete or not currently participating in triathlon • LEAF-Q exclusion criteria by Melin, et al., 2014; pregnant, breastfeeding, chronic illness, use of forms of contraceptive other than oral (i.e., hormonal coil) • Unable to understand study requirements • Current or recent injury (< 6 months) • Unable to provide voluntarily consent • Current diagnosis or undergoing investigation or family history of cardiovascular, respiratory, or metabolic disease • Receiving medical treatment for any condition •Recreational or performance enhancing substance use • Known or diagnosed heart murmur, palpitations, high resting blood pressure, head injury, seizures, epilepsy, fainting, neurological disorder • More than two risk factors for cardiac disease • Previous or current diagnosis for LEA, FHA, DE, or ED

DE, disordered eating; ED, eating disorder; FHA, functional hypothalamic amenorrhea; LEA, low energy availability; LEAF-Q, low energy availability in female's questionnaire.

Table 3.2 outlines the number of individuals recruited, excluded, and included in the final sample for analysis in studies 1-4. Although a large reduction in the original sample size is evident in studies 1-3, the final sample included in analysis in studies 1-3 met the calculated sample size estimation for the cross-sectional studies. Based on a population size of 10,000 registered female triathletes with British Triathlon (British Triathlon, 2021a), a sample size estimation of n=370 was calculated for studies 1-3 with a confidence level of 95% and a 5% margin of error (Qualtrics, London, UK). Although the author of this thesis acknowledges sample size estimation and power analyses should be interpreted with caution as its components can easily be manipulated and are ultimately estimates (Prajapati, et al., 2010; Meyvis & Van Osselaer, 2018). Despite a large reduction in the original sample size, studies 1-3 all fall within the sample size range previously reported in LEA prevalence studies (range 10 to 833; Schaal, et al., 2011a; Logue, et al., 2019 – table 2.2). The author also acknowledges that the larger the study, particularly with prevalence research, the more reliable the results due to small standard error and narrow confidence intervals, therefore resulting in more precise and/or firm conclusions.

The smaller the study, the less reliable the results with large standard error and wide confidence intervals, resulting in imprecise estimate of the effect leading to less precise/no firm conclusions (Hackshaw, 2008). This would be particularly applicable to study 4 (table 3.2) which had a total sample size of n=10 as a smaller sample size makes it difficult to distinguish random variation and a real effect. However, due to the longitudinal design, logistics, and time constraints on both participant and researcher in study 4 the primary goal was to recruit 10 to 15 individuals which was in line with previous studies who directly measured EA (Doyle-Lucas, et al., 2010; Schaal, et al., 2011; Moss, et al., 2020; Zanders, et al., 2021 – table 2.2).

Table 3.2. Sample size for studies 1-4.

N recruited		Excluded	Included
N=878	Study 1	N=36 post-menopausal or non-triathlete N=303 LEAF-Q exclusion (pregnant, breastfeeding, chronic illness, use of forms of contraceptive other than oral; increase false positive; Melin, et al., 2014) N= 146 Female Health Questionnaire incomplete	N=393
	Study 2	N=36 post-menopausal or non-triathlete N=303 LEAF-Q exclusion (pregnant, breastfeeding, chronic illness, use of forms of contraceptive other than oral; increase false positive; Melin, et al., 2014) N= 146 Female Health Questionnaire incomplete	N=393
	Study 3	N=36 post-menopausal or non-triathlete N=303 LEAF-Q exclusion (pregnant, breastfeeding, chronic illness, use of forms of contraceptive other than oral; increase false positive; Melin, et al., 2014) N= 146 Female Health Questionnaire incomplete N=10 self-identified as elite level triathletes and study focused on non-elite triathletes	N=383
N=13	Study 4	N=1 non-compliance/uncomfortable with repeated measures of EI N=1 personal circumstances (bereavement) N=1 medical reasons	N=10

3.5 Questionnaire

In studies 1-3, the 'Female Health Questionnaire' was constructed based on established, validated, and reliable screening tools in addition to demographic variables previously detailed. These included the LEAF-Q (studies 1-4; Melin, et al., 2014), the FAST (studies 1-4; McNulty, et al., 2001), and the EDS-R (studies 1-3; Downs, et al., 2004). The online 'Female Health Questionnaire' was self-administered and was distributed (via a URL) in English using the Qualtrics electronic management system (Qualtrics, London, UK) during recruitment. An adapted version of the online questionnaire was used in study 4 which did not include the EDS-R. In study 4, participants were provided with a URL link to the online questionnaire, which was self-administered, and participants were asked to input their unique participant code for the study.

The items within the instruments included in the questionnaire (LEAF-Q, FAST and EDS-R) need to have internal consistency reliability. This refers to the degree to which the instrument measures exactly what it claims to measure (i.e., the items should all measure the same construct so should be correlated as a group; Taber, 2018). Cronbach's alpha (α; Cronbach, 195) is the most widely used coefficient for assessing internal consistency of the instrument within the recruited sample, being described as "*one of the most important and pervasive statistics in research involving test construction and use*" (Cortina, 1993). Alpha statistics range from 0 to 1 to indicate a lower-bound estimate of reliability (i.e., how much construct items covary; Cronbach, 1951). Higher item covariance produces a higher alpha statistic which is associated with greater confidence in the instrument constructs and conclusions drawn (Cortina, 1993). However, Cronbach's alpha must be used and interpreted with caution.

Alpha scores are interpreted and described differently across the literature and a unanimous threshold for alpha does not exist. Yet it remains many authors consider the arbitrary value of 0.70 as an adequate level of internal consistency (Taber, 2018). Such estimates of alpha cannot be generalised across all situations. A high estimate of alpha may not equate to internal consistency as alpha is influenced by instrument length (i.e., alpha can be improved simply by adding more items). Finally, a maximum alpha of 0.90 is recommended as very high alpha scores may suggest redundancy among the items. Due to these limiting factors of alpha, it is suggested reliability work also include further inferential statistics (i.e., confidence intervals, standard error scores, the Spearman-Brown coefficient or power calculations; Taber, 2018). Additionally, the instruments also need to demonstrate validity which may include concurrent validity and discriminant validity. Concurrent validity is the degree to which an instrument correlated with similar instruments that measure similar constructs (Portney & Watkins, 1998). Discriminant validity refers to the degree to which an instrument offers two distinct results when measuring two different constructs (Portney & Watkins, 1998).

3.5.1 LEAF-Q

The twenty-five item LEAF-Q (Appendix 5) was developed as a self-report screening tool to identify female athletes at risk of developing LEA. Following verification of the relevancy from a collective of clinical experts in medicine, endocrinology, gastroenterology, and sports nutrition, physiological symptoms associated with long-term LEA were included as variables in the LEAF-Q (Melin, et al., 2014). These included injury frequency, gastrointestinal symptoms, and menstrual dysfunction (MD). Questionnaire data were marked and scored by the author of this thesis according to the LEAF-Q scoring key where an individual can score a minimum of 0 and maximum of 49 (Appendix 5). A total score ≥ 8 is considered as at risk of

LEA and a score < 8 is considered low risk. Suggested cut-offs for injuries, gastrointestinal disturbances, and MD are ≥ 2, ≥ 2 and ≥ 4, respectively (Melin, et al., 2014).

The reliability and validity of the LEAF-Q and the self-reported symptoms reported in the LEAF-Q were assessed in a group of endurance athletes (long-distance runners and triathletes), and professional dancers (n=37; Melin, et al., 2014). To assess internal consistency reliability, a test-retest was performed over a 2-week period and Cronbach's α was used to assess LEAF-Q variables. To assess discriminant validity, two sample t-tests were used to assess significant differences between the groups (i.e., low versus high EA), for the mean item score of each variable. Pearson's correlation coefficient (r) was used to assess concurrent validity and associations between the total score, variables, and Triad conditions (Melin, et al., 2014). Overall, the LEAF-Q was validated in female endurance athletes (Cronbach's $\alpha = 0.71$). A total LEAF-Q score ≥ 8 produced a sensitivity of 78% and a specificity of 90% for accurately classifying current EA and/or reproductive function and/or bone health (Melin, et al., 2014).

3.5.2 FAST

The thirty-three item FAST (Appendix 6) was developed specifically for the female athlete to identify DE attitudes and behaviours. The FAST incorporates two different four-item Likert scales, three ranked items, and one dichotomous item. An example question consists of, "During training, I control my fat and calorie intake carefully". Questionnaire data were marked and scored by the author of this thesis according to McNulty, et al., (2001), where questions are scored on a four-point Likert-type scale:

- 1 point = responses of strongly disagree; never; monthly or less; <30 minutes; no significant injuries.
- 2 points = responses of disagree; rarely; weekly; 30-45 minutes; 1-3 times.
- 3 points = responses of agree; sometimes; 2+ times a week; 45-60 minutes.

- 4 points = responses of strongly agree; frequently; daily; 2+ hours responses.

A total FAST score of 79-94 indicates subclinical DE and a score > 94 indicates a clinical ED. An individual can score a minimum of 33 and a maximum of 132 (McNulty, et al., 2001).

The reliability and validity of the FAST were assessed using three established groups: University female athletes diagnosed with ED who participate in competitive athletics (n=12), University female athletes without an ED who participate in competitive athletics (n=14), and University females who did not participate in athletes but were diagnosed with ED (n=15). To correct for learner effect, the FAST was randomly administered alongside three validated psychometric assessment tools (EDE-Q, EDI-2, and the Bulimia Test-Revised) to determine concurrent validity (McNulty, et al., 2001). Overall, the FAST demonstrated internal consistency (Cronbach's $\alpha = 0.87$). Pearson's correlation coefficient (r) demonstrated concurrent validity between the FAST and the EDI-2 and EDE-Q, with higher FAST scores associated with ED as identified by the EDI-2 (0.89, $p < 0.001$) and EDE-Q (0.60, $p < 0.050$). Using a one-way analysis of variance to assess discriminative validity, it was demonstrated the FAST can differentiate the unique characteristics of athletes with ED compared with athletes without DE pathology, and non-athletes with ED ($p < 0.001$; McNulty, et al., 2001). These findings agreed with seminal work by Affenito, et al., (1998).

3.5.3 EDS-R

The twenty-one item self-report EDS-R (Appendix 7) was developed to assess EXD based DSM-IV criteria for substance dependence (Downs, et al., 2004). The EDS-R incorporates the following seven subscales defined by Hausenblas and Downs (2002b):

- Tolerance: defined as "*either a need for increased amounts of exercise to achieve the desired effect or a diminished effect occurs with continued use of the same amount of exercise".*

- Withdrawal: defined as "*manifested by either the characteristic withdrawal symptoms for exercise (e.g., anxiety, fatigue) or the same (or closely related) amount of exercise is taken to relieve or avoid withdrawal symptoms*".
- Continuance: defined as "*exercise is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the exercise (e.g., continued running despite injury)*".
- Lack of Control: defined as "*a persistent desire or unsuccessful effort to cut down or control exercise*".
- Reduction in Other Activities: defined as "*social, occupations, or recreational activities are given up or reduced because of exercise*".
- Time: defined as "*a great deal of time is spent in activities necessary to obtain exercise (e.g., physical activity vacations)*".
- Intention Effects: defined as "*exercise is often taken in larger amounts or over a longer period than was intended*".

Questionnaire data were marked and scored by the author of this thesis according to the 'EDS-21 Manual' by Hausenblas and Downs (2002b), where questions are scored on a 6-point Likert scale ranging from 1 (never) to 6 (always). A higher score reveals more EXD symptoms. Subsequently, participants are classified as at risk for EXD, non-dependent-symptomatic (SY), or non-dependent-asymptomatic (AS) based on the adopted DSM-IV criteria for substance dependence. In line with Hausenblas and Downs (2002b), participants who indicate a score 5-6 on the Likert scale on three or more of the seven subscales are classified as at risk for EXD. Participants who indicate a score 3-4 on the Likert scale on three or more of the subscales, or a combination of at least three criteria in the dependent (5-6) and SY (3-4) range and fail to meet the criteria of at risk of EXD, are classified as SY. Finally, participants who indicate a

score of 1-2 on the Likert scale on three or more of the subscales are classified as AS and recognised as not reporting any EXD symptoms (Hausenblas & Downs, 2002b).

The reliability and validity of the EDS-R were assessed in a group (n=408) of physically active (minimum of three, 1-h exercise sessions) male and female University students. To avoid biased responding, the EDS-R was administered in a counterbalanced order alongside the 'Leisure-Time Exercise Questionnaire' and 'Multidimensional Perfectionism Scale' (Downs, et al., 2004). Overall, the EDS-R demonstrated internal consistency (Cronbach's $\alpha = 0.96$). The seven facets of exercise dependence were internally consistent with coefficient alpha ranges from 0.78 to 0.92 (except for the Reduction in Other Activities subscale [Cronbach's $\alpha = 0.67$] as a result of modifications for the EDS-R). The EDS-R has demonstrated strong psychometric properties (i.e., content, and convergent validity, test-retest, and internal consistency reliability) in previous research (Downs, et al., 2004).

CHAPTER 4

PREVALENCE OF FEMALE TRIATHLETES AT RISK OF LEA

4.1 Introduction

Endurance and ultra-endurance sports worldwide have witnessed an exponential growth in popularity with an increased attraction to the female athlete (Scheer, 2019; Knechtle, Scheer, Nikolaidis & Sousa, 2020; Vitti, Nikolaidis, Villiger & Onywera, et al., 2020). This is reflected by the gradual growth of female athletes competing in triathlon events, ranging from sprint to ultra-distance triathlon, in recent times (Mountjoy, Thomas & Levesque, 2019). Female triathlete participation rates range from 25% to 40% of the triathlon field (Lepers, Rüst, Stapley & Knechtle, 2013a; Wonerow, Rüst, Nikolaidis & Rosemann, et al., 2017; Lepers, 2019). British Triathlon (2021a) recorded a total of 150,000 active racing triathletes in the 2019 season. An abundance of evidence exists supporting the benefits of endurance sport participation, not limited to, improved cardiorespiratory fitness, improved mental health and well-being, weight management, and improved memory and cognition (Ruegsegger & Booth, 2018). Although the health benefits are abundant, evidence exists that over-exercising or under-fueling, occurring intentionally or inadvertently, poses risks for the female endurance athlete that are distinct from their male counterparts (Ackerman, Stellingwerff, Elliot-Sale & Baltzell, et al., 2020).

Previous studies have demonstrated that female athletes from leanness-sports (figure 2.9) have a greater risk of developing LEA (Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). LEA is defined as the imbalance between EI and EEE, resulting in inadequate energy to support bodily function and physiological processes (Loucks, et al., 2011; Mountjoy, et al., 2018). To date, there are two schools of thought and subsequent conceptual models regarding the negative health consequences associated with LEA: Triad (Nattiv, et al., 2007; De Souza, et al., 2014) and RED-S (Mountjoy, et al., 2014; 2018). These models are outlined and discussed in Chapter 2.3.2 of this thesis. Both models call attention to the implications of LEA to health and performance. LEA risk is thought to be greater in leanness sports,

particularly endurance sports, with prevalence rates ranging from 18% to 80% (table 2.2; Muia, et al., 2016; Jesus, et al., 2021). Although limited studies exist within team sports, prevalence rates for team or ball-based sports range from ~12% to 53% (Reed, et al., 2013; Condo, Lohman, Kelly & Carr, 2019; Logue, et al., 2020).

Competing in triathlon may pose an increased risk for the female athlete. Triathlon necessitates large volumes of frequent and intense training across three disciplines (swim, bike, and run). This may result in significantly elevated EEE and therefore requires adequate nutritional replenishment (Vescovi & VanHeest, 2016). Additional risk factors for the female triathlete may be the potential desire for leanness for improved performance or DE behaviour due to body image dissatisfaction (Mountjoy, et al., 2019; Thorpe & Clark, 2020). However, additional data is required for non-elite female triathletes. Considerable research has been undertaken to understand the adverse physiological sequelae documented in female athletes as a result of LEA (Mountjoy, et al., 2014; 2018). Currently, data is equivocal and further research warranted for the determination of accurate LEA cut-offs for the athletic population. However, if a reduction in EI and/or an increase in EEE reduces EA below 30 $kcal \cdot kgLBM^{-1} \cdot day^{-1}$, an array of physiological processes is disrupted as the body systems adjust to reduce energy expenditure (Loucks, et al., 2011). These are discussed in Chapter 2.3.3 and illustrated in figure 2.4. It is widely acknowledged that chronic LEA (from inadequate nutritional practices and/or DE/ED and/or excessive energy expenditure) is the central pathological process leading to the unfavourable health and performance consequences of Triad and RED-S (Ackerman, et al., 2020; Logue, et al., 2020).

Existing studies concerning Triad and RED-S have stated that psychological stress and/or depression manifested as DE represents a large percentage of cases of LEA. It is also acknowledged that other circumstances without such a psychological underpinning may occur such as poor nutritional and training knowledge (Mountjoy, et al., 2014; 2018). Recent work

with long-distance runners has recognised endurance athletes with a proclivity for EXD, may be at increased risk of LEA and they encouraged future work to address the potential association between EXD, LEA, and RED-S (Turton, et al., 2017; Marques, et al., 2019; Logue, et al., 2020). EXD, a behavioural addiction, is the compulsive need to partake in exercise irrespective of harmful health consequences. It is characterised by uncontrollable excessive exercise behaviour, strong exercise withdrawal symptoms, and a compromised social and professional life (Hausenblas & Downs, 2002a; 2002b; Hausenblas, Schreiber & Smoliga, 2017; Çetin, Bulğay, Demir, & Cicioğlu, et al., 2020). Analysis of relevant studies has shown that EXD is often associated with perfectionism and DE/ED pathology in females (Cook, et al., 2016), with endurance athletes at the greatest risk (Marques, et al., 2019). Whether an association exists between EXD risk, DE/ED symptoms and LEA risk among female triathletes is less known.

Previous prevalence studies have investigated elite female middle- and long-distance runners and race walkers (800m – 50km), demonstrating an overall prevalence of 31% for LEA with 37% showing signs of amenorrhea (Heikura, et al., 2018a). Prevalence data amongst elite female endurance athletes found 20% had LEA and 28% were diagnosed with DE/ED (Melin, et al., 2014). Study results from elite and recreational female athletes at the Comrades Marathon revealed 44% were at risk of LEA and one-third were at risk of DE/ED (Folscher, et al., 2015). Although sample size was small (n=15), a cross-sectional study reported 60% of female triathletes were in a calorific deficit consistent with DE pathology and 40% had a history of amenorrhea (Hoch, et al., 2007). Overall prevalence of EXD risk is estimated to range between 2% and 10% across an array of sports (Marques, et al., 2019). A recent study amongst amateur male triathletes indicated the prevalence of risk for EXD was 9% with 60% classified as symptomatic (Tallón & Palomero, 2017). Prevalence data amongst female triathletes at any performance level is lacking.

Furthermore, the majority of EA research has been conducted in elite female athletes, single-sport endurance athletes, high school or University athletes, team sports or dancers (Logue, et al., 2020). Despite the increased scientific attention on Triad and RED-S and the recommendation to determine the severity of the issue across a variety of athletic populations (De Souza, et al., 2014; Mountjoy, et al., 2014; 2018), the number of studies investigating multi-sport endurance events are limited. It could be argued that based on the prevalence of LEA in single-sport endurance events, such as running or cycling, athletes participating in multi-sport endurance events would be at an increased risk due to the increased training demand. Thus, more data are needed in competitive female triathletes examining the prevalence of those at risk of LEA, DE/ED, and EXD. Furthermore, it is important to investigate the potential relationship between these components in female triathletes, as this may have implications for screening and early detection of psychological risk factors for Triad or RED-S. Accordingly, this study aimed to explore LEA, DE/ED, and EXD in competitive female triathletes.

Objectives:

1) Investigate the prevalence of those at risk of LEA, DE/ED risk, and EXD in competitive female triathletes.
2) Investigate possible associations between LEA, DE/ED and EXD in competitive female triathletes.

4.2. Materials and methods

4.2.1 Research design

This cross-sectional, descriptive study required participants to complete an anonymised online questionnaire – the 'Female Health Questionnaire'. The study formed part of a larger investigation, including LEA risk, eating attitudes, and EXD trends regarding age groups (study 2) and performance level (study 3). The study was reviewed and granted ethical approval (Appendix 1) from the University of Sunderland Research Ethics Group and conducted in accordance with the Declaration of Helsinki (2013). All participants took part in the study voluntarily, were provided with information specifying the study details including inclusion and exclusion criteria (table 3.1), provided implied consent for the data to be used in the study and no participation incentives were offered (outlined in Chapter 3.2 – 3.4, Appendices 1-3).

4.2.2 Participants

Recruitment posters for healthy, pre-menopausal, female triathletes, aged 18 or over were arbitrarily advertised via social media platforms (Facebook, Instagram, and Twitter), supported by flyers and word-of-mouth approaches when relevant. Participants were asked to complete the anonymous online 'Female Health Questionnaire' after reading the information sheet (including inclusion and exclusion criteria) and providing implied consent (Appendix 2-3). Table 3.1 (Chapter 3) outlines the inclusion ad exclusion criteria for study 1 – exclusion criteria included:

- Male
- Aged < 18 years
- Menopausal or post-menopausal
- Non-triathlete or not currently participating in triathlon
- LEAF-Q exclusion criteria by Melin, et al., 2014; pregnant, breastfeeding, chronic illness, use of forms of contraceptive other than oral (i.e., hormonal coil) which can lead to false positives

- Incomplete questionnaire
- Unable to understand study requirements
- Unable to provide implied consent

Table 4.1 outlines participant recruitment, exclusion and inclusion into the final study sample for study 1. N=393 individuals were included in the final analysis for study 1 and although a large reduction in the original sample size is evident, a sample size of N=393 met the calculated sample size estimation for the cross-sectional study. Based on a population size of 10,000 registered female triathletes with British Triathlon (British Triathlon, 2021a), a sample size estimation of n=370 was calculated for study 1, with a confidence level of 95% and a 5% margin of error (Qualtrics, London, UK). Study 1 sample size also falls within the sample size range previously reported in LEA prevalence studies (range 10 to 833; Schaal, et al., 2011a; Logue et al., 2019 – table 2.2).

Table 4.1 Sample size for study 1

N recruited	Excluded	Included
N = 878	N=36 post-menopausal or non-triathlete N=303 LEAF-Q exclusion (pregnant, breastfeeding, chronic illness, use of forms of contraceptive other than oral; increase false positive; Melin, et al., 2014) N= 146 Female Health Questionnaire incomplete	N = 393

4.2.3 Data collection

The online 'Female Health Questionnaire' was constructed and distributed (via a URL) using the Qualtrics electronic management system (Qualtrics, London, UK) during recruitment. The self-administered, online questionnaire was accessible for a four-week period

between October 2019 and November 2019 to female triathletes, after which time the URL was deactivated. Questionnaire data were then marked and scored by the author of this thesis between December 2019 and January 202 as outlined in Chapter 3.

4.2.4 Questionnaire data

Demographic data included self-reported age, nationality, self-reported height and weight, self-identified competitive level, and average weekly training time. This is in line with previous research by Folscher, et al., (2014), Melin, et al., (2014) and Slater, et al., (2015). To identify athletes at risk of developing LEA, DE/ED, and EXD, the 'Female Health Questionnaire' incorporated all items of the LEAF-Q, FAST, and EDS-R, respectively.

The twenty-five item LEAF-Q (Chapter 3.4.1) was developed to identify female athletes at risk of developing LEA by utilising subsets of injury frequency, gastrointestinal disturbances, and menstrual function (Melin, et al., 2014). The LEAF-Q was validated in endurance athletes (Cronbach's $\alpha = 0.71$) with a total LEAF-Q score ≥8 producing a sensitivity of 78% and a specificity of 90% for accurately classifying current EA and/or MD and/or bone health (Melin et al, 2014). Based on established cut-offs for the LEAF-Q (Melin et al, 2014), individuals with a total score ≥8 were classified as at risk of LEA. In addition, the individual component scores were analysed to assess increased risk and/or incidence of injury (total subset score ≥2), gastrointestinal disturbances (total subset score ≥2), and menstrual function (total subset score ≥4).

The thirty-three item FAST (Chapter 3.4.2) was developed to identify DE/ED behaviours in athletes (Cronbach's $\alpha = 0.87$; McNulty, et al., 2001). Correlation analysis demonstrated high concurrent validity between the FAST and the EDI-2 and EDE-Q, with higher FAST scores associated with greater incidence of ED as identified by the EDI-2 (0.89, $p < 0.001$) and EDE-Q (0.60, $p < 0.050$). The FAST also demonstrated discriminant validity by differentiating

between the unique characteristics of athletes with ED compared with athletes without DE pathology and non-athletes with ED ($p < 0.001$; McNulty et al, 2001). Based on accepted established cut-offs for the FAST, individuals with a total FAST score < 79 were classified as 'no ED', a score of ≥79 to ≤94 were classified as subclinical DE, and those with a score >94 were classified as clinical ED (McNulty et al, 2001).

The twenty-one item self-report EDS-R (Chapter 3.4.3) was developed to assess EXD based on DSM-IV criteria for substance dependence (Downs, et al., 2004). Seven facets of EXD were assessed on a six-point Likert scale ranging from 1 (*never*) to 6 (*always*): Tolerance (Cronbach's $\alpha = 0.78$), withdrawal (Cronbach's $\alpha = 0.93$), intention effect (Cronbach's $\alpha = 0.92$), lack of control (Cronbach's $\alpha = 0.82$), time (Cronbach's $\alpha = 0.88$), reductions in other activities (Cronbach's $\alpha = 0.67$), and continuance despite physical/psychological consequences ($\alpha = 0.89$). The total summed score to assess global EXD demonstrated high internal consistency (Cronbach's $\alpha = 0.96$; Downs, et al., 2004). Participants were classified as: at risk for EXD, SY or AS.

4.2.5 Statistical analysis

All statistical analyses were performed using SPSS (V.25; IBM Company, SPSS Inc., Chicago, USA). Data normality was assessed by Shapiro-Wilk which is more sensitive than the Kolmogorov-Smirnov test to sample sizes as large as 2000 (Mishra, Pandey, Singh & Gupta, et al., 2019). Normally distributed data were reported as mean ± SD and non-normally distributed data as median (interquartile range [IQ]). Descriptive statistics were calculated for demographic data and self-reported performance. Frequency analysis was undertaken for key components of the LEAF-Q, FAST and EDS-R questionnaire scores. Non-normally distributed data was compared using a Mann-Whitney U test to compare differences in participant characteristics between LEAF-Q groups. Similarly, Kruskal-Wallis tests were used to compare

differences in participant characteristics between FAST and EDS-R groups. For non-normally distributed data, Spearman Rank correlations (S_R) and chi-square tests were used to assess associations between the LEAF-Q, FAST and EDS-R scores. Subsequently, binary logistic regression (BLR) was performed to ascertain the association between eating attitudes (FAST) and exercise behaviour (EDS-R) on the likelihood that participants were low risk for LEA. Plus, BLR was performed to ascertain the association of exercise behaviour (EDS-R) on the likelihood that participants were at risk of DE behaviour (FAST). Statistical significance was set *a priori* at $p \leq 0.05$.

4.3 Results

4.3.1 Participant characteristics

N=393 individuals who met the inclusion criteria (table 3.1 – chapter 3) were included in the final analysis of study 1. Participant characteristics for the LEAFQ-Q, FAST, and EDS-R included in the 'Female Health Questionnaire' are presented in table 4.2. Values of 0.1, 0.3 and 0.5 are commonly indicative of small, moderate, and large effects (Cohen, 1988). Participants classified as at risk for LEA (LEAF-Q) spent significantly more hours training per week, however this was a small effect (Cohen's *d* 0.17 - table 4.2). Participants classified with ED (FAST) were significantly younger than those classified with DE or with no ED, however this was a small effect (Cohen's *d* 0.24 - Table 4.2). Participants classified with ED had a significantly lower body mass than those with DE (small Cohen's *d* 0.23), however, those with DE had a significantly higher BMI than those with no ED (small Cohen's *d* 0.22; Table 4.2). Similarly, participants categorised at risk of EXD (EDS-R) were significantly younger than those classified as symptomatic (SY) or asymptomatic (AS; moderate to large *d* 0.46 - Table 4.2). Finally, those at risk of EXD and those classified as SY spent significantly more time training per week than AS individuals (moderate to large *d* 0.48 - Table 4.2).

Table 4.2. Participant characteristics and training load

	Total	LEAF-Q		FAST			EDS-R		
		At Risk	Not at Risk	No ED	DE	ED	Asymptomatic	Symptomatic	At Risk
		n=165	n=228	n=260	n=99	n=34	n=130	n=229	n=34
Age (years)	36 (13) (18 – 54)	35 (14) (18 – 54)	37 (12) (18 – 52)	36 (12) [*3] (18 – 53)	36 (13) [*2] (18 – 54)	31 (15) [*2; 3] (18 – 48)	39 (12) [*7] (20 – 52)	35 (12) [*6] (18 – 54)	31 (16) [*6; 7] (18 – 48)
Height (m)	1.66 (0.09)	1.65 (0.09)	1.67 (0.08)	1.67 (0.09)	1.65 (0.08)	1.64 (0.11)	1.65 (0.08)	1.67 (0.09)	1.67 (0.09)
Mass (kg)	64.0 (12.0)	63.0 (13.0)	64.0 (11.0)	64.0 (12.0)	67.0 (11.0) [*4]	61.0 (12.5) [*4]	64.0 (14.0)	64.0 (11.0)	60.0 (10.5)
BMI ($kg \cdot m^2$)	23.0 (4.3)	23.0 (5.2)	23.1 (4.0)	22.8 (4.3) [*5]	23.7 (4.4) [*5]	22.7 (6.1)	23.2 (4.7)	23.0 (4.1)	21.9 (4.4)
Training time (h·week)	11.0 (5.5) (2.9 – 42.0)	12.0 (5.5) [*1] (2.9 – 42.0)	10.6 (6.0) [*1] (3.0 – 30.0)	11.0 (6.0) (2.9 – 42.0)	12.0 (5.6) (3.0 – 26.0)	12.5 (5.5) (5.0 – 24.0)	10.0 (5.5) [*8; 9] (2.9 – 33.0)	12.0 (6.0) [*9] (3.0 – 42.0)	12.8 (4.2) [*8] (5.0 – 30.0)

BMI, body mass index; DE, disordered eating; ED, eating disorder; LEAF-Q, low energy availability in female's questionnaire; FAST, female athlete screening tool; EDS-R, exercise dependence scale revised.

Data presented as median (interquartile range) or range in parentheses; n=393.

[*1; 7; 9] Significant difference between groups, p <0.001. [*2; 3; 4; 5; 6; 8] Significant difference between groups, p <0.050.

4.3.2 LEAF-Q scores and key components

LEAF-Q descriptive data are presented in Table 4.3. Nearly half (42%) of participants are classified as being at risk of LEA according to the LEAF-Q (Figure 4.1). When assessing the individual LEAF-Q component scores, over half of all participants (62%) met the component cut-off score ≥2 for increased incidence of injury (Table 4.4). More participants met the component cut-off score ≥2 for increased gastrointestinal disturbances (77%; Table 4.4). 27% of participants met the component cut-off score ≥4 for disrupted menstrual function (Table 4.4).

Of those participants who scored ≥2 for the LEAF-Q injury component (n = 243), the most common reported injuries were muscular strain/tears, knee injuries, Achilles' tendonitis/ankle injuries and stress fractures (Appendix 8.1). Of those participants who scored ≥2 for the LEAF-Q gastrointestinal disturbances component (n = 303), the most common disturbances were related to bloating/gaseous abdomen opposed to cramps or stomach-ache when not menstruating (Appendix 8.1). Finally, of those who scored ≥4 for the LEAF-Q menstrual function component (n = 146), 52% of those who were capable of menstruation reported the cessation of menstruation with heavy training loads (Appendix 8.1).

4.3.3 FAST scores and key components

FAST descriptive data are shown in Table 4.3. 25% of participants were classified with DE and 9% with ED according to FAST scores (Figure 4.1). The percentage of participants who scored ≥3 points for FAST items (described in Chapter 3.4.2) are presented in Appendix 8.2. Sport participation is considered an important facet for their self-esteem according to 78% of participants, with 93% believing they have a lot of good qualities and 65% striving for perfection in all aspects of their life (Appendix 8.2). Most believed triathlon performance was related to their weight with 70% expecting performance improvements with weight reduction

and 74% worried that weight gain would impair performance (Appendix 8.2). Although, most (80%) acknowledged that as an athlete they were very conscious about consuming adequate calories and nutrients on a daily basis (Appendix 8.2).

Diet control during training was reported with 52% of participants controlling fat and calorie intake and 41% limiting carbohydrate intake (Appendix 8.2). 75% recognised they would worry about weight gain if they could not exercise and diet control during periods of injury or absence from training was reported with 70% reporting restricted calorie intake during these periods (Appendix 8.2). However, diet control did not report a high prevalence (10% to 11%) of behaviours typically associated with DE/ED such as, avoidance of food with >3 gram of fat, skipping meals due to alcohol consumption or taking dietary or herbal supplements to increase metabolism or assist in fat burning (Appendix 8.2). Regarding body dissatisfaction, 58% were not happy with their current weight and 60% were concerned about their body fat percentage (Appendix 8.2). Finally, almost 4 in 10 participants have used methods to keep their weight down that they perceive to be unhealthy and 47% believe most female athletes have DE habits (Appendix 8.2).

4.3.4 EDS-R scores

EDS-R descriptive data are shown in Table 4.3. 58% of participants were classified as symptomatic and 9% at risk of EXD according to EDS-R scores (Figure 4.1). Of those considered at-risk of EXD, ‘lack of control’, ‘withdrawal effects’ and ‘time’ (described in Chapter 3.4.3) were the most frequent EDS-R components in those participants classified at risk of EXD (n = 34; Table 4.5).

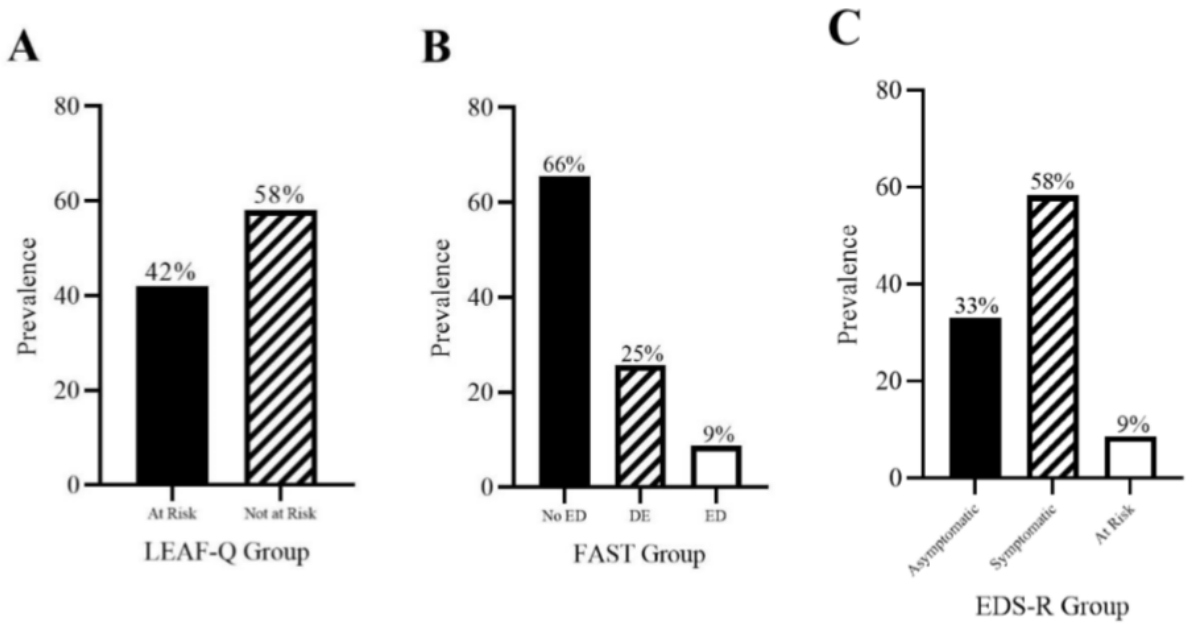


Figure 4.1. Prevalence of risk for LEA (A), DE/ED (B) and EXD (C).

No ED; no eating disorder, DE; disordered eating, ED; eating disorder, LEAF-Q; low energy availability in female's questionnaire, FAST; female athlete screening tool, EDS-R; exercise dependence scale-revised. Data presented as percentages.

Table 4.3. Response data for LEAF-Q, FAST and EDS-R questionnaires scores.

		Mean	STDEV	Median [IQR]	[Min, max]
LEAF-Q	Total (n=393)	7	4	7 [6]	[0; 21]
	At Risk (n=165)	11	3	10 [4]	[8; 21]
	Not at Risk (n=228)	4	2	4 [3]	[0; 7]
FAST	Total (n=393)	73	15	72 [23]	[42; 112]
	No ED (n=260)	65	9	66 [14]	[42; 78]
	DE (n=99)	86	4	86 [6]	[79; 94]
	ED (n=34)	101	5	101 [7]	[95; 112]
EDS-R	Total (n=393)	21	6	20 [7]	[7; 42]
	Asymptomatic (n=130)	15	3	15 [4]	[7; 21]
	Symptomatic (n=229)	23	3	22 [5]	[16; 33]
	At Risk (n=34)	33	4	33 [4]	[24; 42]

STDEV; standard deviation, IQR; interquartile range, DE, disordered eating; ED, eating disorder; LEAF-Q, low energy availability in females' questionnaire; FAST, female athlete screening tool; EDS-R, exercise dependence scale revised.

Table 4.4. Prevalence of risk for LEAF-Q components.

LEAF-Q Component	Cut-off Score	Frequency	Percent
Injury*	At Risk	243	62
	Not at Risk	150	38
Gastrointestinal**	At Risk	303	77
	Not at Risk	90	23
Menstrual***	At Risk	104	27
	Not at Risk	289	74

Cut-off score to be classified as at risk: * ≥2, ** ≥2, and *** ≥4

Table 4.5. Frequency of those who meet the cut-off score (≥5) for the seven subscales of the EXD-R as reported by female triathletes classified as at risk for EXD (n = 34).

EDS-R questionnaire component	Frequency	Percent
1. Withdrawal effects	27	**79**
2. Continuance	23	68
3. Tolerance	23	68
4. Lack of control	30	**88**
5. Reduction of other activities	25	74
6. Time	26	**77**
7. Intention effects	21	62

EDS-R, exercise dependence scale-revised; EXD, exercise dependence

4.3.5 Correlative analysis

As shown in Table 4.6, a weak but significant ($p < 0.001$) positive correlation was observed between LEAF-Q and FAST scores with an R^2 of 11% (6, 18%). Similarly, there was a weak, but significant ($p < 0.001$) positive correlation observed between LEAF-Q and EXD-R scores with an R^2 of 9% (4, 16%) and between FAST and EXD-R scores with an R^2 of 23% (16, 31%) as shown in Table 4.6.

Table 4.6. Summary of bivariate correlations.

Variables	r_s	P	R^2 (%)	R^2 (%) 95% CI lower	R^2 (%) 95% CI upper
LEAF-Q score and FAST score	0.330	< .001	11	6	18
LEAF-Q score and EDS-R score	0.305	< .001	9	4	16
FAST score and EDS-R score	0.480	< .001	23	16	31

CI, confidence interval; LEAF-Q, low energy availability in female's questionnaire; FAST, female athlete screening tool; EDS-R, exercise dependence scale-revised

4.3.6 Cross-tabulation

The cross-tabulation of FAST and LEAF-Q categories (Table 4.7) demonstrated more athletes at risk for developing low EA in the groups with DE and ED than expected under the null hypothesis of no association (count vs. expected count). The cross-tabulation of EXD-R and LEAF-Q categories (Table 4.8) demonstrated more athletes at risk for developing low EA in the symptomatic and at risk of EXD groups than expected under the null hypothesis of no association (count vs. expected count). Finally, the cross-tabulation of FAST and EXD-R categories (Table 4.9) demonstrated more athletes at risk of EXD in the group with ED and more athletes with a symptomatic profile for EXD in the group with DE than expected under the null hypothesis of no association (count vs. expected count; Table 4.9).

Table 4.7. Cross-tabulation of Female Athlete Screening Tool and Low Energy Availability in Female's Questionnaire score categories.

Classification		At Risk of Low EA	Not at Risk of Low EA	Total
No eating disorder	Count	82	178	260
	Expected Count	109.2	150.8	
	Column %	49.7	78.1	66 % of 393
Disordered eating	Count	59	40	99
	Expected Count	41.6	57.4	
	Column %	35.8	17.5	25% of 393
Eating disorder	Count	24	10	34
	Expected Count	14.3	19.7	
	Column %	14.5	4.4	9 % of 393

Pearson chi-square = 35.675; degrees of freed om = 2; p = <.001.

Table 4.8. Cross-tabulation of Exercise Dependence Scale-Revised and Low Energy in Female's Questionnaire score categories.

Classification		At Risk of Low EA	Not at Risk of Low EA	Total
Asymptomatic	Count	40	90	130
	Expected Count	54.6	75.4	
	Column %	24.2	39.5	33% of 393
Symptomatic	Count	102	127	229
	Expected Count	96.1	132.9	
	Column %	61.8	55.7	58% of 393
At Risk of Exercise Dependence	Count	23	11	34
	Expected Count	14.3	19.7	
	Column %	13.9	4.8	9% of 393

Pearson chi-square = 16.521; degrees of freed om = 2; p = <.001.

Table 4.9. Cross-tabulation of Exercise Dependence Scale-Revised and Female Athlete Screening Tool score categories.

Classification		Asymptomatic	Symptomatic	At Risk of Exercise Dependence	Total
No eating disorder	Count	112	134	14	260
	Expected Count	86.0	151.5	22.5	
	Column %	86.2	58.5	41.2	66.2% of 393
Disordered eating	Count	18	34.1	7	99
	Expected Count	32.7	74	8.6	
	Column %	13.8	57.7	20.6	25.2% of 393
Eating disorder	Count	0	21	13	34
	Expected Count	11.2	19.8	2.9	
	Column %	0	9.2	38.2	8.7

Fisher's Exact = 63.012; degrees of freed om = 2; p = .000.

4.3.7 Logistic regression

BLR was performed to ascertain the association of eating attitudes (FAST) and exercise behaviours (EDS-R) on the likelihood that participants were at low risk of low EA. The logistic regression model was statistically significant, χ^2 (2) = 39.916, p < .001. The model explained 13% (Nagelkerke R^2) of the variance in low EA and correctly classified 66% of cases. Sensitivity was 54%, specificity was 75%, positive predictive value was 61% and negative predictive value was 31%. Of the two predictor variables, both eating attitudes and exercise behaviours were statistically significant (Table 4.10). Participants with no ED had 3.375 times higher odds of being low risk of low EA than those with DE/ED. Similarly, participants not at risk of EXD had 2.489 times higher odds of being low risk of low EA than those at risk of EXD.

Further to this, BLR was performed to ascertain the association of exercise behaviour (EDS-R) on the likelihood that participants were considered at risk of DE/ED (FAST). The logistic regression model was statistically significant, χ^2 (1) = 9.745, p < .002. The model explained only 3.4% (Nagelkerke R^2) of the variance in eating attitudes and correctly classified 68% of cases. Exercise behaviour was a statistically significant predictor variable as shown in Table 4.11. Participants considered not at risk of EXD had 3.110 times higher odds of not having DE/ED than those at risk EXD.

Table 4.10. Binary Logistic Regression predicting likelihood of low EA based on eating attitudes (FAST) and exercise behaviours (EDS-R).

							95% CI for Odds Ratio	
		B	SE	Wald	Df	P	Odds Ratio	Lower	Upper
Not at-Risk LEAF-Q	FAST (No DE/ED)	1.216	.226	28.975	1	<.001	3.375	2.167	5.255
	EDS-R (No EXD)	.912	.399	5.217	1	.022	2.489	1.138	5.445

Chi-square = 39.916; degrees of freed om = 2; p = <.001.

Reference category = at risk of low EA

LEAF-Q, low EA in female's questionnaire; DE, disordered eating; ED, eating disorder; FAST, female athlete screening tool; EXD, exercise dependence, EDS-R, EXD scale-revised.

Table 4.11. Binary Logistic Regression predicting the likelihood of disordered eating behaviour (FAST) based on exercise attitudes (EDS-R).

							95% CI for Odds Ratio	
		B	SE	Wald	Df	P	Odds Ratio	Lower	Upper
No DE/ED FAST	EDS-R (No EXD)	1.135	.367	9.583	1	.001	3.110	1.516	6.379

Chi-square = 9.745; degrees of freed om = 1; p = .002.

Reference category = DE/ED (FAST).

DE, disordered eating; ED, eating disorder; FAST, female athlete screening tool; EXD, exercise dependence; EDS-R, exercise dependence scale-revised.

4.4 Discussion

The present study aimed to investigate the prevalence of and potential associations between LEA, DE/ED and EXD in competitive female triathletes. The principle findings were that: 1) 42% of participants were classified as at risk of LEA, 25% with DE and 9% ED symptoms, and 9% were at risk of EXD. 2) Participants were less likely to be at risk of LEA if they were classified as having no DE/ED or they were not at risk of EXD, and 3) participants were more likely to be at risk of EXD if they were classified with ED and participants were more likely to be at risk of being symptomatic for EXD if they were classified with DE. Furthermore, 4) both eating attitudes and exercise behaviour were significant predictors of LEA and EXD was a significant predictor of DE/ED risk in this population.

The findings of study 1 are the first to examine a cohort of female triathletes to investigate the prevalence of LEA, DE/ED, and EXD. Study 1 found female triathletes are a sub-group at risk of developing LEA which may increase the risk of developing Triad or RED-S. LEA in female triathletes may be underpinned by known risk factors, such as, inadequate nutritional practices (with or without DE/ED) or it may be underpinned by excessive energy expenditure (with or without EXD). Current findings suggest the development of LEA is not restricted to elite female athletes or single-sport endurance events.

4.4.1 LEA

In the present study, LEAF-Q scores classified 42% (n=165) of participants as at risk of LEA. Such findings are in line with prevalence data reported by Slater, et al., (2016) who examined the prevalence of LEA in female recreational exercisers (n=109) from both individual and team sports in New Zealand. It was found that 45% (n=49) of participants were classified as at risk of LEA and significantly more participants from individual sports were classified as at risk (70%) compared with team sports (35%). These authors speculated the

higher prevalence observed in individual sports may be due to the emphasis on aesthetics. One difference between the studies was Slater, et al., (2016) not screening for DE behaviour alongside the LEAF-Q, as recommended by Melin, et al., (2014). This makes it difficult to identify the potential cause of the LEA identified as the LEAF-Q alone only investigates the physiological consequences of persistent energy deficiency and not the causes (Melin, et al., 2014). The IOC have also suggested that where screening for LEA should be accompanied by screening for DE/ED and vice versa to facilitate the detection of underlying causes (Mountjoy, et al., 2014; 2018).

Folscher, et al., (2015) reported 44% (n=134) of elite and recreational South-African ultra-marathon runners were classified as at risk of LEA by the LEAF-Q. The authors believed it likely a result of the high volume of training and a lack of knowledge around the implications of inadequate nutritional practices. In contrast to the current study, the mean age (40 years) of participants indicated a mature population and included participants who were classified as post-menopausal, had a history of previous hysterectomy or were using contraceptives other than oral (i.e., hormonal coil or implant) in the analysis. Post-menopausal females may be considered a potential at risk group for the consequences of LEA due to the increased osteoporosis risk resultant from hypo-estrogenic state, however, more research in relation to the additional effects or prevalence of LEA in this population are required (Kataoka, Luo, Chaimani & Onishi, et al., 2020). The LEAF-Q has been validated in endurance athletes, however, its use with post-menopausal females may not be applicable and lead to false positives. It highlights the importance of differential diagnosis to establish the origin of the menstrual function component of the LEAF-Q, via the clinical evaluation of sex hormones and ultrasound by a skilled gynaecologist, to avoid under-or-over estimating LEA risk (Melin, et al., 2014).

The current study reported a high prevalence of individuals considered at risk of LEA reporting an increased incidence of injury and gastrointestinal disturbances. A cross-sectional study of Australian Olympic athletes (male: n=26; female: n=55) by Drew, et al., (2017) supported associations between LEA risk and self-reported injury, illness, and gastrointestinal disturbances. These findings suggest that athletes may display other physiological signs and symptoms of LEA other than those traditionally expected. For example, not all female athletes at risk of LEA will show clinical signs of menstrual dysfunction (MD) and not all female athletes with MD are in a state of LEA. Thus, highlighting the complexity of identifying individuals at risk of LEA (Melin, et al., 2015; Drew, et al., 2017). Taken together with current findings, it is important that female athletes presenting with physiological symptoms beyond that of MD are evaluated for LEA.

