

Low Energy Availability In New Zealand Recreational Athletes

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Abstract

Background:

An insufficient energy intake combined with exercise means the body cannot undergo normal physiological functions. This is termed low energy availability (LEA).

To conserve energy LEA triggers a range of endocrine systems, consequently impairing health and athletic performance.

LEA is thought to be common among athletes, however the prevalence amongst New Zealand athletes is unknown. Determining those *at risk* of LEA may help to maximise prevention, early diagnosis and treatment.

Aim: The aim of this study is to estimate the prevalence of recreational New Zealand athletes *at risk* of LEA.

Methods: Participants aged between 18 and 56 years were recruited (109 female, 61 male) via gyms and fitness centres throughout New Zealand. Participants were all classified as recreational athletes, undertaking at least 2.5 hours of moderate physical activity on weekly basis. Participants completed an anonymous online questionnaire comprising of 98 questions from validated eating disorder (Eating Disorder Inventory 3) and LEA questionnaires (Low Energy Availability in Females Questionnaire).

Outcomes: A total of 33.5% (95% CI 26.5%,41.2%) of participants were classified as *at risk* of LEA. Females had approximately 5.4 times greater probability of being *at risk* of LEA when compared to males ($p < 0.001$). For every one unit decrease in BMI the odds of being *at risk* of LEA were 11% higher (OR 0.89, 95% CI 0.80, 1.00, $p = 0.045$). The dose response relationship between training volume (hours per week) and risk of LEA was found to be significant among female athletes but not males. For every extra hour of exercise female participants undertook per week the odds of being *at risk* of LEA were 1.13 times greater (95% CI 1.02, 1.25, $p = 0.016$). Further, the majority of participants (males 87.5%, 95% CI, 47.3%, 99.7%, $n = 7$, females, 89.8%, 95% CI 77.8%, 96.6%, $n = 44$) who were classified as *at risk* of LEA were considered not *at risk* of an eating disorder.

Conclusion: This study provides important information on the prevalence and predictors of LEA in New Zealand athletes. Considering the high prevalence of New Zealand recreational

athletes *at risk* of LEA this emphasises the importance of prevention and early detection, so treatment can be implemented before health and performance is severely compromised.

Preface

This one year, full time equivalent, Masters by thesis only research was conducted in the Department of Human Nutrition, University of Otago, New Zealand. It contains work from February 2014 to February 2015.

Abstracts from this study were presented at two New Zealand conferences in 2014.

Slater J, Black, K, Cooke R, Brown R. Low energy availability amongst New Zealand Athletes (LEANZ) study – Proposed methods. Postgraduate & Early Career Nutrition Conference, 2014.

Slater J, Black, K, Cooke R, Brown R. Low energy availability amongst New Zealand Athletes (LEANZ) study: Proposed methods. Nutrition Society of New Zealand Conference, Abstract 23, 2014.

The abstract presented at the Nutrition Society of New Zealand Conference, was published in *Nutrients* (Slater et al., 2014).

The candidate's supervisors Dr Katherine Black, Rebecca Cooke and Dr Rachel Brown developed the study concept and obtained ethical approval.

The candidate was responsible for:

- The design of the LEANZ questionnaire – In conjunction with the candidate's supervisors Dr Katherine Black, Rebecca Cooke and Dr Rachel Brown.
- Obtaining ethical approval - In conjunction with the candidate's supervisors Dr Katherine Black, Rebecca Cooke and Dr Rachel Brown.
- Consulting with the University of Otago librarians to obtain the EDI-3 resource.
- Development of the online LEANZ questionnaire.
- Recruitment of study participants - Contacting gyms and fitness centres throughout New Zealand and emailing and delivering advertising posters and information sheets to those that agreed to advertise the LEANZ study (Appendix B).
- Designing the nutrition tip sheet participants received for participating in the LEANZ study (Appendix C).
- Pretested the LEANZ questionnaire to determine the estimated time to complete.

- Distributing the nutrition tip sheet and thank you email to participants.
- Preparing (cleaning and coding) the LEANZ questionnaire data for statistical analysis.
- Conducting all statistical analysis under the supervision of Dr Jill Hazard.
- Providing feedback to participants who were classified as being at high risk of an eating disorder and advising them to contact their General Practitioner (Appendix E).