To our knowledge, this is one of the first studies to focus exclusively on screening a large cohort of competitive female triathletes for the prevalence of LEA. However, an early, small cross-sectional study of club-based female triathletes reported 60% (n=9) were in a calorific deficit and 40% had a history of amenorrhea (Hoch, et al., 2007). Authors proposed the calorific deficit was consistent with DE pathology in this group of athletes. Although a potential acute response to racing opposed to normal behaviour, a cross-sectional study investigating the EB of Ironman distance triathletes reported female participants (n=8) were in a significant negative EB post-race (Kimber, Ross, Mason & Speedy, 2002). Both studies highlighted the high training volume required for triathlon and the importance of adequate fueling. However, this may not be representative of the population of triathletes due to the small sample size recruited in both studies.

Taken together, these studies have suggested a prevalence rate of athletes at risk of LEA ranging from 44% to 69% across a variety of endurance sports, which agrees with the current study. Not all studies have agreed with such a high prevalence of athletes at risk of LEA. For

example, Melin, et al., (2015) found 20% of elite female endurance athletes had LEA status with 28% diagnosed with DE symptomology and Heikura, et al., (2018b) found 31% of elite female middle- and long-distance runners and race walkers were at risk of LEA. Unfortunately, even the lowest prevalence in the reviewed studies at 20% indicates at least one in five athletes are at risk of LEA. The variability in prevalence may be explained by several factors: competitive level (recreational to elite), age, endurance sport category (single-sport or multi-sport), event distance (short to ultra-distance), and the measurement of EA (direct or self-report). The disparity and often small sample sizes recruited may to explain the variability in prevalence rates. Studies with larger sample sizes, as in the current study, may lead to more accurate or representative results of the population.

4.4.2 Eating attitudes

The frequent occurrence of DE and ED in female athletes in comparison to the general population has been long established in the literature (Sundgot-Borgen & Torstveit, 2004). Ackerman, et al., (2020) further acknowledged the association between DE/ED and the increased risk of developing LEA in the athletic population. This is in line with findings in the current study where eating attitudes was a significant predictor of LEA status, with more female triathletes displaying DE/ED considered at risk of LEA according to LEAF-Q scores. This illustrates the importance of this problem and the potential implications on long-term health and performance in female athletes. Plus, highlighting the significance of a multi-disciplinary approach in the screening of athletes for LEA, Triad, or RED-S. However, the aetiology of DE/ED is complex with many causal biopsychosocial factors, such as: age and pubertal maturation, gender, genetics, serotonin and dopamine disturbances, media exposure, pressures for thinness or thin-ideal internalisation, perfectionism, negative urgency, and negative emotionality/neuroticism (Culbert, Racine & Klump, 2015). The interplay between these

recognised biopsychosocial factors may explain the low predictive value of the FAST in the current study.

Estimating the prevalence of DE/ED among athletes remains elusive as many of the validated and standardised self-report DE/ED questionnaires are insensitive to the athletic population. Often under-or-over estimating eating pathology (Joy, et al., 2016). For example, DE/ED may be overestimated in athletes due to rigorous training programmes and eating patterns, which may be essential for optimal performance. Conversely, excessive exercise in the athletic population may reflect an athlete's desire to enhance performance, rather than the desire to burn calories to maintain a minimum body mass (Joy, et al., 2016). Notably, one of the motivational variables used to describe the aetiology of DE/ED and most often utilised in DE/ED questionnaires is a 'drive for thinness'. However, not all athletes strive for 'thinness', rather some athletes strive to reduce body fat while increasing muscle mass to enhance performance or be underpinned by body image dissatisfaction. This 'drive for leanness' or body image dissatisfaction may predispose athletes to DE/ED behaviours (Sears, Tracy & McBrier, 2012).

Nevertheless, in parallel with risk factors for LEA development, previous research has highlighted leanness sports as having an increased risk for DE/ED (Joy, et al., 2016). A study by Knechtle, et al., (2010) suggested body mass as being an important facet of performance in triathletes as an excess of adipose tissue usually requires a greater muscular effort to accelerate legs and, in theory, a higher energy expenditure during the cycle and run. This may lead some athletes to develop an unhealthy fixation towards food and body mass in the pursuit of attaining an 'ideal' physical attractiveness that is associated with enhanced performance, as was found in the current study. Over time this may lead to DE/ED and the onset of LEA.

Using the validated FAST to assess eating attitudes among athletes, the current study found 25% (n=99) of female triathletes were classified with DE symptoms and 9% (n=34) with ED, double that of general population rates (2-4%; Smink & Hoek, 2013; Dahlgren, Wisting & Rø, 2017). Indeed, it is important to note that the current study did not assess the prevalence of clinically defined DE/ED, per se. Future work would be advised to use the current gold standard assessment of the EDE conducted by a trained clinical psychologist (Joy, et al., 2016). A study assessing the prevalence of subclinical and clinical DE in elite female athletes identified 42% of athletes from aesthetic and 24% from endurance sports presented with DE symptoms (Sundgot-Borgen, et al., 2004). However, in a study comparing elite female athletes (n=186) to age-group matched population-based controls (n=145), athletes from leanness sports had a higher prevalence rate (47%) of ED than in non-leanness sports (20%) and controls (21%; Torstveit, et al., 2008). Both studies used the gold standard assessment previously described. Additional studies also reported a similar trend with authors proposing leanness sports as the primary stimulus for body dissatisfaction and DE behaviours among female athletes (Reinking & Alexander, 2005; Kong & Harris, 2015).

Collectively, these studies have suggested a prevalence rate of female athletes at risk of DE pathology (including ED) ranging from 20% to 45% across a variety of leanness sports, which agrees with the current study. However, the prevalence of DE pathology in non-elite athletes, across all ages, in multi-sport endurance events is largely unknown. A study by Mongrain, et al., (2018) investigated the prevalence of DE symptoms and concerns using the EAT-26 in non-elite multi-sport endurance athletes (114 males and 48 females). It was found only 6% of athletes were at risk of DE and represented 13% (n=6) of all female athletes included in the study. The variability in the prevalence of DE/ED symptomatology may be explained, at least partly, by the variability in sample size and in the tools used to determine DE/ED. It is possible that some eating attitude questions were too sensitive and therefore

subclinical and clinical DE symptoms may be underreported. Alternatively, it is possible that athlete age or competitive level may attenuate the preoccupation towards body mass for enhanced performance.

4.4.3 Exercise behaviours

Previous research has recognised the co-occurrence of addictive behaviours (Cook, et al., 2014; Müller, et al., 2015). DE behaviour and LEA in athletes have been associated with psychological factors such as perfectionism, stress, anxiety, and depression (Mountjoy, et al., 2014; 2018). Moreover, recent research by Turton, et al., (2017) have suggested the co-occurrence of EXD in endurance athletes may increase the risk of negative health and performance outcomes associated with LEA and subsequently Triad or RED-S. The current study found 58% (n=229) of female triathletes displayed a symptomatic (SY) profile and 9% (n=34) were classified at risk of EXD. This agrees with the estimated prevalence rates of 3-9% across an array of sports (Marques, et al., 2019). Although prevalence data for EXD in female athletes is lacking, a recent study among male triathletes indicated similar trends with 9% classified with EXD and 60% as symptomatic (Tallón, et al., 2017).

Currently, research is limited on the relationship between EXD, eating attitudes and LEA in the athletic population. Seminal work by Torstveit, et al., (2019) investigated the associations in trained male endurance athletes (cyclists, triathletes, and long-distance runners). It was found higher total EXD scores were associated with DE symptoms, higher training volume with a negative EB, and biomarkers of LEA (RMR, RMR_{ratio}, cortisol, testosterone, insulin, IGF-1, T_3 and glucose). Although these findings related to male athletes from both single-sport and multi-sport endurance events, this is comparable with findings in the current study where female triathletes classified as SY or at risk of EXD were more likely to be at risk of LEA and DE behaviour. Additionally, EXD was a significant predictor of LEA and DE/ED

risk in this population, further suggesting that EXD may increase exposure to the negative health and performance consequences associated with LEA, Triad, and RED-S.

Nevertheless, results should be interpreted with caution as endurance sports require large volumes and long periods of training in the pursuit of performance goals. Thus, making it difficult to distinguish between healthy extreme exercise and pathological excessive exercise (Müller, et al., 2015). Although EXD is not widespread, the aetiology of EXD is complex with many causal factors, such as: a participant's motivations to exercise, body dissatisfaction, stress reduction, perfectionism, and compulsive/addictive personalities (Landolfi, 2013). Back, et al., (2019) found anxiety was the predominant risk factor underpinning EXD and obsessive passion or exercise used as a coping strategy increased the risk of EXD. The interplay between these established causes of EXD may explain the low predictive value of the EXD-R in the current study.

4.4.4 Limitations

The current study should be interpreted with caution. To date there is no standardised or reference guidelines in the literature for the accurate assessment of EA status or prevalence of LEA which may be an impossible task to do accurately in free-living athletes (Mountjoy, et al., 2018; Logue, et al., 2020). However, in line with previous prevalence studies (Folscher, et al., 2015; Melin, et al., 2015; Heikura, et al., 2018a) and the most recent RED-S consensus statement (Mountjoy, et al., 2018), the current study utilised screening tools (LEAF-Q) for physiological symptoms of LEA and associated with Triad and RED-S, alongside an evaluation of eating attitudes (FAST). Although screening may lead to an earlier diagnosis the complexity of predicting health outcomes from screening data is well established (Bahr, 2016). In addition, there is no consensus on which screening tool has the best efficacy, data is reported over various time frames and there is no documented evidence for the proportion of false positives and

negatives of screening tools assessing LEA status (Mountjoy, et al., 2018). Future work would be advised to include both subjective and objective measures, such as biochemical markers of LEA, which cannot be falsified and may increase the sensitivity of measures to determine clinical signs and symptomology of those at risk. Though these measures too can be highly variable between and within individuals.

Alongside prevalence, the current study examined potential associations between eating attitudes and exercise behaviour as risk factors for LEA in female triathletes. However, the sequence of events that led to the findings reported cannot be determined and cannot imply causality. The current study is cross-sectional in design and future longitudinal studies with a sufficient sample size are required to fully understand the development and consequences of LEA. Nevertheless, combined with findings from previous research, the results are valuable in highlighting at risk groups to aid and direct future screening, early detection, and target awareness education.

4.5 Conclusion

Findings from this study showed that among our population of competitive female triathletes a significant proportion were considered at risk of LEA which may be underpinned by DE/ED or EXD. Thus, placing this group of athletes at increased risk for the development of Triad or RED-S and the associated long-term health and performance consequences. It is important we encourage female athletes to continue participating in multi-sport endurance events as the health benefits are well established. However, further research in this area and athletic population (i.e., role of age, performance level) is encouraged to further raise awareness and advocate for early screening and detection. The identification of at-risk groups, as in the current study, will facilitate athlete, coach and parent education and awareness. Coach and parent education is imperative as they may be instrumental in the early detection of LEA, by

recognising external warning signs (i.e., DE patterns, excessive exercise, increased injury, or a decline in performance). Coaches have a responsibility to refer athletes to relevant health professionals. This will safeguard the protection of athlete physical and mental health and performance across all ages and levels of female participation in triathlon.

4.6 Statement of original contribution

- This is the first study to screen a large cohort of female triathletes (n=393) to estimate the prevalence of risk for LEA, DE/ED and EXD.
- This is the first study to examine associations between LEA, DE/ED and EXD risk in a large cohort of female triathletes.

CHAPTER 5

INFLUENCE OF AGE ON THE PREVALENCE OF LEA RISK IN FEMALE TRIATHLETES

5.1 Introduction

The unique endurance sport of triathlon comprises a sequential swim, cycle and run over a variety of distances (see table 2.1) and under a variety of technical constraints. Each discipline is connected by a brief transition (Bentley, et al., 2002; Millet, et al., 2011). Irrespective of distance, competition is held between elite and non-elite athletes. Non-elite athletes, referred to as Age-Groupers in the triathlon field, compete against other athletes within the same 5-year age categories (Vleck, Millet & Alves, 2014). A continual growth in the relative participation of female Age-Groupers has been evident. Over the last decades it appears younger triathletes are more attracted to shorter distance triathlons (i.e., sprint or standard), and master triathletes (aged ≥ 40 years) more attracted to longer distance (i.e., Ironman; Lepers, 2020). It is thought the increased popularity of the shorter distances among younger triathletes, particularly standard distance, may be due to its inclusion as an Olympic sport (Lepers, 2013a). In contrast, the attraction of longer distances with advancing age has been associated with these athletes potentially having more available time, resources, and more financial stability to train and compete (Lepers, 2020).

Current literature in the field of triathlon has concentrated on profiling psychological (Hodges, Augaitis & Crocker, 2016; Peiffer, Abbiss, Sultana & Bernard, et al., 2016), physiological (Brisswalter, Wu, Sultana & Bernard, et al., 2014) and anthropometric (Canda, Castiblanco, Toro & Amestoy, et al., 2014; Rivas, Mielgo-Ayuso, Norte-Navarro & Cejuela, et al., 2015) characteristics of athletes. Similarly, participation and age-related performance trends from single events (i.e., Ironman Hawaii) have been well-described in the literature across triathlon distances and sexes (Wonerow, et al., 2017; Käch, Rüst, Nikolaidis & Rosemann, et al., 2018; Sousa, Nikolaidis, & Knechtle, 2020). To date, little is known about the prevalence and aetiology of LEA in female triathletes. This is despite leanness sports, such as triathlon, being considered at higher risk for the development of LEA as discussed earlier in

this thesis (Loucks, et al., 2011; Mountjoy, et al., 2018). For instance, previous findings reported 42% of competitive female triathletes (aged 18-54 years; n=393) were classified by the LEAF-Q as at-risk of LEA, increasing the risk of developing Triad or RED-S (study 1).

As outlined by the Triad and RED-S models, it is essential for athletes of all ages to maintain an adequate EI to prevent the risk of developing LEA and the associated negative health and performance consequences (Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). It is clear that short and long-term LEA impairs health and performance, with impairments to menstrual function and bone health considered the most serious clinical outcomes (De Souza, et al., 2014). Particularly, the consequences of LEA are likely to be more severe if developed during the critical phase of growth and development in adolescents and young adults. Similarly, if the LEA state with sub-clinical and clinical impairments to menstrual function and bone health are maintained for several years (De Souza, et al., 2014; see Chapter 2.3.3). It is therefore important to consider athlete's age and years of training. This will help further understand factors contributing to the development, onset, and progression of LEA and guide the implementation of prevention programmes across an athlete's lifespan.

Current literature has acknowledged LEA exists across all ages (Mountjoy, et al., 2014; 2018; Ackerman, et al., 2020). Thein-Nissenbaum, (2013) recognised the long-term negative implications of LEA (i.e., low BMD) identified in adolescents and young adults may manifest later in life. Particularly, when females' transition from pre-menopausal to post-menopausal. This relates to the hypometabolic state caused by LEA and/or the E_2 deficiency (hypoestrogenism) caused by menstrual dysfunction (i.e., FHA) that results in impaired bone mass accrual. Impaired bone mass accrual during adolescence and young adulthood will result in a lower PBM being achieved that typically is irreversible. Thus, bone loss in pre-menopausal and post-menopausal females will occur from an already-reduced bone bank (Thein-Nissenbaum, 2013). The co-occurrence of DE/ED with LEA and/or hypoestrogenism has also

been associated with an increased risk for cardiovascular events (i.e., cardiac arrhythmia or impaired endothelial function) and suicidal behaviour (Crow, et al., 2009; American Psychiatric Association, 2013; Smith, et al., 2013). Unfortunately, most of the current studies on LEA or on the Triad and RED-S models have been completed primarily with adolescent, University students or young adults from various sports. This has resulted in limited information regarding the prevalence of LEA risk in different age groups.

It is widely accepted that LEA can occur as a result of DE behaviour and clinical ED (Nattiv, et al., 2007; Mountjoy, et al., 2014). Previous findings reported 25% and 9% of competitive female triathletes (aged 18-54 years; n=393) were classified by the FAST with DE behaviour and clinical ED, respectively. Significant associations between eating attitudes and LEA were identified (study 1). The association between DE behaviour, clinical ED, and EXD has been documented in the literature (Szabo, 2010; Scharmer, et al., 2020). In chapter 4, a significant association between eating attitudes and EXD, as well as LEA risk in competitive female triathletes was found. It was also reported 9% of competitive female triathletes (aged 18-54 years; n=393) were classified by the EDS-R as at risk of EXD (study 1). Comparable to EA research, most of the studies examining DE/ED and EXD have primarily focused on adolescent and young adults and limited information exists on the prevalence of risk and underlying mechanisms across different age groups.

Current estimates suggest that overall, the manifestation of both DE behaviour Mangweth-Matzek & Hoek, 2017; Thompson & Bardone-Cone, 2019) and EXD (Costa, et al., 2013) symptomology decline with age. However, there may be critical transition periods (i.e., puberty and menopause) where body changes occur that may result in the increased risk of DE behaviour or clinical ED (Mangweth-Matzek & Hoek, 2017). In relation to EXD, it has suggested the decrease in symptoms may be associated with older adults being able to regulate emotions, and therefore mood, better than younger counterparts (Costa, et al., 2013).

In light of this, the present study aimed to, 1) investigate the prevalence of competitive female triathletes at risk of LEA, DE/ED, and EXD in different age groups, 2) determine if there were differences in LEA, DE/ED, and EXD scores between age groups, and 3) investigate possible associations between age and LEA, DE/ED, and EXD in competitive female triathletes.

5.2 Materials and methods

5.2.1 Research design

This cross-sectional, descriptive study required participants to complete an anonymised online questionnaire – the 'Female Health Questionnaire'. The study was reviewed and granted ethical approval (Appendix 1) from the University of Sunderland Research Ethics Group and conducted in accordance with the Declaration of Helsinki (2013). All participants took part in the study voluntarily, were provided with information specifying the study details including inclusion and exclusion criteria (table 3.1), provided implied consent for the data to be used in the study and no participation incentives were offered (outlined in Chapter 3.2 – 3.4, Appendices 1-3).

5.2.2 Participants

Recruitment posters for healthy, pre-menopausal, female triathletes, aged 18 or over were arbitrarily advertised via social media platforms (Facebook, Instagram, and Twitter), supported by flyers and word-of-mouth approaches when relevant. Participants were asked to complete the anonymous online 'Female Health Questionnaire' after reading the information sheet (including inclusion and exclusion criteria) and providing implied consent (Appendix 2-3). Table 3.1 (Chapter 3) outlines the inclusion ad exclusion criteria for study 2. Table 5.1 outlines participant recruitment, exclusion and inclusion into the final study sample for study 2. N=393 individuals were included in the final analysis for study 2 and although a large

reduction in the original sample size is evident, a sample size of N=393 met the calculated sample size estimation for the cross-sectional study. Based on a population size of 10,000 registered female triathletes with British Triathlon (British Triathlon, 2021a), a sample size estimation of n=370 was calculated for study 1, with a confidence level of 95% and a 5% margin of error (Qualtrics, London, UK). Study 2 sample size also falls within the sample size range previously reported in LEA prevalence studies (range 10 to 833; Schaal, et al., 2011a; Logue et al., 2019 – table 2.2).

Table 5.1 Sample size for study 2

N recruited	Excluded	Included
N = 878	N=36 post-menopausal or non-triathlete N=303 LEAF-Q exclusion (pregnant, breastfeeding, chronic illness, use of forms of contraceptive other than oral; increase false positive; Melin, et al., 2014) N= 146 Female Health Questionnaire incomplete	N = 393

5.2.3 Data collection and questionnaire data

All the data collection procedures, coding, scoring, and questionnaire data used in the study are outlined in Chapter 4.2.3 and 4.2.4. However, in brief, participants were asked to complete an anonymous online questionnaire, the 'Female Health Questionnaire', which incorporated the LEAF-Q (Melin, et al., 2014), the FAST (McNulty, et al., 2001), and the EDS-R items (Downs, et al., 2004). This was accessible for a four-week period between October 2019 and November 2019 to female triathletes. All data within the 'Female Health Questionnaire' was self-reported.

5.2.4 Statistical analysis

Data normality was assessed using the Shapiro-Wilk test. Descriptive data for age groups was calculated for demographic data and self-reported training load. Frequency analysis

for LEAF-Q, FAST, and EDS-R questionnaire scores was performed across age groups. Non-normally distributed data was compared using Kruskal-Wallis test followed by the Dwass-Steel Crichtlow-Fligner test to compare LEAF-Q, FAST, and EDS-R scores between age groups. Chi-square tests were used to assess associations between groups and the LEAF-Q, FAST, and EDS-R scores. Subsequently, a multinomial logistic regression (MLR) was performed to ascertain the effects of age group on the likelihood that participants would be classified as at risk or not on the prediction model for the EDS-R questionnaires. All statistical analyses were performed using SPSS (V.25; IBM Company, SPSS Inc., Chicago, USA), with the exception of the Kruskal-Wallis test and Dwass-Steel Crichtlow-Fligner analyses which was performed using jamovi (The jamovi project, 2020) version 1.2.2). Statistical significance was set *a priori* at $p \leq 0.05$.

5.3 Results

5.3.1 Participant characteristics

N=393 individuals who met the inclusion criteria (table 3.1 – chapter 3) were included in the final analysis of study 2. Participants were divided in to three age groups based on recommendations in previous literature focusing on age-related differences (Szabo, 2000; Hale, et al., 2010; Costa, et al., 2013): 18-29 years (n = 101), 30-39 years (n = 159), and 40+ years (n = 133). Participant characteristics of the three age groups are presented in Table 5.2. Comparison of age groups found participants aged 18-29 years had significantly lower BMI than those aged 30-39 years and 40+ years, however this was a small effect (Cohen's *d* 0.23 - table 5.2).

Table 5.2. Self-reported age group participant characteristics and training load.

	18 – 29 (years) N=101	30 – 39 (years) N=159	40+ (years) N=133
Height (m)	1.67 (0.08)	1.65 (0.08)	1.66 (0.09)
Mass (kg)	63.0 (9.5)	65.0 (12.0)	64.0 (14.0)
BMI ($kg \cdot m^2$)	22.6 (3.3) *12	23.3 (5.0) *1	23.2 (4.4) *2
Training time (h·week)	11.5 (7.5) (3-30)	11.5 (5.5) (4-42)	11.0 (5.0) (3-22)

BMI, body mass index.
Data presented as median (interquartile range) or range in parentheses; n=393.
*1 Significant difference between groups, p <0.050.

5.3.2 LEAF-Q scores and key components

49% of participants aged 18-29 years were classified as at risk of LEA according to the LEAF-Q (figure 5.1). In comparison, of those aged 30-39 years and 40+ years, 40% and 39% were classified as at risk of LEA, respectively (figure 5.1). When assessing the individual LEAF-Q component scores for age groups, 68% of participants aged 18-29 years met the component cut-off score ≥2 for increased incidence of injury, compared with 62% and 58% of those aged 30-39 years and 40+ years, respectively (table 5.3). Of those aged 18-29 years, 84% met the component cut-off score ≥2 for increased gastrointestinal disturbances. Those aged 30-39 years and 40+ years also reported a high prevalence of gastrointestinal disturbances at 78% and 69%, respectively (table 5.3). Finally, 34% of participants aged 18-29 years met the component cut-off score ≥4 for menstrual dysfunction, compared to 25% of those aged 30-39 years and 40+ years (table 5.3).

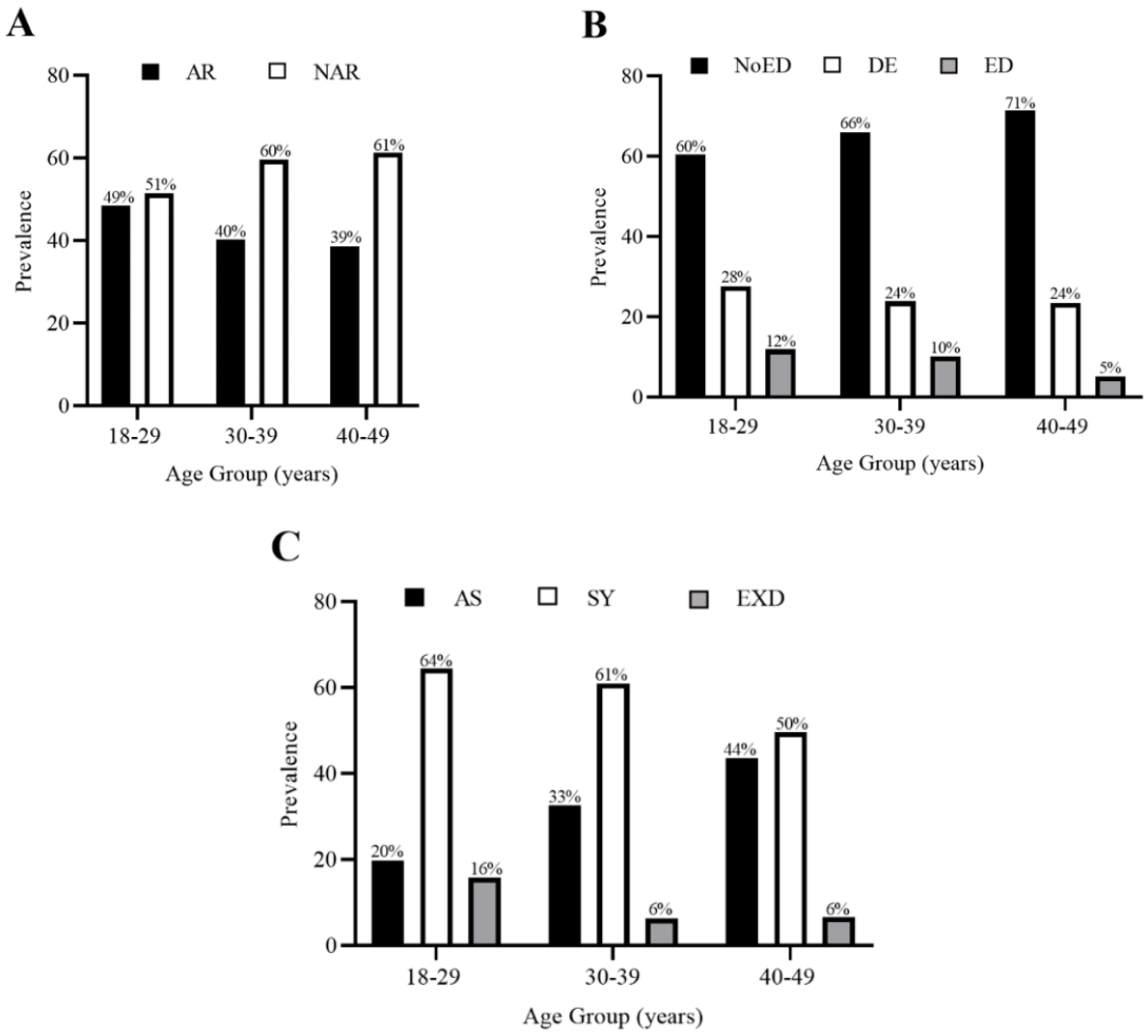


Figure 5.1. Prevalence of risk for LEA (A), DE/ED (B) and EXD (C) by age group.

AR, at risk; NAR, not at risk; No ED, no eating disorder; DE, disordered eating; ED, eating disorder; AS, asymptomatic; SY, symptomatic; EXD, exercise dependent. Data presented as percentage.

Table 5.3. Age group prevalence of risk for LEAF-Q components.

Age Group (years)	Injury		Gastrointestinal		Menstrual	
	At risk*	Not at risk	At risk**	Not at risk	At risk***	Not at risk
18 – 29 (n=101)	68	32	84	16	34	66
30 – 39 (n=159)	62	38	78	22	25	75
40+ (n=133)	66	48	81	33	27	87

*Injury: component cut-off scores for at risk ≥2.
**Gastrointestinal: cut-off scores for at risk ≥2.
***Menstrual: cut-off scores for at risk ≥4.

5.3.3 FAST scores and key components

28% and 12% of participants aged 18-29 years were classified with DE and ED according to the FAST, respectively (figure 5.1). In comparison, 24% and 10% of participants aged 30-39 years and 24% and 9% of participants aged 40+ years were classified with DE and ED (figure 5.1). The percentage of participants from each respective age group who scored ≥3 points for FAST items (outlined in Chapter 3.4.2) are presented in Appendix 9.1. All age groups considered sport participation an important facet for their self-esteem (range: 72% to 87%), with the majority (range: 91% to 94%) of participants from all age groups believing they have a lot of good qualities. However, more participants aged 18-29 years (77%) strive for perfection in all aspects of their life compared with those aged 30-39 years (66%) or 40+ years (56%). Most participants believed their triathlon performance was related to their weight, with more participants aged 40+ years (75%) and 30-39 years (70%) expecting performance improvements with weight reduction, compared to those aged 18-29 years (65%). Although, most (range: 78% to 81%) participants from all age groups acknowledged as an athlete they were very conscious about consuming adequate calories and nutrients on a daily basis (Appendix 9.1).

Diet control during training was reported across all age groups with more participants aged 18-29 years (57%) controlling fat and calorie intake than those aged 30-39 or 40+ years (50%). However, less participants aged 18-29 years (34%) limited carbohydrate intake compared with older participants (range: 42% to 45%). All age groups recognised they would worry about weight gain if they could not exercise, with more participants aged 18-29 years (84%) worried than their older counterparts (range: 70% to 75%). However, diet control did not report a high prevalence across all age groups of behaviours typically associated with DE/ED, avoidance of food with >3 gram of fat, skipping meals due to alcohol consumption, or taking dietary or herbal supplements to increase metabolism or assist in fat burning (range: 9% to 13%; Appendix 9.1).

In regard to body dissatisfaction, over half of participants from all age groups were not happy with their current weight and were concerned about their body fat percentage (range: 56% to 62%) with those participants aged 30-39 years the most concerned. Finally, almost 5 in 10 participants aged 18-29 years have used methods to keep their weight down that they believe are unhealthy compared to the 3 in 10 participants aged 30-39 years or 40+ years. Comparably, more participants aged 18-29 years (55%) believed most female athletes have DE habits compared with older participants (range: 40% to 46%; Appendix 9.1).

5.3.4 EDS-R scores

64% and 16% of participants aged 18-29 years were classified as symptomatic and at risk of EXD according to the EDS-R (figure 5.1). In comparison, 61% and 6% of participants aged 30-39 years and 50% and 6% of participants aged 40+ years were classified as symptomatic and at risk of EXD, respectively (figure 5.1). Of those considered at-risk of EXD, 'lack of control', (described in Chapter 3.4.3) was the most frequently cited EDS-R component for participants aged 18-29 years and 30-39 years (Appendix 9.2). However, 'lack of control',

'time', and 'withdrawal effects' were all equally cited in participants aged 40+ years (Appendix 9.2).

5.3.5 Between Group Comparisons

A Kruskal-Wallis test was conducted to determine if there were differences in LEAF-Q scores between groups that differed in age: 18-29 years, 30-39 years, and 40+ years. Distributions of LEAF-Q scores were similar for all groups, as assessed by visual inspection of a boxplot. Median LEAF-Q scores were statistically significantly different between the age groups, χ^2 (2) = 9.06, p = .010, however this was a small effect (ε^2 = .023). Subsequently, pairwise comparisons were performed using the Dwass-Steel Crichtlow-Fligner test for multiple comparisons. Adjusted p-values are presented. This post-hoc analysis revealed statistically significant differences in median LEAF-Q scores between the 18-29 years and 40-49 years (p = .011) age groups, but not between any other group combination (table 5.4).

A Kruskal-Wallis test was run to determine if there were differences in FAST scores between the three age groups. Distributions of FAST scores were similar for all age groups, as assessed by visual inspection of a boxplot. Median FAST scores were not statistically significantly different between groups, χ^2 (2) = 5.65, p = .059, ε^2 = .014. Finally, a Kruskal-Wallis test was run to determine if there were differences in EDS-R scores between the three age groups. Distributions of EDS-R scores were similar for all groups, as assessed by visual inspection of a boxplot. Median EDS-R scores were statistically significantly different between the age groups, χ^2 (2) = 18.6, p < .001, however this was a small effect (ε^2 = .048). Subsequently, pairwise comparisons were performed using the Dwass-Steel Crichtlow-Fligner test for multiple comparisons. Adjusted p-values are presented. This post-hoc analysis revealed statistically significant differences in median EDS-R scores between the 18-29 years and 40+ years (p = <.001) age groups, but not between any other group combination (table 5.5).

Table 5.4. Dwass-Steel-Critchlow-Fligner Pairwise Comparisons for LEAF-Q.

Age Group (years)		W	P
18 – 29	30 – 39	-3.27	.054
18 – 29	40+	-4.09	.011
30 – 39	40+	-1.21	.671

χ^2 (2) = 9.06, p = .010, ε^2 = .023
LEAF-Q, low energy availability in female's questionnaire

Table 5.5. Dwass-Steel-Critchlow-Fligner Pairwise Comparisons for EDS-R.

Age Group (years)		W	P
18 – 29	30 – 39	-3.30	.051
18 – 29	40+	-6.13	.001
30 – 39	40+	-3.21	.061

χ^2 (2) = 18.6, p < .001, ε^2 = .048
EDS-R, exercise dependence scale-revised

5.3.6 Cross-tabulation

A chi-square test for association was conducted between age and LEAF-Q categories. All expected cell frequencies were greater than five. The test of independence showed that there was no significant association between age and LEAF-Q category, χ^2 (2) = 2.419, p = 0.298 (Appendix 9.3). Similarly, a chi-square test for association showed that there was no significant association between age and FAST category, χ^2 (4) = 5.507, p = 0.239 (Appendix 9.4). A However, there was a statistically significant association between age and EDS-R category, χ^2 (4) = 20.246, p = <.001 (Table 5.6).

Table 5.6. Cross-tabulation of Age Group and EDS-R score categories.

Age Group (years)		Asymptomatic	Symptomatic	At risk EXD	Total
18-29	Count	20	65	16	101
	Expected Count	33.0	58.9	9.1	
	Column %	16.1	29.4	47.1	27% of 393
30-39	Count	52	97	10	159
	Expected Count	52.0	92.7	14.3	
	Column %	41.9	43.9	29.4	42% of 393
40+	Count	58	67	8	119
	Expected Count	44.0	77.5	11.5	
	Column %	44.6	29.3	23.5	31% of 393

Pearson chi-square = 20.246; degrees of freed om = 4; p = <.001.
EXD, exercise dependence; EDS-R, exercise dependence scale revised.

Table 5.7. Multinomial Logistic Regression predicting likelihood of EDS-R based on age group.

								95% CI for Odds Ratio	
		B	SE	Wald	Df	P	Odds Ratio	Lower	Upper
Symptomatic	18-29 years	1.034	.312	10.969	1	<.001	2.813	1.525	5.189
	30-39 years	.479	.248	3.722	1	.054	1.615	.992	2.628
At risk EXD	18-29 years	1.758	.505	12.130	1	<.001	5.800	2.157	15.597
	30-39 years	.332	.511	.422	1	.516	1.394	.512	3.798

Chi-square = 19.828; degrees of freed om = 4; p = <.001.
EDS-R Reference category = Asymptomatic.
Age Group 40+ years = set to zero.
EXD, exercise dependence; EDS-R, exercise dependence scale revised.

5.3.7 Logistic regression

An MLR was performed to predict the likelihood of EXD classification (EDS-R) based on age group. The model was statistically significant, χ^2 (4) = 19.828, p <.001. Using the conventional p ≤ 0.050 threshold, age was a statistically significant predictor for those aged 18-29 years (as shown in Table 5.7). Participants aged 18-29 years had 2.8 times higher odds of being symptomatic and 5.8 times higher odds of being at risk of EXD than their older counterparts.

5.4 Discussion

The present study aimed to investigate the prevalence of LEA, DE/ED, EXD in female triathletes. An additional aim was to examine the influence of age. Accordingly, the main findings were that: 1) the prevalence of those categorised with LEA, DE/ED, and EXD was greater in younger female triathletes compared to their older counterparts 2) There was significant differences in LEA risk and EXD scores between younger and older participants but not for DE/ED scores, however, the prevalence of no ED was higher in older participants than younger. 3) Finally, the only significant association was between age and EXD, with younger participants more likely to be categorised with maladaptive patterns of exercise than older participants.

Taken together, the findings of this study are the first, to our knowledge, to screen a large cohort of female triathletes to examine the influence of age on LEA, DE/ED, and EXD risk. The findings of this study suggest that LEA, DE/ED, and EXD exist in both younger and older female triathletes, however, differences do exist between age groups with prevalence rates declining with age.

5.4.1 Influence of age on LEA

In the current study, the risk of developing LEA existed in significant numbers across all age groups of competitive female triathletes. This aligns with the current Triad and RED-S models (Nattiv, et al., 2007; De Souza et al, 2014; Mountjoy, et al., 2014; 2018). Age-related differences did exist between age groups in the current study with increased prevalence of those at risk of LEA in younger participants, compared with their older counterparts. Although the risk of developing LEA is greater in younger athletes and prevalence may decline with age, the risk does not appear to disappear completely. To date, the majority of studies have focused on the younger athlete (see table 2.2). Current findings support further work being undertaken in the 'older' age groups across athletic populations. The longer an athlete is in a state of LEA, the greater the risk of irreversible impairments to health and performance (Thein-Nissenbaum, 2013). The consequence of these impairments may change throughout an athlete's lifespan. LEA- related impairments to menstrual function (i.e., primary, or secondary amenorrhea) and bone health (i.e., failure to achieve PBM) pose the greatest risk during adolescence and young adulthood as these impairments may manifest later in life (Thein-Nissenbaum, 2013). During adulthood this may manifest as fertility issues due to menstrual dysfunction, whereas the older athlete may experience greater consequences from impaired BMD or impaired endothelial function as they transition through menopause (Thein-Nissenbaum, 2013).

In the present study, 49% of participants aged 18-29 years were at risk of LEA as identified by the LEAF-Q. Previous studies that have recruited adolescent to young adult populations (see table 2.2) have suggested a prevalence rate ranging from 18% to 80%, which agrees with the current study. The wide range in prevalence rates reported is likely a reflection of the variability in EA methods, definitions and thresholds used (see chapter 2.3.1), variability in sports, and performance level examined. Although the influence of age was not directly examined in these studies, it is proposed the higher prevalence evident in younger athletes compared to older may be explained by several factors. For instance, higher physical activity

levels, less work and family commitments, they may be more influenced by the attitudes, behaviours, and health literacy of their coach/trainer/parents, they may not be as emotionally resilient as older athletes, evolving trends in the Western media's portrayal of the 'ideal athletic body', and they are still developing their relationship with food and exercise (Nattiv, et al., 2007; Kroshus, Sherman, Thompson & Sossin, et al., 2014; Staal, Sjödin, Fahrenholtz & Bonnesen, et al., 2018; Civil, Lamb, Loosmore & Ross, et al., 2019). Moreover, these studies have included participants from a variety of sports and performance levels and therefore no direct comparison can be drawn.

Adolescence is widely received as a period of growth and development beginning at puberty and ending at adulthood. Although discrepancies exist in the definition of adolescent age, it has been divided into early adolescence (10-14 years), late adolescence (15-19 years) and young adulthood (20-24 years; Black, Victora & Walker, 2013). Optimal nutrition during this period is crucial to support the changes in body composition, maturation of organ systems, metabolic and hormonal fluctuations, and the formation of nutrient deposits (Sawyer, Afifi, Bearinger & Blakemore, et al., 2012; Das, Salam, Thornburg & Prentice, et al., 2017; Desbrow, Burd, Tarnopolsky & Moore, et al., 2019). Inadequate nutrition at any stage of adolescence may lead to delayed or stunted linear growth and impaired organ remodelling with negative long-term consequences (Das, et al., 2017). In line with the current study's findings for the individual LEAF-Q components, Muia, et al., (2016) recognised the increased prevalence of LEA and subsequent negative health implications in adolescent endurance athletes with high training loads. The most clinically significant symptoms of LEA are its effects on reproductive function and bone formation, as described in Chapter 2.3.3 (Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). Menstrual disturbances as a result of LEA can include functional hypothalamic amenorrhea, oligomenorrhea or delayed onset of menarche (described in Chapter 2.3.3). Adolescent menstrual disturbances may negatively impact on bone health, a

result of hypogonadism (low oestrogen levels), which leads to decreased bone acquisition and increasing the risk of stress fracture (Chapter 2.3.3; Thein-Nissenbaum, 2013; Mountjoy, et al., 2018).

Sustained LEA during this critical time of growth and development is typically irreversible with long-term health consequences. A lack of bone mass accrual during adolescence and early adulthood will result in a depleted bone bank (Thein-Nissenbaum, 2013). As the female athlete ages bone resorption begins to exceed formation, typically after the age of 30, resulting in pre- and post-menopausal women resorbing bone from an already-depleted bone bank (Karlamangla, Burnett-Bowie & Crandall, 2018). Consequently, increasing osteoporosis risk in an already at-risk age group. Although more younger female triathletes were at risk of LEA in the current study, approximately 40% of participants aged 30-39 years and 40+ years were also identified as at risk. As identified in Chapter 4, limited data exists examining the prevalence or effects of LEA in the older athletic populations. However, in line with this study, Folscher, et al., (2015) identified 44% (n = 134) of South-African ultra-marathon runners (mean age: 40 years; range: 21 to 65 years) as at risk of LEA. Although the influence of age was not directly examined, these authors proposed the increased risk may be related to the hypoestrogenic state evident in females as they transition from pre-menopausal to post-menopausal. Therefore, it may be more insightful to further explore any associations between the menopause and the development or effects of current or historical LEA. Alternatively, the high prevalence of LEA in older triathletes in the current study may be explained by the high training loads that were evident in both younger (range: 3-30 h·week) and older athletes (range: 3-22 h·week). This may suggest that some cases of LEA are inadvertent due to an inability match EI to EEE (Nattiv, et al., 2007).

5.4.2 Influence of age on eating attitudes

It is widely received that LEA can occur either intentionally (i.e., DE/ED) or inadvertently (i.e., inability to increase dietary EI to match EEE; Nattiv, et al., 2007; Mountjoy, et al., 2014). DE has been defined as a subclinical spectrum of disruptive eating behaviours, that may lead to EA, whereas ED encompass a psychiatric diagnosis in line with the DSM-5 (Bryne & McLean, 2001; American Psychiatric Association, 2013; Mancine, Kennedy, Stephan & Ley, 2020). DE is accepted in the literature as a subclinical ED and early recognition of symptomatology and identification of at-risk groups are critical in the prevention of long-term physical and mental health consequences (Nattiv, et al., 2007; Vardar, et al., 2007; Quatromoni, 2008).

In the present study, younger athletes aged 18-29 years had a marginally higher prevalence for DE (28%) than those aged 30-39 years and 40+ years (24%). The prevalence of ED in those aged 18-29 years (12%) and 30-39 years (10%) was double that of those aged 40+ years (5%). However, even the 5% of those aged 40+ years is greater than the general population rates (2-4%; Smink & Hoek, 2013; Dahlgren, et al., 2017). No significant differences were found between age groups ($p = 0.07$) which may be a result of small sample sizes per age group, or the age ranges used. As in Chapter 4, it is important to note that the current study did not assess the prevalence of clinically defined DE/ED, per se. It is difficult to compare the results with previous findings due to the inconsistencies in methodologies used to assess eating attitudes, sample sizes, and the variation of ages, performance levels, and sports studies (see table 2.4). In general population studies adopting DSM-5 criteria, the highest incidence rates for DE and ED were in females aged 14-24 years (Stice, et al., 2013; Javaras, Runfola, Thornton & Agerbo, et al., 2015). However, these studies only examined individuals aged 13 to 21 years (Stice, et al., 2013) and 8 to 30 years (Javaras, et al., 2015) with the lowest prevalence rates in those aged 8 to 13 years. Both studies acknowledged the difficulty of assessing the prevalence of clinical ED due to the high mortality associated with some ED (i.e.,

AN and BN). Although data is limited in older populations, the onset of ED after the age of 25 is considered atypical (Stice, et al., 2013).

In the current study, although the prevalence of ED was the lowest in older participants (5%), all age groups reported a prevalence rate greater than general population rates (2-4%; Smink & Hoek, 2013; Dahlgren, et al., 2017). The only consensus in the literature relates to female athletes being more at risk than male athletes. However, the results are relatively consistent with other studies reporting elevated prevalence rates of DE/ED (range: 6% to 45%) in young adult or adult women from leanness sports, compared to general population (Sundgot-Borgen, et al., 2004; Torstveit, et al., 2008; Bratland-Sanda & Sundgot-Borgen, 2013; Hauck, et al., 2020). The current study is also in line with the suggestion that there is a higher prevalence of DE compared to ED (Smink & Hoek, 2013).

In agreement with the current literature, all age groups considered sport participation an important facet for self-esteem which may reduce the risk of the development of DE/ED (de Oliveira Coelho, de Farias, de Mendonça & Lanzillotti, et al., 2013). Yet the pathways to the development of DE/ED in this athletic population may differ depending on age. Adolescence is a critical period in establishing an individual's lifelong relationship with food, which is particularly important in terms of the interplay between diet, exercise, and body image. Particularly as adolescence is a period of rapid changes in body shape and size (i.e., increased body fat; Desbrow, McCormack, Burke & Cox, et al., 2014). It is proposed that younger athletes may be unable to dissociate the relationship between adequate nutrition to support body composition change for improved performance and negative eating attitudes or behaviours aimed to achieve societal "ideals" of physique often portrayed in Western media (Martinsen, et al., 2010). The current study would support this as more younger participants (84%) worry about weight gain compared to older (70% to 75%). 63% of younger participant

confirmed guilt when eating fried foods compared to 53% of older participants and have done things to keep their weight down they believe are unhealthy (48% compared to 30%).

Perfectionism has been associated with the development of DE/ED and younger athletes may be more susceptible to perfectionistic traits (Sundgot-Borgen, et al., 2013). The current study would support this as more participants aged 18-29 years strived for perfection (77%) compared to older participants (56%). Younger athletes may also be more susceptible to strong achievement orientation, increased levels of anxiety or depression, and pressures from their coach/trainer/parents/peers as they transition through puberty (Sundgot-Borgen, et al., 2013). The current study would support these findings as more participants aged 18-29 years (65%) at times feel they are no good at all compared to older participants (45%). More participants aged 18-29 years (27%) try to lose weight to please others compared to older participants (11%). The higher prevalence of younger athletes (55%) compared to older (40%) believing that most female athletes have some DE behaviours may reflect differing social norms and peer influence across the lifespan.

On the other hand, it is thought the peri-menopausal phase may be a critical period of time in the older athlete for the development or exacerbation of DE/ED, which may explain the high prevalence rates of participants aged >30 years in the current study. A result of hormonal changes associated with increases in both body mass and fat mass, and redistribution of body fat which also may negatively impact on mood (Slevec & Tiggemann, 2011; Thompson & Bardone-Cone, 2018; 2019). However, this was not evidenced in the current study as although younger participants had a lower BMI than older, all groups were in the normal range for BMI and had a small effect size (Cohen's *d* 0.23). Nevertheless, the prevalence and aetiology of DE/ED in non-elite, multisport endurance athletes and its relationship with LEA across the lifespan warrants further study.

5.4.3 Influence of age on exercise behaviours

Finally, the current study found a significant association between age and EXD in female competitive triathletes. Comparable to previous findings, it was found that EXD symptoms declined with age as participants aged 40+ years reported lower EXD scores than their younger counterparts (Szabo, 2000; Edmunds, Ntoumanis & Duda, 2006; Hale, Roth, DeLong & Briggs, 2010; Sussman, et al., 2011; Costa, et al., 2013). The age-related decline in EXD symptoms may be a result of older athletes having developed a more balanced lifestyle, have more developed emotional resilience, developed better coping mechanisms which can prevent behavioural disturbance, have lower prevalence rates of DE/ED, and their motivations for exercise may place emphasis on the general health and well-being and social aspects of participation (Szabo, 2000; Costa, et al., 2013; Landolfi, 2013; Back, et al., 2019; Lukács, Sasvári, Varga & Mayer, 2019; Hauck, et al., 2020). In contrast, Hale, et al., (2010) reported no significant differences between age groups for EXD symptoms in male weightlifters. This may be explained by the wide age range used (i.e., young adults: 18-24 years and adults: 25-55 years) or by gender related to differing family responsibilities.

Costa, et al., (2013) reported significantly more adult male gym users (25-44 years) were classified by the EDS-R as at-risk of EXD than young (18-24 years) but no significant age differences in females were reported. It was proposed this was likely due to males potentially displaying a 'drive for muscularity' compared to females displaying a 'drive for thinness'. Costa, et al., (2013) also reported some age differences in the EDS-R subscales. For instance, higher levels of tolerance and time in young and adult groups than middle-aged adults (45-64 years) and young adults had lower intention scores than adults. In the current study, lack of control (i.e., persistent desire or unsuccessful effort to cut down or control exercise) was the most prevalent subscale across all age groups. However, both the 18-29 and 30-39 age groups had a higher prevalence in reduction of other activities than the 40+ group who had the

lowest prevalence: and the two older groups had a higher prevalence of withdrawal effects than the youngest group. The 18-29 and 30-39 age groups had a lower prevalence for intention effects (i.e., engaged with exercise longer than intended) than the 40+ group. These results underline that despite moderate differences in the overall EDS-R score by age, the prevalence of specific EXD symptoms may change by age group.

The current study has addressed the paucity of available evidence in the prevalence of EXD in female multisport endurance athletes across age groups. Although prevalence data in female athletes is generally lacking, Marques, et al., (2019) highlighted the prevalence in male and female athletes from various sports ranged from 1% to 17% when using the EAI or EDS. A further review by Di Lodovico, et al., (2019) reported a prevalence rate of 4% in endurance athletes when using the EDS and 14% when using the EAI. The current study's findings fall within these ranges as across all age groups prevalence rates were 6% to 16% in competitive female triathletes. Nevertheless, the variability in prevalence reported highlights the urgent need for consistency in terminology, definitions, and well-validated instruments used for future screening of EXD. It is important to note that current assessment instruments screen for individuals considered at-risk of EXD, rather than diagnosed EXD and the DSM-5 does not currently consider EXD as a behavioural addiction (Szabo, et al., 2015). In addition, previous studies fail to differentiate between primary and secondary EXD and fail to provide clear descriptions of the populations studied (Mónok, et al., 2012; Landolfi, 2013).

Furthermore, the highest prevalence rates were found in those participants aged 18-29 years (16%) further confirming previous research that the most at-risk groups are adolescent to young adults, and this is a critical period in the development of negative exercise behaviours that may predict the likelihood of athletes also being at risk of LEA. Further longitudinal research is warranted to assess exercise behaviours, EXD symptoms and its association with eating attitudes and EA status from early adolescence into adulthood in this population.

5.4.4 Limitations

The current study should be interpreted with caution. Specific to the current study, although this is one of the few studies to examine the influence of age on LEA, DE/ED and EXD risk, results are based on cross-sectional comparisons of participants in different age groups, rather than observations of change as individuals grow older. The sequence of events that lead to the findings reported in the current study cannot be determined and cannot imply causality. Therefore, the use of longitudinal studies would provide better interpretation in future work, allowing attributions related to cause or direction of effects. As the current study focused on individuals aged 18-54 years, findings cannot be generalised to junior and master triathletes. Future work would be advised to increase recruitment specifically across the 5-year age categories evident in triathlon events to increase ecological validity and to include an age category representative of junior triathletes aged <18 years and master triathletes aged ≥50 years. Finally, the current study did not control for ethnicity which may limit the generalisation of results. Nevertheless, combined with findings from previous research, the results are valuable in highlighting at risk age groups to aid and direct future screening, early detection, and target awareness education.

5.5 Conclusion

In summary, findings from this study showed that the prevalence of those at risk of LEA, DE/ED and EXD was higher in younger competitive female triathletes compared with their older counterparts. Although it is important to note that these concepts existed across all age groups above general population norms. In providing the overall prevalence in different age groups and examining age-related changes, the current study may aid in identifying specific groups of competitive female triathletes who are at greater risk for long-term health consequences. Additionally, identifying the change in specific symptomology of LEA, DE/ED,

and EXD over time may help inform differing education strategies for differing age groups. Thus, helping to target and guide the implementation of early screening and education initiatives across the female athlete's lifespan in the pursuit of protecting athlete physical and mental health. Finally, coach education and awareness of the increased prevalence of LEA, DE/ED, and EXD to the young female triathlete and the associated long-term negative health consequences during this period of growth and development will be imperative for early detection and intervention.

5.6 Statement of original contribution

- This is the first study to screen a cohort of female triathletes (n = 393) to estimate the prevalence of LEA, DE/ED and EXD in different age groups.
- This is the first study to determine if differences exist across age groups in LEA, DE/ED and EXD scores in a large cohort of female triathletes.
- This is the first study to examine associations between age and LEA, DE/ED and EXD in a large cohort of female triathletes.

CHAPTER 6

INFLUENCE OF PERFORMANCE LEVEL ON THE PREVALENCE OF LEA RISK IN FEMALE TRIATHLETES

6.1 Introduction

Whilst the physical and mental benefits of exercise are abundant (Chapter 4.1; Ruegsegger & Booth, 2018), certain athletic populations are considered to be at increased risk of failing to meet EI thresholds to support normal basal physiological function and training (Loucks, et al., 2011). As outlined in studies 1-2, this failure can lead to the development of LEA and lead to the negative health and performance consequences associated with the Triad and RED-S models (Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). Identification and early detection of at-risk individuals has been recognised as critical to prevent individuals reaching the clinical end points of these models, which may be irreversible (De Souza, et al., 2014; Mountjoy, et al., 2018). Findings from study 2 highlighted a greater prevalence of younger athletes at risk of LEA than older, however, there was little difference in training load across age groups. It may be expected that training load may be more related to performance level.

Endurance sports like triathlon require well-developed physical capacities (endurance, power, speed, and flexibility), technical skill and in some regards place emphasis on leanness with athletes exhibiting low fat mass and/or low body mass (Knechtle, et al., 2010; Whyte, 2014). Due to the important role of building endurance capacity across three disciplines, triathlon necessitates large volumes of frequent and intense training which may result in athletes failing to meet the required energy requirements (Vescovi & VanHeest, 2016). An additional desire for leanness, related to either performance or body image issues, may further elevate the risk of LEA in triathletes (Thorpe & Clark, 2020). LEA in this population may be underpinned by DE/ED that may also be related to EXD and/or excessive EEE related to training demands (Torstveit, et al., 2019; Ackerman, et al., 2020). Previous findings from study 1, reported 42% of competitive female triathletes were classified as at risk of LEA (LEAF-Q), 25% and 9% were classified with DE and ED (FAST), and 9% were classified as at risk of

EXD (EDS-R). Further findings from study 2 identified the prevalence of those at risk of LEA, DE/ED and EXD was higher in younger competitive female triathletes compared with their older counterparts.

It has been acknowledged that there is a high prevalence of LEA in Western elite athletic populations from leanness sports (see table 2.2; De Souza, et al., 2014; Mountjoy, et al., 2018; Logue, et al., 2018; 2020). Although not fully established, it is believed non-elite athletes from these sports are a unique subpopulation that may also be at increased risk of LEA, DE/ED and EXD. Slater, et al., (2016) reported 45% of female recreational exercises (defined as non-elite) as at risk of LEA and Torstveit, et al., (2005) reported a higher prevalence of LEA in non-elite female athletes (69%) compared with elite (60%). Irrespective of triathlon distance, competition is held between elite (0.1% of the 2.3 million registered triathletes worldwide - O'Mara, 2019) and non-elite athletes. As highlighted earlier in this thesis, non-elite athletes are referred to as 'age-groupers' in the triathlon field and are considered as competitive recreational athletes as defined by the ITU (Anthony, Rüst, Cribari & Rosemann, et al., 2014; Vleck, et al., 2014). Age-groupers compete against other age-groupers within the same 5-year age categories to be eligible for qualification into the 'Great Britain Age-Group Team' or equivalent for international athletes. Qualification into the National Age-Group team allows age-groupers to compete for European and World Championship medals and titles against fellow age-groupers – classifying these athletes as top-percentile age-groupers (British Triathlon, 2020).

Despite non-elite athletes being a subpopulation that may be at increased risk of developing LEA, the specific risk factors that may differ between non-elite and elite athletes is not fully understood. As performance level improves it is likely that training load increases which increases EEE, thereby increasing the risk of developing LEA if EI is not adequately matched (Nattiv, et al., 2007; Mountjoy, et al., 2014; Wasserfurth, Palmowski, Hahn & Krüger, 2020). Non-elite athletes may be at increased risk due to limited access to nutritional and

training advice and support, and sports medicine personnel (Slater, et al., 2016; Black, et al., 2018; Logue, et al., 2019; Wasserfurth, et al., 2020). This may also apply to non-elite athletic coaches who do not fully understand the importance of nutrition (Wasserfurth, et al., 2020). Non-elite athletes may be more susceptible to current social media diet trends (i.e., high-fat, low carbohydrate diets, clean eating, and veganism) in an effort to enhance performance and/or lose body mass and body fat (Black, et al., 2018). Such trends may lead to the development of DE behaviour (Wasserfurth, et al., 2020). Non-elite athletes are also susceptible to feeling pressure from peers, parents, coaches, and social media to train, eat and look a certain way that may increase the risk of developing DE and thereby, EXD and/or LEA (Slater, et al., 2016; Black, et al., 2018; Logue, et al., 2019; Wasserfurth, et al., 2020).

To date limited information exists on the prevalence of LEA and related factors of DE/ED and EXD among those non-elite female triathletes known as age-groupers. More specifically, the potential differences in risk status between developmental performance levels of those athletes identifying as competitive recreational age-groupers and those identifying as top-percentile age-groupers is largely unknown. It is timely to undertake cross-sectional studies in this population to aid in identifying specific populations and direct resources for the early detection and intervention of LEA, DE/ED and EXD. Therefore, the present study aimed to, 1) investigate the prevalence of competitive female triathletes at risk of LEA, DE/ED, and EXD in different performance levels for athletes classified as age-groupers. 2) Determine if there were differences in LEA, DE/ED, and EXD scores between performance levels, and 3) investigate possible associations between performance level and LEA, DE/ED, and EXD.

6.2 Materials and methods

6.2.1 Research design

This cross-sectional, descriptive study required participants to complete an anonymised online questionnaire – the 'Female Health Questionnaire'. The study was reviewed and granted ethical approval (Appendix 1) from the University of Sunderland Research Ethics Group and conducted in accordance with the Declaration of Helsinki (2013). All participants took part in the study voluntarily, were provided with information specifying the study details including inclusion and exclusion criteria (table 3.1), provided implied consent for the data to be used in the study and no participation incentives were offered (outlined in Chapter 3.2 – 3.4, Appendices 1-3).

6.2.2 Participants

Recruitment posters for healthy, pre-menopausal, female triathletes, aged 18 or over were arbitrarily advertised via social media platforms (Facebook, Instagram, and Twitter), supported by flyers and word-of-mouth approaches when relevant. Participants were asked to complete the anonymous online 'Female Health Questionnaire' after reading the information sheet (including inclusion and exclusion criteria) and providing implied consent (Appendix 2-3). Table 3.1 (Chapter 3) outlines the inclusion ad exclusion criteria for study 3. Table 6.1 outlines participant recruitment, exclusion, and inclusion into the final study sample for study 3. N=383 individuals were included in the final analysis for study 3 and although a large reduction in the original sample size is evident, a sample size of N=383 met the calculated sample size estimation for the cross-sectional study. Based on a population size of 10,000 registered female triathletes with British Triathlon (British Triathlon, 2021a), a sample size estimation of n=370 was calculated for study 1, with a confidence level of 95% and a 5% margin of error (Qualtrics, London, UK). Study 3 sample size also falls within the sample size range previously reported in LEA prevalence studies (range 10 to 833; Schaal, et al., 2011a; Logue et al., 2019 – table 2.2).

Table 6.1 Sample size for study 3

N recruited	Excluded	Included
N = 878	N=36 post-menopausal or non-triathlete N=303 LEAF-Q exclusion (pregnant, breastfeeding, chronic illness, use of forms of contraceptive other than oral; increase false positive; Melin, et al., 2014) N= 146 Female Health Questionnaire incomplete N=10 self-identified as elite level triathletes and study focused on non-elite triathletes	N = 383

The current study focused on non-elite triathletes who are referred to as 'age-groupers' in the triathlon field/community (Anthony, et al., 2014; British Triathlon, 2020). 'Age-groupers' are considered as competitive recreational athletes as defined by the ITU (Anthony, et al., 2014). The ITU has further sub-divided 'age-groupers' into two groups: recreational age-groupers and top-percentile age-groupers as outlined in table 6.2.

Table 6.2. Definitions of non-elite triathlete classification

Classification	Definition (Anthony, et al., 2014; British Triathlon, 2020).
Recreational Age-Group Triathlete	Considered as a competitive triathlete but is not eligible for qualification into European and World Championship events, based on their overall finish time in the age-group category (i.e., 18-24 years) compared to their competitors at qualifying events. Still classed as a non-elite triathlete.
Top-Percentile Age-Group Triathlete	Considered as a competitive triathlete but is eligible for qualification into European and World Championship events, based on their overall finish time in the age-group category (i.e., 18-24 years) compared to their competitors at qualifying events. Still classed as a non-elite triathlete but may be considered as developmental athletes.

6.2.3 Data collection and questionnaire data

All the data collection procedures and questionnaire data used in the study are outlined in Chapter 4.2.3 and 4.2.4. However, in brief, participants were asked to complete an

anonymous online questionnaire, the 'Female Health Questionnaire, which incorporated the LEAF-Q (Melin, et al., 2014), FAST (McNulty, et al., 2001) and EDS-R (Downs, et al., 2004), which was self-administered and accessible for a four-week period between October 2019 and November 2019 to female triathletes.

6.2.4 Statistical analysis

Data normality was assessed using the Shapiro-Wilk test. Descriptive data for performance levels was calculated for demographic data and self-reported training load. Frequency analysis for LEAF-Q, FAST and EDS-R questionnaire scores was performed across performance levels. Non-normally distributed data was compared using a Mann-Whitney U test to compare differences in LEAF-Q, FAST and EDS-R scores between performance levels. Chi-square tests were used to assess associations between performance levels and the LEAF-Q, FAST and EDS-R scores. Subsequently, an MLR was performed to ascertain the effects of performance level on the likelihood that participants would be classified as at risk or not on the prediction model for the EDS-R questionnaire. All statistical analyses were performed using SPSS (V.25; IBM Company, SPSS Inc., Chicago, USA). Statistical significance was set *a priori* at $p \leq 0.050$.

6.3 Results

6.3.1 Participant characteristics

N=383 individuals who met the inclusion criteria (table 3.1 – chapter 3) were included in the final analysis of study 3 with with participants divided in to two self-identified performance level groups: recreational age-groupers (N=293) and top-percentile age-groupers (N=90). Participant characteristics of the two performance levels for Age-Groupers are presented in Table 6.3. Comparisons of performance levels found participants identifying as top-percentile age-groupers were significantly younger (*d* 0.15*)*, had a lower body mass (*d*

0.15) and BMI (*d* 0.26), and spent more hours training per week (*d* 0.24) than those triathletes identifying as recreational age-groupers (table 6.1).

Table 6.3. Performance level participant characteristics, training load and questionnaire data.

	Recreational Age-Grouper (N=293)	Top-percentile Age-Grouper (N=90)
Age (years)	37 (12) [*1]	32 (13) [*1]
Height (m)	1.65 (0.08)	1.67 (0.09)
Mass (kg)	65.0 (12.0) [*2]	62.0 (11.0) [2]
BMI ($kg \cdot m^2$)	23.5 (4.4) [**3]	21.6 (2.8) [**3]
Training time (h·week)	10.7 (5.5) [**4] (3-26)	13.0 (7.0) [**4] (6-30)
LEAF-Q score	6 (6) (0-17)	7 (7) (1-21)
FAST score	73 (21) (42-112)	72 (27) (43-107)
EDS-R score	20 (8) [*5] (7-42)	22 (7) [*5] (9-41)

BMI, body mass index; EDS-R, exercise dependence scale revised; FAST, female athlete screening tool; LEAF-Q, low energy availability in female's questionnaire.
Data presented as median (interquartile range) or range in parentheses; n=383.
[*1, 2, 5] Significant difference between groups, p <0.050.
[**3, 4] Significant differences between groups, p <0.001.

6.3.2 LEAF-Q scores and key components

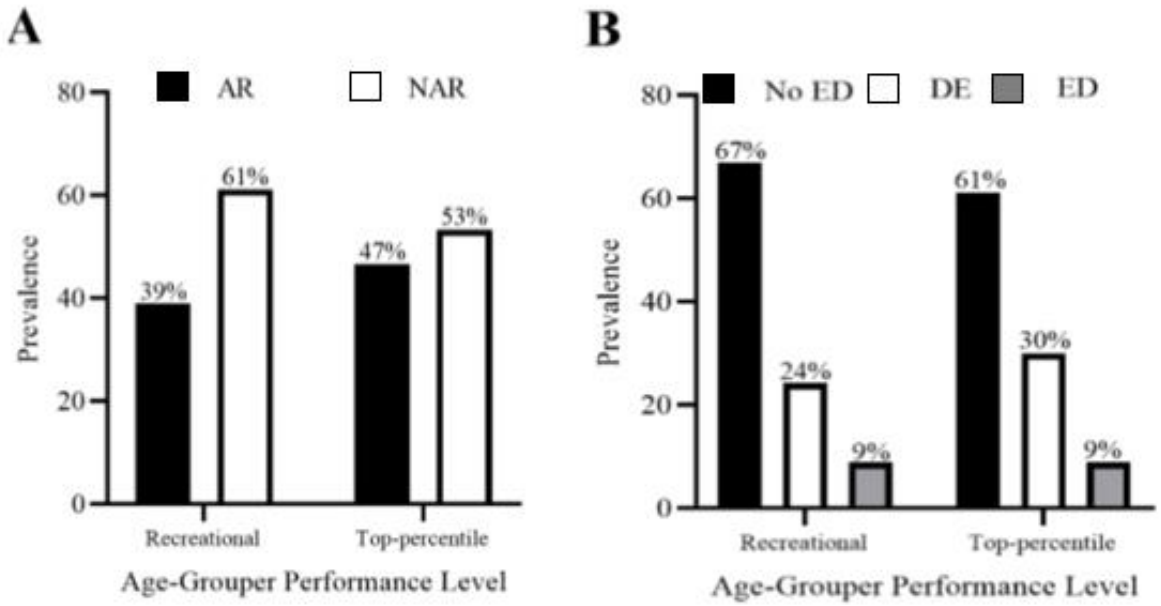
39% of participants who identified as a recreational age-grouper were classified as at-risk of LEA (LEAF-Q) compared with 47% of those who identified as a top-percentile age-grouper (figure 6.1). When assessing the individual LEAF-Q component scores for performance levels, 62% of those identifying as a recreational age-grouper met the component cut-off score ≥2 for increased incidence of injury, compared with 67% of those who identified as a top-percentile age-grouper (table 6.4). 77% of those identifying as a recreational age-grouper met the component cut-off score ≥2 for gastrointestinal disturbances compared with 82% of top-percentile age-groupers (table 6.4). Finally, 25% of those identifying as a

recreational age-grouper met the component cut-off score ≥4 for menstrual dysfunction compared with 32% of top-percentile age-groupers (table 6.4). For every extra hour of exercise participants performed per week, the risk of LEA was 1.05 times more likely to occur (OR = 1.05, 95% CI = 1.00– 1.11, P = 0.035). Body mass (p = 0.56) and BMI (p = 0.43) did not influence the risk of LEA.

Table 6.4. Performance level prevalence of risk for LEAF-Q components.

LEAF-Q component		Recreational age-grouper (N=293)	Top-percentile age-grouper (N=90)
Injury	At risk*	62	67
	Not at risk	38	33
Gastrointestinal	At risk**	77	82
	Not at risk	23	18
Menstrual	At risk***	25	32
	Not at risk	75	68

Injury: component cut-off scores for at risk ≥2.
**Gastrointestinal: cut-off scores for at risk ≥2.
***Menstrual: cut-off scores for at risk ≥4.



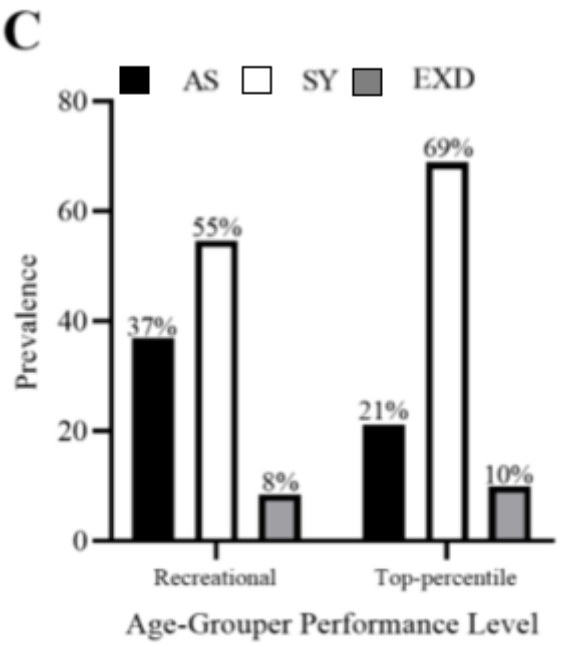


Figure 6.1. Prevalence of risk for LEA (A), DE/ED (B) and EXD (C) by performance level.

AR, at risk; NAR, not at risk; No ED, no eating disorder; DE, disordered eating; ED, eating disorder; AS, asymptomatic; SY, symptomatic; EXD, exercise dependent. Data presented as percentage.

6.3.3 FAST scores and key components

24% of participants who identified as a recreational age-grouper were classified with DE and 9% with ED according to FAST scores (figure 6.1). In comparison, 30% of participants who identified as a top-percentile age-grouper were classified with DE and similarly 9% with ED (figure 6.1). The percentage of participants from each respective performance level who scored ≥3 points for FAST items (i.e., agree to strongly agree) are presented in appendix 10.1. Both recreational (79%) and top-percentile age-groupers (78%) considered sport participation an important facet for their self-esteem, with the majority of participants (93% and 94%, respectively) believing they have a lot of good qualities and ~66% of both groups strive for

perfection in all aspects of their life. More recreational age-groupers believed their triathlon performance was related to their weight with 78% expecting performance improvements with weight reduction and 77% worried that weight gain would impair performance. In comparison, more top-percentile age-groupers worried weight gain would impair performance (62%) than expecting performance improvements with weight reduction (52%). However, most participants from both groups (78% and 87%, respectively) acknowledged that as an athlete they were very conscious about consuming adequate calories and nutrients on a daily basis (appendix 10.1). Diet control during training was reported across both groups with more recreational age-groupers (53%) controlling fat and calorie intake and (44%) limiting carbohydrate intake than top-percentile age-groupers (46% and 32%). Both groups recognised that they would worry about weight gain if they could not exercise, with more recreational age-groupers (80%) worried than top-percentile age-groupers (60%). However, diet control did not report high prevalence across both groups of behaviours typically associated with DE/ED, avoidance of food with >3 gram of fat (12% to 8%), skipping meals due to alcohol consumption (12% to 7%) or taking dietary or herbal supplements to increase metabolism or assist in fat burning (11% to 8%). Although prevalence was slightly greater among recreational age-groupers (appendix 10.1).

In regard to body dissatisfaction, more recreational age-groupers were not happy with their current weight (65%) and were concerned about their body fat percentage (65%), compared with 41% and 44% of top-percentile age-groupers. Almost 4 in 10 participants from both groups have used methods to keep their weight down that they believe are unhealthy. Finally, more top-percentile age-groupers (52%) believed most female athletes have DE habits compared with recreational age-groupers (44%; appendix 10.1).

6.3.4 EDS-R scores

55% of participants who identified as a recreational age-grouper were classified as symptomatic and 8% as at-risk of EXD according to EDS-R scores (figure 6.1). In comparison, 69% of participants who identified as a top-percentile age-grouper were classified as symptomatic and 10% as at-risk of EXD (figure 6.1). Of those considered at-risk of EXD, 'lack of control' (i.e., persistent desire or unsuccessful effort to reduce exercise) was the most frequently cited EDS-R component for participants who identified as a recreational age-grouper (appendix 10.2). However, 'time' (i.e., a great deal of time is spent in activities necessary to obtain exercise such as., exercise holidays) was the most frequently cited EDS-R component for participants who identified as a top-percentile age-grouper (appendix 10.2).

6.3.5 Between group comparisons

A Mann-Whitney U test was run to determine if there were differences in LEAF-Q scores between recreational age-groupers and top-percentile age-groupers. Distributions of the LEAF-Q scores for recreational age-groupers and top-percentile age-groupers were similar, as assessed by visual inspection. Median (interquartile range) LEAF-Q scores for recreational age-groupers (6 (6)) and top-percentile age-groupers (7 (7)) was not statistically significantly different, U = 14 775, z = 1.736, p = 0.083 (table 6.1). Similarly, distributions of the FAST scores for recreational age-groupers and top-percentile age-groupers were similar. Median (interquartile range) FAST scores for recreational age-groupers (73 (21)) and top-percentile age-groupers (72 (27)) was not statistically significantly different, U = 13 174, z = -0.012, p = 0.990 (table 6.1).

However, a Mann-Whitney U test was run to determine if there were differences in EDS-R scores between recreational age-groupers and top-percentile age-groupers. Distributions of the EDS-R scores between groups were similar, as assessed by visual

inspection. Median (interquartile range) EDS-R scores for recreational age-groupers (20 (8)) and top-percentile age-groupers (22 (7)) was statistically significant, U = 15 267, z = 2.270, p = 0.023 (table 6.3). However, this was a small effect (d 0.12).

6.3.6 Cross-tabulation

A chi-square test for association was conducted between performance level and LEAF-Q categories. All expected cell frequencies were greater than five. The test of independence showed that there was no significant association between performance level and LEAF-Q category, χ^2 (1) = 1.717, p = 0.220 (appendix 10.3). Similarly, a chi-square test for association showed that there was no significant association between performance level and FAST category, χ^2 (2) = 1.247, p = 0.538 (appendix 10.4). However, there was a statistically significant association between performance level and EDS-R category, χ^2 (2) = 7.740, p = 0.021 (table 6.5).

6.3.7 Logistic Regression

An MLR was performed to predict the likelihood of EXD classification (ED-R) based on performance level. The model was statistically significant, χ^2 (2) = 8.186, p < .017. Using the conventional p ≤ .050 threshold, performance level was a statistically significant predictor for recreational age-groupers (table 6.6). Participants who were recreational age-gropers were 0.454 times less likely to be classified as symptomatic than top-percentile age-groupers and 0.489 times less likely to be classified as at-risk of EXD.

Table 6.5. Cross-tabulation of performance level and EDS-R score categories.

Performance level		Asymptomatic	Symptomatic	At risk of exercise dependence	Total
Recreational Age-grouper	Count	108	160	25	293
	Expected Count	97.2	169.8	26.0	
	Column %	85.0	72.1	73.5	77% of 383
Top-percentile Age-grouper	Count	19	62	9	90
	Expected Count	29.8	52.2	8.0	
	Column %	15.0	27.9	26.5	24% of 383

Pearson chi-square = 7.740; degrees of freed om = 2; p = .021.
EDS-R, exercise dependence scale-revised

Table 6.6. Multinomial Logistic Regression predicting likelihood of EDS-R based on performance level.

							95% CI for Odds Ratio	
		B	SE	Wald	Df	P	Odds Ratio	Lower	Upper
Symptomatic	Recreational age-grouper	-.790	.290	7.399	1	.007	.454	.257	.802
At risk EXD	Recreational age-grouper	-.716	.462	2.407	1	.121	.489	.198	1.207

Chi-square = 8.186; degrees of freed om = 2; p = .017.
EDS-R Reference category = Asymptomatic.
Top-percentile age-grouper = set to zero.
EXD, exercise dependence.

6.4 Discussion

The present study aimed to investigate the prevalence of LEA, DE/ED, and EXD in athletes performing at different competitive levels. Accordingly, the main findings were that: 1) the prevalence of those classified with LEA, DE/ED, and EXD was greater in those participants who identified as top-percentile age-groupers compared to recreational age-groupers. 2) There were significant differences in EXD scores between recreational and top-percentile age-groupers but not for LEA or DE/ED scores. 3) Finally, the only significant association was between performance level and EXD, with recreational age-groupers less likely to be classified with a symptomatic profile for maladaptive patterns of exercise than top-percentile age-groupers.

Taken together, the findings of the study are the first, to our knowledge, to screen a large cohort of non-elite female triathletes (age-groupers) to examine the influence of performance level on LEA, DE/ED, and EXD risk. The findings of this study suggest that LEA, DE/ED, and EXD risk exist in both recreational and top-percentile age-groupers, however, differences do exist between performance levels with prevalence rates beginning to rise as performance classification improves.

6.4.1 Influence of performance level on LEA

Similar to age-related findings in study 2, the risk of developing LEA existed across both performance levels in non-elite female triathletes (age-groupers) which is consistent with the Triad and RED-S models (Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018; Ackerman et al, 2020). No significant differences in LEAF-Q scores were evident between recreational age-groupers and top-percentile age-groupers, which may reflect the smaller sample size of the latter performance group. The prevalence of those considered at-risk of LEA by the LEAF-Q was marginally higher in top-percentile age-groupers compared with

recreational age-groupers (8% difference). This may reflect that even in the non-elite athletic population, as performance classification improves, the risk of developing LEA may also increase. The prevalence of LEA found in the present study was similar to previous findings in females participating in endurance sports (range: 18% to 80% - see table 2.2: Muia, et al., 2016; Jesus, et al., 2021). There is limited evidence of differing LEA values depending on level of competition in female endurance athletes (Slater, et al., 2016; Black, et al., 2018; Logue, et al., 2019).

In the present study, 39% of participants identifying as recreational age-groupers were considered at risk of LEA by the LEAF-Q, compared with 47% of top-percentile age-groupers. Such findings are in line with data reported by Logue, et al., (2019) who observed a greater prevalence of LEA risk (LEAF-Q) in Irish provincial/inter-county (~47%, n=155) and international (~45%, n=162) athlete groups, than recreationally active individuals (33%, n=235). Although a large sample size was recruited (n=833), participants formed a heterogenous sample from various athletic cohorts (individual and team sports) and therefore no direct comparison can be drawn. Further findings from Meng, et al., (2020) have also reported a significantly higher prevalence of LEA in female Chinese elite athletes (56%, n=52), compared with Chinese recreational athletes (35%, n=114), using the LEAF-Q.

In contrast to the current study, Meng, et al., (2020) surveyed athletes who participated in aesthetic sports which included dance, cheerleading, aerobics, dance sport and rhythmic gymnastics, rather than endurance sports. Comparable to the current study, recruiting developmental to elite level athletes was difficult in these studies and was reflected in the relatively small sample sizes. In addition, both Logue, et al., (2019) and Meng, et al., (2020) used the LEAF-Q to identify those at risk of LEA which has currently only been validated in endurance-trained athletes (Melin, et al., 2014). Due to the variability in the results from the

tools available to measure the components of EA, some like De Souza, et al., (2014) question whether LEA can ever be measured accurately quantitatively.

Two studies reported a prevalence rate similar to the current study when using the LEAF-Q, however, the influence of performance levels was not directly examined. Folscher, et al., (2015) reported 44% of elite and recreational South-African ultra-marathon runners (n=134) were considered at risk, and Slater, et al., (2016) reported 45% of recreationally active individuals in New Zealand (n=109) at risk. In contrast, using different assessment tools to directly measure EA (i.e., LBM, EI, and EEE measurements), Melin, et al., (2015) reported 20% of elite female endurance athletes (n=40) were in a state of LEA and Heikura, et al., 2018a) reported a prevalence of 31% in elite female middle- and long-distance runners and race walkers (n=35).

The current study extends previous work examining the prevalence of LEA that has often focused on elite athletic populations, single-sport endurance events, and aesthetic sports. The current study utilised a homogenous sample where although narrower in focus in regard to the target population, the key advantage is clearer generalisability within a previously under-researched group (Jager, Putnick & Bornstein, 2017). The current study demonstrates that LEA occurs frequently in multi-sport endurance athletes competing as age-groupers, irrespective of the level of competition. Further supporting the requirement for validation of simple and cost-effective screening tools in non-elite groups and to target prevention strategies. The reasons for the marginally higher LEA risk observed between recreational and top-percentile age-groupers or the overall high prevalence in non-elite female triathletes are unclear from the results of the current study.

Nevertheless, non-elite athletic groups may have less access to educational resources regarding appropriate nutritional, training, and health practices. They may also be reliant in

advice from their coach, parent, or General Practitioner rather than specialised sports medicine personnel. The assessment of general and sports nutrition knowledge or knowledge of Triad and RED-S has been difficult to establish due to variability in the use of poorly validated assessment tools. A review by Trakman, et al., (2016) found no differences in coach and athlete nutritional knowledge between various sports or gender and inconsistent findings were found between athletes and non-athletes. However, it was deemed plausible that elite athletes or collegiate athletes receiving funding have greater access to resources and is therefore likely they have greater knowledge than non-elite and non-funded athletes (Trakman, Forsyth, Devlin & Belski., 2016). Folscher, et al., (2015) assessed knowledge of the Triad and the associated health implications in elite and recreational ultra-marathon runners in the 2014 Comrades Marathon (n=134). It was found only 8% of participants head heard of the Triad and of those participants 94% could not name any of its components. Of the few that could (n=7) osteoporosis was the most commonly named health consequence of Triad.

It is widely accepted that the physiological dysfunction outlined in the Triad and RED-S models is a result of LEA and not high training load (Loucks, et al., 1998; Loucks, 2011). The current study found significant differences in training load between groups with top-percentile age-groupers spending more hours per week training than recreational age-groupers. The higher training load will increase EEE and if these athletes do not adequately match EI this could explain the increased risk of developing inadvertent LEA and the higher prevalence's of injury, gastrointestinal disturbances and menstrual dysfunction reported. Participants in the current study were 1.05 times more likely to be considered at risk of LEA for each additional hour of exercise per week. It is also possible that higher training loads may also suppress appetite, increasing the difficulty of matching EI to EEE in endurance athletes (Nattiv, et al., 2007; Mountjoy, et al., 2014; Wasserfurth, et al., 2020). Body mass and BMI did not influence the risk of LEA; however, these components were significantly lower in top-percentile age-

groupers. This may reflect an increased risk of developing DE behaviour that has been associated with high training loads in leanness sports athletes (Logue, et al., 2019; Wasserfurth, et al., 2020). Further research is required to examine the prevalence of LEA across the performance pathway in various athletic cohorts and focus on the role of risk factors such as training and nutritional practices.

6.4.2 Influence of performance level on exercise behaviour

Pollock, et al., (2010) observed significant associations between high training volume with a negative EB and the most clinically significant symptoms of LEA (low BMD and FHA). This has been particularly evident in endurance athletes likely due to the excessive EEE (Pollock, Grogan, Perry & Pedlar, et al., 2010). Although difficult to distinguish, excessive EEE in this particular athletic cohort may be explained as an inadvertent outcome of the high training volume required to achieve performance goals or may be attributed to EXD (Müller, et al., 2015; Marques, et al., 2019; Torstveit, et al., 2019). Similar to findings in the current study previously discussed, Logue, et al., (2019) observed athletic and recreationally active Irish females were 1.06 times more likely to be at risk of LEA for each additional hour of exercise per week. Likewise, Meng, et al., (2020) reported higher training frequency and volumes in elite athletes compared with recreational which may in part explain the higher prevalence of LEA observed. In contrast to the current study, neither study examined the prevalence of EXD.

Current literature has supported the observation that EXD symptomatology may increase as performance level advances (Pierce, McGowan & Lynn, 1993; Szabo, et al., 2013; De La Vega, Parastatidou, Ruíz-Barquín & Szabo, 2016). This may be associated with DE behaviour and higher training loads, thereby increasing the risk of LEA (Szabo, et al., 2010; Torstveit, et al., 2019). In the current study, significant differences in total EDS-R scores were

evident between recreational and top-percentile age-groupers, although only a small difference (2%) was evident in prevalence rates of those considered at risk of EXD. To date, limited literature exists examining the prevalence of EXD observed across athletic populations, performance levels and/or in female athletes. The prevalence rate observed in the current study (8% to 10%) is double that of studies reviewed by Di Lodovico, et al., (2019) who reported a prevalence rate of 4% in studies examining male and female endurance athletes using the EDS.

However, current findings are in line with Magee, et al., (2016) who examined male and female Ironman triathletes (n=345) using the EDS-R and reported a prevalence of 8% at risk of EXD. Similarly, Valenzuela, et al., (2017) reported a prevalence of 9% of male amateur triathletes (n=93) at risk of EXD when using the EDS. Direct comparison between studies is difficult due to the variability inherent with EXD literature related to terms, definitions, and assessment tools used (Szabo, et al., 2015). Magee, et al., (2016) found 'tolerance' and 'time' were the most frequently cited EDS-R components in those classified as symptomatic or at risk of EXD. It was proposed these components suggest physiological dependence which may reflect the high training load and commitment required to complete an Ironman triathlon and may not be problematic. This may be evident in the current study as 'time' was the most frequently cited EDS-R component for top-percentile age-groupers which could simply reflect the physical challenges of competing at a higher level. In contrast, the current study found 'lack of control' and 'withdrawal effects' as the most frequently cited among recreational age-groupers This may suggest this group may have more psychological factors contributing to EXD. For instance, recreational age-groupers had greater body dissatisfaction and concerns about body fat than top-percentile age-groupers. This may suggest that in recreational age-groupers EXD may be secondary in nature (Costa, et al., 2013).

Further research is required examining the prevalence of EXD as to date there is no clear evidence that specific sport structures and levels of performance increases the risk for the

development of EXD or EXD symptomatology (Marques, et al., 2019). This is particularly important as research by Torstveit, et al., (2019) has indicated that the association between training load, DE/ED, and EXD may elevate exposure to the negative health and performance consequences associated with LEA. The reasons for the marginally higher prevalence rates of top-percentile age-groupers considered at risk of EXD in the current study are unclear. However, these differences may be attributed to differences in personality traits, motivation and passion for exercise or competition, body dissatisfaction, perfectionism, vocational dysfunction, psychosomatic problems and coping mechanisms for stress, anxiety, or depression (Landolfi, 2013; Lichtenstein & Hinze, 2020). It is also important to acknowledge such differences reported in the literature may be related to differences in interpretations of the measures used in the assessment of EXD, opposed to greater psychological morbidity across performance levels or athletic cohorts (Szabo, et al., 2015). Nevertheless, the current study extends previous work examining the prevalence of EXD that has been limited in multi-sport endurance athletes and female athletes. It further acknowledges the importance of monitoring training volume and motivations for athletic participation as a method of managing EI with EEE.

6.4.3 Influence of performance level on eating attitudes

Besides the co-dependencies related to EXD that may increase the risk of LEA, there is evidence that eating opportunities may also be reduced due to high training hours observed both in endurance athletes and as performance level improves (Vescovi & VanHeest, 2016; Burke, Castell, Casa & Close, et al., 2019). Although unclear in the current study, the importance of identifying the aetiology of EXD as primary or secondary in nature has been acknowledged in the literature (De Coverley Veale, 1987; Marques, et al, 2019). As research has suggested the development of eating psychopathologies and depression may be greater in

those who exercise excessively (Peñas-Lledó, Leal & Waller, 2002; Landolfi, 2013; Marques, et al., 2019).

Irrespective of the pathway leading to the development of eating psychopathologies, both DE /ED have been acknowledged as a risk factor in the development of LEA in athletes (Ackerman, et al., 2020). Despite no significant differences or associations reported in the current study, which may be in part explained by sample size, it was found 9% of both recreational and top-percentile age-groupers were at risk of ED. Additionally, more top-percentile age-groupers were at risk of DE (30%) compared with recreational (24%), although again, not to a significant level. Existing research examining the prevalence of DE/ED in the athletic population is difficult to surmise and largely inconclusive. Similar to EXD, the literature typically uses a myriad of definitions, inconsistent assessment methods, small sample sizes and often undertaken with elite female athletes, limiting the generalisability of findings (Joy, et al., 2016).

Nevertheless, the current findings agree with previous literature where ED prevalence rates appear to be higher among female athletes compared with general population rates of 2-4% (Sundgot-Borgen, et al., 2004; Smink & Hoek, 2013; Dahlgren, et al., 2017). Smink, et al., (2013) suggested the prevalence of DE was greater than that of ED which also agrees with current findings. To date the relationship between performance level and DE/ED risk has not been systematically investigated, however, Sundgot-Borgen, et al., (2004) reported 42% of Norwegian elite female athletes from aesthetic sports and 24% from endurance sports presented with DE symptoms using the gold standard method of assessment (EDE). Similar to the current study, Folscher, et al., (2015) used the validated FAST and reported 27% of elite and recreational South-African ultra-marathon runners were considered at risk of DE and 5% of ED. In contrast, Mongrain, et al., (2018) used a different validated tool (EAT-26) in non-elite

multi-sport endurance athletes (114 males and 48 females) and found that only 13% of female athletes were at risk of DE.

Collectively, studies examining athletic populations have suggested prevalence rates of DE pathology (including ED) up to 52% (Blaydon, et al., 2002; Joy, et al., 2016) across a variety of leanness sports and performance levels, which agrees with the current study. However, both the prevalence and influence of performance level on the development of eating psychopathologies remains unclear, and true prevalence may be higher when using gold standard methods or future assessment methods developed specifically for athletes. Although no significant differences were found, this may be explained by the relatively small sample size for top-percentile age-groupers and the use of a single self-report questionnaire that is not a formal diagnostic tool, such as the EDE clinical interview (Joy, et al., 2016).

The reasons for the relatively high prevalence's of DE/ED found in both groups may be explained by general risk factors (biology, genetics, age, and pubertal status), psychological (body dissatisfaction, low self-esteem, personality traits (i.e. perfectionism or negative affects), socio-cultural (peer pressure, influence of media, bullying or family history of DE/ED), sport-specific (training load, personality, diet pressure, traumatic events including injuries, rules and regulations of sports or coaching behaviour), and gender-specific risk factors (drive for thinness or muscularity and homosexuality; Bratland-Sanda & Sundgot-Borgen, 2013). Therefore, to mitigate the development of DE behaviour that may lead to the onset of LEA, athletes, coaches, and parents should inform themselves about psychological and physiological changes that may have a negative impact on health and performance (Mountjoy, et al., 2018).

6.4.4 Limitations

The current study should be interpreted with caution. Although this is one of the few studies to examine the influence of performance level on LEA, DE/ED and EXD risk. Results

are based on self-identified performance level, rather than quantifiable performance level. In addition, results are based on cross-sectional comparisons of participants in different performance levels, rather than observations of change as participants progress from recreational age-groupers to top-percentile age-groupers to elite. The sequence of events that lead to the findings reported in the current study cannot be determined and cannot imply causality. The use of longitudinal studies in recreational and developmental athletes would provide better interpretation in future work, allowing attributions related to cause or direction of effects. The n = 10 elite female triathletes excluded highlights the difficulty and challenge of recruiting developmental to elite level athletes, particularly those competing in major championship events. Similar studies conducted in this area would be advised to include larger sample sizes of top-percentile age-groupers and elite level female triathletes. Nevertheless, combined with findings from previous research, the results are valuable in highlighting at risk groups across developmental performance levels for the non-elite athlete and directing future screening, early detection, and target awareness education.

6.5 Conclusion

The current study suggests that self-identified top-percentile age-groupers may have a tendency for higher risk of developing LEA and DE/ED. The risk of EXD appears to be similar, however, the symptomology may differ between performance levels. EXD was associated with performance level with a higher training duration evident in top-percentile age-groupers which may provide some explanation for the elevated risk of LEA. It is important to note that these concepts existed at all levels above general population norms, placing non-elite female triathletes at risk for long term health consequences. It is imperative that additional educational resources on appropriate nutrition, training, and health practices, as well as, signs and symptoms of LEA, DE/ED, and EXD are targeted to non-elite athletes, coaches, and parents. The current study extends previous work examining multi-sport endurance athletes,

emphasising the importance of targeting research to enable implementation of early screening and prevention strategies in the pursuit of athlete health.

6.6 Statement of original contribution

- This is the first study to screen female triathletes (n = 383) to estimate the prevalence of LEA, DE/ED and EXD in different performance levels for athletes classified as age-groupers (non-elite triathletes).
- This is the first study to determine if differences exist between performance levels in LEA, DE/ED and EXD scores in female triathletes.
- This is the first study to examine associations between performance level and LEA, DE/ED and EXD in female triathletes.

CHAPTER 7

CHANGES IN ENERGY AVAILABILITY ACROSS THE SEASON IN FEMALE TRIATHLETES

7.1 Introduction

Although daily fluctuations in nutrient availability occur, dietary energy is fundamental for the optimal functioning of the physiological processes essential for life. Inadequate overall dietary EI may cause sub-optimal functioning of these processes and the prioritisation of some systems over others. This is widely recognised in bioenergetics as EA (Loucks, et al., 2011). Traditionally, nutritional research and practice has focused on the concept of energy balance (EB = EI – TEE; detailed in chapter 2.3.1; Westerterp & Saris, 1991; Westerterp, et al., 1992; Loucks, 2004; Westerterp, 2013). To regulate EB, individuals are required to match EI with the amount of energy expended during the day from physiological processes (i.e., RMR, TEF, NEAT, and EEE). EB is therefore viewed as an *output* from those physiological systems (Loucks, et al., 2011; Areta, et al., 2021).

In contrast, EA is investigated with regards to the effect of a specific metabolic demand on physiological processes such as, exercise training. As demonstrated in studies 1-3, triathlon is characterised by 'large volumes of frequent and intense training' (Vescovi & VanHeest, 2016) which increases the amount of energy expended in locomotion. Longitudinal studies examining training and competition volume, intensity and duration for male and female non-elite triathletes are limited (Vleck, Millet, & Alves, 2014). Shaw, et al., (2004) reported non-elite triathletes (age-groupers; 26% female) on average spent between 8 and 15 hours per week training with a total weekly training distance between 108 and 239 km. As such, endurance athletes are characterised by high fluctuations of TEE due to the variability of EEE both on a micro (i.e., several days) and macro level (i.e., across the season; Heydenreich et al, 2017). EA is defined as EI minus EEE, relative to each kilogram of LBM (Loucks & Verdun, 1998). Unlike EB, EA is viewed as an *input* to those physiological systems as EA is understood as the amount of residual energy for those processes after removing the energy cost of exercise training (Loucks, et al., 2011; Areta, et al., 2021). EB may not be the most appropriate measure

within the athletic population. It is considered unreliable to solely use measures of total or resting energy expenditure to determine the energy available for optimal physiological functioning, as they will underestimate an athlete's energy requirements (Loucks, 2004; Loucks, et al., 2011; Areta, et al., 2021).

As demonstrated in studies 1-3, endurance athletes, such as triathletes, have been identified as an 'at risk' group for the development of LEA and subsequently Triad/RED-S symptoms (Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). Previous findings from study 1 reported 42% of female triathletes were classified as at risk of LEA (LEAF-Q). Further findings from study 5 identified the prevalence of those at risk of LEA was higher in younger female triathletes and study 3 identified a tendency for a higher prevalence of LEA and greater training duration as performance level improved in non-elite female triathletes. Well-controlled laboratory experiments with sedentary eumenorrheic normal weight females have determined EA thresholds associated with energy conservation or disturbances to physiological systems (Loucks & Thuma, 2003; Loucks, 2014). Proposed classifications of EA thresholds in females are optimal EA ≥45 kcal·kgLBM^{-1}·day^{-1}, subclinical LEA 30-45 kcal·kgLBM^{-1}·day^{-1}, and clinical LEA <30 kcal·kgLBM^{-1}·day^{-1} (De Souza, et al., 2014; Loucks, 2014; Melin, et al., 2015), though such distinctions are rather arbitrary.

To date, studies on free-living athletes have been unable to determine clear EA thresholds, however, the three main aetiological factors for the development of LEA have been identified (Nattiv, et al., 2007; De Souza, et al., 2014). These include, 1) clinical ED, 2) intentional but mismanaged efforts to alter body composition to optimise athletic performance that may include DE behaviour, and 3) inadvertent failure to increase dietary EI to match EEE (Nattiv, et al., 2007). Previous findings from study 1 reported 25% and 9% of female triathletes were classified with DE and ED (FAST), with higher levels reported in younger triathletes in

study 2. These risk factors, taken together with the changing training demands, may be associated with periods of lower EI and consequently LEA across various time points of the season in competitive female triathletes.

Much of the research regarding athletic energy status has focused on elite and sub-elite populations from single-sport endurance events (Logue, et al., 2018; 2020). Similarly, an athlete's energy requirements are dependent on seasonal training and competition demands, and in the case of triathlon, dependent on the event type (e.g., sprint triathlon or Iron-distance triathlon). Limited research exists examining EA across the training season in a single cohort of female athletes. Zabriskie, et al., (2019) examined energy expenditure, EB, and body composition over an academic year in Division II collegiate female lacrosse players. Reed, et al., (2013) examined EA and eating attitudes in NCAA Division I female soccer players during the pre-, mid-, and post-season. Zanders, et al., (2018) monitored energy expenditure, EI, and EA across five phases of the competitive season in NCAA Division II female basketball players from September to April. However, the demands of team-sports on energy status are likely different to those of multi-sport endurance athletes.