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List of Abbreviations

| | |
|---------|---|
| ACSM | American College of Sports medicine |
| ADA | American Dietetic Association |
| BEDA-Q | Brief Eating Disorder in Athletes Questionnaire |
| BMD | Bone Mineral Density |
| BMI | Body Mass Index |
| BULIT-R | The Bulimia Test-Revised |
| CI | Confidence Interval |
| CT | Computed tomography scan |
| DEXA | Dual-energy X-ray absorptiometry |
| DMPA | Depot medroxyprogesterone acetate |
| DSM-iii | Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised |
| EAT | EAT-26 Eating Attitudes Test |
| EDE-Q | Eating Disorder Examination Questionnaire |
| EDI | Eating Disorder Inventory |
| EDI-B | Eating Disorder Inventory Bulimia sub-scale |
| EDI-BD | Eating Disorder Inventory Body Dissatisfaction sub-scale |
| EDI-DT | Eating Disorder Inventory Drive for Thinness sub-scale |
| EDI-RC | Eating Disorder Inventory Risk Composite Scale |
| EEE | Exercise energy expenditure |
| EI | Energy intake |
| FAT | Female athlete triad |
| FHA | Functional hypothalamic amenorrhoea |
| FODMAPS | Fermentable oligo-, di-, mono-saccharides and polyols diet |
| FSH | Follicle stimulating hormone |

| | |
|--------|--|
| IOC | International Olympic Committee |
| LBM | Lean Body Mass |
| LEA | Low energy availability |
| LEAF-Q | Low Energy Availability in Females Questionnaire |
| LEANZ | Low energy availability in recreational New Zealand Athletes |
| LH | Luteinising hormone |
| NCEA | National Certificate of Educational Achievement |
| NZ | New Zealand |
| OCP | Oral contraceptive pill |
| OR | Odds ratio |
| PA | Physical Activity |
| RED-S | Relative energy deficiency in sport |
| rT3 | Reverse triiodothyronine |
| SD | Standard deviation |
| T3 | Triiodothyronine |
| T4 | Thyroxine |
| TFEQ | Three-Factor Eating Questionnaire |
| USA | United States of America |
| WHO | World Health Organisation |

1 Introduction

For most individuals participation in sport and exercise provides many health benefits. Unfortunately some individuals do not ingest sufficient energy to meet the demands of training and all physiological functions. This leads to a state of low energy availability (LEA) which can negatively effect bone health, cardiovascular function, reproduction, gastrointestinal and mental health. The prevalence of athletes *at risk* of LEA appears to be high with previous reports of 69.2% amongst recreational athletes (Torstveit & Sundgot-Borgen, 2005). The three most researched health consequences of LEA are the clinical end points eating disorders and disordered eating, menstrual irregularity, and stress fractures. However it is now thought that LEA and its associated health consequences occur on a spectrum from health to disease.

Identifying athletes along the spectrum allows for early detection which may prevent these conditions from progressing and reaching the clinical end points (Nattiv et al., 2007).

LEA can occur with or without an associated eating disorder and although any athlete is at risk of LEA, females appear to be at higher risk than males (De Souza et al., 2014). Symptoms of LEA in female athletes have been documented since the early 1960s however research investigating males is lacking (Erdelyi, 1962). Further, athletes competing in sports that emphasise leanness are more at risk than those that do not (Barrack et al., 2014).

At present the research community is debating the exact definitions of LEA and its components, however, there is continuing progress in determining the aetiology, pathophysiology, diagnosis and management of LEA (De Souza et al., 2014; Mountjoy et al, 2014). Continued advancement of research and knowledge of LEA and associated health consequences will aid development of prevention, early detection and treatment strategies. This will allow athletes of all demographics to enjoy exercise, whilst maintaining good health and maximise their sporting performance.

At present, the extent to which New Zealand athletes are at risk of LEA is unknown. This information is required to help appropriately direct resources into early detection of athletes at risk of LEA, which is crucial in protecting their current and long term health. This study aimed to estimate the prevalence of New Zealand recreational athletes at risk of LEA.