Therefore, the current study followed a cohort of competitive female triathletes across a full triathlon season and thus, the aims of the study were three-fold. 1) Examine changes in EA and eating attitudes across the different phases of the season in competitive female triathletes. 2) Investigate the prevalence of those identified with LEA and those considered at risk of DE/ED, and 3) examine how prevalence rates change across the triathlon.

7.2 Materials and methods

7.2.1 Research design

A longitudinal study design was used. Data collection commenced at the beginning of the pre-competitive season (March 2019) and finishing during the transition/build phase of the

season (January 2020). The primary outcomes for study 4 were to observe and assess changes in EA and eating attitudes (FAST). As such, the core components of EA were observed and assessed for seven consecutive days across eight phases which were separated by two months. The core components of EA include body composition (LBM), EI and EEE. Participants recorded all food and drink consumed using a commercially available food and nutrition tracking application (MyFitnessPal, Under Armour, Baltimore, MD, US) and wore a Polar V800 physical activity monitor with H10 heart rate monitor (Polar Electro, Oy, Kempele, Finland) during all training sessions. To screen for LEA risk and observe and assess changes in eating attitudes, participants were provided with a URL link to a self-administered online questionnaire, using the Qualtrics electronic management system (Qualtrics, London, UK). Participants were sent a link via email or SMS on day 1 of the monitoring week and asked to complete the questionnaire by day 7. A reminder was subsequently emailed to all participants on day 4. Study 4 formed part of a larger study, beyond the scope of this thesis, whereby aerobic fitness was assessed every three months of the triathlon season. For descriptive purposes across the phases of the season, the maximal oxygen consumption ($\dot{V}O_{2max}$) for running and cycling was included in study 4. Figure 7.1 illustrates the research design.

The study was granted ethical approval (Appendix 1) from the University of Sunderland Research Ethics Group and conducted in accordance with the Declaration of Helsinki (2013). All participants took part in the study voluntarily, were provided with information specifying the study details, provided written consent for their data to be used in the study, and no incentives were offered (Chapter 3.2-3.3; Appendices 1-3). One of the key ethical issues of study 4 was the repeated measures of dietary analysis and body composition, particularly in individuals with a history of DE/ED or FHA. As a result, a health history questionnaire was administered and individuals with a previous or current diagnosis of DE/ED, menstrual irregularities and/or FHA were not included. All participants completed a familiarisation week.

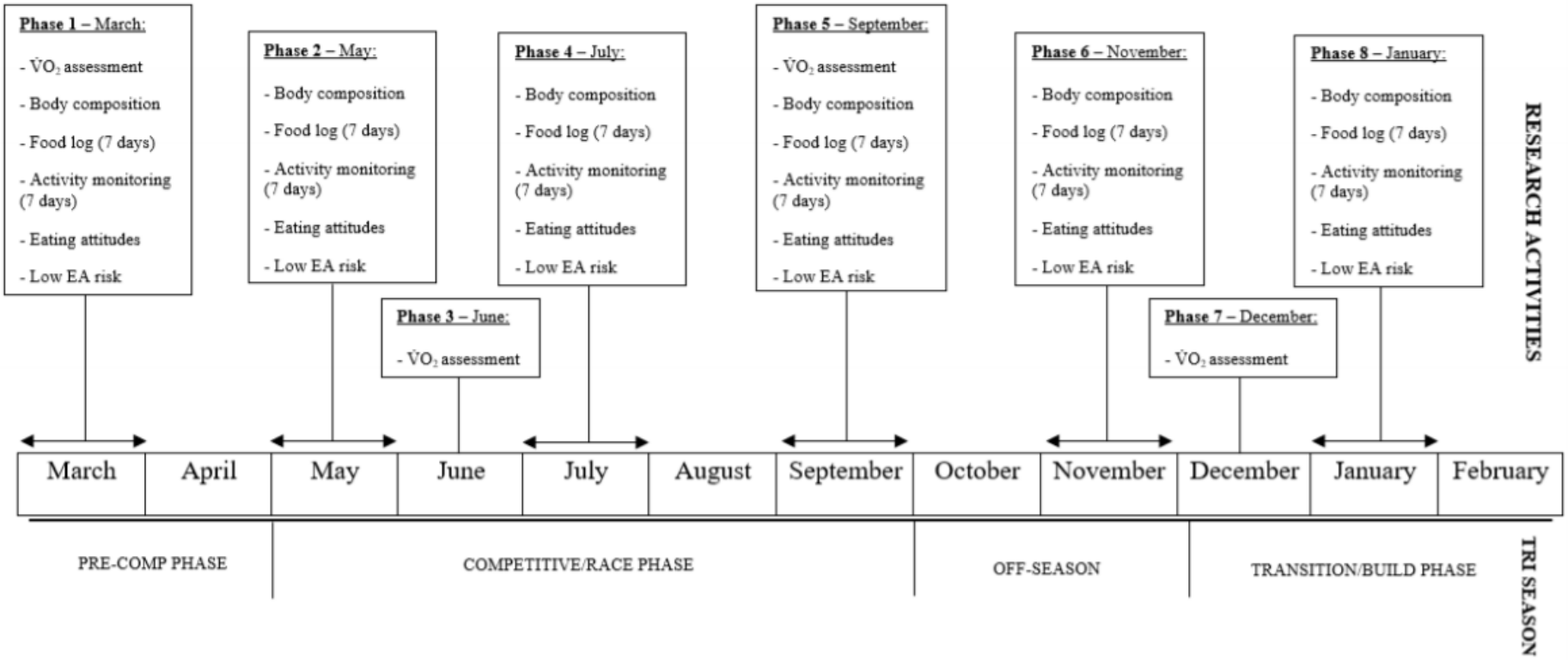


Figure 7.1 Overview of research design.

7.2.2 Participants

A total of ten, female triathelets completed the study (mean ± SD: 27.7 ± 8.6 years; 1.67 ± 0.04 m; 62.2 ± 3.2 kg; 22.6 ± 1.3 BMI; 20.1 ± 4.5% body fat). Participants were recruited from registered British Triathlon clubs (which included University triathlon clubs) in the North-East of England via an emailed recruitment poster and word-of-mouth. During an initial visit, study details and participation requirements were explained, written informed consent and completion of an Institutional Review Board-approved pre-participation health screening medical form (Appendix 4) was obtained. Table 3.1 (Chapter 3) outlines the inclusion ad exclusion criteria for study 3. Table 7.1 outlines participant recruitment, exclusion, and inclusion into the final study sample for study 4. Due to the longitudinal design, logistics, and time constraints on both participant and researcher in study 4 the primary goal was to recruit 10 to 15 individuals which was in line with previous studies who directly measured EA (Doyle-Lucas, et al., 2010; Schaal, et al., 2011; Moss, et al., 2020; Zanders, et al., 2021 – table 2.2). The final sample included ten participants without significant injury or pregnancy who were fully participating in triathlon training during each assessment phase.

Table 7.1 Sample size for study 4

N recruited	Excluded	Included
N = 13	N=1 non-compliance/uncomfortable with repeated measures of EI N=1 personal circumstances (bereavement) N=1 medical reasons	N = 10

7.2.3 Energy availability and eating attitudes (primary outcome measures)

7.2.3.1 Anthropometry

During each phase, with participants unshod, wearing a t-shirt and gym shorts body mass was recorded to the nearest 0.1 kg with a digital scale (703, Seca, Germany) and stature

was measured to the nearest 0.1 cm using a digital stationary stadiometer (264, Seca, Germany). Applying these variables, body mass index (BMI) was calculated as: $\frac{bodymass(kg)}{stature(m)^2}$. A flexible measuring tape (201, Seca, Germany) was used to obtain two measurements of waist and hip circumference to the nearest 1.0 cm consistent with the International Society for the Advancement of Kinanthropometry guidelines described by Norton (2018). Waist circumference was measured at a level midway between the lowest rib and the anterior superior iliac crest. Hip circumference was measured at the level of the greatest posterior protuberance of the buttocks, without compressing the skin. Participants were stood upright with feet together for even weight distribution, arms hanging freely at the sides with measurements conducted at the end of a normal expiration.

Body composition including body fat percentage and LBM were determined by bioelectrical impedance analysis (BIA). A non-invasive, indirect method using a two-compartment model, at a single frequency of 50 KHz (Bodystat 1500, Bodystat, Isle of Man) which has been validated against the DXA method (Batterham, Tapsell & Jenkins, 2002). Although field methods such as BIA are inherently prone with estimation errors, comparisons of BIA devices with two component models in a variety of athletes have produced valid results with "*r* values > 0.67, standard error of estimate values < 4.3% bodyfat, and total errors < 4.6% bodyfat and 2.4 kg of FFM" (Moon, 2013). Participants were in a supine position with arms ⩾30 degrees away from their torso and legs separated. After cleaning with alcohol, self-adhesive electrodes were placed on the right hand and foot. Proximal electrodes were placed on the dorsal surface at the ulnar styloid process at the wrist and on the dorsal surface between the malleoli at the ankle. Distal electrodes were placed on the dorsal surface of the metacarpal phalangeal joint, 1 cm proximal to the knuckle of the third finger on the hand and on the dorsal surface of the metatarsal phalangeal joint, 1 cm proximal to the joint of the second toe on the

foot. All measurements were obtained after participants had rested in the supine position for 5 minutes (Moon, Stout, Smith & Tobkin, et al., 2010).

To ensure the comfort of the participant, verbal consent was given prior to each measurement, verbal cues were given throughout, a private room was used, the measurement was taken by a member of the sex, and the option to have a second member of the research team present who also was of the same sex was offered. BIA is prone to estimation errors due to biological factors such as, hydration status, recent food and drink intake, training load, and recent exercise activity (Moon, 2013). In an effort to control such factors participants were advised to hydrate adequately (~2L·day) in the 24-h prior to the test, eat and drink similar foods in the 24-h prior to the test, and to avoid high-intensity exercise 24-h prior to the test (Moon, 2013). Acknowledging the limitations discussed, BIA was used in study 4 due to the relatively non-invasive and time-efficient benefits of the protocol.

7.2.3.2 Dietary energy intake

EI was assessed during each phase with diet records kept for seven consecutive days with participants asked to record all weighed food and drink consumed, including alcohol. Participants were provided a familiarisation session where they were asked to download the MyFitnessPal application to their mobile device and login with the details provided by the principal investigator. During the session participants were given in-depth written (Appendix 11.1) and verbal instructions on the functionality of the application, dietary reporting strategies, instructed on how to alter serving sizes within the application, and provided links to educational materials related to serving sizes when weighing of food or drink was not possible (Appendix 11.1). Participants were also asked to record photographs of any meals that could not be weighed where they utilised the serving size resources provided (i.e., eating out at a restaurant). Participants were instructed to maintain and follow their normal eating pattern. All diet records

were reviewed, and participants were contacted via email or telephone when further clarity or supplementary information was needed following completion of each phase. All participants completed a minimum of one familiarisation session of recording a seven-day diet record during January 2019 and two participants completed a second. During phases 6 and 8, one participant was unable to utilise the application due to technical difficulties and therefore kept a paper diet record which was given to the principal investigator to upload. Nutrient data from the diet records were coded and analysed using Nutritics analysis software (Nutritics Ltd 2020, Co. Dublin, Ireland) for energy (kilocalorie), macronutrients (carbohydrates, fat, and protein) for each phase and expressed as a daily average for total and relative intakes. Nutritics is based on McCance and Widdowson 7th edition augmented with directly sourced information from manufacturers.

7.2.3.3 Exercise energy expenditure

Alongside the seven-day diet records, EEE was assessed where participants kept a training record and wore a HR monitor during each phase. Participants were instructed to maintain and follow their normal training regime. During the familiarisation session participants were asked to download a commercially available exercise tracking application (Strava Inc, San Francisco, CA, US) to their mobile device and share their diary with the principle investigator. The Strava application was used in study 4 as all participants currently used this application as a training log and it is heavily used within the triathlon community, giving a real-world application to the study. During the session participants were given in-depth written (Appendix 11.1) and verbal instructions on the functionality of the application and training reporting strategies. Participants were also educated on the setup, functionality, pairing of the HR sensor and training recording strategies specific to the Polar V800 watch. Prior to each phase of testing, the Polar V800 watches were set according to each individual's current age, height, weight, HR_{max}, sex, $\dot{V}O_{2max}$ and paired with the corresponding HR sensor.

The algorithm used for the estimation of EEE are pre-fixed by Polar Electro and not indicated in the manual, however, the Polar V800 has been validated previously in estimating EE in aerobic activity (Hernández-Vicente, Santos-Lozano, De Cocker & Garatachea, 2016). Participants were instructed to wear the watch on the left wrist and the HR monitor around the chest. Participants were instructed to describe each session in as much detail as possible, including, type, duration, distance, intensity, and RPE (explained during familiarisation) of the session and to wear the HR monitor at all sessions, including swimming and during cycling (training and transportation). All participants completed a minimum of one familiarisation session of recording a seven-day diet and training record during January 2019. Training data were saved on both the Strava application and watch which was later synchronized using the Polar Flow online software (Polar Electro Oy, Kempele, Finland) to analyse EEE (kilocalories) and training time (hours) for each phase and expressed as the mean daily EEE for described exercise sessions and mean weekly training time.

7.2.3.4 Energy availability calculations

Measures of EI and EEE were assessed during the same seven consecutive days, during each phase, to enable the calculation of EA. EA was observationally defined as EI minus EEE relative to kilograms of LBM (kcal·kgLBM^{-1}·day^{-1}) (Loucks et al, 2011; Loucks & Thuma, 2003). Measures of LBM were obtained during one of the three consecutive days prior to the start of the seven-day data collection phase when participants received the Polar V800 watch. The mean daily EI and mean daily EEE was used for the calculation of current EA. To control for the potential underestimation of EA, EEE was corrected for the participant's resting EE (kcal) without EEE estimated from the Polar HR monitors, that would have occurred during the equivalent time period (Heikura, et al., 2018a; Loucks & Thuma, 2003). To estimate resting EE (kcal·day·$^{-1}$), predictive resting metabolic rate (pRMR) was calculated using the Cunningham (1980) equation (500 + (22 x LBM)). This is accepted as the most applicable for

endurance athletes (Staal, et al., 2018; Thompson & Manore, 1996) and has previously been used with male triathletes (Torstveit, et al., 2019). To identify the validity of self-reported nutritional data, the EI : $_{p}$RMR ratio was calculated (Black, et al., 2000). Low validity of EI was identified with a physical activity level < 1.6 which has previously been used with endurance athletes (Melin, et al., 2015).

7.2.3.5 Questionnaire data

Questionnaire data used in the study are outlined in Chapter 3.4 and Chapter 4.2.4 of this thesis. During each phase, a self-administered online questionnaire was constructed and distributed (via a URL) in English using the Qualtrics electronic management system (Qualtrics, London, UK) with participants receiving a unique participant code to input. Participants were provided the URL link on day 1 of the monitoring week and instructed to complete the questionnaire by the end of week on day 7. All participants received a mid-week questionnaire completion reminder. In brief, the online questionnaire incorporated the LEAF-Q (Melin, et al., 2014) to assess LEA risk and the FAST (McNulty, et al., 2001) to assess eating attitudes.

7.2.4 Aerobic fitness (secondary, descriptive measure)

Study 4 formed part of a larger study where aerobic fitness (running and cycling) was assessed every three months of the triathlon season. During the larger study, beyond the scope of this thesis, a submaximal graded exercise test (GXT) was used to determine a lactate profile, subsequently followed by a maximal ramp protocol to determine $\dot{V}O_{2max}$. This protocol has previously been validated in the healthy adult population used in this study (Bennett, Parfitt, Davison & Eston, 2015; Beltz, Gibson, Janot & Kravitz et al, 2016). For descriptive purposes in study 4, the maximal oxygen consumption ($\dot{V}O_{2max}$) was used as a marker of aerobic fitness for running and cycling across the season, lactate threshold was not used in study 4. If study 4

were to be repeated, a maximal ramp protocol of 1-min continuous stages would have been used as a marker of aerobic fitness or as an alternative time trials (i.e., 20 or 30-min) for running and cycling performance would have been used (AIS, Tanner & Gore, 2013). However, for the purpose of this thesis a full description of the protocol used in the larger study is detailed accordingly.

Each of the $\dot{V}O_{2max}$ tests were conducted on separate days within a ten-day period with a minimum of 24 hours between each test to optimise recovery. Participants were asked to refrain from exercise in the 24 hours prior to each test and to report for testing at the same time of day within a 2-hour limit. Participants were also instructed not to ingest food in the 3 hours preceding testing and to avoid caffeine and alcohol in the 24 hours prior to arrival in the laboratory. Volume calibration of the Cortex Metalyser 3B (Cortex, Biophysik, Leipzig, Germany) was performed manually with a 3-L syringe and gas calibration conducted against ambient air and a known gas concentration (5% CO_2, 15% O_2, and 80% N_2) prior to each individual test. Preceding each test, three blood pressure readings, using an automated sphygmomanometer (OMRON, M3, Hoofddorp, Netherlands), were recorded after a supine rest period of 5 minutes, each separated by 2 minutes. A heart rate monitor (H10, Polar Electro, Finland) was used throughout all tests to measure heart rate (HR).

7.2.4.1 Treadmill test

The test consisted of a submaximal GXT that consisted of 4-6 x 3-min discontinuous exercise stages at a fixed gradient of 1% on a motorised treadmill (Desmo Pro, Woodway Inc, Wisconsin, USA) to determine a lactate profile. The exercise intensity was increased by 1 $km \cdot h^{-1}$ until the onset of blood lactate accumulation (LT_{OBLA}), an exercise intensity corresponding to 4 $mmol \cdot l^{-1}$ (Santos-Concejero, Granados, & Irazusta et al, 2014). Once LT_{OBLA} was attained, participants continued the test with a maximal ramp protocol to determine

$\dot{V}O_{2max}$ that consisted of 1-min continuous exercise stages at a fixed gradient of 1%, with exercise intensity increasing by 1 $km \cdot h^{-1}$ until volitional exhaustion.

7.2.2.2 Cycle ergometer test

The test consisted of a submaximal GXT that consisted of 4-6 x 3-min continuous exercise stages at a cadence of 70 RPM on an electronically braked cycle ergometer (Lode, Excalibur, Groningan, Netherlands) to determine a lactate profile. The exercise intensity was increased by 30W until LT_{OBLA}, an exercise intensity corresponding to 4 $mmol \cdot l^{-1}$ (Santos-Concejero et al, 2014). Once LT_{OBLA} was attained, participants continued the test with a maximal ramp protocol to determine $\dot{V}O_{2max}$ that consisted of 1-min continuous exercise stages, with exercise intensity increasing by 30W until volitional exhaustion.

7.2.4.3 Physiological data

$\dot{V}O_{2max}$ and RER was acquired with data stored on the inherent database (Metasoft version 5.1.0, Cortex Biophysik GmbH, Leipzig, Germany). The primary criterion for establishing $\dot{V}O_{2max}$ is a plateau, in the event that a plateau is not achieved secondary criteria is used. Confirmation of $\dot{V}O_{2max}$ was based on achieving at least three of the following criteria previously recommended for use in female triathletes (Snoza, Berg & Slivka, 2014): 1) ± 10 $beats.min^{-1}$ of age-predicted maximum HR (Nes, Janszky & Wisloff et al, 2013); 2) RER >1.10 (Midgley, McNaughton, Polman & Marchant, 2007); 3) plateau of <2.1 $ml \cdot kg^{-1} \cdot min^{-1}$ in $\dot{V}O_2$ (Millet, Dreano & Bentley, 2003); 4) RPE >17 (Beltz, et al., 2016); 5) blood lactate concentration ≥8 $mmol \cdot l^{-1}$ (Edvardsen, Hem & Anderssen, 2014). Offline analyses then determined $\dot{V}O_{2max}$ as the highest 15 second average (Robergs & Burnett, 2003). Rating of perceived exertion (RPE) was assessed using the Borg 6-20 scale at the end of every stage during the submaximal and maximal GXT (Borg, 1970). HR was recorded during the last 15

seconds of the final minute of each exercise stage throughout the protocol (Liguori & ACSM, 2020).

7.2.5 Statistical analysis

All statistical analyses were performed using SPSS (V.25; IBM Company, SPSS Inc., Chicago, USA) and $p \leq 0.05$ was considered statistically significant. Data normality was assessed using the Shapiro-Wilk test. All data are presented as means ± standard deviation (SD). Frequency analysis was undertaken for EA thresholds and key components of the LEAF-Q and FAST questionnaire scores. To examine changes across the phases, repeated measures analysis of variance (ANOVA) was performed. When indicated, Bonferroni *post hoc* corrections were used to identify significantly different phases.

7.3 Results

7.3.1 Difference between phases (participant characteristics)

Changes in descriptive data for all participants (n=10) across all eight phases are shown in table 7.2. Body mass, BMI, fat mass, run $\dot{V}O_{2max}$, training time or training HR remained relatively constant across the season ($p > 0.05$). Statistically significant differences in body fat were detected across the season, F (5, 45) = 2.642, p = .035. A large effect was observed as a n^2_p of 0.227 indicates that 22.7% of the between-subjects variance was accounted for by body fat. The assumption of sphericity was met as assessed by Mauchly's test of sphericity, χ^2 (14) = 21.200, p = .113 but post hoc analysis with a Bonferroni adjustment revealed no significant differences between phases ($p > .05$). Statistically significant differences in LBM were detected across the season, F (5, 45) = 2.524, p = .043. A large effect was observed as a n^2_p of 0.219 indicates that 21.9% of the between-subjects variance was accounted for by LBM. The assumption of sphericity was met, χ^2 (14) = 15.627, p = .364 and post hoc analysis with Bonferroni adjustment revealed that LBM was statistically significantly higher in Phase 4

(July) than Phase 8 (January; 1.630 (95% CI, .039 to 3.221 kg, p = .043) but not between any other phases (p > .05). Statistically significant differences in cycle $\dot{V}O_{2max}$ were detected across the season, *F* (3, 15) = 3.690, p = .036. A large effect was observed as a n^2_p of 0.425 indicates that 42.5% of the between-subjects variance was accounted for by cycle $\dot{V}O_{2max.}$ The assumption of sphericity was met, χ^2 (5) = 2.070, p = .844 but post hoc analysis with a Bonferroni adjustment revealed no significant differences between phases (p > .05).

50% of participants reported taking oral contraceptives during the duration of the study. Of the five participants not taking oral contraceptives, three reported regular menstrual cycles of 26-35 days in length, one reported oligomenorrhoeic cycles of 36-90 days in length, and one reported secondary amenorrhoea throughout the duration of the study.

7.3.2 EI, EEE & $_p$RMR

Self-reported EI, EEE, $_p$RMR, and EI : $_p$RMR ratio across the season are shown in table 7.3. Participants recorded ingesting 1988 ± 44 kcals·day^{-1} on average throughout the season with no significant differences in self-reported EI detected across the season which may be due to lower power (p = 0.728). EI : $_p$RMR ratio highlighted participants consistently under-reported EI across the season, however no significant changes across the season were detected which may be due to lower power (p > .05). Statistically significant differences in $_p$RMR were detected across the season, *F* (5, 45) = 2.507, p = .044. A large effect was observed as a n^2_p of 0.218 indicates that 21.8% of the between-subjects variance was accounted for by $_p$RMR, however this is based on a predictive equation rather than measured RMR. The assumption of sphericity was met, χ^2 (14) = 15.726, p = .358 but post hoc analysis with a Bonferroni adjustment revealed no significant differences between phases (p > .05). Statistically significant differences were detected in EEE across the season, *F* (2.922, 26.299) = 3.042, p = .048.

Table 7.2. Differences in descriptive characteristics and anthropometrics of female triathletes (n = 10) across the season.

	Phase 1 (March)	Phase 2 (May)	Phase 3 (June)	Phase 4 (July)	Phase 5 (September)	Phase 6 (November)	Phase 7 (December)	Phase 8 (January)	P
Age (years)	28 ± 9	-	-	-	-	-	-	-	-
Height (m)	1.67 ± 0.04	-	-	-	-	-	-	-	-
Mass (kg)	62.2 ± 3.2	62.4 ± 3.2	-	63.3 ± 2.5	62.5 ± 2.2	63.1 ± 2.6	-	62.3 ± 2.3	0.879
BMI ($kg \cdot m^2$)	22.5 ± 1.6	22.5 ± 1.3	-	22.7 ± 1.4	22.6 ± 1.2	22.8 ± 1.3	-	22.6 ± 1.5	0.554
Body fat (%)	20.1 ± 4.5	21.8 ± 4.2	-	21.9 ± 5.8	20.7 ± 5.3	21.9 ± 4.9	-	23.9 ± 4.0	0.035†
Fat mass (kg)	12.6 ± 3.2	13.6 ± 2.8	-	13.9 ± 3.9	13.0 ± 3.4	13.9 ± 3.3	-	14.9 ± 2.6	0.134
Lean body mass (kg)	49.7 ± 2.7	48.8 ± 3.1	-	49.5 ± 3.6[1]	49.9 ± 4.0	49.2 ± 3.0	-	47.7 ± 3.0	0.043*
Run $\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	45.3 ± 4.4	-	45.3 ± 4.2	-	41.4 ± 4.5	-	41.9 ± 4.3	-	0.185
Cycle $\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	44.5 ± 3.3	-	45.6 ± 5.6	-	39.7 ± 5.5	-	40.3 ± 6.1	-	0.036†
Training Time (hours·week)	6.3 ± 2.4	6.2 ± 3.1	-	5.8 ± 1.6	5.1 ± 2.7	4.6 ± 2.2	-	5.5 ± 2.4	0.066
Training HR ($beats \cdot min^{-1}$)	145 ± 9	142 ± 17	-	145 ± 11	146 ± 16	138 ± 14	-	140 ± 13	0.175

Data is presented as mean ± SD. † Significant effect of Phase via Repeated Measures ANOVA but pairwise comparisons for each phase p >.05. * Significant effect of Phase via Repeated Measures ANOVA with significant pairwise comparison. [1] Significantly different from Phase 8.

A large effect was observed as a n^2_p of 0.253 indicates that 25.3% of the between-subjects variance was accounted for by EEE. The assumption of sphericity was not met, χ^2 (14) = 30.773, p = .008. Epilson (Ɛ) was .584, as calculated according to Greenhouse and Geisser (1959) and used to correct the one-way repeated measures ANOVA. Post hoc analysis with Bonferroni adjustment revealed that EEE was statistically significantly lower in Phase 6 (November) than in Phase 5 (September; 39.4 (95% CI, 8.1 to 70.7) kcal, p = .011) but not between any other phases (p > .05).

Table 7.3 Changes in self-reported EI, EEE, $_p$RMR in female triathletes (n=10) across season.

	EI (kcal·day^{-1})	EI (kJ·day^{-1})	EEE (kcal·day^{-1})	$_p$RMR (kcal·day^{-1})	EI : $_p$RMR
Phase 1 (March)	2038 ± 284	8445 ± 1247	359 ± 135	1594 ± 60	1.27 ± 0.2
Phase 2 (May)	1978 ± 437	8311 ± 1841	370 ± 217	1573 ± 69	1.26 ± 0.3
Phase 3 (June)	-	-	-	-	-
Phase 4 (July)	2035 ± 457	8548 ± 1928	327 ± 160	1569 ± 59	1.28 ± 0.3
Phase 5 (September)	1933 ± 324	8122 ± 1375	343 ± 226	1609 ± 91	1.21 ± 0.2
Phase 6 (November)	1948 ± 329	8186 ± 1394	242 ± 157[1]	1577 ± 66	1.24 ± 0.2
Phase 7 (December)	-	-	-	-	-
Phase 8 (January)	1998 ± 385	8391 ± 1630	304 ± 139	1549 ± 67	1.29 ± 0.2
P	.728	.783	.048*	.044*	.691

Data is presented as mean ± SD. EEE, exercise energy expenditure; EI, energy intake; Kcal, kilocalorie; kJ, kilojoule; g, gram; $_p$RMR = predictive resting metabolic rate.* Significant effect of Phase via Repeated Measures ANOVA with significant pairwise comparison. [1] Significantly different from Phase 5.

7.3.3 EA and LEA risk (LEAF-Q)

Figure 7.2 presents the EA of individual female triathletes across the season. No significant differences in EA were detected across the season (p = .591). LEA (<30

kcal·kgLBM^{-1}·day^{-1}) was observed in 2 of 10 (20%) participants during phase 1 (March) and phase 6 (November), and in 3 of 10 (30%) during phases 2 to 5 (May to September). Of those participants demonstrating LEA, one participant presented with LEA during all phases across the season and another participant across five phases from March to November. One participant presented with LEA during phases 4 to 5 (July to September) and in phase 8 (January), and two further participants presented with LEA each in separate phases (phase 2 and phase 8). Two participants (20%) displayed one instance of optimal EA (>45 kcal·kgLBM^{-1}·day^{-1}) throughout the season and all other phases were considered subclinical LEA (30-45 kcal·kgLBM^{-1}·day^{-1}). Subclinical LEA was observed by most participants and maintained throughout the season.

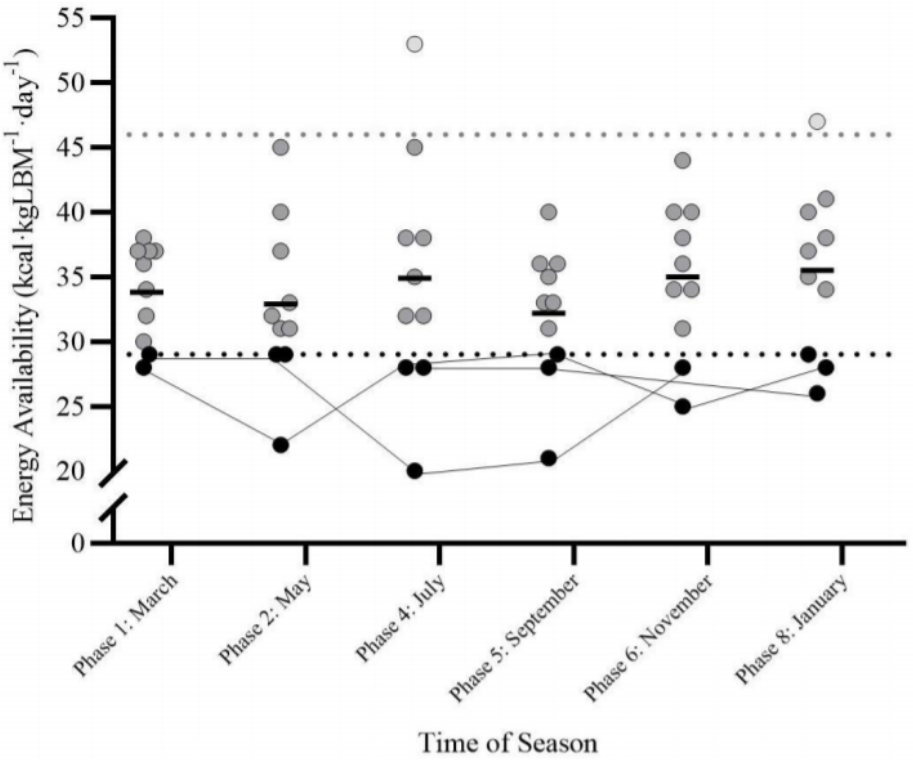


Figure 7.2. Energy availability of individual competitive female triathletes across the season.

Solid black circles represent EA of individuals with LEA (<30 kcal·kgLBM^{-1}·day^{-1}) across the season. Open dark grey circles represent EA of individuals with subclinical LEA (30-45 kcal·kgLBM^{-1}·day^{-1}). Open light grey circles represent EA of individuals with optimal to high EA (>45 kcal·kgLBM^{-1}·day^{-1}). Solid black bars represent the mean EA (kcal·kgLBM^{-1}). Dashed black line represents a threshold of LEA (<30 kcal·kgLBM^{-1}·day^{-1}) and dashed grey line represents a threshold of optimal EA (>45 kcal·kgLBM^{-1}·day^{-1}).

Figure 7.3 presents LEA risk (LEAF-Q) and the EA of individual female triathletes across the season. Statistically significant differences in LEA risk identified by the LEAF-Q were detected across the season, F (5, 45) = 1.629, p = .011. A large effect was observed as a n^2_p of 0.274 indicates that 27.4% of the between-subjects variance was accounted for by the LEAF-Q. The assumption of sphericity was met, χ^2 (14) = 14.625, p = .432 but post hoc analysis with a Bonferroni adjustment revealed no significant differences between phases (p > .05). Five of 10 (50%) participants were classified at risk of LEA by the LEAF-Q in phases 1 (March) and 5 (September), in 3 of 10 (30%) during phase 2 (May), in 4 of 10 (40%) during phases 4 (July) and 6 (November), and in 1 of 10 (10%) during phase 8 (January).

Of those participants who presented with subclinical LEA (30-45 kcal·kgLBM^{-1}·day^{-1}), one participant was considered at risk of LEA by the LEAF-Q during all phases across the season, another was considered at risk of LEA throughout phases 1 to 6 (March to November), and another participant was considered at risk of LEA throughout phases 4 to 6 (July to November). Of those participants who presented with LEA (<30 kcal·kgLBM^{-1}·day^{-1}), one participant who presented with LEA across five phases of the season (March to November) was only considered at risk of LEA by the LEAF-Q during phase 1 (March). Another participant who presented with LEA throughout the season was considered by the LEAF-Q at low risk of LEA during all phases.

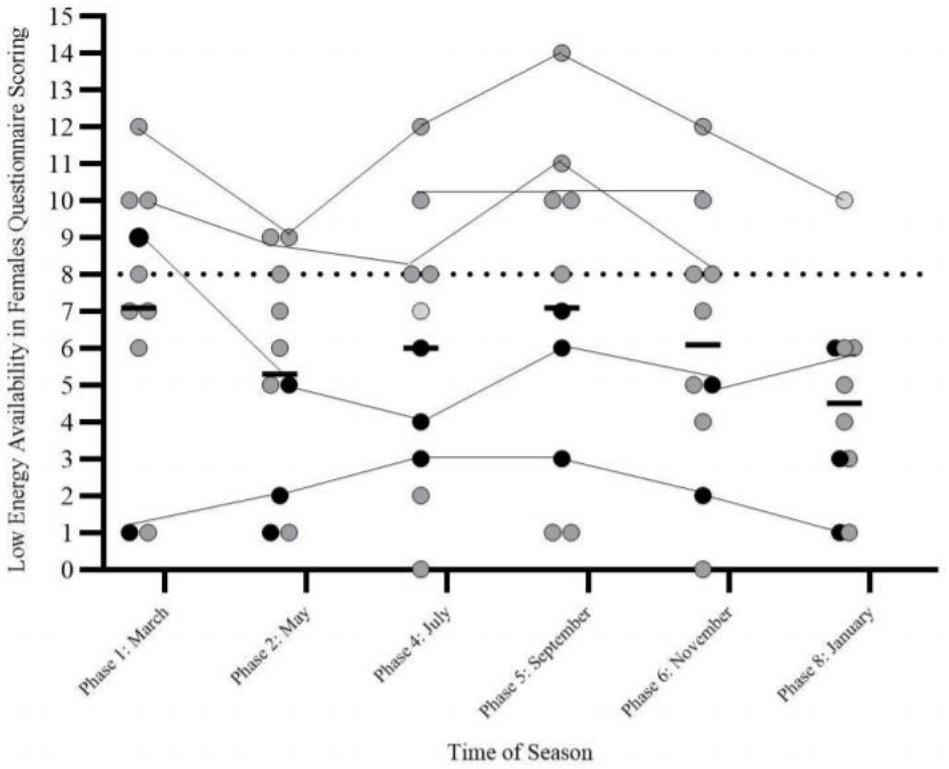


Figure 7.3. Low energy availability risk (LEAF-Q) and energy availability of individual competitive female triathletes across the season.

Dashed black line represents a threshold of those considered at risk of developing LEA by the LEAF-Q (≥ 8). Solid black circles represent EA of individuals with LEA (<30 kcal·kgLBM^{-1}·day^{-1}) across the season. Open dark grey circles represent EA of individuals with subclinical LEA (30-45 kcal·kgLBM^{-1}·day^{-1}). Open light grey circles represent EA of individuals with optimal to high EA (>45 kcal·kgLBM^{-1}·day^{-1}). Solid black bars represent the mean LEAF-Q total score.

7.3.4 Eating attitudes (FAST)

Figure 7.4 presents eating attitudes (FAST) and the EA of individual female triathletes across the season. No significant differences in eating attitudes were detected across the season ($p = .524$). Four of 10 (40%) participants were considered at risk of DE during phases 1 (March), 6 (November), and 8 (January. Two of 10 (20%) participants were considered at risk of DE during phases 2 to 5 (May to September). No participants were considered at risk of ED (FAST total score >94). One participant was considered at risk of DE during all phases of the season and subclinical LEA (30-45 kcal·kgLBM^{-1}·day^{-1}) was observed across all phases.

Another participant was considered at risk of DE in five phases of the season (March and July to January) and LEA was observed in five phases (March to November).

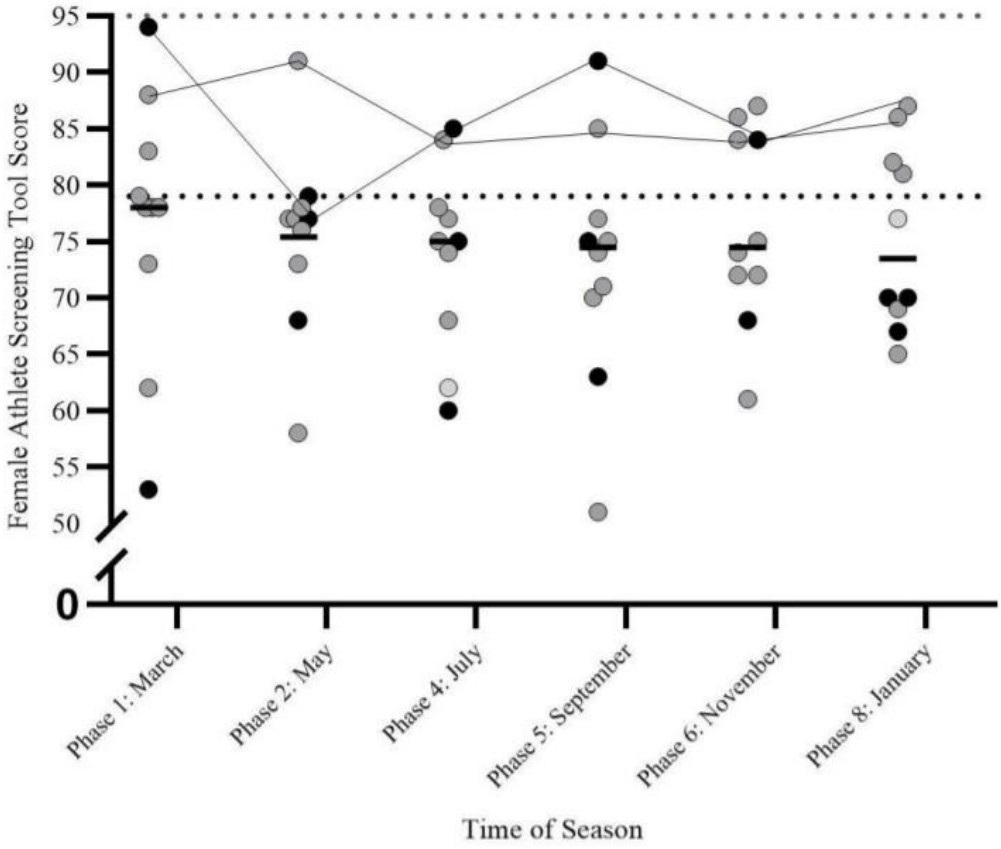


Figure 7.4. Eating attitudes (FAST) and energy availability of individual competitive female triathletes across the season.

The two dashed lines represent a threshold of DE (total FAST score 79-94). Solid black circles represent EA of individuals with LEA (<30 kcal·kgLBM^{-1}·day^{-1}) across the season. Open dark grey circles represent EA of individuals with subclinical LEA (30-45 kcal·kgLBM^{-1}·day^{-1}). Open light grey circles represent EA of individuals with optimal to high EA (>45 kcal·kgLBM^{-1}·day^{-1}). Solid black bars represent the mean FAST total score.

7.4 Discussion

The present study aimed to follow a cohort of female triathletes across a full triathlon season and examine the changes in EA and eating attitudes. An additional aim was to examine the prevalence of those considered at risk of LEA and of DE/ED by the LEAF-Q and FAST. Accordingly, the main findings were that: 1) no significant differences in mean measures of

EA or eating attitudes were detected across the season. 2) Significant differences were detected in LEA risk identified by the LEAF-Q, however, post hoc analysis revealed no significant differences between phases of the season. 3) The prevalence of LEA was higher in the competitive season but 4) the prevalence of those who met the cut-off score for DE was higher in the pre-and-post competitive season.

Taken together, the findings of the study are the first, to our knowledge, to follow a single cohort of female triathletes across the season to examine the changes in EA and eating attitudes. Although few significant differences were observed, findings of this study suggest that female triathletes may be in a state of subclinical LEA across the duration of the season. Despite mean EA remaining relatively stable across the season, some variation in the prevalence rates of LEA were observed. This may indicate that certain phases of the season (i.e., competitive) may contribute to the overall LEA risk in some athletes, but not all.

7.4.1 EA

The mean EA across the season remained relatively consistent in competitive female triathletes ranging from 32 to 36 kcal·kgLBM^{-1}·day^{-1}. Loucks and Thuma (2003) examined the dependence of LH pulsatility on EA in sedentary, eumenorrheic, normal weight females in a randomised, repeated-measures clinical study. It was reported the habitual mean EI was 48 ± 7 kcal·kgLBM^{-1}·day^{-1} (Loucks & Thuma, 2003). No statistically significant difference (repeated measures ANOVA with *post hoc* analysis) in LH pulsatility were reported whilst EA was maintained at an EB equating to ~45 kcal·kgLBM^{-1}·day^{-1} but when EA was decreased to ≤30 kcal·kgLBM^{-1}·day^{-1}, disruption in LH pulsatility was observed (Loucks & Thuma, 2003). It was therefore proposed that below a threshold of 30 kcal·kgLBM^{-1}·day^{-1}, menstrual disturbances, and other Triad sequalae would be induced (Loucks & Thuma, 2003). However,

there is no current consensus regarding clear EA threshold in female athletes (De Souza, et al., 2019).

The threshold of <30 kcal·kgLBM^{-1}·day^{-1} was unsubstantiated in recent work by Williams, et al., (2015) and Lieberman, et al., (2018). In these studies, a randomised control trial was used to examine varying degrees of EA on menstrual status, by manipulating EI and EEE in previously eumenorrheic females. These studies could not identify an absolute threshold of EA that induced menstrual disturbances. A result of many females displaying menstrual disturbances above the threshold of 30 kcal·kgLBM^{-1}·day^{-1} and menstrual disturbances failing to be induced in some females below a threshold of 30 kcal·kgLBM^{-1}·day^{-1} (Williams, et al., 2015; Lieberman, et al., 2018). This is in line with findings from the current study as one individual presented with subclinical LEA (30-45 kcal·kgLBM^{-1}·day^{-1}) throughout the season and reported secondary amenorrhea. Another participant presented with clinical LEA across five phases of the season (March to November) and reported oligomenorrheic cycles of 36-90 days in length. Such findings tend to indicate individual variability in the EA threshold at which menstrual disturbances and other Triad/RED-S sequalae are observed (De Souza, et al., 2019).

Although Williams, et al., (2015) and Lieberman, et al., (2018) could not substantiate an absolute threshold of EA, it was observed that a linear increase in menstrual disturbances existed as EA decreased. In addition, the predicted probability of observing menstrual disturbances was >50% when EA was <30 kcal·kgLBM^{-1} (Williams, et al., 2015; Lieberman, et al., 2018). As a result of these findings, De Souza, et al., (2019) proposed a dose-continuum may exist between EA, menstrual disturbances, and other Triad/RED-S sequalae, but more studies are needed to further elucidate this concept.

Despite the lack of consensus around absolute thresholds of EA, it has been suggested that a threshold of <30 kcal·kgLBM^{-1}·day^{-1} when examining large groups of females may be useful in determining an at-risk profile for Triad/RED-S (Reed, et al., 2015). In contrast, rather than use a single absolute threshold of EA, it has been suggested that a combination of EA measures, body mass and composition, eating behaviours, and other measures of metabolic status (i.e., measured RMR) be used in free-living athletes (De Souza, et al., 2019). Though no consensus exists regarding an absolute threshold of EA that induces menstrual disturbances, there is agreement that to ensure the optimal functioning of physiological systems and processes, physically active females should aim for ≥45 kcal·kgLBM^{-1} of EI to ensure adequate EA (Loucks & Thuma, 2003; De Souza, et al., 2014; Loucks, 2014; De Souza, et al., 2019).

Continued work by Loucks, et al., (2011) has further suggested that subclinical LEA of 30-45 kcal·kgLBM^{-1} may be tolerated for short periods during a well-constructed weight-loss programme. In the current study, the prevalence of subclinical/clinical LEA (<45 kcal·kgLBM^{-1}) was worrying in this group of female triathletes as optimal EA was only observed once across the season in two participants. Although likely related to the small sample size (n=10), low power and methodological limitations (see Chapter 2.3.1.2), worryingly the prolonged nature of subclinical LEA in this cohort may begin to induce subclinical abnormalities in physiological function, if EA is not restored outside of the competitive season. While a worrying prevalence of subclinical LEA was observed in the current study across the season, this is not unprecedented. Melin, et al., (2015) reported similar findings with 63% of elite female endurance athletes (n = 40) reporting clinical/subclinical LEA (<45 kcal·kgLBM^{-1}). Zabriskie, et al., (2019) also reported a consistent state of clinical/subclinical LEA across five phases of the season in twenty female NCAA Division II lacrosse athletes (range: 23-30 kcal·kgLBM^{-1}). Although EA was not examined, a cross-sectional study (n=15) by Hoch, et al., (2007) reported 60% of club-based female triathletes were in a calorific deficit consistent

with DE pathology and 40% had a history of amenorrhea. In contrast, a cross-sectional study by Hoch, et al., (2009) reported 36% of varsity female athletes (n=80) had clinical/subclinical LEA, compared with 39% of sedentary female control students (n=80).

Although the mean EA remains relatively stable and above 30 kcal·kgLBM^{-1} across all phases of the season in the present study, subtle changes to the prevalence of LEA across the season is evident. It was observed that 30% of participants were in a state of clinical LEA (<30 kcal·kgLBM^{-1}) predominantly in the competitive season and 20% of participants during the pre-competitive/off-season. This may suggest that certain phases of the season may contribute to the overall LEA risk observed in some athletes. The increased risk may be an inadvertent inability to adequately compensate for increased energy needs. In the current study, overall training time was considerably lower than previously reported in Chapters 4-6 and no significant differences were observed between phases of the season. This could reflect poor validity of EEE measures and self-reported training duration (Borresen & Lambert, 2006; Mujika, 2017), where participants in Chapters 4-6 could be over-reporting and in the current study participants may be under-reporting. Although the potential increase in energy needs was not observed in the current study with regards to training duration, there were significant differences in cycle $\dot{V}O_{2max}$ which accounted for 42.5% of the variance observed. It was observed that cycle $\dot{V}O_{2max}$ peaked at the height of the competitive season (June) which may reflect the cumulative training effects outside of the seven-day monitoring period. This highlights that single assessments of EA in free-living athletes may not be reflective of overall training and nutritional practices (Burke, et al., 2018), and/or the varied taper patterns and recovery periods used by athletes during the competitive season, when participating in multiple competitions (Mujika, 2011).

Owing to the methodological difficulties associated with measured EA in free-living athletes (see Chapter 2.3.1.2), the number of comparable studies is small albeit growing.

Previous observational work by Melin, et al., (2015) in elite female endurance athletes (n=40) observed fifteen (37%) participants with optimal current EA (≥ 45 kcal·kgLBM^{-1}), seventeen (43%) with subclinical LEA (30 to 45 kcal·kgLBM^{-1}), and eight (20%) with clinical LEA (< 30 kcal·kgLBM^{-1}). Reed, et al., (2013) examined changes in EA across the season (pre-mid-and-post) in Division I female soccer players (n=19) where a 19% reduction in EA was observed from pre to mid-season, followed by a 35% improvement of EA at post-season. Similar to the current study, mean EA across all time points remained above 30 kcal·kgLBM^{-1} and 29% of participants displayed clinical LEA at the pre and/or mid-season time point (Reed, et al., 2013). Across all studies and in line with current findings, the observation of subclinical/clinical LEA in participants was attributed to both EI-related and EE-driven causes. Such observations may also be explained by under-reporting of dietary EI, particularly in the current study where sample size was small (n=10) and poor validity of diet records was observed (mean EI:$_{p}$RMR = 1.26 ± 0.2; Black, et al., 2000).

Furthermore, it is important to note Melin, et al., (2015) recruited thirteen national team level endurance athletes from Denmark and Sweden, and Reed, et al., (2013) recruited nineteen American Division I female soccer players. In both instances the demands of elite level sport, single-sport endurance events, and team-sports on energy status and potential risk factors are likely to be different to competitive (non-elite) female triathletes. In reference to Chapter 6 of this thesis, the participants recruited in the current study would be considered as recreational age-groupers (see Chapter 6.2.1). In contrast to the current study, Melin, et al., (2015) only observed participants at one time point and Reed, et al., (2013) observed across three. Unlike the current study, these studies do not provide a longitudinal overview of changes in energy status.

Direct comparison is also difficult between studies due to methodological differences in determining EI, EEE, and EA. Melin, et al., (2015) examined body composition and bone

health using the gold standard assessment of DXA, measured RMR was recorded, EEE was derived from training records and individual prediction equations from measured HR and corresponding EE during an incremental $\dot{V}O_{2max}$, a complete gynecological assessment was used to assess reproductive function, and blood samples were drawn to analyse biomarkers of energy deficiency. Though these clinical methods have greater accuracy, reliability, and validity for determining energy status and to monitor risk, they may not be feasible, cost-effective, user-friendly, non-invasive, accessible, or generalised to "real-life" situations to a large percentage of the non-elite athletic population (both coaches and athletes). Additionally, as covered previously in this thesis and Chapter 4, more subjective, qualitative measures may be more sensitive than the quantitative measures for LEA, Triad and/or RED-S.

Despite the current study not finding any significant differences in measured EA (likely due to lower power), significant differences were found in LEA risk with the LEAF-Q accounting for 27.4% of variance observed across the season. These findings further suggest that for some athletes there may be certain phases of the season that contribute to the overall LEA risk observed. In the current study, the mean percentage of those considered at risk of LEA across the season in competitive female athletes was 37% (range: 10% to 50%). Such findings are similar to those reported previously in this thesis. For example, the LEAF-Q classified 42% of competitive female triathletes at risk of LEA (Chapter 4), 49% of competitive female triathletes aged 18-29 years were classified at risk (Chapter 6), and finally Chapter 7 identified 39% of recreational age-groupers as at risk of LEA. Overall, the current study's findings are in line with both previous findings of this thesis and previous studies examining female endurance sport athletes (range: 18% to 80% - see table 2.2: Muia, et al., 2016; Jesus, et al., 2021).

However, of those participants that were considered at risk of LEA by the LEAF-Q in the present study (mean 37%, range 10-50%), the majority displayed subclinical LEA (30-45

kcal·kgLBM^{-1}). Currently, there is no documented evidence for the percentage of false positives and negatives of the LEAF-Q (Mountjoy, et al., 2018). The current study highlights that LEAF-Q data needs to be interpreted with caution until further validation studies are available, as the LEAF-Q may over-estimate LEA in comparison to actual measures of LEA. However, it should be noted that due to small sample size the current study included participants on oral contraceptives which may increase the likelihood of false positives (Melin, et al., 2014).

7.4.2 Eating attitudes (FAST)

Eating attitudes across the season remained relatively consistent in competitive female triathletes with a total FAST score ranging from 73 to 77, which remains below the DE threshold of 79 (McNulty, et al., 2001). These observations of relative stability in eating attitudes across the season are consistent with past research (Doughty & Hausenblas, 2005; Krentz & Warschburger, 2013; Thompson, Petrie & Anderson, 2017). As discussed in Chapters 4-6, current studies within the athletic population have documented the prevalence of DE/ED behaviours. However, these studies have all been cross-sectional in nature involving single measures which has limited our understanding of the development and progression of DE/ED behaviours over time in athletes. Longitudinal designs within the athletic population would allow the examination of DE/ED pathology and determine if DE/ED classifications or behaviours develop, remain stable, or change over time. Comparison of the current findings is therefore difficult as to date limited studies of this nature exist.

Reed, et al., (2013) reported similar overall findings of eating attitudes as the current study in Division I female soccer players, as no differences between the mid and post season in body dissatisfaction, drive for thinness, or bulimia scores were observed. Direct comparison to the current study is difficult as Reed, et al., (2013) used the EDI-2 focusing specifically on

the three aforementioned subscales previously related to chronic energy deficiency in female athletes (Cobb, et al., 2003; Gibbs, Williams, Scheid & Toombs, et al., 2011; Reed, et al., 2011). Although Reed, et al., (2013) observed no significant changes across the season, the clinical LEA observed in some athletes (29%) may be explained by the negative relationship observed between EA and body dissatisfaction and drive for thinness. No significant differences were observed in the current study across the season which may be explained by small sample size, structure of the FAST, and the lack of anonymity provided with the online questionnaire (unlike in Chapters 4-6). However, 40% of participants were considered at risk of DE outside of the competitive season (May to September) and 20% were at risk of DE during the competitive season. This tends to indicate that some participants, but not all, may experience some degree of pressure related to weight and body shape as they transition in and out of the main competitive season. This could provide explanation to some of the subclinical/clinical LEA (<45 kcal·kgLBM^{-1}) observed throughout the season.

The current findings also corroborate that despite DE/ED underpinning a large percentage of LEA cases in athletes, other situations may also contribute to the development of subclinical/clinical LEA (Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; Melin, et al., 2015). For example, in the current population subclinical/clinical LEA may inadvertent due to poor nutritional knowledge, a mismanaged programme to reduce body mass and/or fat, other psychological stress, EXD, or it could also be explained by the under-reporting of EI and DE/ED symptoms.

7.4.3 Limitations

The current study should be interpreted with caution. One of the primary limitations was the small sample size and the low power to distinguish differences between phases and/or groups with low versus higher EA, or those with no DE versus DE. Another limitation is the

lack of non-athletic control participants to assess whether there are more pronounced differences across the season, or greater prevalence in these conditions in a population of competitive triathletes. It too would be beneficial to examine athletes with greater training loads (e.g., higher competitive level or triathletes participating in half-ironman/ironman distances), where distinct changes between training phases would have been more marked. However, the longitudinal nature of the study and the demands of the seven-day diet and training records, assessment of body composition and $\dot{V}O_{2max}$, plus an online questionnaire likely caused substantial participant and researcher burden.

As addressed in Chapter 4.4.2 there remains no standardised or reference guidelines for the assessment of EA and the methodological limitations associated with the assessment of EI, EEE, and body composition are slowly being addressed in the literature (Mountjoy, et al., 2018). Limitations include the use of seven-day diet records to assess EI which are frequently fraught with misreported and under-reported information (Capling, et al., 2017). Despite all participants being provided with training and familiarisation sessions on how to accurately record EI, poor validity of diet records was evident across all participants. Until more accessible and simpler methods are available, this will continue to be a barrier in assessing measured EA in free-living athletes. Similar self-report methods were used in the assessment of EEE with seven-day training records, although these typically contain less misreported information but may explain the low training duration observed (Neilson, Robson, Friedenreich & Csizmadi, 2008).

The use of the Polar V800 HR monitor with built in accelerometer may too be a limiting factor, as although validated, they too are frequently fraught with over-and-under estimation of EE with EE algorithms generally unreleased to the scientific community (Hernández-Vicente, et al., 2016). In regard to the analysis of body composition, there is a risk that changes in hydration status occurred between measurements (i.e., hypo- and hyperhydration) which can

alter the electrolyte balance and subsequently influence BIA measurements (Mialich, Sicchieri & Junior, 2014). Nutritional status, circadian rhythm, and acute training status could also have influenced BIA measurements (Campa, Toselli, Mazzilli & Gobbo, et al., 2021). The lack of objective measurements related to biomarkers of energy deficiency (discussed in Chapter 4.4.2) and clinical assessment of reproductive function and bone health also limit the findings of the current study. Another limiting factor is the lack of control for menstrual cycle phase to assess if different phases of the menstrual cycle influence total EA in competitive female triathletes.

Similar to Chapter 4-6, the sequence of events that led to the findings reported cannot be determined and cannot imply causality. Assessment of EA at one point in time across the various phases of the season may provide results not representative of an individual's true habitual EI and EEE, or account for micro-cycles of periodised training. However, with the logistics and participant demands of the study considered, the current study demonstrated that female triathletes may be at increased risk of reduced EA and the associated negative health implications across the season. The current study should primarily be viewed as explorative due to the increased risk of type 2 errors and requires additional studies of this kind with larger sample sizes to be conducted for verification of findings. Acknowledging the prior points are limitations to the data, this study was designed as a longitudinal, observational study employing validated methodologies accessible to free-living athletes. This approach is commonly used by field practitioners and increases the external validity of the study by incorporating a real-world pragmatic aspect. The current study experienced similar methodological limitations previously reported in the literature and future work would be recommended to incorporate clinical laboratory-based assessments to more accurately corroborate or refute the current findings.

7.5 Conclusion

The current study is the first, to our knowledge, to examine energy availability and eating attitudes in a single cohort of free-living, female triathletes, across the triathlon season.

Current findings indicated that female triathletes in the study consistently failed to match dietary EI to their levels of EE throughout the season as a worrying percentage of individuals were observed with subclinical and/or clinical LEA. No statistically significant changes in EA were observed across the season and mean EA remained above the EA threshold previously associated with negative health outcomes but below the optimal level of EA. Subclinical LEA has been found to only be tolerated for short periods. The current study observed this state across the season which may increase the risk of subclinical abnormalities in physiological function in these athletes if EA is not restored post-season. No statistically significant changes were observed in eating attitudes, but the prevalence of DE may explain the reduced EA observed in some athletes. Consequently, female triathletes should not be overlooked as a population at risk of subclinical and/or clinical LEA or negative eating attitudes. The results further validate the requirement of additional resources for non-elite athletic populations (including coaches, NGBs and parents) focusing on optimal nutritional strategies and periodisation of both nutrition and training for multi-sport endurance athletes. Further studies are required to examine changes in EA and eating attitudes and other behaviours (i.e., EXD) that may contribute to reduced EA across the various triathlon distances.

7.6 Statement of original contribution

- This is the first study to follow a single cohort of free-living, competitive female triathletes and non-elite, female endurance athletes across the triathlon season to examine changes in EA and eating attitudes.

CHAPTER 8

GENERAL DISCUSSION

8.1 Introduction

Female athletes from leanness sports (i.e., endurance, aesthetic, or weight-class) have been identified as a group that may be at increased risk of developing LEA and the associated negative health and performance consequences (Nattiv, et al., 2007; De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). However, there has been limited focus on athletes from multi-sport endurance events, such as triathlon, that are often characterised by high training loads over a sustained period (Vescovi & VanHeest, 2016; Etxebarria, et al., 2019). This has led to findings from single-sport endurance events, such as running or cycling, being generalised, and applied to multi-sport endurance athletes. The main purpose of this thesis was to investigate the prevalence of risk of LEA and associated risk factors (i.e., DE behaviour, clinical ED, and EXD) in female triathletes. Using self-report screening tools (i.e., LEAF-Q, FAST, and EDS-R) and direct measures of EA (i.e., LBM, EI, and EEE), a series of studies were conducted to establish the prevalence of LEA risk.

Studies 1 to 3 were cross-sectional, descriptive studies using self-report screening tools. Study 1 examined the prevalence of risk for LEA, DE/ED and EXD, and potential associations in female triathletes. Study 2 examined the influence of age on the prevalence of risk and examined potential associations between age and LEA, DE/ED, and EXD in female triathletes. Study 3 examined the influence of performance level on the prevalence of risk and examined potential associations between performance level and LEA, DE/ED, and EXD in non-elite female triathletes. Study 4 was a longitudinal study design using direct measures of EA and self-report screening tools to examine changes in EA and eating attitudes across different phases of the season in female triathletes, explore prevalence rates for LEA risk and DE/ED risk, and examine how prevalence rates change across the season.

8.2 Key Findings

Study 1 (n = 393):

- 42% of female triathletes aged 18-54 years were considered at risk of LEA by the LEAF-Q.
- 25% were considered at risk of DE behaviour and 9% considered at risk of clinical ED by the FAST.
- 58% were considered as symptomatic for EXD and 9% were considered at risk of EXD by the EDS-R.
- Eating attitudes and exercise behaviour were significant predictors of LEA and exercise behaviour was a significant predictor of eating attitudes in female triathletes aged 18-54 years.

Study 2 (n = 393):

- The prevalence of those considered at risk of LEA, DE behaviour, clinical ED, and EXD was higher in younger female triathletes (aged 18-29 years) compared with older (30-39 years and 40-49 years).
- Younger female triathletes were more likely to be considered as symptomatic for EXD than older.

Study 3 (n = 383):

- The prevalence of those considered at risk of LEA, DE behaviour, and EXD was higher in competitive female triathletes identifying as a top-percentile age-grouper compared to those identifying as a recreational age-grouper.
- Recreational age-groupers were less likely to be considered as symptomatic for EXD than top-percentile age-groupers.

Study 4 (n = 10):

- No statistically significant differences in measured EA or eating attitudes (FAST) were observed across the season in a cohort of female triathletes.
- The prevalence of subclinical LEA across the duration of the season was high in female triathletes with the prevalence of LEA highest during the competitive phase. The prevalence of DE behaviour was higher in the pre-and-post competitive phase.

8.3 Significance of findings

8.3.1 Prevalence of risk

In study 1 (n = 393), it was shown that a significant proportion of female triathletes were considered at risk of LEA (42%). The prevalence observed was in line with previous studies examining endurance sports athletes who reported a prevalence rate ranging from 18% to 80% (table 2.2; Muia, et al., 2016; Jesus, et al., 2021). It has been proposed that athletes from leanness sports, particularly endurance sports, are at a greater risk of developing LEA than those from non-leanness sports (Nattiv, et al., 2007; Mountjoy, et al., 2014). In the current study, the prevalence rate of risk observed (42%) for female triathletes was in the upper percentile of the prevalence rates previously reported in team or ball-based sports (range: 12% to 53%; Reed, et al., 2013; Condo, et al., 2019; Logue, et al., 2020). Importantly, female triathletes were identified as a group that should be considered at increased risk of Triad or RED-S and the associated negative consequences, which had not been previously explored.

LEA may occur intentionally or inadvertently (Nattiv, et al., 2007). This study provided important information regarding the prevalence of risk of DE behaviour, clinical ED, and EXD with greater prevalence's observed in female triathletes than previously reported in the general population (Sundgot-Borgen & Torstveit, 2004; Smink & Hoek, 2013; Mónok, et al., 2012; Sussman, et al., 2011). Additionally, this study was the first to investigate associations between LEA, eating attitudes, and exercise behaviours in female triathletes. In this population, the risk of LEA may be underpinned by known risk factors (i.e., DE/ED; Nattiv, et al., 2007; Mountjoy, et al., 2014) as it was identified that eating attitudes were significant predictors of LEA. However, it was also identified that exercise behaviours (EXD) were significant predictors of LEA and eating attitudes which agrees with seminal research by Torstveit, et al., (2019). This highlights the multifactorial nature of underlying, psychological risk factors that need to be considered in research and clinical practice when assessing LEA, Triad and/or RED-S.

8.3.2 Age

In alignment with current Triad and RED-S models (Nattiv, et al., 2007; Mountjoy, et al., 2018), study 2 (n = 393) evidenced high prevalence's of LEA risk in female triathletes, irrespective of age (range: 39% to 49%). Importantly, it was identified LEA risk was greatest in younger athletes and appeared to decline with age, albeit it did not disappear completely. This was comparable to findings for DE/ED and EXD across age groups. This has not previously been explored in this athletic population and the majority of work to date has centred on younger athletic populations (see table 2.2 and 2.3). The higher prevalence's evident across age groups in study 2 supports the need for continued work, to further elucidate LEA, DE/ED, and EXD both as risk factors for LEA and as individual constructs in older athletic populations. Thein-Nissenbaum (2013) acknowledged the long-term consequences of LEA, whereby the longer the duration of LEA, the greater the risk of irreversible health consequences. Again, this is similar for DE/ED and EXD behaviours (American Psychiatric Association, 2013). However, the prevalence of injury, gastrointestinal disturbances, and menstrual disturbances were higher in younger athletes compared with older in study 2.

It was acknowledged that the associated negative consequences of LEA may change across an athlete's lifespan, however, the most critical period for the development of LEA is during adolescence and young adulthood (Thein-Nissenbaum, 2013). This is due to it being a critical period of growth and development, particularly in relation to bone health, and impairments during this time will manifest negatively later in life. Study 2 also highlighted the specific aetiology and symptomology of LEA, DE/ED, and EXD may differ dependent on age which may have implications for screening, diagnosis, treatment, and the implementation of educational initiatives.

8.3.3 Performance level

In study 3 (n = 383), there were no significant differences in LEA and DE/ED risk between performance levels in non-elite female triathletes. This may reflect the smaller sample size evident in top-percentile age-groupers compared with recreational age-groupers. However, there was a tendency for top-percentile age-groupers to display higher prevalence rates of LEA and DE risk than recreational age-groupers. This highlighted that LEA and DE/ED exists in non-elite athletic populations but as performance level/classification improves, the risk of developing LEA and/or DE behaviour increases. This agrees with the current Triad and RED-S models (Nattiv, et al., 2007; Mountjoy, et al., 2018). The aetiology of why LEA and DE behaviour risk may increase with performance level is unclear from study 3 findings. Similar to findings by Logue, et al., (2019), there were significant differences in training load with top-percentile age-groupers spending more hour's training per week. This may suggest inadvertent LEA resulting from athletes failing to match EI to elevated EEE and/or appetite suppression (Wasserfurth, et al., 2020).

Importantly, study 3 reported significant differences in EXD scores between performance levels and a significant association between EXD and performance level was detected, which has not previously been explored in this population. It has been proposed that the differences observed in EXD scores between performance levels may relate to differences in EXD symptomology (Szabo, et al., 2013; De La Vega, et al., 2016). For instance, top-percentile age-groupers may exhibit more physiological factors of EXD compared to recreational age-groupers who may exhibit more psychological factors. This highlights the importance of research differentiating between primary and secondary EXD and its relation to the development of LEA (Costa, et al., 2013).

8.3.4 Seasonal changes

Study 4 (n = 10) showed that there were no significant differences in EA and eating attitudes across the season in a single cohort of female triathletes, which has not previously been explored. It has been suggested that athletes aim for ≥ 45 kcal·kgLBM^{-1} of EI to ensure adequate EA for optimal physiological functioning, however, there is no consensus regarding clear EA thresholds for athletic populations (Loucks & Thuma, 2003; De Souza, et al., 2019). Although no significant changes were evident across the season, it was highlighted that the mean EA across the season ranged from 32 kcal·kgLBM^{-1}·day^{-1} to 36 kcal·kgLBM^{-1}·day^{-1}. The high prevalence of subclinical LEA observed is worrying as current research has suggested prolonged subclinical LEA (30-45 kcal·kgLBM^{-1}) may result in the development of subclinical impairments to physiological functioning if EA is not restored (Loucks, et al., 2011).

Additionally, it was observed that for some, but not all, certain phases of the season may contribute to overall LEA risk. For instance, it was observed that for some athletes the prevalence of LEA was greater in the competitive season and the risk of DE behaviour may be greater in the non-competitive season for some athletes. This may indicate some female triathletes experience some degree of pressure related to body mass and shape as they transition between phases of the triathlon season. Or it could also be related to physiological factors such as exercise intensity. This may explain the continued subclinical EA evident throughout the season. Study 7 also highlights other factors beyond that of eating attitudes may contribute to the development of subclinical and clinical LEA that may be inadvertent (Nattiv, et al., 2007; Mountjoy, et al., 2014; Melin, et al., 2015). Importantly, study 7 highlights the difficulties associated with recruiting large sample sizes for longitudinal studies, the methodological limitations associated with measured EA, and the implications for under-reporting of EI, DE/ED symptoms, and training duration. Study 7 also highlighted that data derived from both the LEAF-Q and FAST should be interpreted with caution and further validation and reliability

studies are required with regards to false positives and negatives. Study 7 highlighted that the LEAF-Q may over-estimate LEA in comparison to actual measures of LEA.

8.4 Implications for research and clinical practice

- Current evidence suggests that female triathletes, across all ages and performance levels, should be screened for LEA to facilitate the prevention or early detection of Triad/RED-S. LEA is a significant area of concern to female triathletes as it may result in acute and chronic health problems (i.e., cardiovascular, immunological, gastrointestinal), negative impact on bone health by reducing BMD which may lead to increased risk of stress fracture or osteoporosis, increased risk of menstrual dysfunction leading to fertility issues and negatively impact on bone health, increased risk of psychological harm (i.e., DE/ED, EXD, anxiety and depression), metabolic disturbances which may cause acute and chronic issues with body composition, and there is potential for acute and chronic negative impacts on training response and/or capacity. Risk factors for elite athletic populations are likely different to non-elite athletes, however, the risk of developing LEA and the health and performance consequences of LEA to both groups of athletes are the same. However, it is fair to assume that most non-elite triathletes have less access to training and nutritional education and sports-specific medical personnel who have a degree of understanding of the signs and symptoms of LEA. Therefore, raising awareness of the risk and screening non-elite populations is warranted.
- In female triathletes, LEA, DE/ED, and EXD can occur together or in isolation. Identification of one requires the assessment of the others. It is important to screen for both current and historical LEA, DE/ED, and EXD to further understand the implications to past and current health status. It is important to consider the differences in aetiological factors and symptomology of LEA, DE/ED, and EXD across different age groups and performance levels when developing treatment strategies and educational resources. Athletes going

through key transitional periods, such as puberty or menopause, may be at increased risk and should be provided with extra support to transition in a health and safe manner. LEA is difficult to assess, particularly in field settings as evidenced in study 4, and often a high index of suspicion is required for the individual at risk. In the screening of LEA, a balance is required so that information regarding the health of the individual is acquired whilst reducing the risk of causing harm, distress, or burden to the individual (i.e., individuals becoming more aware of eating behaviours or body composition). Based on evidence from the current body of work, it is proposed that individuals are screened annually as part of an overarching health examination, on commencement of the season or if an individual presents with any significant risk factors (e.g., DE/ED, menstrual dysfunction, recurrent injury/illness etc.,) at any time point across the season. Single, direct assessments of EA may not be reflection of overall training and nutritional practices, may not reflect acute or chronic EA, and often have low validity of self-reported EI/EEE measures. The current body of work agrees that current tools and measures available for the assessment of EA are insufficient in conducting an accurate assessment of EA in free-living athletes and therefore it is irresponsible to make a universal recommendation to female triathletes to measure EA. It is suggested that current screening tools, such as the LEAF-Q, are used as a means to screen for individuals potentially at risk of developing LEA and to consult guidance). published by the IOC (Mountjoy, et al., 2014; 2018). However, as highlighted in the current body of work the current version of the LEAF-Q excludes a large percentage of females from being screened for LEA due to the sensitivity of items (e.g., pregnancy, menopause, chronic illness etc.,). For the assessment of DE/ED, the gold standard measure EDE-16 interview is advised.

- Screening for risk of LEA not only increases awareness of the prevalence of risk in athletic populations but is the first step towards diagnosis and treatment. Annual screening with a

self-report questionnaire is recommended, and where individuals are identified at risk of LEA and/or DE/ED, a more in-depth clinical evaluation should follow (De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). Screening should include: a full medical history of menstrual health, medication, stress fractures, critical comments relating to body composition or eating behaviours, psychological and personality factors such as, depression or perfectionism, dieting or pressure to lose body mass, overtraining, and recurrent or non-healing injuries. A more in-depth clinical examination may include assessment of BMI, body composition, physical signs of DE/ED (i.e., Russell's sign, orthostatic hypotension), pelvic examination to exclude other gynecological pathologies, exclusion of other health issues, psychological assessment, and assessment of bone health via DXA. In-depth clinical evaluation should be conducted by a multi-disciplinary team that may include a General Practitioner, sports dietician, exercise physiologist and/or mental health professional. This pathway of screening and diagnosis is currently advocated by the ACSM and IOC (De Souza, et al., 2014; Mountjoy, et al., 2014; 2018). It is also suggested to utilise the RED-S clinical assessment tool (Mountjoy, et al., 2014; 2018) for guidance on clinical assessment and return to play. Following clinical assessment, individuals are classified into red-light, amber-light, and green-light categories based on current health status (figure 8.1). Individuals categorised as high risk in the red-light category are not cleared to participate in sport, moderate risk yellow light individuals are cleared for supervised participation with a medical treatment plan with regular re-assessment, and low risk green light individuals can engage in full sports participation (Mountjoy, et al., 2014; 2018).

Table 1 Relative Energy Deficiency in Sport risk assessment model for sport participation (modified from Skårderud *et al*)[140]

High risk: no start red light	Moderate risk: caution yellow light	Low risk: green light
▸ Anorexia nervosa and other serious eating disorders ▸ Other serious medical (psychological and physiological) conditions related to low energy availability ▸ Extreme weight loss techniques leading to dehydration induced haemodynamic instability and other life-threatening conditions	▸ Prolonged abnormally low % body fat measured by DXA or anthropometry using The International Society for the Advancement of Kinanthropometry ISAK[141] or non-ISAK approaches[142] ▸ Substantial weight loss (5–10% body mass in 1 month) ▸ Attenuation of expected growth and development in adolescent athlete	▸ Healthy eating habits with appropriate energy availability
	▸ Abnormal menstrual cycle: FHA amenorrhoea >6 months ▸ Menarche >16 years ▸ Abnormal hormonal profile in men	▸ Normal hormonal and metabolic function
	▸ Reduced BMD (either from last measurement or Z-score < −1 SD). ▸ History of 1 or more stress fractures associated with hormonal/menstrual dysfunction and/or low EA	▸ Healthy BMD as expected for sport, age and ethnicity ▸ Healthy musculoskeletal system
	▸ Athletes with physical/psychological complications related to low EA/ disordered eating - ECG abnormalities- Laboratory abnormalities ▸ Prolonged relative energy deficiency ▸ Disordered eating behaviour negatively affecting other team members ▸ Lack of progress in treatment and/or non-compliance	

BMD, bone mineral density; DXA, dual-energy X-ray absorptiometry; EA, energy availability; FHA, functional hypothalamic amenorrhoea; ISAK, International Society for the Advancement of Kinanthropometry

Figure 8.1. RED-S return to play guidance from Mountjoy, et al., (2014).

8.5 Future work

- The work described in this thesis examined the prevalence of risk for LEA in competitive female triathletes. Future research should account for the differing demands associated with the different triathlon distances (i.e., sprint, standard, half ironman, and ironman) and to examine the potential differences in the prevalence of risk for LEA and associated risk factors (i.e., DE/ED and EXD).
- The current work described in this thesis focused on the prevalence of risk for LEA. Future work in competitive female triathletes would be advised to explore the health and performance impairments associated with LEA (i.e., menstrual dysfunction, bone health, endothelial function etc.,) and the influence of menstrual cycle phase.
- Study 2 identified the prevalence of LEA was greater in younger competitive triathletes than older and acknowledged the long-term consequences of developing LEA during adolescence and young adulthood. To date limited research exists with junior female triathletes. Future research should explore the prevalence of risk for LEA, DE/ED and EXD in this population. Additionally, it may be beneficial to explore associated impairments to health and to monitor changes over time.

- Similarly, study 2 identified the risk of developing LEA and associated risk factors does not disappear as athletes age. Future research should explore the prevalence of risk for LEA, DE/ED and EXD in master female triathletes. Additionally, future research needs to explore associations between perimenopause, menopause, and post-menopause and LEA, DE/ED, and EXD risk and associated health and performance impairments.
- Throughout studies 1-4 it was acknowledged that the risk of LEA may occur inadvertently due to inadequate awareness and knowledge related to nutrition, training, LEA, Triad and/or RED-S. Future work would be advised to explore these concepts in athletes, coaches, parents, and health professionals.

8.6 Limitations

Accordingly, limitations applicable to the individual studies have previously been detailed in studies 1-4. General limitations that comprise all studies within the present thesis are discussed as follows. Overall, sample size for the cross-sectional studies presented in studies 1-3 met the calculated sample size estimation of n=370 and falls within the sample size previously reported in LEA prevalence studies (range 10 to 833; Schaal, et al., 2011a; Logue et al., 2019 – table 2.2). Larger studies are required to enable greater statistical power to confirm current observations. However, studies 1-3 highlighted the current version of the LEAF-Q is not inclusive of all females which leads to a large percentage of females being excluded from screening (N=303 studies 1-3). This is due to the increased risk of false positives due to the sensitivity of items for individuals who may have chronic illness, pregnant, menopausal or use oral contraceptives etc. This also leads to a significant loss in statistical powers and bias as not all females are eligible for screening using the LEAF-Q. Future developments in current or new screening tools are needed to include all females and fully elucidate prevalence. The same is applicable to study 4, sample size was n = 10 which limited statistical power; however, this is not unusual for longitudinal studies of this nature due to logistics, financial and time

constraints to participant and researcher. Across studies 1-4 where questionnaires were utilised, data is self-reported and dependent on the honesty of completion, retrospective memory and understanding of the questions. Participants may have found some questions of a sensitive nature and thus modified their response, resulting in an underestimation of prevalence. However, honesty was encouraged by ensuring anonymity. The 147 incomplete questionnaires excluded from analysis may have been incomplete due to time constraints or due to the sensitivity of some questions or the length of the questionnaire. Although the study excluded post-menopausal women there was no screening for perimenopausal women which may have resulted in overestimation of prevalence with regards to the menstrual function component of the LEAF-Q.

Within this thesis female athletes were studied exclusively for consistency throughout. It was evident from study 1 that few studies have investigated female athletes and controls when investigating the prevalence of risk for LEA. This has led to difficulties in defining the magnitude of the problem within the athletic population and warrants more research to define the scope of the problem in non-athletes in comparison to athletic populations. Additionally, it was evident from reviewing the literature that few studies have investigated the scope of the problem with male athletes, particularly in triathlon where male participation rates are high. The current RED-S model has acknowledged that male athletes from leanness sports are a population at risk of LEA (Mountjoy, et al., 2014, 2018), however due to difficulties in comparing males and females the current study focused on females. This does not neglect the fact that a paucity of literature exists in males and is a population that warrants much more research.

8.7 Conclusion

Through a series of studies, this thesis has provided further knowledge concerning LEA risk in female triathletes. 1) A significant proportion of female triathletes are considered at risk of LEA and associated risk factors of DE/ED, and EXD. 2) Eating attitudes and exercise behaviour are significant predictors of LEA and 3) exercise behaviour is a significant predictor of eating attitudes in female triathletes. 4) The prevalence of risk of LEA, DE/ED, and EXD was greater in younger female triathletes compared with their older counterparts. However, 5) the risk still exists in significant proportions in older female triathletes. 6) Significant associations existed between age and EXD with younger athletes more likely to be symptomatic for EXD. 7) The prevalence of risk of LEA, DE/ED, and EXD tended to be greater in female triathletes identifying as top-percentile age-groupers than recreational. 8) Significant associations existed between performance level and EXD. 9) No significant differences were detected in EA or eating attitudes across the triathlon season and, 10) the prevalence of subclinical LEA was high across the duration of the season. Together, the findings from this thesis have advanced our understanding of the prevalence of risk and the influence of age, performance level, and season changes on LEA and associated risk factors in female triathletes.

Ackerman, K.E., Cano Sokoloff, N., & De Nardo Maffazioli, G., et al., (2015). Fractures in relation to menstrual status and bone parameters in young athletes. *Medicine & Science in Sports & Exercise*, *47*(8), pp. 1577–86.

Ackerman, K.E., Holtzman, B., & Cooper, K.M., et al., (2019). Low energy availability surrogates correlate with health and performance consequences of relative energy deficiency in sport. *British Journal of Sports Medicine*, 53, pp. 628–33.

Ackerman, K.E., Nazem, T., Chapko, D., Russell, M., Mendes, N., & Taylor, A.P., et al., (2011). Bone microarchitecture is impaired in adolescent amenorrheic athletes compared with eumenorrheic athletes and nonathletic controls. *Journal of Clinical Endocrinology & Metabolism*, *96*(10), pp. 3123–33.

Ackerman, K.E., Putman, M., Guereca, G., Taylor, A.P., Pierce, L., & Herzog, D.B., et al., (2012). Cortical microstructure and estimated bone strength in young amenorrheic athletes, eumenorrheic athletes and non-athletes. *Bone*, *51*(4), pp. 680–7.

Ackerman, K.E., Stellingwerff, T., Elliot-Sale, K.J., Baltzell, A., Cain, M., Goucher, K., Fleshman, L., & Mountjoy, M.L., (2020). REDS (Relative Energy Deficiency in Sport): time for a revolution in sports culture and systems to improve athlete health and performance. *British Journal of Sports Medicine*, 0, pp. 1-2.

Adams, J., & Kirkby, R.J. (2002). Excessive exercise as an addiction: A review. *Addiction Research and Theory*, *10*(5), pp. 415–38.

Adams, J., (2009). Understanding exercise dependence. *Journal of Contemporary Psychotherapy*, *39*(4), pp. 231.

Affenito, S.G., Yeager, K.A., Rosman, J.L., Ludemann, M.A., Adams, C.H., & Welch, G.W., (1998). Development and validation of a screening tool to identify eating disorders in the female athlete. *Journal of the American Dietetic Association*, *98*(9), pp A78.

Aidman, E.V., & Woollard, S., (2003). The influence of self-reported exercise addiction on acute emotional and physiological responses to brief exercise deprivation. *Psychology of Sport and Exercise*, *4*(3), pp. 225-36.

Ainsworth, B. E., Haskell, W. L., Herrmann, S. D., Meckes, N., Bassett, D. R. Jr., Tudor-Locke, C., & Leon, A. S., (2011). Compendium of physical activities: A second update of codes and MET values. *Medicine & Science in Sports & Exercise*, *43*, pp. 1575–81.

Allegre, B., Souville, M., Therme, P., & Griffiths, M., (2006). Definitions and measures of exercise dependence. *Addiction Research and Theory*, 14(6), pp. 631–646.

Allen, A.M., McRae-Clark, A.L., Carlson, S., Saladin, M.E., Gray, K.M., et al., (2016). Determining menstrual cycle phases in human bio-behavioural research: A review with recommendations. *Experimental & Clinical Psychopharmacology*, *24*(1), 1-11.

American Psychiatric Association, (2013). *Diagnostic and Statistical Manual of Mental Disorders (*5th ed.). Arlington, VA.

American Psychiatric Association., (2000). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington, DC: Author.

American Society for Reproductive Medicine Practice Committee, (2006). Current evaluation of amenorrhea. *Fertility and Sterility*, *86*(5), pp. S148-S155.

American Society of Reproductive Medicine Practice Committee, (2004). Current evaluation of amenorrhea. *Fertil. Sterility*, *82*, pp. 266–72.

Amorim, T., Koutedakis, Y., Nevill, A., Wyon, M., Maia, J., & Machado, J., et al., (2017). Bone mineral density in vocational and professional ballet dancers. *Osteoporosis International*, *28*, pp. 2903–12.

Anthony, D., Rüst, C.A., Cribari, M., Rosemann, T., Lepers, R., & Knechtle, B., (2014). Differences in participation and performance trends in age group half and full marathoners. *Chinese Journal of Physiology*, 57, pp. 209-219.

Arcelus, J., Mitchell, A.J., & Wales, J., et al., (2011). Mortality rates in patients with anorexia nervosa and other eating disorders. A meta-analysis of 36 studies. *Archives of General Psychiatry*, *68*, pp. 724–31.

Areta, J.L., Taylor, H.L., & Koehler, K., (2020). Low energy availability: history, definition and evidence of its endocrine, metabolic and physiological effects in prospective studies in females and males. *European Journal of Applied Physiology*, pp.1-21.

Arthur-Cameselle, J.N., Sossin, K., & Quatromoni, P.A., (2017). A qualitative analysis of factors related to eating disorder onset in female collegiate athletes and non-athletes. *Eating Disorders*, *25*(3), pp. 199–215.

Australian Institute of Sport, Tanner, R., & Gore, C., (2013). *Physiological tests for elite athletes.* Human Kinetics.

Bachmann, G.A., & Kemmann, E., (1982). Prevalence of oligomenorrhea and amenorrhea in a college population. *American Journal of Obstetrics and Gynecology*, *144*(1), pp. 98–102.

Back, J., Josefsson, T., Ivarsson, A., & Gustafsson, H., (2019). Psychological risk factors for exercise dependence. *International Journal of Sport and Exercise Psychology*, pp.1-12.

Baekeland, F., (1970). Exercise deprivation: sleep and psychological reactions. *Archives of General Psychiatry*, 22, pp. 365–9.

Bahr, R., (2016). Why screening tests to predict injury do not work – and probably never will: a critical review. *British Journal of Sports Medicine* ¸50 (13), pp. 776-780.

Bailey, C.A., & Brooke-Wavell, K., (2008). Exercise for optimising peak bone mass in women. *Proceedings of the Nutrition Society*, *67*(1), pp. 9-18.

Bailey, D.A., McKay, H.A., & Mirwald, R.L., *et al.*, (1999). A six-year longitudinal study of the relationship of physical activity to bone mineral accrual in growing children: the University of Saskatchewan bone mineral accrual study. *Journal of Bone & Mineral Research*, *14*, pp. 1672–79.

Balasch, J., & Vanrell, J.A., (1987). Corpus luteum insufficiency and infertility, a matter of controversy. *Human Reproduction*, *2*, pp. 557–67.

Bamber, D.J., Cockerill, I.M., & Carroll, D., (2000). The pathological status of exercise dependence. *British Journal of Sports Medicine*, *34*(2), pp. 125–32.

Bamber, D.J., Cockerill, I.M., Rodgers, S., & Carroll, D., (2003). Diagnostic criteria for exercise dependence in women. *British Journal of Sports Medicine*, *37*(5), pp. 393.

Bardone-Cone, A.M., Wonderlich, S.A., Frost, R.O., Bulik, C.M., Mitchell, J.E., & Uppala S., et al., (2007). Perfectionism and eating disorders: current status and future directions. *Clinical Psychology Review*, *27*(3), pp. 384–405.

Barrack, M.T., Gibbs, J.C., De Souza, M.J., Williams, N.I., Nichols, J.F., & Rauh, M.J., et al., (2014). Higher incidence of bone stress injuries with increasing female athlete Triad-related risk factors: a prospective multisite study of exercising girls and women. *American Journal of Sports Medicine*, *42*(4), pp. 949-58.

Barrow, G.W., & Saha, S., (1988). Menstrual irregularity and stress fractures in collegiate female distance runners. *American Journal of Sports Medicine*, *16*(3), pp. 209-16.

Batterham, M.J., Tapsell, L.C., & Jenkins, A.B., (2002). A comparison of bioelectrical impedance and near infrared interactance with dual energy x-ray absorptiometry for the determination of body fat. *Nutrition & Dietetics*,59, pp. 120–126.

Baxter-Jones, A.D., Faulkner, R.A., & Forwood, M.R., et al., (2011). Bone mineral accrual from 8 to 30 years of age: an estimation of peak bone mass. *Journal of Bone & Mineral Research, 26*, pp. 1729Y39.

Beals, K.A., & Hill, A.K., (2006). The prevalence of disordered eating, menstrual dysfunction, and low bone mineral density among US collegiate athletes. *International journal of sport nutrition and exercise metabolism, 16*(1), pp. 1-23.

Beals, K.A., & Manore, M.M., (2000). Behavioral, psychological, and physical characteristics of female athletes with subclinical eating disorders. *International Journal of Sport Nutrition & Exercise Metabolism, 10*(2), pp. 128–43.

Beals, KA, & Manore, M.M., (2002). Disorders of the female athlete triad among collegiate athletes. *International Journal of Sport Nutrition & Exercise Metabolism, 12*, pp. 281–93.

Beltz, N.M., Gibson, A.L., Janot, J.M., Kravitz, L., Mermier, C.M., & Dalleck, L.C., (2016). Graded exercise testing protocols for the determination of VO2max: historical perspectives, progress, and future considerations. *Journal of Sports Medicine, 2016.*

Bennell, K.L., Malcolm, S.A., Thomas, S.A., Reid, S.J., Brukner, P.D., & Ebeling, P.R., et al., (1996). Risk factors for stress fractures in track and field athletes. A twelve-month prospective study. *American Journal of Sports Medicine, 24(6), pp. 810-18.*

Bennett, H., Parfitt, G., Davison, K., & Eston, R., (2016). Validity of submaximal step tests to estimate maximal oxygen uptake in healthy adults. *Sports Medicine, 46(*5), pp.737-750.

Bentley, D.J., Millet, G.P., Vleck, V.E., & MacNaughton, L.R., (2002). Specific aspects of contemporary triathlon. *Sports Medicine, 32*, pp. 1-15.

Berczik, K., Szabó, A., Griffiths, M.D, Kurimay, T., Kun, B., Urbán, R., & Demetrovics, Z., (2012). Exercise Addiction: Symptoms, Diagnosis, Epidemiology, and Etiology. *Substance Use & Misuse, 47(*4), pp. 403–417.

Black, A.E., (2000). Critical evaluation of energy intake using the Goldberg cut-off for energy intake: basal metabolic rate. A practical guide to its calculation, use and limitations. *International Journal of Obesity & Related Metabolic Disorders, 24*(9), pp. 1119–30.

Black, D.R., Larkin, D.C., Coster, D.C., Leverenz, L.J., & Abood, D.A., (2003). Physiologic screening test for eating disorders/disordered eating among female collegiate athletes. *Journal of Athletic Training, 38*(4), pp. 286–97.

Black, K., Slater, J., Brown, R.C., & Cooke, R., (2018). Low energy availability, plasma lipids, and hormonal profiles of recreational athletes. *Journal of Strength & Conditioning Research, 32*(10), pp. 2816-24.

Black, R.E., Victora, C.G., & Walker, S.P.S, (2013). Maternal and child under nutrition and overweight in low-income and middle-income countries. *Lancet*, 382, pp. 427–451.

Blaydon, M. J., Lindner, K. J., & Kerr, J. H., (2002). Metamotivational characteristics of eating-disordered and exercise-dependent triathletes: An application of reversal theory. *Psychology of Sport & Exercise, 3*(3), pp. 223–36.

Bonci, C.M., Bonci, L.J., & Granger, L.R, et al., (2008). National athletic trainers' association position statement: preventing, detecting, and managing disordered eating in athletes. *Journal of Athletic Training*, 43, pp. 80–108.

Bonjour, J.P., & Rizzoli, R., (2001). Bone acquisition in adolescence. In *Osteoporosis*, vol I, end 2. Edited by Marcus R, Kelsey J, Feldman D. San Diego: Academic Press, pp. 621 – 38.

Boreham, C.A., & McKay, H.A., (2011). Physical activity in childhood and bone health. *British Journal of Sports Medicine, 45*, pp. 877Y9.

Borg, G., (1970). Perceived exertion as an indicator of somatic stress. *Scandinavian Journal of Rehabilitation Medicine, 2*(2), pp. 92-8.

Borresen, J., & Lambert, M., (2006). Validity of self-reported training duration. *International Journal of Sports Science & Coaching*, *1*(4), pp.353-359.

Boudin, E., & Van Hul, W., (2017). Genetics of human bone formation. *European Journal of Endocrinology*, *177*(2), pp. R69-83.

Braakhuis, A.J., Meredith, K., Cox, G.R., Hopkins, W.G., & Burke, L.M., (2003). Variability in estimation of self-reported dietary intake data from elite athletes resulting from coding by different sports dietitians. *International Journal of Sports Nutrition & Exercise Metabolism*, *13*(2), pp. 152–65.

Brage, S., Brage, N., Franks, P.W., Ekelund, U., & Wong, M.Y., et al., (2004) Branched equation modeling of simultaneous accelerometry and heart rate monitoring improves estimate of directly measured physical activity energy expenditure. *Journal of Applied Physiology*, *96*, pp. 343–51.

Brage, S., Westgate, K., Franks, P.W., Stegle, O., Wright, A., & Ekelund, U., et al., (2015). Estimation of Free-Living Energy Expenditure by Heart Rate and Movement Sensing: A Doubly-Labelled Water Study. *PLOS ONE*,10, pp. e0137206.

Bratland-Sanda, S., & Sundgot-Borgen, J., (2013). Eating disorders in athletes: overview of prevalence, risk factors and recommendations for prevention and treatment. *European Journal of Sport Science, 13*(5), pp. 499-508.

Bratland-Sanda, S., Martinsen, E.W., & Rosenvinge, J.H., et al., (2011). Exercise dependence score in patients with longstanding eating disorders and controls: the importance of affect regulation and physical activity intensity. *European Eating Disorders Review*, *19*, pp. 249–55.

Bratland-Sanda, S., Mathisen, T.F., Sundgot-Borgen, J., & Rosenvinge, J.H., (2019). Defining compulsive exercise in eating disorders: acknowledging the exercise paradox and exercise obsessions. *Journal of Eating Disorders*, *7*(1), pp. 1-3.

Brehm, B.J., & Steffen, J.J., (1998). Relation between obligatory exercise and eating disorders. *American Journal of Health Behaviour, 22*(2), pp. 108-19.

Brisswalter, J., Wu, S.S.X., Sultana, F., Bernard, T., & Abbiss, C.R., (2014). Age difference in efficiency of locomotion and maximal power output in well-trained triathletes. *European Journal of Applied Physiology*, 114, pp. 2579-86.

British Triathlon (2020). https://www.britishtriathlon.org/about-us/faqs?category=age-group. Accessed on: 18.08.2020.

British Triathlon, (2021a): https://www.britishtriathlon.org/media/statistics. (Accessed on: 23.02.2021).

British Triathlon, (2021b). https://www.britishtriathlon.org/about-us/governance/equality-diversity-inclusion/gender. (Accessed on: 23.02.2021).

Bronson, F.H., (1985). Mammalian reproduction: an ecological perspective. *Biology of Reproduction*, 32, pp. 1–26.

Bronson, F.H., (1989). *Mammalian reproductive biology*. University of Chicago Press, Chicago.

Brown, I.A., (1997). Theoretical model of the behavioural addictions – applied to offending. *In: Hodge JE, McMurran M, Hollin RC, editors. Addicted to Crime. 1st ed. Chichester, West Sussex, England: Wiley*, pp. 13–65.

Brown, M.A., Howatson, G., Quin, E., Redding, E., & Stevenson, E.J., (2017). Energy intake and energy expenditure of pre-professional female contemporary dancers. *PLOS ONE, 12*(2), pp. e0171998.

Buckley, G.L., Hall, L.E., & Lassemillante, A.C.M., et al., (2019). Retired athletes and the intersection of food and body: a systematic literature review exploring compensatory behaviours and body change. *Nutrients,* 11, pp. 1395.

Burke, L.M., Castell, L.M., Casa, D.J., & Close, G.L., et al., (2019). International Association of Athletics Federations Consensus Statement 2019: nutrition for athletics. *International Journal of Sports Nutrition & Exercise Metabolism*, 29(2), pp. 73–84.

Burke, L.M., Cox, G.R., Cummings, N.K., & Desbrow, B., (2001). Guidelines for daily carbohydrate intake: Do athletes achieve them? *Sports Medicine*, *31*(4), pp. 267–299.

Burke, L.M., Lundy, B., & Fahrenholtz, I.L., et al., (2018). Pitfalls of conducting and interpreting estimates of energy availability in free- living athletes. *International Journal of Sports Nutrition & Exercise Metabolism*, 28, pp. 350–63.

Burrows, T.L., Ho, Y.Y., Rollo, M.E., & Collins, C.E., (2019). Validity of dietary assessment methods when compared to the method of doubly labeled water: a systematic review in adults. *Frontiers in Endocrinology*, *10*, pp. 850.

Byrne, S., & Mclean, N., (2001). Eating disorders in athletes: a review of the literature. *Journal of Science & Medicine in Sport*, *4*, pp. 145–59.

Byrne, S., & McLean, N., (2002). Elite athletes: effects of the pressure to be thin. *Journal of Science & Medicine in Sport*, *5*(2), pp. 80-94.

Campa, F., Toselli, S., Mazzilli, M., Gobbo, L.A., & Coratella, G., (2021). Assessment of Body Composition in Athletes: A Narrative Review of Available Methods with Special Reference to Quantitative and Qualitative Bioimpedance Analysis. *Nutrients*, 13, pp. 1620-45.