2 Review of Literature

2.1 Introduction

Physical activity (PA), sport and exercise are promoted as part of a healthy lifestyle. It is known that PA promotes bone health, improves blood lipid profiles and helps maintain muscle mass (Pate et al., 1995). However, there are certain situations in which exercise can have a negative effect on health, specifically bone health, lipid profiles and hormones, ironically some of the very beneficial health outcomes that exercise promotes (Mountjoy et al., 2014). These effects can occur when an energy deficit exists to such an extent that the body has insufficient energy available to meet the needs of training and normal physiological functioning.

This condition was originally only described in female athletes and was previously known as the Female Athlete Triad (Yeager et al., 1993). Since then the description and definition of this condition has undergone a number of modifications and alterations from the original definition put forward by the American College of Sports Medicine (ACSM) (Yeager et al., 1993) in the early 1990s. At present, debate continues surrounding the description of sub-optimal energy intakes which are now termed Low Energy Availability (LEA) or Relative Energy Deficit in Sport (RED-S) (Mountjoy et al., 2014; Loucks et al., 2011).

This literature review will provide an overview of the history of LEA, as well as discuss literature related to the prevalence, epidemiology, pathophysiology as well as screening and treatment of LEA, both in male and female athletes.

2.2 Low Energy Availability (LEA): A Historical Perspective

2.2.1 Introduction

It is over 50 years since concern was first raised in the published literature regarding the potentially negative influence of excessive exercise in combination with low dietary intakes on the health and performance of athletes (Erdelyi, 1962). Following this first publication no further literature was published until the 1970s and it wasn't until the 1980's and 1990's that a significant body of evidence arose investigating bone health, menstrual function and disordered eating behaviours amongst athletes (Warren et al., 1986, Howat et al., 1989, Marcus et al., 1985, Drinkwater et al., 1990). It was not until 1993 that this state of energy insufficiency in women was termed the "*Female Athlete Triad*" (Yeager et al., 1993). Recently, studies have indicated inadequate energy intake also detrimentally effects males, and thus it has been proposed the condition should be more accurately termed "*Relative Energy Deficiency in Sport*" (RED-S) (Mountjoy et al., 2014) to encompass both males and females.

2.2.2 1960s and 1970s

During the early 1960s, concerns were being raised about the disruption of patterns and timing of the onset of menarche amongst female athletes, particularly those participating at a competitive level (Erdelyi, 1962).

Gyula Erdelyi, a Hungarian Medical Doctor undertook the first large cross sectional study in the area (Erdelyi, 1962) in response to his anecdotal observations of female athletes reporting altered menarche. One specific aim of the study was to answer the question "*what is the influence of sports activities on the menstrual period and menstrual cycle of the female athletes?*". Researchers used questionnaires, personal interviews and menstrual cycle charts to collect data on 729 Hungarian female athletes participating in competitive sport.

It was observed that participation in competitive sport did not alter the timing of onset of menarche. Participants were found to have their first menstrual period at a mean age of 13.6 years, which matched the mean age for the onset of menarche of the wider Hungarian population. It is possible at this time period even female athletes at a competitive level were not doing excess exercise compared to their peers at this age and therefore were at no more risk of LEA and menstrual disturbances.

A subset of these athletes (n=557) were required to report on changes to their menstrual cycle whilst participating in sport, 467 (83.8%) indicated no changes, 28 (5.0%) reported

“*favorable changes*” and 62 (11.1%) detailed “*unfavorable changes*” to their menstrual cycle.

These findings led Erdelyi to state “.... *That we should exclude every pathologic factor which may cause these changes before we should consider sports as the causative factor. However, we may find menstrual disorders that may be associated with too much sports activities.*”. The author suggested that in cases of unfavorable menstrual changes, the athlete should temporarily decrease training load.