Canda, A.S., Castiblanco, L.A., Toro, A.N., Amestoy, J.A., & Higueras, S., (2014). Morphological characteristics of the triathlete according to sex, category and competitive level. *Apunts Sports Medicine*, 49, pp. 75-84.

Capling, L., Beck, K.L., Gifford, J.A., Slater, G., Flood, V.M., & O'Connor, H., (2017). Validity of dietary assessment in athletes: A systematic review. *Nutrients, 9*(12), pp. 1313.

Casadevall, A., & Fang, F.C., (2016). Rigorous Science: a How-To Guide. MBio, *7*(6), e01902-16.

Casadevall, A., & Fang, F.C., (2018). Making the scientific literature fail-safe. Journal of Clinical. Investigation, *128*(10), pp. 4243–4.

Çetin, E., Bulğay, C., Demir, G.T., Cicioğlu, H.İ., Bayraktar, I. and Orhan, Ö., (2020). The Examination of the Relationship Between Exercise Addiction and Performance Enhancement in Elite Athletes. *International Journal of Mental Health & Addiction*, pp.1-12.

Chapa, D.A.N., Hagan, K.E., & Forbush, K.T., et al., (2018). The athletes' relationships with training scale (art): a self- report measure of unhealthy training behaviors associated with eating disorders. *International Journal of Eating Disorders*, *51*, pp. 1080–9.

Chapman, C.L., De Castro, J.M., (1990). Running addiction: measurement and associated psychological characteristics. *Journal of Sports Medicine & Physical Fitness*, *30*(3), pp. 283–290.

Chen, W.J., (2016). Frequent exercise: A healthy habit or a behavioral addiction? *Chronic Diseases & Translational Medicine, 2*(4), pp. 235-240.

Chesney, E., Goodwin, G.M., & Fazel, S., (2014). Risks of all- cause and suicide mortality in mental disorders: a meta- review. *World Psychiatry*, *13*, pp. 153–60.

Chin, E.L., Hoggatt, M., & McGregor, A.J., et al., (2016). Sex and Gender Medical Education Summit: a roadmap for curricular innovation. Biology of Sex Differences, *7*(1), pp. 52.

Civil, R., Lamb, A., Loosmore, D., Ross, L., Livingstone, K., Strachan, F., Dick, J.R., Stevenson, E.J., Brown, M.A., & Witard, O.C., (2019). Assessment of dietary intake, energy status, and factors associated with RED-S in vocational female ballet students. *Frontiers in Nutrition*, 5, p.136.

Cobb, K.L., Bachrach, L.K., Greendale, G., Marcus, R., Neer, R.M., Nieves, J., Sowers, M.F., Brown, B.W., Gopalakrishnan, G., & Leutters, C., et al., (2003). Disordered eating, menstrual irregularity and bone mineral density in female athletes. *Medicine & Science in Sports & Exercise*, *35*, pp. 711–19.

Cohen, J., (1988). *Statistical power analysis for the behavioural sciences.* 2nd Ed. Hillside, NJ: Lawrence Erlbaum Associates.

Colledge, F., Cody, R., Buchner, U.G., Schmidt, A., Puehse, U., Gerber, M., Wiesbeck, G., Lang, U.E. and Walter, M., 2020. Excessive exercise–a meta-review. *Frontiers in Psychiatry*, *11*, p.1288.

Combs, C.E., Nicholls, J.J, Duncan Bassett, J.H., & Williams, G.R., (2011). Thyroid hormones and bone development. *Minerva Endocrinology*, *36*(1), pp. 71–85.

Condo, D., Lohman, R., Kelly, M., & Carr, A., (2019). Nutritional intake, sports nutrition knowledge and energy availability in female Australian rules football players. *Nutrients*, 11 (5), pp.971.

Conn, P.M., & Crowley, W.F., (1994). Gonadotropin-releasing hormone and its analogs. *Annual Review of Medicine*, 45, pp. 391–405.

Cook, B., Hausenblas, H., & Freimuth, M., (2014). Exercise addiction and compulsive exercising: Relationship to eating disorders, substance use disorders, and addictive disorders. *In: Brewerton TD, Baker Dennis A, editors. Eating Disorders, Addictions and Substance Use Disorders. New York: Springer*, pp.127–144.

Cook, B.J., & Hausenblas, H.A., (2008). The role of exercise dependence for the relationship between exercise behavior and eating pathology mediator or moderator? *Journal of Health Psychology*, 13(4), pp. 495–502.

Cook, B.J., Wonderlich, S.A., & Mitchell, J.E., et al., (2016). Exercise in eating disorders treatment: systematic review and proposal of guidelines. *Medicine & Science in Sports & Exercise*, *48*, pp. 1408–14.

Cornish, J., Callon, K.E., Bava, U., Lin, C., Naot, D., & Hill, B.L., et al., (2002). Leptin directly regulates bone cell function in vitro and reduces bone fragility in vivo. *Journal of Endocrinology*, *175*(2), pp. 405–15.

Cortina, J.M., (1993). What is coefficient alpha? An examination of theory and applications. *Journal of Applied Psychology*, 78 (1), pp. 98–104.

Costa, S., Hausenblas, H. A., Oliva, P., Cuzzocrea, F., & Larcan, R., (2013). The role of age, gender, mood states and exercise frequency on exercise dependence. *Journal of Behavioural Addictions*, *2*(4), pp. 216– 223.

Cronbach, L. J., (1951). Coefficient alpha and the internal structure of tests. *Psychometrika*, 16, pp. 297-333.

Crotty, N.M., Boland, M., Mahony, N., Donne, B., & Fleming, N., (2021). Reliability and Validity of the Lactate Pro 2 Analyzer. *Measurement in Physical Education & Exercise Science*, pp.1-10.

Crow, S.J., Peterson, C.B., & Swanson, S.A., et al., (2009). Increased mortality in bulimia nervosa and other eating disorders. *American Journal of Psychiatry*, *166*, pp. 1342–6.

Culbert, K.M., Racine, S.E., & Klump, K.L., (2015). Research Review: What we have learned about the causes of eating disorders–a synthesis of sociocultural, psychological, and biological research. *Journal of Child Psychology & Psychiatry*, *56*(11), pp.1141-1164.

Cunningham, H.E., Pearman, S., & Brewerton, T.D., (2016). Conceptualizing primary and secondary pathological exercise using available measures of excessive exercise. *International Journal of Eating Disorders*, *49*(8), pp. 778–792.

Cunningham, J.J., (1980). A reanalysis of the factors influencing basal metabolic rate in normal adults. *American Journal of Clinical Nutrition*, *33*(11), pp. 2372-74.

Curry, E.J., Logan, C., Ackerman, K.E., McInnis, K.C., & Matzkin, E.G., (2015). Female athlete triad awareness among multi-specialty physicians. *Sports Medicine – Open*, *1*(1), pp.38.

Dahlgren, C.L., Wisting, L., & Rø, Ø., (2017). Feeding and eating disorders in the DSM-5 era: a systematic review of prevalence rates in non-clinical male and female samples. *Journal of Eating Disorders*, *5*(1), p.56.

Dale, E., Gerlack, D.H., & Wilhite, A.L., (1979). Menstrual dysfunction in distance runners. *Obstetrics Gynecology*, *54*, pp. 47–53.

Dalle Grave, R., Calugi, S., & Marchesini, G., (2008). Compulsive exercise to control shape or weight in eating disorders: prevalence, associated features, and treatment outcome. *Comprehensive Psychiatry*, *49*(4), pp. 346-52.

Das, J.K., Salam, R.A., Thornburg, K.L., Prentice, A.M., Campisi, S., Lassi, Z.S., Koletzko, B., & Bhutta, Z.A., (2017). Nutrition in adolescents: physiology, metabolism, and nutritional needs. *Annals of the New York Academy of Sciences*, 1393(1), pp.21-33.

De La Vega, R., Parastatidou, I.S., Ruíz-Barquín, R., & Szabo, A., (2016). Exercise addiction in athletes and leisure exercisers: The moderating role of passion. *Journal of Behavioral Addictions*, *5*(2), pp.325-331.

De Oliveira Coelho, G.M., de Farias, M.L.F., de Mendonça, L.M.C., de Mello, D.B., Lanzillotti, H.S., Ribeiro, B.G., & de Abreu Soares, E., (2013). The prevalence of disordered eating and possible health consequences in adolescent female tennis players from Rio de Janeiro, Brazil. *Appetite*, 64, pp.39-47.

De Souza, M.J., & Williams, N.I., (2004). Physiological aspects and clinical sequelae of energy deficiency and hypoestrogenism in exercising women. Human Reproduction. Update, *10*(5), pp. 433–48.

De Souza, M.J., (2003). Menstrual disturbances in athletes, A focus on luteal phase defects. *Medicine & Science in Sport & Exercise*, *35*, pp. 1553–63.

De Souza, M.J., Hontscharuk, R., Olmsted, M., Kerr, G., & Williams, N.I., (2007). Drive for thinness score is a proxy indicator of energy deficiency in exercising women. *Appetite*, *48*(3), pp. 359–67.

De Souza, M.J., Koltun, K.J., Etter, C.V., & Southmayd, E.A., (2017). Current status of the female athlete triad: update and future directions. *Current Osteoporosis Reports*, *15*(6), pp.577-87.

De Souza, M.J., Koltun, K.J., Strock, N.C., & Williams, N.I., (2019). Rethinking the concept of an energy availability threshold and its role in the female athlete triad. *Current Opinion in Physiology*, *10*, pp. 35-42.

De Souza, M.J., Miller, B.E., & Loucks, A.B., et al., (1998). High frequency of luteal phase deficiency and anovulation in recreational women runners: blunted elevation in follicle-stimulating hormone observed during luteal-follicular transition. *Journal of Clinical Endocrinology & Metabolism*, 83, pp. 4220–32.

De Souza, M.J., Nattiv, A., Joy, E., Misra, M., Williams, N.I., Mallinson, R.J., Gibbs, J.C., Olmsted, M., Goolsby, M., & Matheson, G., (2014). 2014 Female Athlete Triad Coalition Consensus Statement

on treatment and return to play of the female athlete triad: 1st International Conference held in San Francisco, California, May 2012 and 2nd International Conference held in Indianapolis, Indiana, May 2013. *British Journal of Sports Medicine*, 48(4), pp. 289-298.

De Souza, M.J., Toombs, R.J., & Scheid, J.L., et al., (2010). High prevalence of subtle and severe menstrual disturbances in exercising women: confirmation using daily hormone measures. Human Reproduction, *25*(2), pp. 491–503.

De Souza, M.J., West, S.L., Jamal, S.A., Hawker, G.A., Gundberg, C.M., & Williams, N.I., (2008). The presence of both an energy deficiency and estrogen deficiency exacerbate alterations of bone metabolism in exercising women. *Bone*, *43*(1), pp. 140–8.

De Souza, M.J., Williams, N.I., & Nattiv, A., et al., (2014). Misunderstanding the female athlete triad: refuting the IOC consensus statement on Relative Energy Deficiency in Sport (RED-S). British Journal of Sports Medicine, *48*(20), pp. 1461–5.

Delaisse, J.M., (2014). The reversal phase of the bone-remodeling cycle: cellular prerequisites for coupling resorption and formation. *Bone Key Reports*, 3.

Desbrow, B., Burd, N.A., Tarnopolsky, M., Moore, D.R., & Elliott-Sale, K.J., (2019). Nutrition for special populations: Young, female, and master's athletes. *International Journal of Sport Nutrition & Exercise Metabolism*, 29(2), pp.220-227.

Desbrow, B., McCormack, J., Burke, L.M., Cox, G.R., Fallon, K., Hislop, M., Logan, R., Marino, N., Sawyer, S.M., Shaw, G., & Star, A., (2014). Sports Dietitians Australia position statement: sports nutrition for the adolescent athlete. *International Journal of Sport Nutrition & Exercise Metabolism*, 24 (5), pp. 570-584.

Di Lodovico, L., Poulnais, S., & Gorwood, P., (2019). Which sports are more at risk of physical exercise addiction: a systematic review. *Addictive Behaviors, 93*, pp. 257-262.

DiPietro, L., & Stachenfeld, N.S., (2006). The myth of the female athlete triad. British Journal of Sports Medicine, *40*(6), pp. 490–3.

Dittmer, N., Jacobi, C., & Voderholzer, U., (2018). Compulsive exercise in eating disorders: Proposal for a definition and a clinical assessment. *Journal of Eating Disorders*, 6, pp. 42.

Dolan, E., McGoldrick, A., Davenport, C., Kelleher, G., Byrne, B., & Tormey, W., et al., (2012). An altered hormonal profile and elevated rate of bone loss are associated with low bone mass in professional horse-racing jockeys. *Journal of Bone & Mineral Metabolism, 30*(5), pp. 534–42.

Doughty, J., & Hausenblas, H., (2005). A longitudinal examination of disordered eating correlates in collegiate gymnasts. *Women in Sport & Physical Activity Journal*, 14(1), pp. 52-61.
Downs, D.S., Hausenblas, H.A., &Nigg, C.R., (2004). Factorial validity and psychometric examination of the Exercise Dependence Scale-Revised. *Measurement in Physical Education & Exercise Science*, *8*(4), pp. 183-201.

Doyle-Lucas, A.F., Akers, J.D., & Davy, B.M., (2010). Energetic efficiency, menstrual irregularity, and bone mineral density in elite professional female ballet dancers. *Journal of Dance Medicine & Science*, *14*(4), pp. 146-154.

Drew, F.L, (1961). The epidemiology of secondary amenorrhea. *Journal of Chronic Diseases*, *14*, pp. 396–401.

Drew, M.K., Vlahovich, N., Hughes, D., & Appaneal, R., et al. (2017). A multifactorial evaluation of illness risk factors in athletes preparing for the Summer Olympic Games. *Journal of Science & Medicine in Sport*, 20, pp. 745–750.

Drinkwater, B.L., Bruemner, B., & Chesnut, C.H., (1990). Menstrual History as a Determinant of Current Bone Density in Young Athletes. *JAMA: Journal of the American Medical Association*, *263*(4), pp. 545

Drinkwater, B.L., Nilson, K., Chesnut, C.H., Bremner, W.J., Shainholtz, S., & Southworth, M.B., (1984). Bone mineral content of amenorrheic and eumenorrheic athletes. *New England Journal of Medicine, 311*(5), pp. 277– 81.

Drinkwater, B.L., Nilson, K., Ott, S., & Chesnut, C.H., (1986). Bone Mineral Density After Resumption of Menses in Amenorrheic Athletes. *JAMA: Journal of the American Medical Association, 256*(3), pp. 380.

Duckham, R.L., Peirce, N., Meyer, C., Summers, G.D., Cameron, N., & Brooke-Wavell, K., (2012). Risk factors for stress fracture in female endurance athletes: a cross-sectional study. *British Medical Journal Open, 2*(6).

Edmunds, J., Ntoumanis, N., & Duda, J.L., (2006). Examining exercise dependence symptomatology from a self-determination perspective. *Journal of Health Psychology, 11*, pp. 887–903.

Edvardsen, E., Hem, E., & Anderssen, S.A., (2014). End criteria for reaching maximal oxygen uptake must be strict and adjusted to sex and age: a cross-sectional study. *PLOS ONE,* 9 (1), p.e85276.

Egorov, A.Y., & Szabo, A., (2013). The exercise paradox: An interactional model for a clearer conceptualization of exercise addiction. *Journal of Behavioral Addictions, 2*(4), pp. 199-208.

Elliott-Sale, K.J., Tenforde, A.S., Parziale, A.L., Holtzman, B., & Ackerman, K.E., (2018). Endocrine effects of relative energy deficiency in sport. *International Journal of Sport Nutrition & Exercise Metabolism, 28*(4), pp. 335-49.

epidemiological examples across the life course. Current Epidemiology Reports, *3*(2), pp. 161–80.

Etxebarria, N., Mujika, I., & Pyne, D.B., (2019). Training and competition readiness in triathlon. *Sports, 7*(5), pp. 101.

Fairburn, C.G., Cooper, Z., & O'Connor, M., (2014). Eating disorder examination, version 17. Oxford: CREDO.

Feicht, C.B., Johnson, T.S., Martin, B.J., Sparkes, K.E., & Wagner, W.W., (1978). Secondary amenorrhea in athletes. *Lancet, 2*, pp. 1145–46.

Folscher, L.L., Grant, C.C., Fletcher, L., & van Rensberg, D.C.J., (2015). Ultra-marathon athletes at risk for the female athlete triad. *Sports Medicine - Open, 1*(1), pp. 1-8.

Freimuth, M., Moniz, S., & Kim, S.R., (2011). Clarifying exercise addiction: Differential diagnosis, co-occurring disorders, and phases of addiction. *International Journal of Environmental Research & Public Health, 8*(10), pp. 4069–4081.

Fröhlich, M., Balter, J., Pieter, A., Schwarz, M., & Emrich, E., (2013). Model-theoretic optimization approach to triathlon performance under comparative static conditions–Results based on the Olympic Games 2012. *International Journal of Kinesiology & Sports Science*, 1(3).

Garner, D.M., (1991). Eating Disorder Inventory-2. Professional Manual. Odessa, FL: Psychological Assessment Research, Inc.

Garner, D.M., (2004). Eating Disorder Inventory-3. Professional Manual. Lutz, FL: Psychological Assessment Resources, Inc.

Garner, D.M., Olmsted, M.P., & Polivy, J., (1983). Development and validation of a multidimensional EDI for anorexia nervosa and bulimia. *International Journal of Eating Disorders, 2*, pp. 15–34.

Garner, D.M., Olmsted, M.P., Bohr, Y., & Garfinkel, P.E., (1982). The eating attitudes test: psychometric features and clinical correlates. *Psychological medicine, 12*(4), pp. 871-78.

Gibbs, J. C., Williams, N. I., Scheid, J. L., Toombs, R. J., & De Souza, M. J., (2011). The association of a high drive for thinness with energy deficiency and severe menstrual disturbances: confirmation in

a large population of exercising women. *International Journal of Sport Nutrition & Exercise Metabolism*, 21, pp. 280–290.

Gibbs, J.C., Williams, N.I., & De Souza, M.J, (2013). Prevalence of individual and combined components of the female athlete triad. Medicine & Science in Sports & Exercise,*45*(5), pp. 985–96.

Goldberg, G.R., Black, A.E., Jebb, S.A., Cole, T.J., Murgatroyd, P.R., Coward, W.A., & Prentice, A.M., (1991). Critical evaluation of energy intake data using fundamental principles of energy physiology: 1. Derivation of cut-off limits to identify under-recording. *European Journal of Clinical Nutrition*, *45*(12), pp. 569–81.

Goodman, A., (1990). Addiction: definition and implications. *British Journal of Addiction*, 85(11), pp. 1403–1408.

Goolsby, M.A., & Boniquit, N., (2017). Bone health in athletes: the role of exercise, nutrition, and hormones. *Sports Health*, *9*(2), pp. 108-17.

Goolsby, M.A., & Boniquit, N., (2017). Bone health in athletes: The role of exercise, nutrition, and hormones. *Sports Health*, *9*(2), pp. 108-117.

Gordon, C.M., Ackerman, K.E., Berga, S.L., Kaplan, J.R., Mastorakos, G., Misra, M., Murad, M.H., Santoro, N.F., & Warren, M.P., (2017a). Functional hypothalamic amenorrhea: an endocrine society clinical practice guideline. *Journal of Clinical Endocrinology & Metabolism*, *102*(5), pp. 1413-39.

Gordon, C.M., Zemel, B.S., Wren, T.A., Leonard, M.B., Bachrach, L.K., Rauch, F., Gilsanz, V., Rosen, C.J., & Winer, K.K., (2017b). The determinants of peak bone mass. *Journal of Pediatrics*, *180*, pp. 261-69.

Grandi, S., Clementi, C., Guidi, J., Benassi, M., & Tossani, E., (2011). Personality characteristics and psychological distress associated with primary exercise dependence: An exploratory study. *Psychiatry Research*, *189*(2), pp. 270–275.

Greenleaf, C., Petrie, T.A., Carter, J., & Reel, J.J., (2009). Female collegiate athletes: Prevalence of eating disorders and disordered eating behaviors. *Journal of American College Health*, *57*(5), pp. 489-96.

Guebels, C.P., Kam, L.C., Maddalozzo, G.F., & Manore, M.M., (2014). Active women before/after an intervention designed to restore menstrual function: Resting metabolic rate and comparison of four methods to quantify energy expenditure and energy availability. *International Journal of Sports Nutrition & Exercise Metabolism*, *24*(1), pp. 37–46.

Gulker, M.G., Laskis, T.A., & Kuba, S.A., (2001). Do excessive exercisers have a higher rate of obsessive-compulsive symptomatology? *Psychology, Health & Medicine*, *6*(4), pp. 387–398.

Guntur, A.R., & Rosen, C.J., (2013). IGF-1 regulation of key signaling pathways in bone. *Bone Key Reports*, *2*, pp. 437.

Hackshaw, A., (2008). Small studies: strengths and limitations. *European Respiratory Journal*, *32*(5), pp. 1141-43.

Hailey, B.J., & Bailey, L.A., (1982). Negative addiction in runners: a quantitative approach. *Journal of Sports Behaviour*, *5*(3), pp. 150–54.

Hale, B.D., Roth, A.D., DeLong, R.E., & Briggs, M.S., (2010). Exercise dependence and the drive for muscularity in male bodybuilders, power lifters, and fitness lifters. *Body Image*, 7(3), pp. 234-239.

Hall, K.D., & Kahan, S., (2018). Maintenance of lost weight and long-term management of obesity. *Medical Clinics of North America*, 102, pp. 183–97.

Hamer, M., & Karageorghis, C.I., (2007). Psychobiological mechanisms of exercise dependence. *Sports Medicine*, *37*(6), pp. 477–484.

Hamilton-Fairley, D., & Taylor, A., (2003). ABC of subfertility: Anovulation. *BMJ: British Medical Journal*, *327*(7414), pp. 546.

Harris, E.C., & Barraclough, B., (1998). Excess mortality of mental disorder. *British Journal of Psychiatry*, 173, pp. 11–53.

Harris, H.R., Babic, A., Webb, P.M., Nagle, C.M., Jordan, S.J., Risch, H.A., Rossing, M.A., Doherty, J.A., Goodman, M.T., Modugno, F., & Ness, R.B., (2018). Polycystic ovary syndrome, oligomenorrhea, and risk of ovarian cancer histotypes: evidence from the Ovarian Cancer Association Consortium. *Cancer Epidemiology & Prevention Biomarkers*, *27*(2), pp. 174-82.

Hart, S., Drevets, K., Alford, M., Salacinski, A., & Hunt, B.E., (2013). A method-comparison study regarding the validity and reliability of the Lactate Plus analyser. *British Medical Journal - Open*, 3 (2).

Hauck, C., Schipfer, M., Ellrott, T., & Cook, B., (2020). The relationship between food addiction and patterns of disordered eating with exercise dependence: in amateur endurance athletes. *Eating & Weight Disorders-Studies on Anorexia, Bulimia & Obesity*, *25*(6), pp. 1573-82.

Hausenblas, H.A., & Downs D.S., (2002a). Exercise dependence: a systematic review. *Psychology of Sport & Exercise*, 3, pp. 89–123.

Hausenblas, H.A., & Downs, D.S., (2002b). How much is too much? The development and validation of the exercise dependence scale. *Psychology & Health*, *17*(4), pp. 387–404.

Hausenblas, H.A., & Fallon, E.A., (2006). Exercise and body image: A meta-analysis. *Psychology & Health*, *21*(1), pp. 33–47.

Hausenblas, H.A., & Giacobbi, P.R., (2004). Relationship between exercise dependence symptoms and personality. *Personality & Individual Differences*, *36*(6), pp. 1265–73.

Hausenblas, H.A., Schreiber, K. & Smoliga, J.M., (2017). Addiction to exercise. *British Medical Journal*, 357, p.p. 1745.

Havill, L.M., Mahaney, M.C., Binkley, T.L., & Specker, B.L., (2007). Effects of genes, sex, age, and activity in BMC, bone size, and areal and volumetric BMD. *Journal of Bone & Mineral Research*, *22*(5), pp. 737-46.

Heikura, I.A., Burke, L.M., Bergland, D., Uusitalo, A.L., Mero, A.A., & Stellingwerff, T., (2018b). Impact of Energy Availability, Health, and Sex on Hemoglobin-Mass Responses Following Live-High–Train-High Altitude Training in Elite Female and Male Distance Athletes. *International Journal of Sports Physiology & Performance*, *13*(8), pp. 1090-96.

Heikura, I.A., Uusitalo, A.L., Stellingwerff, T., Bergland, D., Mero, A.A., & Burke, L.M., (2018a). Low energy availability is difficult to assess but outcomes have large impact on bone injury rates in elite distance athletes. *International Journal of Sport Nutrition & Exercise Metabolism*, *28*(4), pp. 403-11.

Hernández-Vicente, A., Santos-Lozano, A., De Cocker, K., & Garatachea, N., (2016). Validation study of Polar V800 accelerometer. *Annals of Translational Medicine*, 4 (15).

Hill, R.J., & Davies, P.S., (2001). The validity of self-reported energy intake as determined using the doubly labelled water technique. *British Journal of Nutrition*, *85*(4), pp. 415–30.

Hilton, L.K., & Loucks, A.B., (2000). Low energy availability, not exercise stress, suppresses the diurnal rhythm of leptin in healthy young women. *American Journal of Physiology Endocrinology & Metabolism*, 278, pp. E43–9.

Hoch, A.Z., Pajewski, N.M., Moraski, L., Carrera, G.F., Wilson, C.R., Hoffmann, R.G., Schimke, J.E., & Gutterman, D.D., (2009). Prevalence of the female athlete triad in high school athletes and sedentary students. *Clinical Journal of Sport Medicine: Official Journal of the Canadian Academy of Sport Medicine*, *19*(5), pp. 421.

Hoch, A.Z., Papanek, P., Szabo, A., Widlansky, M.E., Schimke, J.E., & Gutterman, D.D., (2011). Association between the female athlete triad and endothelial dysfunction in dancers. *Clinical Journal of Sport Medicine: Official Journal of the Canadian Academy of Sport Medicine*, *21*(2), pp.119.

Hoch, A.Z., Stavrakos, J.E., & Schimke, J.E., (2007). Prevalence of female athlete triad characteristics in a club triathlon team. *Archives of Physical Medicine & Rehabilitation*, 88(5), pp. 681–2.

Hodges, N.J., Augaitis, L., & Crocker, P.R., (2016). Sport commitment and deliberate practice among male and female triathletes. *International Journal of Sports Psychology*, 47, pp. 652-665.

Hofseth, L.J., (2018). Getting rigorous with scientific rigor. Carcinogenesis, 39(1), pp. 21–5.

Holland, G., & Tiggemann, M., (2017). "Strong beats skinny every time": Disordered eating and compulsive exercise in women who post fitspiration on Instagram. *International Journal of Eating Disorders*, *50*(1), pp. 76-9.

Hulley, A.J., & Hill, A.J., (2001). Eating disorders and health in elite women distance runners. *International Journal of Eating Disorders*, *30*(3), pp. 312-17.

Hunt, J.E., (2019). In Search of a Meaningful Story: Oral History and Triathlon Memory in Australia. *International Journal of the History of Sport*, *36*(13-14), pp. 1218-1233.

Ihle, R., & Loucks, A.B., (2004). Dose-response relationships between energy availability and bone turnover in young exercising women. *Journal of Bone & Mineral Research*, 19, pp. 1231–40.

International Society for Clinical Densitometry Writing Group for the ISCD Position Development Conference, (2004). Diagnosis of osteoporosis in men, women, and children. *Journal of Clinical Densitometry*, *7*, pp. 17–26.

James-Todd, T.M., Chiu, Y.H., & Zota, A.R., (2016). Racial/ethnic disparities in environmental endocrine disrupting chemicals and women's reproductive health outcomes.

Javaras, K.N., Runfola, C.D., Thornton, L.M., Agerbo, E., Birgegård, A., & Norring, C., (2015). Sex- and age-specific incidence of healthcare-register-recorded eating disorders in the complete Swedish 1979-2001 birth cohort. *International Journal of Eating Disorders*, 48, pp. 1070–81.

Jeacocke, N.A., & Beals, K.A., (2015). Eating disorders and disordered eating in athletes. In: Burke L, Deakin V, eds. *Clinical Sports Nutrition*. Australia: McGraw- Hill Education, pp. 213–33.

Jesus, F., Castela, I., Silva, A.M., Branco, P.A., & Sousa, M., (2021). Risk of Low Energy Availability among Female and Male Elite Runners Competing at the 26th European Cross-Country Championships. *Nutrients*, *13*(3), pp. 873.

Johnson, C., Powers, P.S., & Dick, R., (1999). Athletes and eating disorders: the National Collegiate Athletic Association study. *International Journal of Eating Disorders*, *26*(2), pp. 179-88.

Jones, G.S., (1976). The luteal phase defect. *Fertility & Sterility, 27,* pp. 351–56.

Jonnavithula, S., Warren, M., Fox, R., & Lazaro, M., (1993). Bone density is compromised in amenorrheic women despite return of menses: a 2-year study. *Obstetrics & Gynecology*, *81*(5), pp. 669-74.

Joy, E., De Souza, M.J., Nattiv, A., Misra, M., Williams, N.I., Mallinson, R.J., Gibbs, J.C., Olmsted, M., Goolsby, M., & Matheson, G., (2014). Female athlete triad coalition consensus statement on treatment and return to play of the female athlete triad. *Current Sports Medicine Reports*, 13, 219–232.

Joy, E., Kussman, A., & Nattiv, A., (2016). Update on eating disorders in athletes: a comprehensive narrative review with a focus on clinical assessment and management. *British Journal of Sports Medicine, 50*(3), pp. 154–62.

Käch, I.W., Rüst, C.A., Nikolaidis, P.T., Rosemann, T., & Knechtle, B., (2018). The age-related performance decline in Ironman triathlon starts earlier in swimming than in cycling and running. *Journal of Strength & Conditioning Research, 32*(2), pp.379-395.

Kaiser, U.B., et al., (1997). Studies of gonadotropin-releasing hormone (GnRH) action using GnRH receptor-expressing pituitary cell lines. *Endocrinology Review*, 18, pp. 46–70.

Karlamangla, A.S., Burnett-Bowie, S.A.M. and Crandall, C.J., 2018. Bone health during the menopause transition and beyond. *Obstetrics & Gynecology Clinics*, 45(4), pp.695-708.

Kataoka, Y., Luo, Y., Chaimani, A., Onishi, A., Kimachi, M., Tsujimoto, Y., Murad, M.H., Li, T., Cipriani, A. & Furukawa, T.A., (2020). Cumulative network meta-analyses, practice guidelines, and actual prescriptions for postmenopausal osteoporosis: a meta-epidemiological study. *Archives of Osteoporosis*, 15(1), pp.1-10.

Keen, A.D., & Drinkwater, B.L., (1997). Irreversible bone loss in former amenorrheic athletes. *Osteoporosis International*, *7*(4), pp. 311-15.

Khan, A.A.D., Hanley, J.P., Bilezikian, N., & Binkley, N., et al., (2006). Standards for performing DXA in individuals with secondary causes of osteoporosis. *Journal of Clinical Densitometry*, 9, pp. 47–57.

Khan, A.A.L., Bachrach, J.P., Brown, D.A., & Hanley, R.G., et al., (2004). Standards and guidelines for performing central dual-energy x-ray absorptiometry in premenopausal women, men, and children. *Journal of Clinical Densitometry, 7,* pp. 51–64.

Khan, K.M., Liu-Ambrose, T., & Sran, M.M., et al., (2002). New criteria for female athlete triad syndrome? As osteoporosis is rare, should osteopenia be among the criteria for defining the female athlete triad syndrome? British Journal of Sports Medicine, *36*(1), pp. 10–3.

Kimber, N.E., Ross, J.J., Mason, S.L., & Speedy, D.B., (2002). Energy balance during an ironman triathlon in male and female triathletes. *International Journal of Sports Nutrition & Exercise Metabolism*, 12(1), pp. 47–62.

Klein, D.A., & Walsh, B.T., (2004). Eating disorders: clinical features and pathophysiology. *Physiology & Behaviour*, *81*(2), pp. 359-74.

Klein, D.A., Paradise, S.L., & Reeder, R.M., (2019). Amenorrhea: a systematic approach to diagnosis and management. *American Family Physician*, *100*(1), pp.39-48.

Knapp, J., Aerni, G., & Anderson, J., (2014). Eating disorders in female athletes: use of screening tools. *Current Sports Medicine Reports*, *13*(4), pp. 214-18.

Knechtle, B., Scheer, V., Nikolaidis, P.T., & Sousa, C.V., (2020). Participation and performance trends in the oldest 100-km ultramarathon in the world. *International Journal of Environmental Research & Public Health*, 17(5), pp. 1705-1719.

Knechtle, B., Wirth, A., Baumann, B., Knechtle, P., & Rosemann, T., (2010). "Personal Best Time, Percent Body Fat, and Training Are Differently Associated with Race Time for Male and Female Ironman Triathletes." *Research Quarterly for Exercise & Sport*, 81 (1), pp. 62-68.

Koehler, K., Achtzehn, S., Braun, H., Mester, J., & Schaenzer, W., (2013). Comparison of self-reported energy availability and metabolic hormones to assess adequacy of dietary energy intake in young elite athletes. *Applied Physiology, Nutrition, & Metabolism*, 38(7), pp. 725–33.

Koehler, K., Williams, N.I., Mallinson, R.J., Southmayd, E.A., Allaway, H.C.M., & De Souza, M.J., (2016a). Low resting metabolic rate in exercise associated amenorrhea is not due to a reduced

proportion of highly active metabolic tissue compartments. *American Journal of Physiology Endocrinology & Metabolism*, 311, pp. E480–87.

Kong, P., & Harris, L.M., (2015). The sporting body: body image and eating disorder symptomatology among female athletes from leanness focused and non-leanness focused sports. *Journal of Psychology, 149*(2), pp. 141-60.

Kosmiski, L., Schmiege, S.J., Mascolo, M., Gaudiani, J., & Mehler, P.S., (2014). Chronic starvation secondary to anorexia nervosa is associated with an adaptive suppression of resting energy expenditure. *Journal of Clinical Endocrinology & Metabolism*, 99, pp. 908–14.

Kovacsik, R., Griffiths, M. D., Pontes, H. M., Soós, I., de la Vega, R., Ruíz-Barquín, R., Demetrovics, Z., & Szabo, A. (2018). The role of passion in exercise addiction, exercise volume, and exercise intensity in long-term exercisers. *International Journal of Mental Health Addiction.*

Krentz, E.M., & Warschburger, P., (2013). A longitudinal investigation of sports-related risk factors for disordered eating in aesthetic sports. *Scandinavian Journal of Medicine & Science in Sports, 23*(3), pp. 303–10.

Kroshus, E., Sherman, R.T., Thompson, R.A., Sossin, K., & Austin, S.B., (2014). Gender differences in high school coaches' knowledge, attitudes, and communication about the female athlete triad. *Eating Disorders*, 22(3), pp.193-208.

Lagowska, K., & Kapczuk, K., (2016). Testosterone concentrations in female athletes and ballet dancers with menstrual disorders. *European Journal of Sport Science, 16*(4), pp.490-97.

Landolfi, E., (2013). Exercise addiction. *Sports Medicine, 43*(2), pp. 111-19.

Larson-Meyer, D.E., Woolf, K., & Burke, L., (2018). Assessment of nutrient status in athletes and the need for supplementation. *International Journal of Sports Nutrition & Exercise Metabolism, 28*(2), pp. 139–158.

Lejoyeux, M., Avril, M., Richoux, C., Embouazza, H., & Nivoli, F., (2008). Prevalence of exercise dependence and other behavioral addictions among clients of a Parisian fitness room. *Comprehensive Psychiatry, 49*(4), pp. 353–358.

Lejoyeux, M., Guillot, C., Chalvin, F., Petit, A., & Lequen, V., (2012). Exercise dependence among custumers from a parisian sport shop. *Journal of Behavioural Addiction, 1*(1), pp. 28–34.

Lepers, R., (2019). Sex difference in triathlon performance. *Frontiers in Physiology*, 10, pp. 973.

Lepers, R., (2020). *The Master Triathlete.* In Triathlon Medicine (pp. 195-205). Springer, Cham.

Lepers, R., Rüst, C.A., Stapley, P.J., & Knechtle, B., (2013a). Relative improvements in endurance performance with age: evidence from 25 years if Hawaii ironman racing. *Age*, 35, pp. 953-962.

Levine, J.A., (2004). Non-exercise activity thermogenesis (NEAT): environment and biology. *American Journal of Physiology-Endocrinology & Metabolism*, 286, pp. E675–E85.

Levine, J.A., (2005). Measurement of energy expenditure. *Public Health Nutrition*, 8(7a), pp. 1123-1132.

Lichtenstein, M.B. & Hinze, C.J., (2020). Exercise addiction. In: *Adolescent Addiction* (pp. 265-288). Academic Press.

Lichtenstein, M.B., & Jensen, T.T., (2016). Exercise addiction in CrossFit: Prevalence and psychometric properties of the Exercise Addiction Inventory. *Addictive Behaviors Reports, 3*, pp. 33-37.

Lichtenstein, M.B., Hinze, C.J., & Emborg, B., et al., (2017). Compulsive exercise: links, risks and challenges faced. *Psychology Research & Behaviour Management*, 10, pp. 85–95.

Lieberman, J.L., De Souza, M.J., Wagstaff, D.A., & Williams, N.I., (2018). Menstrual disruption with exercise is not linked to an energy availability threshold. *Medicine & Science in Sports & Exercise*, *50*(3), pp. 551.

Liguori, G., & American College of Sports Medicine, (2020). *ACSM's Guidelines for Exercise Testing and Prescription*. Lippincott Williams & Wilkins.

Logue, D., Madigan, S.M., Delahunt, E., Heinen, M., Mc Donnell, S.J., & Corish, C.A., (2018). Low energy availability in athletes: a review of prevalence, dietary patterns, physiological health, and sports performance. *Sports Medicine*, *48*(1), pp.73-96.

Logue, D.M., Madigan, S.M., Heinen, M., McDonnell, S.J., Delahunt, E., & Corish, C.A., (2019). Screening for risk of low energy availability in athletic and recreationally active females in Ireland. *European Journal of Sport Science*, *19*(1), pp.112-122.

Logue, D.M., Madigan, S.M., Melin, A., Delahunt, E., Heinen, M., Donnell, S.J.M. & Corish, C.A., (2020). Low Energy Availability in Athletes 2020: An Updated Narrative Review of Prevalence, Risk, Within-Day Energy Balance, Knowledge, and Impact on Sports Performance. *Nutrients*, 12 (3), p.835.

Long, C.G., Smith, J., Midgley, M., & Cassidy, T., (1993). Over-exercising in anorexic and normal samples: Behaviour and attitudes. *Journal of Mental Health*, *2*(4), pp. 321–327.

Loucks, A.B., & Callister, R., (1993). Induction and prevention of low-T3 syndrome in exercising women. *American Journal of Physiology-Regulatory Integrative & Comparative Physiology*, 264, pp. R924–30.

Loucks, A.B., & Heath, E.M., (1994a). Induction of low-T3 syndrome in exercising women occurs at a threshold of energy availability. *American Journal of Physiology*, 266, pp. R817–23.

Loucks, A.B., & Horvath, S.M., (1985). Athletic amenorrhea: a review. *Medicine & Science in Sports & Exercise*, *17*, pp. 56–72.

Loucks, A.B., & Redman, L.M., (2004). The effect of stress on menstrual function. Trends in Endocrinology & Metabolism, *15*(10), pp. 466–71.

Loucks, A.B., & Thuma, J.R., (2003). Luteinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating women. Journal of Clinical Endocrinology & Metabolism, *88*(1), pp. 297–311.

Loucks, A.B., & Verdun, M., (1998). Slow restoration of LH pulsatility by refeeding in energetically disrupted women. *American Journal of Physiology*, 275, pp. R1218–26.

Loucks, A.B., (2004). Energy balance and body composition in sports and exercise. *Journal of Sports Sciences*, *22*(1), pp.1 -14.

Loucks, A.B., (2007). Low energy availability in the marathon and other endurance sports. *Sports Medicine*, *37*(4–5), pp. 348–52.

Loucks, A.B., (2020). Exercise training in the normal female: effects of low energy availability on reproductive function. In: Hackney AC, Constantini NW (eds) *Endocrinology of Physical Activity & Sport*. Springer International Publishing, Cham, pp 171–191.

Loucks, A.B., Kiens, B., & Wright, H.H., (2011). Energy availability in athletes. *Journal of Sports Sciences*, *29*(s1), pp. S7-S15.

Loucks, A.B., Verdun, M., & Heath, E.M., (1998). Low energy availability, not stress of exercise, alters LH pulsatility in exercising women. *Journal of Applied Physiology*, 84, pp. 37–46.

Loumidis, K.S., & Wells, A., (1998). Assessment of beliefs in exercise dependence: the development and preliminary validation of the exercise beliefs questionnaire. *Personality & Individual Differences*, *25*(3), pp. 553–567.

Lukács, A., Sasvári, P., Varga, B., & Mayer, K., (2019). Exercise addiction and its related factors in amateur runners. *Journal of Behavioral Addictions,* 8 (2), pp.343-349.

Macintyre, R.I., Heron, K.E., Howard, L.M., & Symons Downs, D., (2020). Gender and Black–White Race Differences in Young Adult Exercise Dependence Prevalence and Associated Disordered Eating. *Research Quarterly for Exercise & Sport*, pp. 1-9.

Maeda, K., Ohkura, S., Uenoyama, Y., Wakabayashi, Y., Oka, Y., Tsukamura, H., & Okamura, H., (2010). Neurobiological mechanisms underlying GnRH pulse generation by the hypothalamus. *Brain Research*, *1364*, pp. 103–15.

Magee, C.A., Buchanan, I., & Barrie, L., (2016). Profiles of exercise dependence symptoms in Ironman participants. *Psychology of Sport & Exercise*, *24*, pp. 48–55.

Mallinson, R.J., Williams, N.I., Gibbs, J.C., Koehler, K., Allaway, H.C., & Southmayd, E., et al., (2016). Current and past menstrual status is an important determinant of femoral neck geometry in exercising women. *Bone, 88*, pp. 101–12.

Mallinson, R.J., Williams, N.I., Hill, B.R., & De Souza, M.J., (2013). Body composition and reproductive function exert unique influences on indices of bone health in exercising women. *Bone*, *56*(1), pp. 91–100.

Mancine, R., Kennedy, S., Stephan, P., & Ley, A., (2020). Disordered Eating and Eating Disorders in Adolescent Athletes. *Spartan Medical Research Journal*, 4(2), p.11595.

Markus, G., & Arimany, A., (2019). ITU Triathlon History. *Triathlon Medicine*, pp. 1-2.

Marques, A., Peralta, M., Sarmento, H., Loureiro, V., Gouveia, É.R., & de Matos, M.G., (2019). Prevalence of risk for exercise dependence: A systematic review. *Sports Medicine*, *49*(2), pp. 319-330.

Marr, J.W., & Heady, J.A., (1986). Within-and between-person variation in dietary surveys: number of days needed to classify individuals. *Human Nutrition & Applied Nutrition*, *40*(5), pp. 347-364.

Martinsen, M., & Sundgot-Borgen, J., (2013). Higher prevalence of eating disorders among adolescent elite athletes than controls. *Medicine & Science in Sports & Exercise*, *45*, pp. 1188–97.

Martinsen, M., Bratland-Sanda, S., Eriksson, A.K., & Sundgot-Borgen, J., (2010). Dieting to win or to be thin? A study of dieting and disordered eating among adolescent elite athletes and non-athlete controls. *British Journal of Sports Medicine*, *44*(1), pp.70-76.

Martinsen, M., Holme, I., Pensgaard, A.M., Torstveit, M.K., & Sundgot-Borgen, J., (2014). The development of the Brief Eating Disorder in Athletes questionnaire. *Medicine & Science in Sports & Exercise*, *46*(8), pp. 1666-75.

Matkovic, V., Jelic, T., & Wardlaw, G.M., et al., (1994). Timing of peak bone mass in Caucasian females and its implication for the prevention of osteoporosis. *Journal of Clinical Investigations.*, *93*, pp. 799-808.

McKenzie, B.L., Coyle, D.H., Santos, J.A., Burrows, T., Rosewarne, E., Peters, S.A., Carcel, C., Jaacks, L.M., Norton, R., Collins, C.E., & Woodward, M., (2021). Investigating sex differences in the accuracy of dietary assessment methods to measure energy intake in adults: a systematic review and meta-analysis. *American Journal of Clinical Nutrition*.

McKinney, J., Velghe, J., Fee, J., Isserow, S., & Drezner, J.A., (2019). Defining athletes and exercisers. *American Journal of Cardiology*, *123*(3), pp. 532-35.

McNamara, J., & McCabe, M.P., (2012). Striving for success or addiction? Exercise dependence among elite Australian athletes. *Journal of Sports Sciences*, *30*(8), pp. 755–766.

McNeely, M.J., & Soules, M.R., (1988). The diagnosis of luteal phase deficiency. A critical review. *Fertility & Sterility*, *50*, pp. 1–15.

McNulty, K.Y., Adams, C.H., Anderson, J.H., & Affenito, S.G., (2001). Development and validation of a screening tool to identify eating disorders in female athletes. *Journal of American Dietetic Association*, *101*(8), pp. 886-92.

Meczekalski, B., Katulski, K., Czyzyk, A., Podfigurna-Stopa, A., & Maciejewska-Jeske, M., (2014). Functional hypothalamic amenorrhea and its influence on women's health. *Journal of Endocrinological Investigation*, *37*(11), pp. 1049-1056.

Mehler, P.S., & Brown, C., (2015). Anorexia nervosa—medical complications. *Journal of Eating Disorders*, *3*, pp.11.

Melin, A., Tornberg, A.B., Skouby, S., Faber, J., Ritz, C., Sjödin, A., & Sundgot-Borgen, J., (2014). The LEAF questionnaire: A screening tool for the identification of female athletes at risk for the female athlete triad. *British Journal of Sports Medicine*, *48*, 540–545.

Melin, A., Tornberg, Å.B., Skouby, S., Møller, S.S., Faber, J., Sundgot-Borgen, J., & Sjödin, A., (2016). Low-energy density and high fiber intake are dietary concerns in female endurance athletes. *Scandinavian Journal of Medicine & Science in Sports*, *26*(9), pp. 1060-71.

Melin, A., Tornberg, Å.B., Skouby, S., Møller, S.S., Sundgot-Borgen, J., Faber, J., Sidelmann, J.J., Aziz, M., & Sjödin, A., (2015). Energy availability and the female athlete triad in elite endurance athletes. *Scandinavian Journal of Medicine & Science in Sports*, *25*(5), pp. 610-22.

Meng, K., Qiu, J., Benardot, D., Carr, A., Yi, L., Wang, J., & Liang, Y., (2020). The risk of low energy availability in Chinese elite and recreational female aesthetic sports athletes. *Journal of the International Society of Sports Nutrition*, *17*(1), pp. 1-7.

Metzger, C.E., Baek, K., & Swift, S.N., et al., (2016). Exercise during energy restriction mitigates bone loss but not alterations in estrogen status or metabolic hormones. Osteoporosis. International, *27*(9), pp. 2755–64.

Meyvis, T., & Van Osselaer, S.M., (2018). Increasing the power of your study by increasing the effect size. *Journal of Consumer Research*, *44*(5), pp. 1157-73.

Mialich, M.S., Sicchieri, J.F., & Junior, A.J., (2014). Analysis of body composition: a critical review of the use of bioelectrical impedance analysis. *International Journal of Clinical Nutrition*, *2*(1), pp.1-10.

Midgley, A.W., McNaughton, L.R., Polman, R., & Marchant, D., (2007). Criteria for determination of maximal oxygen uptake. *Sports Medicine*, 37 (12), pp. 1019-1028.

Millet, G.P., Dreano, P., & Bentley, D.J., (2003). Physiological characteristics of elite short-and long-distance triathletes. *European Journal of Applied Physiology*, 88 (4-5), pp. 427-430.

Millet, G.P., Vleck, V.E., & Bentley, D.J., (2011). Physiological requirements in triathlon. *Journal of Human Sport & Exercise*, 6, pp. 184–204.

Mishra, P., Pandey, C.M., Singh, U., Gupta, A., Sahu, C., & Keshri, A., (2019). Descriptive statistics and normality tests for statistical data. *Annals of Cardiac Anaesthesia*, *22*(1), p.67.

Mitchell, J.H., Haskell, W., Snell, P., & Van Camp, S.P., (2005). Task Force 8: Classification of sports. *Journal of the American College of Cardiology*, *45*(8), pp. 1364-67.