In 1977 Bloomberg (1977) reported findings from Dr Kenneth Foreman, a distinguished running coach who collected cross sectional data between 1971 and 1973 to build on the research of Erdelyi (Bloomberg, 1977; Erdelyi, 1962). A pool of 47 female athletes at the National Amateur Athletic Union women’s cross country championships in America completed a questionnaire regarding their menstrual patterns and training load. The results of the questionnaire revealed that 27 athletes had what Dr Foreman described as “*regular*” menstrual patterns (<6 days variation in their menstrual cycle), 9 athletes had “*irregular*” menstrual patterns (variation of 1-4 weeks in menstrual cycle) and 11 athletes had “*very irregular*” menstrual patterns (<2 menstrual cycles per year or not at all). The average number of miles run per week was considerably higher for the participants with irregular and very irregular menstrual patterns compared to those with regular menstrual patterns (79.6 miles and 63.8 miles respectively ($\approx 128\text{km}$ and $\approx 103\text{km}$)). The authors concluded that the harder women train the more likely they are to have irregular menstrual periods.

In 1977 Malina et al., undertook another cross sectional study (n=222) in America, however this time as well as surveying college athletes, they also included high school athletes and non-athletes about the timing of the onset of menarche (Malina et al., 1977). Researchers developed a questionnaire which included self-reported information on the participant’s menstrual history and training schedule. Contrary to earlier findings by Erdelyi (Erdelyi, 1962) this study concluded that athletes at high school, college and Olympic level attained menarche significantly later than non-athletes (mean age, 13.02, 13.05, 14.18 and 12.29 years respectively). It is possible this difference in findings is due to changing attitudes towards female involvement in competitive sports over the previous 16 years. The Title IX amendments of 1972, allowed both sexes equal access to sporting opportunities and funding. Title IX is a comprehensive federal law in the United States of America (USA), that allows equal rights to males and females in any federally funded education programme or activity. The impact of Title IX on the number of females competing in sport was evident at the elite

level. Thirteen percent (13.2%) of participants at the 1964 Tokyo Olympics were females, by 1976 this had increased to 20.7% (International Olympic Committee, 2014). The equalisation of access to sporting opportunities and funding afforded by Title IX may have resulted in females increasing the amount and intensity of training at a younger age, and thereby increasing their risk of being in a state of LEA before or at the time of menarche.

Observations from these early studies suggested female athletes were at risk of experiencing hormonal changes which could influence development and timing of menstruation. However, due to the cross sectional nature of these studies it was not possible to determine the mechanism/s at work. Findings from these observational studies provoked research into the issues surrounding exercise and dietary energy intakes and investigation of possible concerns for athletes overall health.

2.2.3 1980s

A significant body of evidence emerged in the 1980s and early 1990s connecting disordered eating behaviors, amenorrhoea and reduced bone mass (Table 2.1.) (Warren et al., 1986; Howat et al., 1989; Marcus et al., 1985; Drinkwater et al., 1990). In 1984, two case control studies conducted in the USA investigated the bone mineral content of amenorrhoeic and eumenorrhoeic athletes (Drinkwater et al., 1984; Cann et al., 1984).

Drinkwater et al., compared the bone mineral density of 14 amenorrhoeic women runners and rowers to 14 eumenorrhoeic controls matched for sport, age, body mass and height as well as frequency and duration of daily training schedules. Cann et al., did not specifically recruit athletes, instead recruiting 36 amenorrhoeic women from a Reproductive Endocrinology Center, 11 of which had functional hypothalamic amenorrhoea (FHA), however, 10 of these were “regular participants in vigorous exercise programs”. The researchers in this study compared their bone mineral density to 50 eumenorrhoeic matched controls (according to age, but not exercise).

Both studies found that although the radius, which is mostly comprised of cortical bone was unaffected by menstrual status, the vertebrae, which has a higher content of trabecular bone was found to have significantly lower bone mineral content in amenorrhoeic women when compared to eumenorrhoeic women (Cann et al., amenorrhoeic 126.8 (SD 6.8) mg.cm³, controls 165.8 (SD 4.2) mg.cm³, p<0.01, Drinkwater et al., amenorrhoeic 1.12 (SD 0.04) g.cm², eumenorrhoeic 1.30 (SD 0.03) g.cm², p<0.01) .