Mongrain, J., Masson, G., Bégin, C., & Lamarche, B., (2018). Prevalence of disordered eating among non-elite multisport endurance athletes. *Journal of Sports Science*, *6*, pp. 1-8.

Mónok, K., Berczik, K., Urbán, R., Szabo, A., Griffiths, M.D., Farkas, J., Magi, A., Eisinger, A., Kurimay, T., Kökönyei, G., Kun, B., (2012). Psychometric properties and concurrent validity of two exercise addiction measures: A population wide study. *Psychology of Sport & Exercise*, *13*(6), pp. 739-746.

Moon, J.R., (2013). Body composition in athletes and sports nutrition: an examination of the bioimpedance analysis technique. *European Journal of Clinical Nutrition*, 67 (1), pp. S54-S59.

Moon, J.R., Stout, J.R., Smith, A.E., Tobkin, S.E., Lockwood, C.M., & Kendall, K.L, (2010). Reproducibility and validity of bioimpedance spectroscopy for tracking changes in total body water: implications for repeated measurements. *British Journal of Nutrition*, 104, pp. 1384–1394.

Mooney, M., Charlton, P.C., Soltanzadeh, S., & Drew, M., (2017). Who 'owns' the injury or illness? Who 'owns' performance? Applying systems thinking to integrate health and performance in elite sport. *British Journal of Sports Medicine*, pp. 1-2.

Morgan, J.F., Reid, F., & Lacey, J.H., (2000). The SCOFF questionnaire. *Western Journal of Medicine*, *172*(3), pp. 164.

Morgan, W.P., (1979). Negative addiction in runners. *Physician Sports Medicine*, *7*(2), pp. 57–70.

Moss, S.L., Randell, R.K., Burgess, D., Ridley, S., Ó Caireallain, C., Allison, R., & Rollo, I., (2020). Assessment of energy availability and associated risk factors in professional female soccer players. *European Journal of Sport Science*, pp.1-10.

Mountjoy, M., Sundgot-Borgen, J., Burke, L., Carter, S., Constantini, N., Lebrun, C., Meyer, N., Sherman, R., Steffen, K., Budgett, R., & Ljungqvist, A., (2014). The IOC consensus statement: beyond the female athlete triad—relative energy deficiency in sport (RED-S). *British Journal of Sports Medicine*, *48*(7), pp. 491-497.

Mountjoy, M., Sundgot-Borgen, J., Burke, L., Carter, S., Constantini, N., Lebrun, C., Meyer, N., Sherman, R., Steffen, K., & Budgett, R., et al (2015a). RED-S CAT. Relative Energy Deficiency in Sport (RED-S) Clinical Assessment Tool (CAT). *British Journal of Sports Medicine*, *49*(7), pp. 421-3.

Mountjoy, M., Sundgot-Borgen, J., Burke, L., Carter, S., Constantini, N., Lebrun, C., Meyer, N., Sherman, R., Steffen, K., Budgett, R., & Ljungqvist, A., (2015b). Authors' 2015 additions to the IOC consensus statement: Relative Energy Deficiency in Sport (RED-S). *British Journal of Sports Medicine*, *49*(7), pp. 417-420.

Mountjoy, M., Sundgot-Borgen, J.K., & Burke, L.M., et al., (2018). IOC consensus statement on relative energy deficiency in sport (RED- S): 2018 update. *British Journal of Sports Medicine*, 52, pp. 687–97.

Mountjoy, M., Thomas, A., & Levesque, J., (2019). The female triathlete. *Triathlon Medicine*, pp.207.

Muia, E. N., Wright, H. H., Onywera, V. O., & Kuria, E. N., (2016). Adolescent elite Kenyan runners are at risk for energy deficiency, menstrual dysfunction and disordered eating. *Journal of Sports Sciences*, *34*(7), pp. 598–606.

Mujika, I., (2011). Tapering for triathlon competition. *Journal of Human Sport & Exercise*, *6*(2), pp.264-270.

Mujika, I., (2017). Quantification of training and competition loads in endurance sports: methods and applications. *International Journal of Sports Physiology & Performance*, *12*(s2), pp.S2-9.

Müller, A., Cook, B., Zander, H., Herberg, A., Müller, V., & de Zwaan, M., (2014). Does the German version of the Exercise Dependence Scale measure exercise dependence? *Psychology of Sport & Exercise*, *15*(3), pp. 288–292.

Müller, A., Loeber, S., & Söchtig, J., et al., (2015). Risk for exercise dependence, eating disorder pathology, alcohol use disorder and addictive behaviors among clients of fitness centers. *Journal of Behavioural Addiction*, *4*, pp. 273–80.

Müller, M.J., & Bosy-Westphal, A., (2013). Adaptive thermogenesis with weight loss in humans. *Obesity*, 21, pp. 218–28.

Murakami, H., Kawakami, R., Nakae, S., Nakata, Y., Ishikawa-Takata, K., Tanaka, S., & Miyachi, M., (2016). Accuracy of wearable devices for estimating total energy expenditure: comparison with metabolic chamber and doubly labeled water method. *JAMA Internal Medicine*, *176*(5), pp.702-03.

Murakami, H., Kawakami, R., Nakae, S., Yamada, Y., Nakata, Y., Ohkawara, K., Sasai, H., Ishikawa-Takata, K., Tanaka, S., & Miyachi, M., (2019). Accuracy of 12 wearable devices for estimating physical activity energy expenditure using a metabolic chamber and the doubly labeled water method: validation study. *JMIR mHealth & uHealth*, *7*(8), p.e13938.

Muros, J.J., Ávila-Alche, Á., Knox, E., & Zabala, M., (2020). Likelihood of suffering from an eating disorder in a sample of Spanish cyclists and triathletes. *Journal of Eating Disorders*, *8*(1), pp. 1-7.

Myburgh, K.H., Hutchins, J., Fataar, A.B., Hough, S.F., & Noakes, T.D., (1990). Low bone density is an etiologic factor for stress fractures in athletes. *Annals of Internal Medicine*, *113*(10), pp. 754-59.

Nagel, D.L., Black, D.R., Leverenze, L.J., & Coster, D.C., (2000). Evaluation of a screening test for female college athletes with eating disorders and disordered eating. *Journal of Athletic Training*, *35*(4), pp. 431–40.

Nana, A., Slater, G.J., Hopkins, W.G., Halson, S.L., Martin, D.T., West, N.P., & Burke, L.M., (2016). Importance of standardized DXA protocol for assessing physique changes in athletes. *International Journal of Sport Nutrition & Exercise Metabolism*, *26*, pp. 259–67.

Nattiv, A., Loucks, A.B., & Manore, M.M., et al, (2007). American College of Sports Medicine position stand. The female athlete triad. *Medicine & Science in Sports & Exercise*, 39, pp. 1867–82.

Neidel, P., Wolfram, P., Hotfiel, T., Engelhardt, M., Koch, R., Lee, G., & Zwingenberger, S., (2019). Cross-sectional investigation of stress fractures in german elite triathletes. *Sports*, *7*(4), pp.88.

Neilson, H.K., Robson, P.J., Friedenreich, C.M., & Csizmadi, I., (2008). Estimating activity energy expenditure: How valid are physical activity questionnaires? *American Journal of Clinical. Nutrition*, 87, pp. 279–291.

Nes, B.M., Janszky, I., Wisløff, U., Støylen, A., & Karlsen, T., (2013). Age-predicted maximal heart rate in healthy subjects: The HUNT Fitness Study. *Scandinavian Journal of Medicine & Science in Sports*, 23 (6), pp. 697-704.

Nichols, J.F., Rauh, M.J., Barrack, M.T., Barkai, H.S., & Pernick, Y., (2007). Disordered eating and menstrual irregularity in high school athletes in lean-build and non-lean-build sports. *International Journal of Sports Nutrition Exercise & Metabolism*, *17*, pp. 364–77.

Nichols, J.F., Rauh, M.J., Lawson, M.J., Ji, M., & Barkai, H.S., (2006). Prevalence of the female athlete triad syndrome among high school athletes. *Archives of Pediatrics & Adolescent Medicine*, *160*(2), pp. 137-42.

Norton, K.I., (2018). *Standards for Anthropometry Assessment*. Kinanthropometry and Exercise Physiology: Volume One: Anthropometry, p.68.

O'Mara, K., (2019). The Truth About Triathlon Participation in the United States. Available at: https://www.triathlete.com/culture/news/the-truth-about-triathlon-participation-in-the-united-states/. (Accessed on: 25.07.2020).

Oberle, C.D., Watkins, R.S., & Burkot, A.J., (2018). Orthorexic eating behaviors related to exercise addiction and internal motivations in a sample of university students. *Eating & Weight Disorders-Studies on Anorexia, Bulimia & Obesity*, *23*(1), pp. 67-74.

Ogden, J., Veale, D., & Summers, Z., (1997). The development and validation of the exercise dependence questionnaire. *Addiction Research*, *5*(4), pp. 343–355.

Olympic Charter, 2019. *International Olympic Committee*. Lausanne, Switzerland, 2019. https://stillmed.olympic.org/media/Document%20Library/OlympicOrg/General/EN-Olympic-Charter.pdf (accessed on 13 Jul 2020).

Olympic Movement Medical Code. In force as from 1 Jul 2019. http://www.ioc-preventionconference.org/atpc/2019/wp-content/uploads/2019/07/S1.3_Budgett_Olympic-Movement-Medical-Code.pdf (accessed on 13 Jul 2020).

Otis, C.L., Drinkwater, B., & Johnson, M., et al, (1997). American College of Sports Medicine position stand. The female athlete triad. *Medicine & Science in Sports & Exercise*, 29, pp. i–ix.

Ozier, A.D., & Henry, B.W., (2011). Position of the American dietetic association: nutrition intervention in the treatment of eating disorders. *Journal of American Dietetics Association, 111*, pp. 1236–41.

Papageorgiou, M., Elliott-Sale, K.J., Parsons, A., Tang, J.C.Y., Greeves, J.P., & Fraser, W.D., et al., (2017). Effects of reduced energy availability on bone metabolism in women and men. *Bone*, 105, pp. 191–9.

Paradis, K.F., Cooke, L.M., Martin, L.J., & Hall, C.R., (2013). Too much of a good thing? Examining the relationship between passion for exercise and exercise dependence. *Psychology of Sport & Exercise, 14*(4), pp. 493–500.

Parastatidou, I.S., Doganis, G., Theodorakis, Y., & Vlachopoulos, S P. (2012). Exercising with passion: Initial validation of the passion scale in exercise. *Measurement in Physical Education & Exercise Science*, *16*(2), pp. 119–134.

Peiffer, J., Abbiss, C.R., Sultana, F., Bernard, T. & Brisswalter, J., (2016). Comparison of the influence of age on cycling efficiency and the energy cost of running in well-trained triathletes. *European Journal of Applied Physiology*, 116, pp. 195-201.

Peñas-Lledó, E., Vaz Leal, F.J., & Waller, G., (2002). Excessive exercise in anorexia nervosa and bulimia nervosa: relation to eating characteristics and general psychopathology. *International Journal of Eating Disorders*, 31 (4), pp. 370-375.

Pernick, Y., Nichols, J.F., Rauh, M.J., Kern, M., Ji, M., Lawson, M.J., & Wilfley, D., (2006). Disordered eating among a multi-racial/ethnic sample of female high-school athletes. *Journal of Adolescent Health, 38*(6), pp. 689-95.

Petrie, T.A., & Greenleaf, C.A., (2007). Eating disorders in sport: From theory to research to intervention. In G. Tenenbaum & R. C. Eklund (Eds.), *Handbook of Sport Psychology* (pp. 352–378). John Wiley & Sons, Inc.

Pettersen, I., Hernæs, E., & Skårderud, F., (2016). Pursuit of performance excellence: a population study of Norwegian adolescent female cross-country skiers and biathletes with disordered eating. *BMJ - Open Sport & Exercise Medicine*, *2*(1), pp. e000115.

Petterson, F., Fires, H., & Nillius, S.J., (1973). Epidemiology of secondary amenorrhea, prevalance and incidence rates. *American Journal of Obstetrics & Gynecology*, 117, pp. 80–86.

Pierce, E. F., McGowan, R. W., & Lynn, T. D., (1993). Exercise dependence in relation to competitive orientation of runners. *Journal of Sports Medicine & Physical Fitness,* 33(2), pp. 189–193.

Pollock, N., Grogan, C., Perry, M., Pedlar, C., Cooke, K., Morrissey, D., & Dimitriou, L. (2010). Bone-mineral density and other features of the female athlete triad in elite endurance runners: A longitudinal and cross-sectional observational study. *International Journal of Sport Nutrition & Exercise Metabolism*, 20(5), pp. 418–426.

Portney, L.G., & Watkins, M.P., (1998). *Foundations of clinical research; applications to practice.* Appleton & Lange, Norwalk, CT.

Prajapati, B., Dunne, M., & Armstrong, R., (2010). Sample size estimation and statistical power analyses. *Optometry Today*, *16*(7), pp. 10-18.

Quah, Y.V., Poh, B.K., Ng, L.O., & Noor, M.I., (2009). The female athlete triad among elite Malaysian athletes: prevalence and associated factors. *Asia Pacific Journal of Clinical Nutrition*, *18*(2), pp. 200-08.

Qualtrics, (2022). https://www.qualtrics.com/blog/calculating-sample-size/. *Accessed on:* 06.03.2022.

Quatromoni, P.A., (2008). Clinical observations from nutrition services in college athletics. *Journal of the American Dietetic Association*, *108*(4), pp. 689-694.

Ravussin, E., & Bogardus, C., (1989). Relationship of genetics, age, and physical fitness to daily energy expenditure and fuel utilization. *American Journal of Clinical Nutrition*, *49*(5), pp. 968-975.

Reardon, C.L., Hainline, B., & Aron, C.M., et al., (2019). Mental health in elite athletes: international Olympic Committee consensus statement (2019). *British Journal of Sports Medicine*, *53*, pp. 667–99.

Recker, R.R., Davies, K.M., Hinders, S.M., Heaney, R.P., Stegman, M.R., & Kimmel, D.B., (1992). Bone gain in young adult women. *JAMA*, *268*, pp. 2403–8.

Reed, J.L., Bowell, J.L., Hill, B.R., Williams, B.A., De Souza, M.J., & Williams, N.I., (2011). Exercising women with menstrual disturbances consume low energy dense foods and beverages. *Applied Physiology Nutrition & Metabolism*. 36(3), pp. 382–94.

Reed, J.L., De Souza, M.J., & Williams, N.I., (2013). Changes in energy availability across the season in Division I female soccer players. *Journal of Sports Sciences*, *31*(3), pp. 314-24.

Reed, J.L., De Souza, M.J., Mallinson, R.J., Scheid, J.L., & Williams, N.I., (2015). Energy availability discriminates clinical menstrual status in exercising women. *Journal of International Society Sports Nutrition, 12*, pp. 1-11.

Reinking, M.F., & Alexander, L.E., (2005). Prevalence of disordered eating behaviours in undergraduate female collegiate athletes and nonathletes. *Journal of Athletic Training*, 40, pp. 47–51.

Rion, S., & Kawecki, T.J., (2007). Evolutionary biology of starvation resistance: what we have learned from Drosophila: Starvation resistance in Drosophila. *Journal of Evolutionary Biology*, 20, pp.1655–64.

Rivas, L.G., Mielgo-Ayuso, J., Norte-Navarro, A., Cejuela, R., Cabañas, M.D., & Martínez-Sanz, J.M., (2015). Body composition and somatotype in university triathletes. *Nutricion Hospitalaria*, 32, pp. 799-807.

Robbeson, J.G., Kruger, H.S., & Wright, H.H., (2015). Disordered eating behavior, body image, and energy status of female student dancers. *International Journal of Sport Nutrition & Exercise Metabolism*, *25*(4), pp. 344-52.

Robergs, R.A., & Burnett, A.F., (2003). Methods used to process data from indirect calorimetry and their application to VO2 max. *Journal of Exercise Physiology Online*, 6(2).

Rosendahl, J., Bormann, B., Aschenbrenner, K., Aschenbrenner, F., & Strauss, B., (2009). Dieting and disordered eating in German high school athletes and non-athletes. *Scandinavian Journal of Medicine & Science in Sports*, *19*(5), pp. 731-39.

Rousselet, M., Guérineau, B., Paruit, M.C., Guinot, M., Lise, S., Destrube, B., Ruffio-Thery, S., Dominguez, N., Brisseau-Gimenez, S., Dubois, V., & Mora, C., (2017). Disordered eating in French high-level athletes: association with type of sport, doping behavior, and psychological features. *Eating & Weight Disorders-Studies on Anorexia, Bulimia & Obesity*, *22*(1), pp. 61-68.

Ruegsegger, G.N., & Booth, F.W., (2018). Health benefits of exercise. *Cold Spring Harbor Perspectives in Medicine*, 8(7), pp. a029694.

Sale, C., & Elliott-Sale, K.J., (2019). Nutrition and athlete bone health. *Sports Medicine, 49*(2), pp. 139-51.

Sanborn, C.F., Martin, B.J., & Wagner, W.W., (1982). Is athletic amenorrhea specific to runners? *American Journal of Gynecology, 143*, pp. 859–63.

Santos-Concejero, J., Granados, C., Irazusta, J., Bidaurrazaga-Letona, I., Zabala-Lili, J., Tam, N., & Gil, S.M., (2014). OBLA is a better predictor of performance than Dmax in long and middle-distance well-trained runners. *Journal of Sports Medicine & Physical Fitness*, 54 (5), pp.553-8.

Sawyer, S.M., Afifi, R.A., Bearinger, L.H., Blakemore, S.J., Dick, B., Ezeh, A.C., & Patton, G.C. (2012). Adolescence: A foundation for future health. *Lancet*, 379(9826), 1630–1640.

Schaal, K., Tafflet, M., Nassif, H., Thibault, V., Pichard, C., Alcotte, M., Guillet, T., El Helou, N., Berthelot, G., Simon, S., &Toussaint, J.F., (2011b). Psychological balance in high level athletes: gender-based differences and sport-specific patterns. *PLOS ONE*, *6*(5), pp. e19007.

Schaal, K., Tiollier, E., Le Meur, Y., Casazza, G., & Hausswirth, C., (2017). Elite synchronized swimmers display decreased energy availability during intensified training. *Scandinavian Journal of Medicine & Science in Sports*, *27*(9), pp. 925-34.

Schaal, K., van Loan, M.D., & Casazza, G.A., (2011a). Reduced catecholamine response to exercise in amenorrheic athletes. *Medicine & Science in Sports & Exercise*, *43*(1), pp. 34–43.

Scharmer, C., Gorrell, S., Schaumberg, K., & Anderson, D., (2020). Compulsive exercise or exercise dependence? Clarifying conceptualizations of exercise in the context of eating disorder pathology. *Psychology of Sport & Exercise*, *46*, pp .101586.

Scheer, V., (2019). Participation trends of ultra-endurance events. *Sports Medicine & Arthroscopy Review*, 27(1), pp. 3-7.

Scheid, J. L., & De Souza, M. J., (2010). Menstrual Irregularities and Energy Deficiency in Physically Active Women: The Role of Ghrelin, PYY and Adipocytokines. Cytokines, Growth Mediators and Physical Activity in Children during Puberty. *Basel: KARGER*.

Schneider, J., & Wade, G., (1989). Availability of metabolic fuels controls estrous cyclicity of Syrian hamsters. *Science*, 244, pp. 1326–28.

Schneider, J.E., & Wade, G., (1990). Decreased availability of metabolic fuels induces anestrus in golden hamsters. *American Journal of Physiology-Regulatory Integrative & Comparitive Physiology*, 258, pp. R750–55.

Schreiber, K., & Hausenblas, H.A., (2015). *The truth about exercise addiction. Understanding the dark side of thinspiration.* London, UK: Rowman & Littlefield.

Schtscherbyna, A., Soares, E.A., de Oliveira, F.P., & Ribeiro, B.G., (2009). Female athlete triad in elite swimmers of the city of Rio de Janeiro, Brazil. *Nutrition*, *25*(6), pp. 634-39.

Schwartz, B., Cumming, D.C., Riordan, E., Selye, M., Yen, S.C.C., & Rebar, R.W., (1981). Exercise-associated amenorrhea, a distinct entity? *American Journal of Obstetrics & Gynecology*, *141*, pp. 662–70.

Scofield, K.L., & Hecht, S., (2012). Bone health in endurance athletes: runners, cyclists, and swimmers. *Current Sports Medicine Reports*, *11*(6), pp. 328–34.

Sears, L.A., Tracy, K.R. & McBrier, N.M., (2012). Self-esteem, body image, internalization, and disordered eating among female athletes. *Athletic Training & Sports Health Care*, *4*(1), pp.29-37.

Serier, K.N., Smith, J.E., Lash, D.N., Gianini, L.M., Harriger, J.A., Sarafin, R.E., & Wolfe, B.L., (2018). Obligatory exercise and coping in treatment-seeking women with poor body image. *Eating & Weight Disorders-Studies on Anorexia, Bulimia & Obesity*, *23*(3), pp. 331-38.

Shanmugam, V., Jowett, S., & Meyer, C., (2014). Interpersonal difficulties as a risk factor for athletes' eating psychopathology. *Scandinavian Journal of Medicine & Science in Sports*, *24*(2), pp. 469–76.

Shaw, T., Howat, P., Trainor, M., & Maycock, B., (2004). Training patterns and sports injuries in triathletes. *Journal of Science & Medicine in Sport*, 7 (4), pp. 446-450.

Sherman, R.T., & Thompson, R.A., (2006). Practical use of the International Olympic Committee Medical Commission Position Stand on the Female Athlete Triad: A case example. *International Journal of Eating Disorders*, *39*(3), pp. 193-201.

Shroff, H., Reba, L., Thornton, L.M., Tozzi, F., Klump, K.L., Berrettini, W.H., Brandt, H., Crawford, S., Crow, S., Fichter, M.M., & Goldman, D., (2006). Features associated with excessive exercise in women with eating disorders. *International Journal of Eating Disorders*, *39*(6), pp. 454-61.

Silva, M.R., & Paiva, T., (2015). Low energy availability and low body fat of female gymnasts before an international competition. *European Journal of Sport Science*, *15*(7), pp. 591-99.

Singh, K.B., (1981). Menstrual disorders in college students. *American Journal of Obstetrics & Gynecology*, *140*(3), pp. 299–302.

Slater, J., McLay-Cooke, R., Brown, R., & Black, K., (2016). Female Recreational Exercisers at Risk for Low Energy Availability. *International Journal of Sport Nutrition & Exercise Metabolism, 26(*5*),* pp. 421–27.

Slevec, J. H., & Tiggemann, M., (2011). Predictors of body dissatisfaction and disordered eating in middle-aged women. *Clinical Psychology Review*, 31, pp. 515–524.

Smink, F.H.D., & Hoek, H.W., (2013). Epidemiology, course and outcome of eating disorders. *Eating Disorders*, 26(6), pp. 543-548.

Smith, A.R., Fink, E.L., & Anestis, M.D., et al., (2013). Exercise caution: over-exercise is associated with suicidality among individuals with disordered eating. *Psychiatry Research*, 206, pp. 246–55.

Smith, K.S., Lenton, E.A., Landgren, B.M., & Cooke, I.D., (2006). Is the short luteal phase a defective luteal phase? *Annals of the NY Academy of Sciences*, *442*, pp. 387-90.

Snoza, C.T., Berg, K.E., & Slivka, D.R., (2016). Comparison of VO2peak and achievement of VO2peak criteria in three modes of exercise in female triathletes. *Journal of Strength & Conditioning Research*, 30 (10), pp. 2816-2822.

Solberg, E.E., Borisson, S., Sharma, M., Papadakis, M., Willhelm, J.A., et al, (2016). Sudden cardiac arrest in sports—need for uniform registration: a position paper from the sport cardiology section of the European Association for Cardiovascular Prevention and Rehabilitation. *European Journal of Preventative Cardiology*, 23 (2016), pp. 657-667.

Sousa, C.V., Nikolaidis, P.T., & Knechtle, B., (2020). Ultra-triathlon—Pacing, performance trends, the role of nationality, and sex differences in finishers and non-finishers. *Scandinavian Journal of Medicine & Science in Sports*, *30*(3), pp.556-563.

Southmayd, E.A., Mallinson, R.J., Williams, N.I., Mallinson, D.J., & De Souza, M.J., et al., (2016). Current status of the female athlete triad: update and future directions. *Current Osteoporosis Research*, 15, pp. 577-87.

Sowers, M., Randolph, J.F., & Crutchfield, M., et al., (1998). Urinary ovarian and gonadotropin hormone levels in premenopausal women with low bone mass. *Journal of Bone & Mineral* Research, 13, pp. 1191–202.

Spano, L., (2001). The relationship between exercise and anxiety, obsessive-compulsiveness, and narcissism. *Personality & Individual Differences*, *30*(1), pp. 87–93.

Staal, S., Sjödin, A., Fahrenholtz, I., Bonnesen, K., & Melin, A.K., (2018). Low RMRratio as a surrogate marker for energy deficiency, the choice of predictive equation vital for correctly identifying male and female ballet dancers at risk. *International Journal of Sport Nutrition & Exercise Metabolism*, 28(4), pp.412-418.

Stice, E., South, K., & Shaw, H., (2012). Future directions in etiologic, prevention, and treatment research for eating disorders. *Journal of Clinical Child & Adolescent Psychology*, *41*(6), pp. 845–55.

Stirling, A., & Kerr, G., (2012). Perceived vulnerabilities of female athletes to the development of disordered eating behaviours. *European Journal of Sport Science*, *12*(3), pp. 262–73.

Strath, S.J., Brage, S., & Ekelund, U., (2005). Integration of physiological and accelerometer data to improve physical activity assessment. *Medicine & Science in Sports & Exercise*, *37*, pp. S563–71.

Ströhle, A., (2019). Sports psychiatry: mental health and mental disorders in athletes and exercise treatment of mental disorders. *European Archives of Psychiatry & Clinical Neuroscience*, *269*, pp. 485–98.

Stunkard, A.J., & Messick, S., (1985). The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of Psychosomatic Research*, *29*(1), pp. 71-83.

Sundgot-Borgen, J., & Garthe, I., (2011). Elite athletes in aesthetic and Olympic weight-class sports and the challenge of body weight and body compositions. *Journal of Sports Science*, *29*(1), pp. S101–14.

Sundgot-Borgen, J., & Torstveit, M.K., (2004). Prevalence of eating disorders in elite athletes is higher than in the general population. Clinical Journal of Sport Medicine, *14*(1), pp. 25–32.

Sundgot-Borgen, J., & Torstveit, M.K., (2010). Aspects of disordered eating continuum in elite high-intensity sports. *Scandinavian Journal of Medicine & Science in Sports*, *20*, pp. 112-21.

Sundgot-Borgen, J., (1993). Prevalence of eating disorders in elite female athletes. *International Journal of Sport Nutrition & Exercise Metabolism*, *3*(1), pp. 29-40.

Sundgot-Borgen, J., (1994). Risk and trigger factors for the development of eating disorders in female elite athletes. *Medicine & Science in Sports & Exercise*, *26*(4), pp. 414–19.

Sundgot-Borgen, J., Meyer, N.L., & Lohman, T.G., et al., (2013). How to minimise the health risks to athletes who compete in weight- sensitive sports review and position statement on behalf of the AD hoc research Working group on body composition, health and performance, under the auspices of the IOC medical Commission. *British Journal of Sports Medicine*, 47, pp. 1012–22.

Sussman, S., Lisha, N., & Griffiths, M. D., (2011a). Prevalence of the addictions: A problem of the majority or the minority? *Evaluation & the Health Professions*, *34*(1), pp. 3–56.

Sygo, J., Coates, A.M., Sesbreno, E., Mountjoy, M.L., & Burr, J.F., (2018). Prevalence of indicators of low energy availability in elite female sprinters. *International Journal of Sport Nutrition & Exercise Metabolism*, *28*(5), pp. 490-96.

Szabo, A., (1995). The impact of exercise deprivation on well-being of habitual exercisers. *The Australian Journal of Science & Medicine in Sport, 27*, pp. 68–75.

Szabo, A., (2000). *Physical activity and psychological well-being*. London: Routledge. Physical activity as a source of psychological dysfunction. In S. J. Biddle K. R Fox & S. H. Boutcher (Eds.) p. pp. 130–153.

Szabo, A., (2010). *Addiction to exercise: a symptom or a disorder?* New York: Nova Science Publishers.

Szabo, A., Griffiths, M.D., Marcos, R.D.L.V., Mervó, B., & Demetrovics, Z., (2015). Focus: addiction: methodological and conceptual limitations in exercise addiction research. *The Yale Journal of Biology & Medicine*, *88*(3), pp. 303.

Szabo, A., Vega Rde.L., Ruiz-Barquin, R., & Rivera, O., (2013). Exercise addiction in Spanish athletes: investigation of the roles of gender, social context and level of involvement. *Journal of Behavioural Addictions*, *2*(4), pp. 249–252.

Taber, K.S., (2018). The use of Cronbach's Alpha when developing and reporting research instruments in science education. *Research in Science Education*, 48, 99. 1273-1296.

Tallón, P.L.V., & Palomero, F.A., (2017). Risk of exercise addiction among male amateur triathletes and its relationship with training variables. *RICYDE*, 13(48), pp.162-171.

Tenforde, A.S., Barrack, M.T., & Nattiv, A., et al., (2016). Parallels with the female athlete triad in male athletes. Sports Medicine, *46*(2), pp. 171–82.

Terry, A., Szabo, A., & Griffiths, M., (2004). The exercise addiction inventory: a new brief screening tool. *Addiction Research & Theory*, *12*(5), pp. 489–499.

Thein-Nissenbaum, J., (2013). Long term consequences of the female athlete triad. *Maturitas*, *75*(2), pp.107-112.

Thein-Nissenbaum, J.M., Rauh, M.J., Carr, K.E., Loud, K.J., & McGuine, T.A., (2011). Associations between disordered eating, menstrual dysfunction, and musculoskeletal injury among high school athletes. *Journal of Orthopaedic & Sports Physical Therapy*, *41*(2), pp.60-69.

Thompson, A., Petrie, T., & Anderson, C., (2017). Eating disorders and weight control behaviors change over a collegiate sport season. *Journal of Science & Medicine in Sport*, 20 (9), pp. 808-813.

Thompson, J. K., & Pasman, L., (1991). The obligatory exercise questionnaire. *The Behavior Therapist*, *14*, 137.

Thompson, J., & Manore, M.M., (1996). Predicted and measured resting metabolic rate of male and female endurance athletes. *Journal of American Dietetics Association*, 96, pp. 30–34.

Thompson, J.K., & Blanton, P., (1987). Energy conservation and exercise dependence: a sympathetic arousal hypothesis. *Medicine & Science in Sports & Exercise*, *19*(2), pp. 91-9.

Thompson, K.A., & Bardone-Cone, A.M., (2018). Menopausal status and disordered eating and body image concerns among middle-aged women. *International Journal of Eating Disorders*, 52, pp. 314–318.

Thompson, K.A., & Bardone-Cone, A.M., (2019). Disordered eating behaviors and attitudes and their correlates among a community sample of older women. *Eating Behaviors*, *34*, pp.101301.

Thorpe, H., & Clark, M., (2020). Gut feminism, new materialism and sport women's embodied health: the case of RED-S in endurance athletes. *Qualitive Research in Sport, Exercise & Health*, 12(1), pp. 1-17.

Tomten, S.E., Falch, J.A., & Birkeland, K.I., et al., (1998). Bone mineral density and menstrual irregularities. A comparative study on cortical and trabecular bone structures in runners with alleged normal eating behavior. *International Journal of Sports Medicine*, 19, pp.92–7.

Toro, J., Galilea, B., Martinez-Mallén, E., Salamero, M., Capdevila, L., Mari, J., Mayolas, J., & Toro, E., (2005). Eating disorders in Spanish female athletes. *International journal of sports medicine*, *26*(8), pp. 693-700.

Torstveit, M.K., & Sundgot-Borgen, J., (2011). Are under- and overweight female elite athletes thin and fat? A controlled study. *Medicine & Science in Sports & Exercise*, 44, pp. 949–57.

Torstveit, M.K., Fahrenholtz, I.L., Lichtenstein, M.B., Stenqvist, T.B., & Melin, A.K., (2019). Exercise dependence, eating disorder symptoms and biomarkers of Relative Energy Deficiency in Sports (RED-S) among male endurance athletes. *BMJ Open Sport & Exercise Medicine*, 5 (1).

Torstveit, M.K., Rosenvinge, J.H., & Sundgot-Borgen, J., (2008). Prevalence of eating disorders and the predictive power of risk models in female elite athletes: a controlled study. *Scandinavian Journal of Medicine & Science in Sports, 18*(1), pp. 108-18.

Trakman, G.L., Forsyth, A., Devlin, B.L., & Belski, R., (2016). A systematic review of athletes' and coaches' nutrition knowledge and reflections on the quality of current nutrition knowledge measures. *Nutrients, 8*(9), p.570.

Tsutsumi, R., & Webster, N.J., (2009). GnRH pulsatility, the pituitary response and reproductive dysfunction. *Endocrine Journal, 56*(6), pp.729-37.

Turton, R., Goodwin, H., & Meyer, C., (2017). Athletic identity, compulsive exercise and eating psychopathology in long-distance runners. *Eating Behaviour, 26*, pp.129–32.

USA Triathlon (2016). USA Triathlon Membership Survey Report. Available at: https://www.teamusa.org/USA-Triathlon/News/Articles-and-Releases/2017/December/18/USA-Triathlon-Membership-Survey-Report. Accessed 14 June 2020.

USA Triathlon, (2019). https://www.teamusa.org/USA-Triathlon/News/Articles-and-Releases/2019/March/08/USA-Triathlon-Unveils-Refreshed-Strategic-Plan-Through-2020. (Accessed on: 23.02.2021).

Valenzuela, P.L., & Arriba-Palomero, F., (2017). Risk of exercise addiction among male amateur triathletes and its relationship with training variables. *Revista Internacional Ciencas Deporte, 13* (48), pp.162–71.

Vallerand, R.J., Blanchard, C., Mageau, G.A., Koestner, R., Ratelle, C., Léonard, M., & Marsolais, J., (2003). Les passions de l'ame: On obsessive and harmonius passion. *Journal of Personality & Social Psychology, 85*(4), pp. 756–767.

VanHeest, J.L., Rodgers, C.D., Mahoney, C.E., & De Souza, M.J. (2014). Ovarian Suppression Impairs Sport Performance in Junior Elite Female Swimmers. *Medicine & Science in Sports & Exercise, 46(1),* pp. 156–166.

Vardar, E., Vardar, S.A., & Kurt, C., (2007). Anxiety of young female athletes with disordered eating behaviors. *Eating Behaviors, 8*(2), pp. 143-47.

Vasikaran, S., Cooper, C., Eastell, R., Griesmacher, A., Morris, H.A., & Trenti, T., et al., (2011). Markers of bone turnover for the prediction of fracture risk and monitoring of osteoporosis treatment: a need for international reference standards. *Osteoporosis International, 22*, pp. 391–420

Vasikaran, S.D., (2008). Utility of biochemical markers of bone turnover and bone mineral density in management of osteoporosis. *Critical Reviews in Clinical Laboratory Sciences, 45*(2), pp. 221–58.

Veale, D.M.W.C., (1987). Exercise dependence. *British Journal of Addiction, 82*, pp. 735–40.

Vescovi, J.D., & VanHeest, J.L., (2016). Case study: impact of inter-and intra-day energy parameters on bone health, menstrual function, and hormones in an elite junior female triathlete. *International Journal of Sport Nutrition & Exercise Metabolism, 26*(4), pp. 363-369.

Villablanca, P.A., Alegria, J.R., Mookadam, F., Holmes, D.R., Wright, R.S., & Levine, J.A., (2015). Non-exercise activity thermogenesis in obesity management. *Mayo Clinic Proceedings*, 90, pp. 509–19.

Villella, C., Martinotti, G., & Di Nicola, M., et al., (2011). Behavioural addictions in adolescents and young adults: results from a prevalence study. *Journal of Gambling Studies, 27*(2), pp. 203–214.

Viner, R.T., Harris, M., Berning, J.R., & Meyer, N.L., (2015). Energy Availability and Dietary Patterns of Adult Male and Female Competitive Cyclists with Lower than Expected Bone Mineral Density. *International Journal of Sport Nutrition & Exercise Metabolism*, *25*(6), pp. 594–602.

Vitti, A., Nikolaidis, P.T., Villiger, E., Onywera, V., & Knechtle, B., (2020). The "New York City Marathon": participation and performance trends of 1.2M runners during half-century. *Research in Sports Medicine,* 28(1), pp. 121-137.

Vleck, V., Millet, G.P., & Alves, F.B., (2014). The impact of triathlon training and racing on athletes' general health. *Sports Medicine*, *44*(12), pp. 1659-1692.

Wagner, A.J., Erickson, C.D., & Tierney, D.K., et al., (2016). The diagnostic accuracy of screening tools to detect eating disorders in female athletes. *Journal of Sport Rehabilitation*, *25*, pp. 395–8.

Walker, S., & Lloyd, C., (2011). Barriers and attitudes health professionals working in eating disorders experience. *International Journal of Therapy & Rehabilitation*, 18, pp. 383–90.

Warren, M.P., Brooks-Gunn, J., Hamilton, L.H., Warren, L.F., & Hamilton, W.G., (1986). Scoliosis and fractures in young ballet dancers. Relation to delayed menarche and secondary amenorrhea. *New England Journal of Medicine, 314*(21), pp. 1348-53.

Wasserfurth, P., Palmowski, J., Hahn, A., & Krüger, K., (2020). Reasons for and Consequences of Low Energy Availability in Female and Male Athletes: Social Environment, Adaptations, and Prevention. *Sports Medicine-Open*, *6*(1), pp.1-14.

Weaver, C.M., Gordon, C.M., Janz, K.F., Kalkwarf, H.J., Lappe, J.M., & Lewis, R., et al., (2016). The National Osteoporosis Foundation's position statement on peak bone mass development and lifestyle factors: a systematic review and implementation recommendations. *Osteoporosis International*, *27*(4), pp. 1281-386.

Weitzmann, M.N., & Pacifici, R., (2006). Estrogen deficiency and bone loss: an inflammatory tale. *Journal of Clinical Investigation*, *116*(5), pp. 1186-94.

Wells, K.R., Jeacocke, N.A., Appaneal, R., Smith, H.D., Vlahovich, N., Burke, L.M., & Hughes, D., (2020). The Australian Institute of Sport (AIS) and National Eating Disorders Collaboration (NEDC) position statement on disordered eating in high performance sport. *British Journal of Sports Medicine*, *54*(21), pp. 1247-58.

Westerterp, K.R., & Saris, W.H., (1991). Limits of energy turnover in relation to physical performance, achievement of energy balance on a daily basis. *Journal of Sports Sciences*, *9*(S1), pp. 1-15.

Westerterp, K.R., (2013). Physical activity and physical activity induced energy expenditure in humans: measurement, determinants, and effects. *Frontiers in Physiology*, *4*, p.90.

Wewege, M.A., & Ward, R.E., (2018). Bone mineral density in pre-professional female ballet dancers: a systematic review and meta-analysis. *Journal of Science & Medicine in Sport, 21*, pp. 783-8.

Whiting, S.J., Vatanparast, H., Baxter-Jones, A., Faulkner, R.A., Mirwald, R., & Bailey, D.A., (2004). Factors that affect bone mineral accrual in the adolescent growth spurt. *Journal of Nutrition, 134*, pp. *696S*-700S.

Whyte, G., (2014). Age, sex and (the) race: gender and geriatrics in the ultra-endurance age. *Extreme Physiology. Med*, 3 (1), pp. 1.

Williams, N.I., Helmreich, D.L., Parfitt, D.B., Caston-Balderrama, A., & Cameron, J.L., (2001). Evidence for a causal role of low energy availability in the induction of menstrual cycle disturbances during strenuous exercise training. Journal of Clinical Endocrinology & Metabolism, *86*(11), pp. 5184–93.

Williams, N.I., Koltun, K.J., Strock, N.C., & De Souza, M.J., (2019). Female athlete triad and relative energy deficiency in sport: A focus on scientific rigor. *Exercise & Sport Sciences Reviews, 47*(4), pp.197-205.

Williams, N.I., Leidy, H.J., & Hill, B.R., et al., (2015). Magnitude of daily energy deficit predicts frequency but not severity of menstrual disturbances associated with exercise and caloric restriction. American Journal of Physiology Endocrinology & Metabolism, *308*(1), pp. E29–39.

Williams, N.I., Mallinson, R.J., & De Souza, M.J., (2019). Rationale and study design of an intervention of increased energy intake in women with exercise-associated menstrual disturbances to improve menstrual function and bone health: the REFUEL study. Contemporary Clinical Trials Communications, 14, pp. E100325.

Wilson, G., Hill, J., Sale, C., Morton, J.P., & Close, G.L., (2015). Elite male flat jockeys display lower bone density and lower resting metabolic rate than their female counterparts: implications for athlete welfare. *Applied Physiology Nutrition & Metabolism, 40*(12), pp. 1318–20.

Wonerow, M., Rüst, C.A., Nikolaidis, P.T., Rosemann, T., & Knechtle, B., (2017). Performance trends in age group triathletes in the Olympic distance triathlon at the world championships 2009-2014. *Chinese Journal of Physiology*, 60, pp. 137-150.

Woodruff, S.J., & Meloche, R.D., (2013). Energy availability of female varsity volleyball players. *International Journal of Sport Nutrition & Exercise Metabolism*, *23*(1), pp. 24-30.

World Medical Association, (2013). World Medical Association Declaration of Helsinki ethical principles for medical research involving human subjects. JAMA: Journal of the American Medical Association, 310(20), 2191–2194.

Yates, A., Shisslak, C., Crago, M., & Allender, J., (1994). Overcommitment to sport: Is there a relationship to the eating disorders?. *Clinical Journal of Sport Medicine*, *4*(1), pp. 39-46.

Yeager, K.K., Agostini, R., Nattiv, A., & Drinkwater, B., (1993) The female athlete triad: disordered eating, amenorrhea, osteoporosis. Medicine & Science in Sports & Exercise, *25*(7), pp. 775–77.

Young, S., Rhodes, P., Touyz, S., & Hay, P., (2013). The relationship between obsessive-compulsive personality disorder traits, obsessive-compulsive disorder and excessive exercise in patients with anorexia nervosa: a systematic review. *Journal of Eating Disorders*, *1*, pp.1–13.

Zabriskie, H.A., Currier, B.S., Harty, P.S., Stecker, R.A., Jagim, A.R., & Kerksick, C.M., (2019). Energy status and body composition across a collegiate women's lacrosse season. *Nutrients, 11*(2), pp. 470.

Zanders, B.R., Currier, B.S., Harty, P.S., Zabriskie, H.A., Smith, C.R., Stecker, R.A., Richmond, S.R., Jagim, A.R., & Kerksick, C.M., (2021). Changes in Energy Expenditure, Dietary Intake, and Energy Availability Across an Entire Collegiate Women's Basketball Season. *Journal of Strength & Conditioning Research*, *35*(3), pp. 804-10.

Ethical approval letters studies 1-4:

University of Sunderland

Downloaded: 26/05/2021
Approved: 15/08/2018

Anna Brassell
School of Nursing and Health Sciences
Programme: Doctor of Philosophy

Dear Anna

PROJECT TITLE: Menstrual Cycle, Performance and Recovery in Female Endurance Athletes
APPLICATION: Reference Number 002640

On behalf of the University ethics reviewers who reviewed your project, I am pleased to inform you that on 15/08/2018 the above-named project was **approved** on ethics grounds, on the basis that you will adhere to the following documentation that you submitted for ethics review:

- University research ethics application form 002640 (form submission date: 01/08/2018); (expected project end date: 30/09/2019).
- Participant information sheet 1005086 version 2 (01/08/2018).
- Participant consent form 1005087 version 2 (01/08/2018).

If during the course of the project you need to deviate significantly from the above-approved documentation please email ethics.review@sunderland.ac.uk

For more information please visit: https://www.sunderland.ac.uk/research/governance/researchethics/

Yours sincerely

Karen Robson
Ethics Administrator
University of Sunderland

University of Sunderland

Downloaded: 26/05/2021
Approved: 07/10/2019

Anna Brassell
School of Nursing and Health Sciences
Programme: Doctor of Philosophy

Dear Anna

PROJECT TITLE: Energy Availability in Female Triathletes
APPLICATION: Reference Number 004594

On behalf of the University ethics reviewers who reviewed your project, I am pleased to inform you that on 07/10/2019 the above-named project was **approved** on ethics grounds, on the basis that you will adhere to the following documentation that you submitted for ethics review:

- University research ethics application form 004594 (form submission date: 21/08/2019); (expected project end date: 31/03/2020).
- Participant information sheet 1009035 version 2 (21/08/2019).
- Participant consent form 1009036 version 2 (21/08/2019).

The following optional amendments were suggested:

Please add, in case of complaint, Independent Contact, John Fulton, Chair, UoS Research Ethics Group (john.fulton@sunderland.ac.uk) Again, it is no longer the Research Ethics Committee, but the UoS Research Ethics Group Could you consider adding to the participant documentation that some of the questions (e.g. frequency and character of bowel movement, contraception practices) are personal in nature - do you think it enough to say that participants may omit questions? Again, could you consider adding the safeguard of support services (even if no more than 'see your GP') in the event of distress or concern arising from engagement with the questionnaire?

If during the course of the project you need to deviate significantly from the above-approved documentation please email ethics.review@sunderland.ac.uk

For more information please visit: https://www.sunderland.ac.uk/research/governance/researchethics/

Yours sincerely

Callum Williams
Ethics Administrator
University of Sunderland

Participant information sheets studies 1-3:

University of Sunderland

Female Health Questionnaire

You are being invited to participate in a research study titled Energy Availability in Female Triathletes. This study is being conducted by Anna Brassell from the University of Sunderland.

The purpose of the study is to investigate energy availability in female triathletes. One of the key factors considered to impact upon health and performance parameters in female endurance athletes is energy availability. Currently little research has been conducted in this area on female multisport endurance athletes. Therefore, we are conducting a survey-based study that will take approximately 15 to 25 minutes to complete, firstly to look at the prevalence of those at risk of low energy availability and secondly to investigate the awareness of associated conditions resultant from low energy availability. Could all female triathletes (from all performance levels [recreational to elite/professional]) who read this please answer the survey – not just those who feel they might have an issue with their energy availability.

In order to participate in this study, you must be:

- Female
- 18 years or older
- Pre-menopausal
- Participate in triathlon (all levels of performance accepted)

By completing this survey, you are giving consent for the information you provide to be included in this study. Your participation is voluntary and is specific to this study and shall not be taken to imply consent to participate in any subsequent experiment or deviation from that detailed. All information will remain confidential as to your identity. It will not be possible to withdraw your data once the survey is submitted due to the anonymous approach taken. You do not have to answer any questions you do not want to.

We believe there are no known risks associated with this research study; however, as with any online related activity the risk of a breach is always possible. To the best of our ability your participation in this study will remain confidential, and only anonymised data will be published. All information and data collected during the study will be accessed, stored and analysed by the lead researcher and supervisory team, respectively. Data will be maintained in password protected files on a personal computer which is also password protected. Data will be held for the purposes of publication for 5 years, after which it will be destroyed (paper documents will be shredded and electronic data will be securely erased in line with the University's Information Handling Policy).

If there is anything you do not understand or wish to ask questions about, please feel free to ask.

Thank you for participating.

Anna Brassell

Email: bg13pw@student.sunderland.ac.uk

Ethical Approval

This study has been approved by the School of Nursing and Health Sciences, Ethics Sub-Committee for Sport. Should you need to, you may contact the Chair of the Committee, John Fulton.

Name: John Fulton
Chairperson of Research Ethics Sub-Committee
School of Nursing and Health Sciences
University of Sunderland
Sunderland
Email: john.fulton@sunderland.ac.uk

Participant information sheets study 4:

University of Sunderland

CHANGES IN SLEEP, ENERGY AVAILABILITY AND TRAINING LOAD ACROSS THE TRAINING SEASON

Dear Participant,

I am writing to invite you to participate in a research study which will help identify the changes in sleep, energy availability and training load, across the training and competitive season, in female multisport athletes. Daily athlete monitoring of fatigue, stress and recovery is often employed to optimise athletic performance and minimise the risk of non-functional overreaching, injury and illness. The study will monitor repeated measures of sleep pattern and quality, energy availability, dietary energy intake, exercise energy expenditure, eating attitudes, aerobic fitness, anthropometry and demographics will be obtained. Health history questionnaires to assess training status and menstrual status will also be administered.