2.2.4 1990s

In 1990, Drinkwater et al., investigated 97 female American athletes, to determine the relationship between previous menstrual irregularities and current menstrual status to current bone density measured via single and dual photon absorptiometry. Participants were asked to complete a self-reported questionnaire to determine their current and past menstrual status. Current menstrual status was confirmed via four blood tests, seven days apart that were assayed for oestradiol and progesterone levels (Drinkwater et al., 1990).

Participants were divided into three groups: (1) women who had always had regular menses; (2) women who had periods of oligomenorrhoea or amenorrhoea as well as periods of regular menses; and (3) women who are amenorrhoeic now and had been amenorrhoeic or oligomenorrhoeic in the past. The investigators observed a significant linear relationship between the women's current vertebral density and their past and present menstrual patterns. Athletes with a history of regular menses had significantly higher lumbar densities (1.27 g.cm^2) than those with a history of oligomenorrhoea/amenorrhoea combined with regular periods (1.18 g.cm^2). Women who never experienced regular menses had significantly lower lumbar densities compared to those with a history of oligomenorrhoea/amenorrhoea combined with regular menses (1.05 g.cm^2). These results suggest that menstrual irregularities were impacting on bone health, which could potentially lead to severe health problems such as increased fracture risk and osteoporosis.

A further finding from this study was a significant positive correlation between body mass and bone density. This relationship however was also affected by menstrual status, as normal oestrogen levels appeared to negate the adverse effect decreased body mass had on bone density. As menstrual irregularities became more severe the negative association between body mass and bone health became stronger. This is suggestive of an important interaction between menstrual pattern, body mass, and vertebral density and points to a potential role for energy balance plays in menstrual dysfunction (Drinkwater et al., 1990)

Two years later in 1992 the American College of Sports Medicine (ACSM) convened a group of highly regarded researchers, well versed in women's issues, to address this growing area of medical concern within the sporting community. It was during this workshop the term 'the Female Athlete Triad' (FAT) was coined. The FAT was defined as "the combination of disordered eating, amenorrhoea and osteoporosis found in physically active girls and women" (Figure 1.).

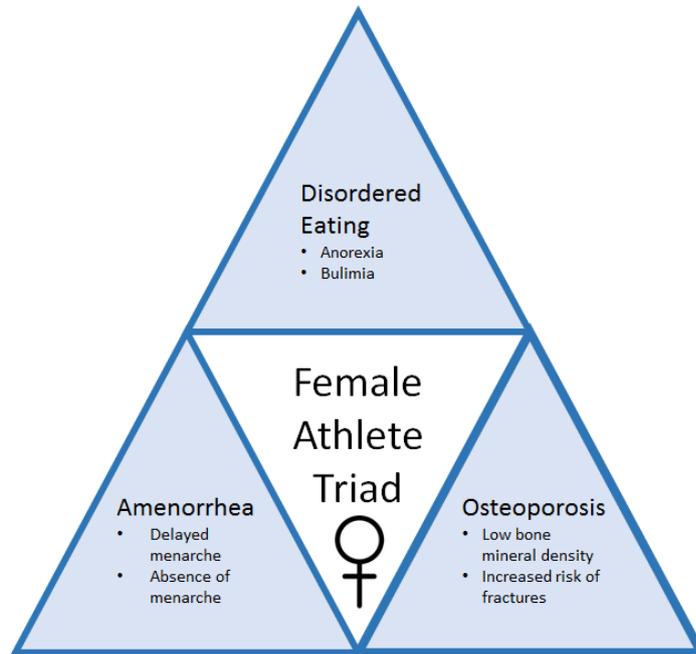


Figure 1 The Female Athlete Triad original model, adapted from “American College of Sports Medicine position stand. The female athlete triad”, by Otis et al., 1997 *Med Sci Sports Exerc.*29(5), 1-4.

Both the medical and practical concerns associated with the FAT were addressed at this meeting. The expert group covered topics including prevention, screening, risk profiling, diagnostic parameters, training parameters, training dynamics, treatment, educational gaps and research needs.

The expert group acknowledged that at this point their understanding of the FAT was limited but that it was a complex medical concern that needed to be researched further.