Before deciding whether or not you would like to participate in the project, it is important for you to understand what the project will involve. There is an enclosed 'Information for Participants' explaining the study in greater detail. If you do decide you are interested in taking part in the project, please contact the researchers detailed in the information sheet that will provide you with more details.

I have enclosed further information about the project and I hope that you wish to take part. If you would like any further information, do not hesitate to contact me on the contact details below.

With best wishes,

Anna Brassell

Department of Sport and Exercise Science
University of Sunderland
School of Nursing and Health Sciences
City Campus
Sunderland SR1 3SD
Tel: 07810800411
Email: anna.brassell@yahoo.co.uk / bg13pw@student.sunderland.ac.uk

INFORMATION FOR PARTICIPANTS:
CHANGES IN SLEEP, ENERGY AVAILABILITY AND TRAINING LOADS ACROSS THE TRAINING SEASON

Purpose of the Study

The aim of this research is to examine the changes in sleep pattern and quality, energy availability and training load across the training season in female multisport athletes and factors such as eating attitudes. If an imbalance between appropriate training overload and adequate recovery periods develops this can lead to an abnormal training response impacting on athlete health and performance. Daily athlete monitoring of fatigue, stress and recovery is often employed to optimise athletic performance minimise the risk of non-functional overreaching, injury and illness. It is important to monitor the sleep, energy availability and training load of these athletes across the season to determine their risk of low energy availability and to characterise the sleeping quality of these athletes in relation to fluctuations in training load. It is important to understand the inter-relationship between these constructs and how this ultimately impacts on athlete health, and its influence on outcomes of athletic success and failure, if we are to educate individuals on the importance of recovery for athlete health.

What are you expected to do?

Throughout the training season, repeated measures of sleep pattern, sleep quality, energy availability, dietary energy intake, exercise energy expenditure, eating attitudes, aerobic fitness and anthropometry measurements will be taken.

On the first visit to the exercise physiology laboratory at the University of Sunderland, you will be introduced to staff and familiarised with equipment, protocols and the questionnaire. During this visit you will complete a baseline health history questionnaire to assess demographics, training status and menstrual status. You will also complete the initial measurements of body composition and aerobic fitness (VO_{2max}). The VO_{2max} test will involve you completing a standardised, multi-stage, incremental treadmill test which will include a 10-min warm-up. Treadmill gradient will be set at 1% throughout the test, with speed increasing by 1km/h every 3-minutes until exhaustion. An additional test may also be completed on a cycle ergometer (Lode Excalibur Sport), whereby, power will be increased 30W every 3-minutes. Ventilatory data (VO_2, VCO_2, RER and V_E) and heart rate will be collected continuously and in the final minute of each workload, a 0.3µl capillary blood sample will be taken to analyse blood lactate concentration and rating of perceived exertion will be recorded. Measures of body composition will be repeated every two months and aerobic fitness (VO_{2max}) will be repeated every 3 months throughout the training season.

Measurements of dietary energy intake, exercise energy expenditure and sleep pattern will be recorded daily from day 1 of the study for seven consecutive days, and repeated every two months throughout your training season. For energy intake you will be required to complete a detailed online, weighed diet log using the MyFitnessPal app. You will be encouraged to weigh and record all foods, supplements and beverages where possible and record time of day, location and meal type within the app. For exercise energy expenditure you will be required to wear a heart rate monitor for all training sessions and to complete a detailed online, training log using the Strava app which will include type of activity, duration and intensity. For sleep pattern you will be required to complete a simple sleep diary recording

bedtime, wake-up time and daytime nap duration. You will also wear a wrist activity monitor, like a watch, daily throughout the training season (except when swimming and showering) to determine the amount and quality of sleep you are getting.

Finally, at the end of each seven-day recording period you will complete an online questionnaire that will ask you questions related to your sleep quality, stress-recovery state and dietary habits and behaviours throughout that month of training. You will be given a 7-day period to complete this questionnaire.

Risks and Discomforts

- Aerobic fitness tests require highly vigorous exercise which is not without risk; there is a risk of sudden cardiac events, which in rare cases may be fatal.
- Temporary, post-exercise lower limb muscular discomfort, likely to peak 48h post-exercise then disappear.
- You may temporarily damage ("pull) a muscle or ligament.
- Capillary blood sampling (finger-prick) is invasive and raises the risk of cross-infection from blood borne disease (e.g. Hepatitis and HIV).
- If you score particularly high in any component of the validated screening tools assessing the risk of disordered eating behaviours and/or low energy availability, you will receive a letter from the research team suggesting that you make an appointment with your GP.

These risks will be minimised by:

- Completing a comprehensive, pre-participation medical screening questionnaire.
- To minimise any injury, you will be required to complete a 10-minute warm-up with light stretched.
- Collection of blood samples will be performed by trained and competent research students with Hepatitis B vaccination.
- Employment of University of Sunderland standard laboratory techniques for blood sampling, which conform to the British Association of Sport and Exercise (BASES) guidelines (2007)
- The screening tools used in this study do not offer a diagnosis of disordered eating and you would be recommended to speak to a qualified health care professional.

Exclusion Criteria

To ensure your safety we have a clear set of exclusion criteria, listed below.

- Current smokers
- Those with a current or previous diagnosis for cardiovascular, respiratory or metabolic disease (ACSM, 2013)
- Those undergoing investigation for any cardiovascular, respiratory or metabolic disease
- Those with signs and symptoms of cardiovascular, respiratory or metabolic disease (ACSM, 2013) which include:
    - Pain or discomfort in the chest, neck, jaw or arms
    - Shortness of breath at rest or with mild exertion
    - Unusual fatigue/shortness of breath with usual activities
- Those with a family history of cardiovascular, respiratory or metabolic disease (ACSM, 2013)
- Those receiving medical treatment for any medical condition
- Current diagnosis or history of head injury, seizures, epilepsy, sudden fainting, dizziness or other neurological disorder
- Aged under 18 years or over 50 years
- Currently diagnosed or experiencing symptoms of the menopause or perimenopause
- Unable to understand study requirements
- Unable to voluntarily provide consent
- Current or recent (<6-months) lower limb injury
- Novice multisport athlete with <1-years' experience
- Recreational or performance enhancing substance use (illegal or legal)
- Known/Diagnosed sleep-related disorder
- Known/Diagnosed palpitations/tachycardia (resting heart rate >100beats.min^{-1})
- Known/Diagnosed heart murmur
- Resting blood pressure (systolic/diastolic) >140/90mmHg (either measure or both)
- More than two risk factors for cardiac disease (ACSM, 2013)
- Pregnant women or lactating women or those who suspect they may be pregnant (self-reported)
- Male multisport endurance athletes

All will be assessed via the pre-participation health screening questionnaire.

Inclusion Criteria

- Female
- Aged 18-50 years
- Multisport endurance athlete
- Age-group level
- A minimum of 1-year experience in multisport endurance training and events prior to testing
- Performed exercise 4-5 times per week prior to testing
- Intent to complete one race in 2019 season
- Non-smokers
- Apparently healthy (determined from self-report pre-participation health questionnaire)

Responsibilities of the Participant

Your health and safety during the study is of utmost importance to us. It is your responsibility to fully inform the research team if there is any reason why you should not take part. You must provide details of your current and past health status and inform us of any previous adverse responses to high intensity exercise, for example, dizziness, fainting, palpitations, injuries, which would exclude you from participating. It is your responsibility to immediately report any discomfort or unpleasant feelings during the exercise tests. Please inform us of any medications (prescription or non-prescription), nutritional supplements, and performance enhancing and/or recreational substances that you are using (legal or illegal) prior to your participation.

Benefits

The results from this study will be used to guide your future training and will identify your individual responses to multisport endurance training with respect to changes in sleep, energy availability, training load, body composition and aerobic fitness throughout your training season in the lead up to competition. Results will inform coaches and athletes of changes in performance and recovery in female multisport endurance athletes throughout the training season and potential impacts of such a training load on components of athlete health and performance.

Further Information

Any questions about this research study may be directed to the lead researcher or any member of the research supervisory team. Questions are encouraged at any point in time; before, during or on completion of the study.

Anna Brassell (Lead Researcher)

Department of Sport and Exercise Science
University of Sunderland
School of Nursing and Health Sciences
City Campus
Sunderland SR1 3SD
Tel: 07810800411
Email: anna.brassell@yahoo.co.uk / bg13pw@student.sunderland.ac.uk
Dr Lisa Board (Director of Studies)

Department of Sport and Exercise Science
University of Sunderland
School of Nursing and Health Sciences
City Campus
Sunderland SR1 3SD
Tel: 0191 515 2115
Email: lisa.board@sunderland.ac.uk

Use of Data

All information and data collected during the study will be treated as confidential. It will be included in the consent form that participants will have the right to withdraw from the study, without the need to provide reason, at any point during the study. They will be given the option to take any findings that have been taken to dispose of how they deem appropriate and participant results will not be included in the final data analysis or write up of the project. To maintain the participants' anonymity and as an identity protection, all participants will be given a unique identification (ID) number and asked to provide a memorable word or number for if they wish to withdraw their data, known only to the lead researcher and supervisory team. Identification of participants will not be possible to an outside party and data will only be made available to members of the research team. Whereby, third party online apps are utilised, despite the apps having independent data protection regulations in line with current GDPR legislation, there is still a potential for online data breach and it can't be guaranteed that all participant data stored will be deleted. Participant data will remain confidential by removing identifying information from reports and presentations of the data and results will be reported as means ± standard deviation in the final write up of the data. Results will be used for post-doctoral theses and may be presented at National or International scientific conferences or submitted for publication in a scientific journal. Data will be held for the purposes of publication for 10 years, after which it will be destroyed (paper documents will be shredded and electronic data will be securely erased in line with the University's Information Handling Policy). Only group data, not individual data, will be presented to maintain full anonymity and confidentiality. The data is the property of the client and can be withdrawn at any time, without the need to provide reason.

Ethical Approval

This study has been approved by the School of Nursing and Health Sciences, Ethics Sub-Committee for Sport. Should you need to, you may contact the Chair of the Committee, John Fulton.

Name: John Fulton
Chairperson of Research Ethics Sub-Committee
School of Nursing and Health Sciences
University of Sunderland
Sunderland
Email: john.fulton@sunderland.ac.uk

Participant consent studies 1-3 (in addition to appendix 2):

1. By completing this questionnaire, you are providing informed consent to participate in this research. Please email bg13pw@student.sunderland.ac.uk if you would like to be sent the information sheet on this study.
Yes, I provide informed consent to participate in this research.
If did not answer then stop, you have finished the survey.

Participant consent study 4:

University of Sunderland

PARTICIPANT CONSENT FORM (PAGE 1)
CHANGES IN SLEEP, ENERGY AVAILABILITY AND TRAINING LOADS ACROSS THE TRAINING SEASON

Name of Researcher: Anna Brassell

Please circle the appropriate response:

I confirm that I have read and understand the information sheet for the above research project and have had the opportunity to ask questions.	YES / NO
I understand, and agree, to have and complete the stated measurements undertaken.	YES / NO
I agree to fill out questionnaires enquiring about my family history, lifestyle and about my current physical activity levels.	YES / NO
I understand, and agree to fill out questionnaires enquiring about my menstrual status, eating behaviours, dietary habits, sleep quality and stress-recovery state.	YES / NO
I agree to take part in repeated maximal exercise tests. I have been informed of the associated risks.	YES / NO
I agree to have capillary blood samples taken from a fingertip or ear lobe.	YES / NO
I agree to take part in repeated body composition assessments. I have been informed of the associated risks.	YES / NO
I agree to take part in repeated measurements of dietary energy intake, exercise energy expenditure and sleep.	YES / NO
I understand that any questionnaire-based screening tools do not offer a medical diagnosis of respective conditions.	YES / NO
I understand that undergraduate and postgraduate students (under the supervision of academic staff) will assist in the undertaking of my assessments and that they have been given appropriate training.	YES / NO
I understand that all data collected throughout the study will be kept safely and securely.	YES / NO
I understand that my results will remain anonymous unless the researchers need to contact my GP in relation to my results.	YES / NO
I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason.	YES / NO
I understand that upon my request any personal data will be removed from the study database should I wish to withdraw my participation.	YES / NO
I consent that my personal data can be retained in a database by the study researchers for the purposes of research and statistical analysis.	YES / NO

PARTICIPANT CONSENT FORM (PAGE 2)
CHANGES IN SLEEP, ENERGY AVAILABILITY AND TRAINING LOADS ACROSS THE TRAINING SEASON

I understand that in the use of online apps, despite apps having data protection regulations in line with GDPR, there is still potential for online data breach and it can't be guaranteed data stored will be deleted.	YES / NO
I understand that my data may be made available with my anonymity protected to research students for the purposes of fulfilling their research projects.	YES / NO
I understand that the data collected from my participation in this programme can be published in academic/professional journals, and can also be presented at conferences.	YES / NO
I understand that my anonymity will be protected at all times and no individual names will be ascribed to any publication.	YES / NO
I agree that any personal information about me will remain locked in filing cabinets at the University of Sunderland and will be destroyed after 10 years of the conclusion of the study.	YES / NO
I agree to participate fully and understand all responsibilities and requirements that are necessary for the study.	YES / NO
I agree that my personal information may be passed on to emergency medical services (First-aider, paramedic, doctors) should the need arise during the testing.	YES / NO
I understand that the data collected from my participation can be withdrawn at any time, without the need to provide reason.	YES / NO

Name of Participant (print name) Date Participant Signature

....

Name of person taking consent Date Researcher Signature

(Researcher name)

....

Health screening study 4:

University of Sunderland

PRE-PARTICIPATION HEALTH QUESTIONNAIRE (PAGE 1)

This information will not be treated as confidential in a laboratory session. This information regarding research and external clients will be treated as confidential and only members of staff employed by the University of Sunderland, Sport and Exercise Science will have access.

Your safety during participation in this research study is of paramount importance to us. We ask you to complete the following pre-participation medical screening questionnaire. Please answer the questions honestly and thoroughly.

PERSONAL INFORMATION

NAME: **DATE OF BIRTH:**

MEDICAL CONDITIONS:

If you circle/tick 'yes' to any of the following questions please provide further details in the space provided at the end of the questionnaire and please list all current medications.

1. Have you had to consult your Doctor within the last six months?	YES / NO
2. Do you have diabetes?	YES / NO
2a. Insulin Dependent Diabetes Mellitus (IDDM)	
2b. Non-insulin dependent diabetes mellitus (NIDDM)	
2c. How long have you had diabetes?	years
3. Do you have asthma?	YES / NO
3a. Do you have an asthma inhaler?	YES / NO
4. Do you have a chronic obstructive pulmonary disease, ex interstitial lung disease or cystic fibrosis?	YES / NO
5. Has your doctor ever told you that you have heart trouble?	YES / NO
6. Has your doctor ever told you that you have a heart murmur?	YES / NO
7. Has your doctor ever told you that you have circulation problems?	YES / NO
8. Do you have, or have you ever had, high blood pressure?	YES / NO
9. Have you ever had a stroke?	YES / NO
10. Is there a history of heart disease in your family?	YES / NO
11. Do you have, or have you ever had, seizures or epilepsy?	YES / NO
12. Do you have, or have you ever had, any form of illness or injury to the head?	YES / NO
13. Do you have, or have you ever had, any form of liver disorder?	YES / NO
14. Do you have, or have you ever had, any form of kidney disorder?	YES / NO
15. Do you have, or have you ever had, any form of thyroid disorder?	YES / NO
16. Do you have, or have you ever had, any form of cancer?	YES / NO
17. Do you have osteoporosis?	YES / NO
18. Do you have any form of arthritis?	YES / NO
19. Are you, or do you have reason to believe, you may be pregnant?	YES / NO
20. Do you currently have any form of muscle, ligament or joint injury?	YES / NO

SIGNS AND SYMPTOMS:

21. Do you ever have pains in your chest or surrounding areas, especially during exercise?	YES / NO
22. Do you ever get that feeling your heart is beating abnormally, racing, or skipping beats, either at rest or during exercise?	YES / NO
23. Do you ever get pains in your calves, buttocks, or at the backs of your legs during exercise which is not due to fatigue or stiffness?	YES / NO
24. Do you ever feel faint or have spells of severe dizziness, particularly with exercise?	YES / NO
25. Do you ever experience swelling or accumulation of fluid around the ankles?	YES / NO
26. Do you ever experience unusual fatigue or shortness of breath at rest or with mild exertion?	YES / NO
27. Have you ever had an attack of shortness of breath that came on after you stopped exercising?	YES / NO
28. Have you been awakened in the night by shortness of breath?	YES / NO
29. Do you ever have chest tightness when not exercising?	YES / NO
30. Have you ever had chest tightness, cough or wheezing which made it difficult for you to perform in sports?	YES / NO
31. Have you ever been treated or hospitalised for asthma?	YES / NO
32. Have you ever been told to give up sports because of health problems?	YES / NO
33. Have you ever been told you have high blood pressure?	YES / NO
34. Have you ever been told you have high cholesterol?	YES / NO
35. Do you have trouble breathing or do you cough during or after activity?	YES / NO
36. Have you in the last 6 months had an ECG or 24h heart rate trace taken?	YES / NO

PHYSICAL ACTIVITY & LIFESTYLE:

PHYSICAL ACTIVITY DEFINITIONS

Sedentary: No physical activity sessions of 30mins duration or longer per week

Low: Some physical activities on 1-2 days per week but activity sessions are generally less than 30mins

Moderate: Regular physical activity sessions of 30mins or more on 3-5days per week

High: Regular physical activity sessions of at least 30mins on 6-7 days per week

37. Using the definitions above, how would you describe your present level of physical activity?

Sedentary Low Moderately Active Highly Active

38. How would you describe your present level of fitness?

Very unfit Unfit Moderately fit Very fit Elite fitness

39. How would you describe your present body weight?

Underweight Ideal weight Overweight

40. Do you smoke or vape? *If no, please go to question 43.*	YES / NO
41. How many cigarettes do you smoke, on average, per day?	
42. How long have you smoked?	
43. Do you drink alcohol? *If no, please go to question 46.*	YES / NO
44. How many united of alcohol, on average, do you drink per day during the week? (1 unit = ½ pint lager/beer, one shot spirits, one small glass wine)	
45. Have you drunk any alcohol in the last 24 hours?	YES / NO
46. Have you ingested any substances in the last 48/72 hours?	YES / NO

MEDICATIONS:

47. Are you currently taking any form of medications? *If yes, please list all current medications, doses and purposes below.*	YES / NO

Name of medication	Dose	Purpose

Please use the following space to tell us about your medical conditions in more detail.

I confirm that:

(a) I am willing to take part in the research project as a volunteer subject.
(b) I have had no significant illness since my last medical examination.

I understand that:

(a) The member of staff will explain the nature and purpose of each practical session and will inform me of any foreseeable risk to my health as a result of my participation.
(b) I agree to terminate any practical activity if the member of staff in charge feels it is advisable to do so.
(c) I have/will inform the member of staff in charge of any permanent and/or temporary medical condition from which I am suffering or have suffered recently, which might be made worse by physical activity participation.

I authorise the member of staff in charge to inform my general practitioner should he/she feel that any significant untoward event occurs during or after the practical session, which might be a result of my participation.

Name (Participant)	.....	Name (Researcher)	.....
Signature	.....	Signature	.....
Date	.....	Date	.....

GP Contact Details & Emergency Contact Details:

GP Name	.....	Emergency Contact Name	.....
GP Contact Address	.....	Emergency Contact Relationship	.....
GP Contact Telephone	.....	Emergency Contact Telephone	.....

LEAF-Q + scoring key studies 1-4:

1. Injuries **Mark the response that most accurately describes your situation**

A: Have you had absences from your training, or participation in competitions during the last year due to injuries?

☐ No, not at all ☐ Yes, once or twice ☐ Yes, three or four times ☐ Yes, five times or more

A1: If yes, for how many days absence from training or participation in competition due to injuries have you had in the last year?

☐ 1-7 days ☐ 8-14 days ☐ 15-21 days ☐ 22 days or more

A2: If yes, what kind of injuries have you had in the last year? _____

_____

Comments or further information regarding injuries: _____

_____

_____

2. Gastro intestinal function

A: Do you feel gaseous or bloated in the abdomen, also when you do not have your period?

☐ Yes, several times a day ☐ Yes, several times a week

☐ Yes, once or twice a week or more seldom ☐ Rarely or never

B: Do you get cramps or stomach ache which cannot be related to your menstruation?

☐ Yes, several times a day ☐ Yes, several times a week

☐ Yes, once or twice a week or more seldom ☐ Rarely or never

C: How often do you have bowel movements on average?

☐ Several times a day ☐ Once a day ☐ Every second day

☐ Twice a week ☐ Once a week or more rarely

D: How would you describe your normal stool?

☐ Normal (soft) ☐ Diarrhoea-like (watery) ☐ Hard and dry

Comments regarding gastrointestinal function: _____

_____

3. Menstrual function and use of contraceptives

3.1 Contraceptives

Mark the response that most accurately describes your situation

A: Do you use oral contraceptives?

☐ Yes ☐ No

A1: If yes, why do you use oral contraceptives?

☐ Contraception ☐ Reduction of menstruation pains ☐ Reduction of bleeding

☐ To regulate the menstrual cycle in relation to performances etc..

☐ Otherwise menstruation stops

☐ Other_____

_____

A2: If no, have you used oral contraceptives earlier?

☐ Yes ☐ No

A2:1 If yes, when and for how long?_____

_____

_____

B: Do you use any other kind of hormonal contraceptives? (e.g. hormonal implant or coil)

☐ Yes ☐ No

B1: If yes, what kind?

☐ Hormonal patches ☐ Hormonal ring ☐ Hormonal coil ☐ Hormonal implant ☐ Other

_____

3.2 Menstrual function

Mark the response that most accurately describes your situation

A: How old were when you had your first period?

☐ 11 years or younger ☐ 12-14 years ☐ 15 years or older ☐ I don't remember

☐ I have never menstruated (If you have answered "I have never menstruated" there are no further questions to answer)

_____

B: Did your first menstruation come naturally (by itself)?

☐ Yes ☐ No ☐ I don't remember

B1: If no, what kind of treatment was used to start your menstrual cycle?

☐ Hormonal treatment ☐ Weight gain

☐ Reduced amount of exercise ☐ Other

_____

C: Do you have normal menstruation?

☐ Yes ☐ No **(go to question C6)** ☐ I don't know **(go to question C6)**

C2: If yes, when was your last period?

☐ 0-4 weeks ago ☐ 1-2 months ago ☐ 3-4 months ago ☐ 5 months ago or more

C2: If yes, are your periods regular? (Every 28th to 34th day)

☐ Yes, most of the time ☐ No, mostly not

C3: If yes, for how many days do you normally bleed?

☐ 1-2 days ☐ 3-4 days ☐ 5-6 days ☐ 7-8 days ☐ 9 days or more

C4: If yes, have you ever had problems with heavy menstrual bleeding?

☐ Yes ☐ No

C5: If yes, how many periods have you had during the last year?

☐ 12 or more ☐ 9-11 ☐ 6-8 ☐ 3-5 ☐ 0-2

_____

3.2 Menstrual function **Mark the response that most accurately describes your situation**

C6: If no or "I don't remember", when did you have your last period?

☐ 2-3 months ago ☐ 4-5 months ago ☐ 6 months ago or more

☐ I'm pregnant and therefore do not menstruate

D: Have your periods ever stopped for 3 consecutive months or longer (besides pregnancy)?

☐ No, never ☐ Yes, it has happened before ☐ Yes, that's the situation now

---

E: Do you experience that your menstruation changes when you increase your exercise intensity, frequency or duration?

☐ Yes ☐ No

E1: If yes, how? (Check one or more options)

☐ I bleed less ☐ I bleed fewer days ☐ My menstruations stops

☐ I bleed more ☐ I bleed more days

---

1. A: **0** No, not at all, **1** Yes, once or twice, **2** Yes, three or four times, **3** Yes, five times or more

1. A1: **1** 1-7 days, **2** 8-14 days, **3** 15-21 days, **4** 22 days or more

2. A: **3** Yes, several times a day, **2** Yes, several times a week, **1** Yes, once or twice a week or more seldom, **0** Rarely or never

2. B: **3** Yes, several times a day, **2** Yes, several times a week, **1** Yes, once or twice a week or more seldom, **0** Rarely or never

2. C: **1** Several times a day, **0** Once a day, **2** Every second day, **3** Twice a week, **4** Once a week or more rarely

2. D: **0** Normal, **1** Diarrhoea-like, **2** Hard and dry

3.1 A1: **0** Contraception, **0** Reduction of menstruation pains, **0** Reduction of bleeding, **0** To regulate the menstrual cycle in relation to performances etc.., **1** Otherwise menstruation stops

3.2 A: **0** 11 years or younger, **0** 12-14 years, **1** 15 years or older, **0** I don't remember, **8** I have never menstruated

3.2 B: **0**Yes, **1** No, **1** I don't remember

3.2 B1: **1** Hormonal treatment, **1** Weight gain, **1** Reduced amount of exercise, **1** Other

3.2 C: **0** Yes, **2** No (go to question 3.2 C6), **1** I don't know (go to question 3.2 C6)

3.2 C1: **0** 0-4 weeks ago, **1** 1-2 months ago, **2** 3-4 months ago, **3** 5 months ago or more

3.2 C2: **0** Yes, most of the time, **1** No, mostly not

3.2 C3: **1** 1-2 days, **0** 3-4 days, **0** 5-6 days, **0** 7-8 days, **0** 9 days or more

3.2 C4: **0** Yes, **0** No

3.2 C5: **0** 12 or more, **1** 9-11, **2** 6-8, **3** 3-5, **4** 0-2

3.2 C6: **1** 2-3 months ago, **2** 4-5 months ago, **3** 6 months ago or more **0** I'm pregnant and therefore do not menstruate

3.2 D: **0** No, never, **1** Yes, it has happened before, **2** Yes, that's the situation now

3.2 E: **1** Yes, **0** No

3.2 E1: **1** I bleed less, **1** I bleed fewer days, **2** My menstruations stops, **0** I bleed more, **0** I bleed more days

FAST + scoring key studies 1-4:

Key:

Exercise= Physical Activity ≥20 minutes

Practice= Schedule time allotted by coach to work as a team or individually in order to improve performance

Training= Intense physical activity. The goal is to improve fitness level in order to perform optimally.

1. **I participate in additional physical activity ≥ 20 minutes in length on days that I have practice or competition.**
   1) **Frequently**
   2) **Sometimes**
   3) **Rarely**
   4) **Never**
2. **If I cannot exercise, I find myself worrying that I will gain weight.**
   1) **Frequently**
   2) **Sometimes**
   3) **Rarely**
   4) **Never**
3. **I believe that most female athletes have some form of disordered eating habits.**
   1) **Strongly agree**
   2) **Agree**
   3) **Disagree**
   4) **Strongly disagree**
4. **During training, I control my fat and calorie intake carefully.**
   1) **Frequently**
   2) **Sometimes**
   3) **Rarely**
   4) **Never**
5. **I don't not eat foods that have more than 3 grams of fat.**
   1) **Strongly agree**
   2) **Agree**
   3) **Disagree**
   4) **Strongly disagree**
6. **My performance would improve if I lose weight.**
   1) **Strongly agree**
   2) **Agree**
   3) **Disagree**
   4) **Strongly disagree**
7. **If I got on the scale tomorrow and gained 2 pounds, I would practice or exercise harder or longer than usual.**
   1) **Frequently**
   2) **Sometimes**
   3) **Rarely**
   4) **Never**
8. **I weight myself _____.**
   1) **Daily**
   2) **2 or more times a week**
   3) **Weekly**
   4) **Monthly or less**
9. **If I chose to exercise on a day of competition (game/meet), I exercise for**
   1) **2 or more hours**
   2) **45 minutes to 1 hour**
   3) **30-45 minutes**
   4) **less than 30 minutes**
10. **If I know that I will be consuming alcoholic beverages, I will skip meals on that day or the following day.**
    1) **Frequently**
    2) **Sometimes**
    3) **Rarely**
    4) **Never**
11. **I feel guilty if I chose fried foods for a meal.**
    1) **Frequently**
    2) **Sometimes**
    3) **Rarely**
    4) **Never**
12. **If I were injured, I would still exercise even if I was instructed not to do so by my athletic trainer or physician.**
    1) **Strongly agree**
    2) **Agree**
    3) **Disagree**
    4) **Strongly disagree**

13. I take dietary or herbal supplements in order to increase my metabolism and/or to assist in burning fat.
    1) Frequently
    2) Sometimes
    3) Rarely
    4) Never
14. I am concerned about my percent body fat.
    1) Frequently
    2) Sometimes
    3) Rarely
    4) Never
15. Being an athlete, I am very conscious about consuming adequate calories and nutrients on a daily basis.
    1) Frequently
    2) Sometimes
    3) Rarely
    4) Never
16. I am worried that if I were to gain weight, my performance would decrease.
    1) Strongly agree
    2) Agree
    3) Disagree
    4) Strongly disagree
17. I think that being thin is associated with winning.
    1) Strongly agree
    2) Agree
    3) Disagree
    4) Strongly disagree
18. I train intensely for my sport so I will not gain weight.
    1) Frequently
    2) Sometimes
    3) Rarely
    4) Never
19. During season, I choose to exercise on my one day off from practice or competition.
    1) Frequently
    2) Sometimes
    3) Rarely
    4) Never
20. My friends tell me that I am thin, but I feel fat.
    1) Frequently
    2) Sometimes
    3) Rarely
    4) Never
21. I feel uncomfortable eating around others.
    1) Frequently
    2) Sometimes
    3) Rarely
    4) Never
22. I limit the amount of carbohydrates I eat.
    1) Frequently
    2) Sometimes
    3) Rarely
    4) Never
23. I try to lose weight to please others.
    1) Frequently
    2) Sometimes
    3) Rarely
    4) Never
24. If I were unable to compete to my sport, I would not feel good about myself.
    1) Strongly agree
    2) Agree
    3) Disagree
    4) Strongly disagree
25. If I were injured and unable to exercise, I would restrict my caloric intake.
    1) Strongly agree
    2) Agree
    3) Disagree
    4) Strongly disagree
26. In the past 2 years I have been unable to compete due to an injury.
    1) 7 or more times
    2) 4-6 times
    3) 1-3 times
    4) no significant injuries
27. During practice I have trouble concentrating due to feelings of guilt about what I have eaten that day.
    1) Frequently
    2) Sometimes
    3) Rarely
    4) Never
28. I feel that I have a lot of good qualities.
    1) Strongly agree
    2) Agree
    3) Disagree
    4) Strongly disagree
29. At times I feel that I am no good at all.
    1) Strongly agree
    2) Agree
    3) Disagree
    4) Strongly disagree
30. I strive for perfection in all aspects of my life.
    1) Strongly agree
    2) Agree
    3) Disagree
    4) Strongly disagree
31. I avoid eating meat in order to stay thin.
    1) Strongly agree
    2) Agree
    3) Disagree
    4) Strongly disagree
32. I am happy with my present weight.
    1) Yes
    2) No
33. I have done things to keep my weight down that I believe are unhealthy.
    1) Frequently
    2) Sometimes
    3) Rarely
    4) Never

Scoring

4 pts= frequently, 3 pts= sometimes, 2 pts= rarely, 1 pt= never (Reverse #15, 28, 32)

70-84= subclinical disordered eating

>84= clinical eating disorder

McNULTY, K. Y., Adams, C. H., Anderson, J. M., & Affenito, S. G. (2001). Development and validation of a screening tool to identify eating disorders in female athletes. *Journal of the American Dietetic Association*, *101*(8), 886-892.

EDS-R + scoring studies 1-3:

1	2	3	4	5	6
Never					Always

1. I exercise to avoid feeling irritable._____
2. I exercise despite recurring physical problems._____
3. I continually increase my exercise intensity to achieve the desired effects/benefits._____
4. I am unable to reduce how long I exercise._____
5. I would rather exercise than spend time with family/friends._____
6. I spend a lot of time exercising._____
7. I exercise longer than I intend._____
8. I exercise to avoid feeling anxious._____
9. I exercise when injured._____
10. I continually increase my exercise frequency to achieve the desired effects/benefits._____
11. I am unable to reduce how often I exercise._____
12. I think about exercise when I should be concentrating on school/work._____
13. I spend most of my free time exercising._____
14. I exercise longer than I expect._____
15. I exercise to avoid feeling tense._____
16. I exercise despite persistent physical problems._____
17. I continually increase my exercise duration to achieve the desired effects/benefits._____
18. I am unable to reduce how intense I exercise._____
19. I choose to exercise so that I can get out of spending time with family/friends._____
20. A great deal of my time is spent exercising._____
21. I exercise longer than I plan._____

Scale Scoring

The proposed scoring procedure for the *Exercise Dependence Scale* is computer based which allows for immediate and accurate scoring. The computer scoring of the *Exercise Dependence Scale* is based on the SPSS (Statistic Package for the Social Sciences). A syntax file has been developed (see below) by the authors that enables immediate feedback to the *Exercise Dependence Scale* responses once the items are entered into SPSS. The syntax enables:

1. Computing a total and subscale mean scores for *Exercise Dependence Scale-21*. A higher score indicates more exercise dependent symptoms.

2. Categorizing participants into either at-risk for exercise dependent, nondependent-symptomatic, or nondependent-asymptomatic groups. The categorization into one of the three groups is generated by a scoring manual that consists of flowchart decision rules, in which items or combinations or items determine if an individual would be classified in the dependent, symptomatic, or asymptomatic range on each of the 7 DSM criteria. Individuals who are classified into the dependent range on 3 or more of the DSM criteria are classified as exercise dependence. The dependent range is operationalized as indicating a score of 5 or 6 for that item. Individuals who scored in the 3 to 4 range are classified as symptomatic. These individuals may theoretically be considered at-risk for exercise dependence. Finally, individuals who score in the 1-2 range are classified as asymptomatic.

Exercise Dependence Scale (EDS-21) Component Scoring

Component	Item Numbers
Withdrawal Effects	1,8,15
Continuance	2,9,16
Tolerance	3,10,17
Lack of Control	4,11,18
Reduction in Other Activities	5,12,19
Time	6,13,20
Intention Effects	7,14,21

Study 1 supplementary data:

Appendix 8.1. Responses of those female triathletes who are considered at risk for the key components of the LEAF-Q (Injury [cut-off score ≥2] (n = 243); Gastrointestinal Disturbances [cut-off score ≥2] (n = 303); Menstrual function [cut-off score ≥4] (n = 104)).

LEAF questionnaire component	Frequency	Percent
1. Injury history		
Number of days lost from participation due to injury in past year:		
1-7	78	32
8-14	46	19
15-21	43	18
≥22	76	31
Most common injuries reported (respondent can choose more than one):		
Muscular strain/tear	56	23
Iliotibial band friction syndrome (ITB)	17	7
Knee injury	42	17
Hamstring injury	12	5
Achilles tendonitis/ankle injury	41	17
Plantar fasciitis/foot injury	22	9
Stress fractures	23	9
Shoulder injury	13	5
Back injury	21	9
2. Gastrointestinal Disturbances		
Abdominal bloated/gaseous when not having periods:		
Daily - weekly	92	30
Seldom	130	43
Rarely or never	81	27
Cramps/stomach-ache not related to your menstruation:		
Daily - weekly	38	13
Seldom	119	39
Rarely or never	146	48
3. Menstrual Function		
Exercise-related menstrual changes:		
Bleed less	32	42
Menstruation stops	40	52
Bleed more	77	7

LEAF-Q; Low energy availability in female's questionnaire

Appendix 8.2. Responses to the female athlete screening tool as reported by female triathletes (n = 393).

FAST questionnaire component (scores ≥3)	Frequency	Percent
1. I participate in additional physical activity ≥20 minutes in length on days that I have practice or competition.	225	57
2. If I cannot exercise, I find myself worrying that I will gain weight.	295	75
3. I believe that most female athletes have some form of disordered eating habits.	184	47
4. During training, I control my fat and calorie intake carefully.	206	52
5. I do not eat foods that have more than 3 grams of fat.	44	11
6. My performance would improve if I lose weight.	275	70
7. If I got on the scale tomorrow and gained 2 pounds, I would practice or exercise harder or longer than usual.	176	45
8. I weigh myself… (daily or two or more times a week)	122	31
9. If I chose to exercise on the day of competition (game/meet), I exercise for… (2+ hours or 45 minutes to an hour)	37	9
10. If I know that I will be consuming alcoholic beverages, I will skip meals on that day or the following day.	41	10
11. I feel guilty if I choose fried foods for a meal.	222	57
12. If I were to be injured, I would still exercise even if I was instructed not to do so by my athletic trainer or physician.	141	36
13. I take dietary or herbal supplements in order to increase my metabolism and/or to assist in burning fat.	42	11
14. I am concerned about my percent body fat.	234	60
15. Being an athlete, I am not very conscious about consuming adequate calories and nutrients on a daily basis.	78	20
16. I am worried that if I were to gain weight, my performance would decrease.	290	74
17. I think that being thin is associated with winning.	147	37
18. I train intensely for my sport so I will not gain weight.	177	45
19. During season, I choose to exercise on my one day off from practice or competition.	146	37
20. My friends tell me that I am thin but I feel fat.	185	47
21. I feel uncomfortable eating around others.	78	20
22. I limit the amount of carbohydrates that I eat.	161	41
23. I try to lose weight to please others.	61	16
24. If I were unable to compete in my sport, I would not feel good about myself.	307	78
25. If I were injured and unable to exercise, I would restrict my calorie intake.	274	70
26. In the past 2 years I have been unable to compete due to an injury… (7+ or 4 to 6 times)	33	8
27. During practice I have trouble concentrating due to feelings of guilt about what I have eaten that day.	53	14
28. I feel that I don't have a lot of good qualities.	27	7
29. At times I feel that I am no good at all.	210	53
30. I strive for perfection in all aspects of my life.	257	65
31. I avoid eating meat in order to stay thin.	34	9
32. I am not happy with my present weight.	228	58
33. I have done things to keep my weight down that I believe are unhealthy	148	38

FAST, female athlete screening tool

Appendix 8.3. Frequency of individuals at risk across the LEAF-Q, FAST and EDS-R in female triathletes (n=393).

Questionnaire Risk Category			Frequency	Percent
LEAF-Q	FAST	EDS-R		
At risk low EA	ED	At risk EXD	12	3
At risk low EA	DE	At risk EXD	5	1
At risk low EA	No ED	At risk EXD	6	2
At risk low EA	ED	SY	12	3
At risk low EA	DE	SY	44	11
At risk low EA	No ED	SY	46	12
At risk low EA	ED	AS	0	0
At risk low EA	DE	AS	10	3
At risk low EA	No ED	AS	30	8
Not at-risk low EA	ED	At risk EXD	1	0
Not at-risk low EA	DE	At risk EXD	2	1
Not at-risk low EA	No ED	At risk EXD	8	2
Not at-risk low EA	ED	SY	9	2
Not at-risk low EA	DE	SY	30	8
Not at-risk low EA	No ED	SY	88	22
Not at-risk low EA	ED	AS	0	0
Not at-risk low EA	DE	AS	8	2
Not at-risk low EA	No ED	AS	82	21

AS, asymptomatic; DE, disordered eating; EA, energy availability; ED, eating disorder; EDS-R, exercise dependence scale revised; EXD, exercise dependence; FAST, female athlete screening tool; LEAF-Q, low energy availability in female's questionnaire; SY, symptomatic.

Study 2 supplementary data:

Appendix 9.1. Responses to key components of the FAST questionnaire as reported by female triathletes (n = 379)

FAST questionnaire component (scores ≥3)	18 – 29 (years) N=101	30 – 39 (years) N=159	40 – 49 (years) N=119
1. I participate in additional physical activity ≥20 minutes in length on days that I have practice or competition.	62	59	53
2. If I cannot exercise, I find myself worrying that I will gain weight.	84	70	75
3. I believe that most female athletes have some form of disordered eating habits.	55	46	40
4. During training, I control my fat and calorie intake carefully.	57	50	50
5. I do not eat foods that have more than 3 grams of fat.	13	11	10
6. My performance would improve if I lose weight.	65	70	75
7. If I got on the scale tomorrow and gained 2 pounds, I would practice or exercise harder or longer than usual.	55	43	39
8. I weigh myself… (daily or two or more times a week)	23	33	35
9. If I chose to exercise on the day of competition (game/meet), I exercise for… (2+ hours or 45 minutes to an hour)	8	9	11
10. If I know that I will be consuming alcoholic beverages, I will skip meals on that day or the following day.	13	10	9
11. I feel guilty if I choose fried foods for a meal.	63	54	53
12. If I were to be injured, I would still exercise even if I was instructed not to do so by my athletic trainer or physician.	44	33	34
13. I take dietary or herbal supplements in order to increase my metabolism and/or to assist in burning fat.	9	13	9
14. I am concerned about my percent body fat.	56	62	59
15. Being an athlete, I am not very conscious about consuming adequate calories and nutrients on a daily basis.	19	20	22
16. I am worried that if I were to gain weight, my performance would decrease.	78	74	72
17. I think that being thin is associated with winning.	46	32	38
18. I train intensely for my sport so I will not gain weight.	52	44	44
19. During season, I choose to exercise on my one day off from practice or competition.	50	40	25
20. My friends tell me that I am thin but I feel fat.	56	47	41
21. I feel uncomfortable eating around others.	26	18	19
22. I limit the amount of carbohydrates that I eat.	34	42	46
23. I try to lose weight to please others.	27	12	11
24. If I were unable to compete in my sport, I would not feel good about myself.	87	72	77
25. If I were injured and unable to exercise, I would restrict my calorie intake.	77	68	65
26. In the past 2 years I have been unable to compete due to an injury… (7+ or 4 to 6 times)	13	4	8
27. During practice I have trouble concentrating due to feelings of guilt about what I have eaten that day.	20	13	10
28. I feel that I don't have a lot of good qualities.	9	6	6
29. At times I feel that I am no good at all.	65	54	45
30. I strive for perfection in all aspects of my life.	77	66	56
31. I avoid eating meat in order to stay thin.	16	6	8
32. I am not happy with my present weight.	56	61	58
33. I have done things to keep my weight down that I believe are unhealthy.	48	35	30

FAST, female athlete screening tool.
Data presented as percentage.

Appendix 9.2. Frequency of those who meet the cut-off score (≥5) for the seven subscales of the EDS-R as reported by female triathletes classified as at risk for EXD across age groups.

EDS-R questionnaire component	18 – 29 (years) N=16	30 – 39 (years) N=10	40 – 49 (years) N=8
Withdrawal effects	69	90	**88**
Continuance	63	80	63
Tolerance	69	60	75
Lack of control	**88**	**100**	**88**
Reduction of other activities	75	90	50
Time	75	70	**88**
Intention effects	56	60	75

EXD, exercise dependence; EDS-R, exercise dependence scale revised. Data presented as percentage.

Appendix 9.3. Cross-tabulation of Age Group and LEAF-Q score categories.

Age Group (years)		At risk (LEAF-Q)	Not at risk (LEAF-Q)	Total
18-29	Count	49	52	101
	Expected Count	42.4	58.6	
	Column %	30.8	23.6	27% of 379
30-39	Count	64	95	159
	Expected Count	66.7	92.3	
	Column %	40.3	43.2	42% of 379
40-49	Count	46	73	119
	Expected Count	49.9	69.1	
	Column %	28.9	33.2	31% of 379

Pearson chi-square = 2.506; degrees of freed om = 2; p = .286.
LEAF-Q, low energy availability in female's questionnaire.

Appendix 9.4. Cross-tabulation of Age Group and FAST score categories

Age Group (years)		No ED (FAST)	DE (FAST)	ED (FAST)	Total
18-29	Count	61	28	12	101
	Expected Count	66.9	25.1	9.1	
	Column %	24.3	29.8	35.3	27% of 379
30-39	Count	105	38	16	159
	Expected Count	105.3	39.4	14.3	
	Column %	41.8	40.4	47.1	42% of 379
40-49	Count	85	28	6	119
	Expected Count	78.8	29.5	10.7	
	Column %	33.9	29.8	17.6	31% of 379

Pearson chi-square = 4.695; degrees of freed om = 4; p = .322.
DE, disordered eating; ED, eating disorder; FAST, female athlete screening tool.

Study 6 supplementary data:

Appendix 10.1. Responses to key components of the FAST questionnaire as reported by female triathletes (n = 383).

FAST questionnaire component (item scores ≥ 3 points, i.e., agree to strongly agree)	Recreational Age-grouper N=293	Top-Percentile Age-grouper N=90
1. I participate in additional physical activity ≥20 minutes in length on days that I have practice or competition.	57	58
2. If I cannot exercise, I find myself worrying that I will gain weight.	80	60
3. I believe that most female athletes have some form of disordered eating habits.	44	52
4. During training, I control my fat and calorie intake carefully.	55	46
5. I do not eat foods that have more than 3 grams of fat.	12	8
6. My performance would improve if I lose weight.	78	52
7. If I got on the scale tomorrow and gained 2 pounds, I would practice or exercise harder or longer than usual.	44	12
8. I weigh myself… (daily or two or more times a week)	32	9
9. If I chose to exercise on the day of competition (game/meet), I exercise for… (2+ hours or 45 minutes to an hour)	9	12
10. If I know that I will be consuming alcoholic beverages, I will skip meals on that day or the following day.	12	7
11. I feel guilty if I choose fried foods for a meal.	61	46
12. If I were to be injured, I would still exercise even if I was instructed not to do so by my athletic trainer or physician.	38	31
13. I take dietary or herbal supplements in order to increase my metabolism and/or to assist in burning fat.	11	8
14. I am concerned about my percent body fat.	65	44
15. Being an athlete, I am not very conscious about consuming adequate calories and nutrients on a daily basis.	22	13
16. I am worried that if I were to gain weight, my performance would decrease.	77	62
17. I think that being thin is associated with winning.	39	33
18. I train intensely for my sport so I will not gain weight.	49	38
19. During season, I choose to exercise on my one day off from practice or competition.	39	33
20. My friends tell me that I am thin but I feel fat.	52	36
21. I feel uncomfortable eating around others.	21	19
22. I limit the amount of carbohydrates that I eat.	44	32
23. I try to lose weight to please others.	17	12
24. If I were unable to compete in my sport, I would not feel good about myself.	79	78
25. If I were injured and unable to exercise, I would restrict my calorie intake.	71	67
26. In the past 2 years I have been unable to compete due to an injury… (7+ or 4 to 6 times)	7	12
27. During practice I have trouble concentrating due to feelings of guilt about what I have eaten that day.	15	11
28. I feel that I don't have a lot of good qualities.	7	6
29. At times I feel that I am no good at all.	56	47
30. I strive for perfection in all aspects of my life.	66	65
31. I avoid eating meat in order to stay thin.	9	8
32. I am not happy with my present weight.	65	41
33. I have done things to keep my weight down that I believe are unhealthy.	39	37

FAST, female athlete screening tool. Data presented as percentages.

Appendix 10.2. Frequency of those who meet the cut-off score (≥5) for the seven subscales of the EDS-R as reported by female triathletes classified as at risk for EXD across performance levels.

EDS-R questionnaire component	Recreational Age Grouper N=25	Top-Percentile Age Grouper N=9
Withdrawal effects	84	67
Continuance	68	67
Tolerance	64	78
Lack of control	**92**	78
Reduction of other activities	72	78
Time	72	**89**
Intention effects	56	78

EXD, exercise dependence; EDS-R, exercise dependence scale revised. Data presented as percentage.

Appendix 10.3. Cross-tabulation of performance level and LEAF-Q score categories

Performance Level		At risk (LEAF-Q)	Not at risk (LEAF-Q)	Total
Recreational Age-grouper	Count	114	179	293
	Expected Count	119.3	173.7	
	Column %	73.1	78.9	77% of 383
Top-percentile Age-grouper	Count	42	48	90
	Expected Count	36.7	53.3	
	Column %	26.9	21.1	24% of 383

Pearson chi-square = 1.717; degrees of freed om = 1; p = .220.
LEAF-Q, low energy availability in female's questionnaire.

Appendix 10.4. Cross-tabulation of performance level and FAST score categories

Performance Level		No ED (FAST)	DE (FAST)	ED (FAST)	Total
Recreational Age Grouper	Count	196	71	26	293
	Expected Count	192.0	75.0	26.0	
	Column %	78.1	72.4	76.5	77% of 383
Top-percentile Age Grouper	Count	55	27	8	90
	Expected Count	59.0	23.0	8.0	
	Column %	21.9	27.6	23.5	24% of 383

Pearson chi-square = 1.247; degrees of freed om = 2; p = .538.
No ED, no eating disorder; DE, disordered eating; ED, eating disorder; FAST, female athlete screening tool.