Some of the points deemed a priority by this expert group were;

- 1) *“To prepare educational materials for athletes, parents, coaches, trainers and administrators.*
- 2) *To educate all sports related healthcare providers, coaches and trainers on the signs symptoms and possible health issues involved with the FAT.*
- 3) *To identify physicians and scientists who are able to address lay and professional audiences on the issues.*
- 4) *To prepare guidelines for team and primary care physicians to follow for pre-participation examinations on female athletes.*

- 5) *To prepare guidelines for the team and primary care physicians to follow regarding prevention, identification, and treatment of these disorders.*
- 6) *To prepare a position paper endorsed by the ACSM establishing a standard of conduct for those responsible for coaching and/or training of female athletes.*
- 7) *To work with the appropriate medical specialty groups to address preventive measures and appropriate treatment of these disorders.*
- 8) *Research needs were identified in the following areas: prevalence, prevention surveillance, body composition, weight management, disordered eating, menstrual dysfunction and osteoporosis*

(Yeager et al., 1993).”

Over the next five years researchers attempted to address some of these priorities.

Further investigations were conducted to assess the prevalence of eating disorders amongst athletes, (Davis & Cowles, 1989; Pasman & Thompson, 1988; Rosen & Hough, 1988) (Table 2.1). Unfortunately the methods used to determine prevalence varied greatly, with unclear definitions of study populations, absent or inappropriate control groups, a narrow spectrum of sports, and non-standard diagnostic criteria for an eating disorder (Table 2.1). Some studies investigated the prevalence of athletes using pathogenic weight control methods or disordered eating behaviours (Sundgot-Borgen & Larsen, 1993; Benson et al., 1990; Rosen & Hough, 1988) whilst others estimated the prevalence of athletes with clinical eating disorders (Sundgot-Borgen, 1993). Therefore it is not surprising the prevalence rates varied substantially between studies, with reported prevalence ranging from 1%-62%.

Rosen & Hough, (1988) carried out a study in the USA on 42 female collegiate gymnasts (Rosen & Hough, 1988). Participants completed the Michigan State University weight control survey, which identifies individuals using pathogenic weight control methods. Participants were classified as using pathogenic weight control methods if they had used one of the following techniques at least twice weekly for three or more months over the past year – self-induced vomiting, use of laxatives, or diuretics for weight loss, regular use of diet pills, fasting for more than one day and/ or fluid restriction for at least one day per week. Over half (62%) of participants reported using at least one form of pathogenic weight control indicating disordered eating behaviours.

Another study, also conducted in the USA (Petrie & Stoeber, 1993), utilised The Bulimia Test-Revised a validated eating disorder questionnaire to detect the presence of bulimic symptoms

in 218 female high school gymnasts. The investigators reported while only 4% of athletes had bulimia, 57.3% were exercising ≥ 2 hours/day, 28.4% were fasting or going on strict diets ≥ 4 x in the past year and 6% vomited ≥ 2 x/month. The results of these studies indicate that serious eating disturbances exist even in the absence of diagnosable eating disorders.

Despite inconsistencies in the methodology used in these studies, trends were emerging (Table 2.1). Researchers reported athletes had comparable frequencies of eating disorders when compared to non-athletes (Sundgot-Borgen, 1993; Sundgot-Borgen & Larsen, 1993; Davis & Cowles, 1989; Pasman & Thompson, 1988). Researchers often grouped athletes from sports with similar characteristics, in particular athletes participating in sports that emphasise leanness or promote a low body mass, such as aesthetic sports or sports that require body revealing clothing. It appeared these athletes had a higher prevalence of disordered eating behaviours than their athletic counterparts competing in sports that do not emphasise leanness (Davis & Cowles, 1989; Borgen & Corbin, 1987; Warren et al., 1990).

Also consistent with non-athlete literature (Leichner, 1986), sex appeared to be a major risk factor in developing an eating disorder, with females having a significantly higher risk than males (Pasman & Thompson, 1988, Wilkins & Boland, 1991; Johnson et al., 1999). Wilkins and Boland (1991), investigated a large sample of male (n=99) and female (n=78) athletes and compared their risk of suffering from an eating disorder to nonathletic controls (39 males, 78 females). Multiple screening tools were used to measure participants risk of an eating disorder including using two validated eating disorder questionnaires - the Eating Attitudes Test – 26 (EAT-26) and the Eating Disorder Inventory (EDI) drive for thinness scale, participants also rated the degree to which they felt pressure to maintain a lean body weight and the degree to which they used dieting and exercise to control their body weight. The results indicated that sex was the most important variable in predicting indices of eating disorders with females at higher risk.

Another study, using the EDI, showed no significant difference in risk of suffering an eating disorder between amenorrhoeic female runners (n=7), eumennorrhoeic female runners (n=9) and non-athlete controls (n=6) (Klock & DeSouza, 1995). Similarly, Rosenvinge et al., (1993) found no significant differences in risk of an eating disorder between 31 elite Norwegian swimmers (19 males and 12 females) and 33 controls (20 males and 13 females) athletes and controls for any of the three EAT-26 scales.

In 1993, Sundgot-Borgen et al., invited Norwegian female athletes (n=603) to participate in a study aimed at determining the prevalence of pathogenic weight control methods and self-reported eating disorders among elite female athletes and non-athlete controls (Sundgot-Borgen et al, 1993). A pool of 522 athletes consented to take part and an equal number of matched controls were recruited. Athlete participants represented 35 different sports, which were divided into six groups: technical sports, endurance sports, aesthetic sports, weight-dependent sports, ballgames and power sports. Participants were required to complete a self-administered questionnaire developed by the study investigators which included questions regarding weight history, menstrual history, physical activity patterns, dieting history, nutritional habits and the use of pathogenic weight control methods. Participants also completed the EDI.

The results of the study showed no significant difference ($p=0.08$) between athletes and controls in the prevalence of participants at risk of an eating disorder with 22% of athletes and 26% of controls meeting the criteria. However a significantly higher percentage of athletes competing in aesthetic sports and weight-dependent sports, were at risk of an eating disorder compared to athletes competing in ballgames and power sports (where leanness is considered less important). A significantly lower percentage of athletes competing in endurance sports were at risk of an eating disorder compared to those competing in aesthetic sports and weight-dependent sports. More athletes than controls reported the use of at least one pathogenic weight control method such as vomiting or inappropriate use of diuretics and laxatives ($p<0.05$). Specifically the prevalence of pathogenic weight control methods was significantly higher in athletes participating in the sports that emphasise leanness including aesthetic sports (16%) and weight-dependent sports (17%) compared to those competing in ballgames (8%) and power sports (6%) and compared to non-athlete controls (7%) ($p<0.05$).

These results suggest important trends, however it is important to note a significant difference in prevalence of eating disorders was found between the sports categorised in the endurance group. This ranged between 0% and 33.3% (95% CI, 31.4, 34.5) (Sundgot-Borgen, 1993). Such results suggest this more general grouping of sports may cause these more subtle yet important differences to be lost and this subjective grouping of sports may not be reliable. It is possible if the sports in the endurance group had been considered as separate groups, some of these may have shown significantly higher scores on the EDI compared with non-athletes. It is also important to acknowledge that in some instances sample sizes were small (sample sizes ranging from n=30 (Benson et al. 1990) and n=60 (Abraham, 1996) to n=19 males and 12 females

(Rosenvinge 1993) and n= 7 amenorrheic runners, 9 eumenorrheic runners, and 6 eumenorrheic sedentary women (Klock & DeSouza, 1995) and may not have been large enough to detect any effect with statistical power therefore limiting the ability to draw firm conclusions (Klock & DeSouza, 1995; Benson et al. 1990; Rosenvinge 1993; Abraham, 1996).

2.2.5 Late 1990s and Early 2000s

The ACSM published the first position stand on the Female Athlete Triad in 1997 (Otis, 1997). The major aim of this document was to inform physicians, trainers, and other healthcare providers about the triad by providing scientific information relating to screening, diagnosis, prevention and treatment.

As discussed at the 1992 ACSM FAT meeting the diagnostic criteria for having the FAT was presence of disordered eating behaviours, amenorrhoea and osteoporosis occurring in sequence (Yeager et al, 1993). Amenorrhoea was defined as a “persistent absence of menstrual cycles” for at least three months. Disordered eating was described as occurring on a continuum, ranging from abnormal eating behaviours at a subclinical level, through to clinical eating disorders including anorexia nervosa, bulimia nervosa and eating disorders not otherwise specified. Osteoporosis was described as having a bone mineral density more than 2.5 SD below the mean of young adults. A further purpose of the position stand was to highlight the serious short and long term health consequences of the triad for female athletes. The 1997 ACSM position statement not only documented the current situation with respect to the FAT, but highlighted the need for further research investigating the prevalence, causes, prevention and treatment of the syndrome.

However, even by this stage, discussions were emerging in the literature regarding the definitions of the FAT components, and in particular concern about the FAT only including the clinical end points of each component. There was concern that many female athletes with less severe manifestations of the triad components had the potential to go unnoticed, yet still faced significant health implications.

Although research had been carried out on the ovulatory status of exercising females in the late 1980s (Loucks et al, 1989) researchers became increasingly interested in the prevalence of menstrual abnormalities less obvious than the female affected by an absence of menses.

De Souza *et al* (1998) undertook a prospective observational study on moderately active females to estimate the prevalence of luteal and ovulatory abnormalities. Participants were

included if they were between the ages of 18 and 36 years and had consecutive, asymptomatic, regular menstrual cycles 24-36 days in length. Women with oligomenorrhoea (menstrual cycle 39-90 days) or amenorrhoea were excluded. Of the 46 females screened, 35 met the inclusion criteria. Participants were originally split into two groups, inactive (n=11) and exercising (recreational runners n=24). Participants recorded the first and last day of menses for each menstrual cycle throughout the study and collected 8-hour urine samples on day two, three or four of each menstrual cycle. Urine was analysed for creatinine, total follicle stimulating hormone (FSH), luteinising hormone (LH), pregnanediol-3-glucuronide and oestrogen conjugates, from which ovulatory status was determined. The results showed 91% of the inactive females were ovulatory with the remaining 9% demonstrating luteal phase deficiency. However, less than half (42%) of the exercising females were ovulatory, 42% demonstrated luteal phase deficiency and 16% were anovulatory.

A group of Canadian researchers undertook a cross sectional study in the early 1990s investigating the association between asymptomatic menstrual disturbances (anovulation and luteal phase deficiency) and bone mineral density (Prior et al, 1990). They recruited 66 premenopausal women aged 21-42 years. The study was carried out over a year and participants were required to provide a blood sample during their first and last menstrual cycle of the study year. These were analysed for LH, FSH, progesterone, testosterone, estradiol, prolactin, cortisol and triiodothyroine. Bone mineral density was measured twice via quantitative computed tomography at a mean \pm SD interval of 12.0 ± 1.8 months apart. Results showed over a one year period subclinical ovulatory disturbances, including anovulation and a short luteal phase, were associated with loss of spinal trabecular bone. Further they found the mean luteal phase length over the year was the best predictor of change in trabecular bone.

This finding of an association between sub clinical menstrual disturbances and bone health as well as consistently high estimations of the prevalence of more subtle menstrual disturbances in exercising women raised the question whether luteal suppression occurred somewhere on the continuum between regular menstrual cycles and amenorrhoea.

Similar research investigating the prevalence of bone mineral density in athletes suggested osteoporosis is very rare in athletes, although the prevalence of osteopenia was increased (Micklesfield et al., 1995; Carmichael & Carmichael, 1995; Lauder et al., 1999) Young et al, investigated menstrual disturbances in 44, 17 y old school students at an elite ballet school in Melbourne, Australia. Twenty two percent of dancers met the World Health Organisation

(WHO) diagnostic criteria for osteopenia while no dancers met the criteria for osteoporosis (Young, 1994). This led the research community to conclude that osteoporosis can, and does, occur in athletes, however because of the very low prevalence rates, by including osteoporosis as part of the diagnostic criteria for the FAT, the number of female athletes who can be clearly defined as suffering from FAT is severely limited (Frusztajer et al., 1990; Rencken et al., 1996). Furthermore, the definition of the FAT at this time highlighted the role of disordered eating in the development and maintenance of amenorrhoea and associated poor bone health in physically active females. As cross sectional studies started to incorporate multiple aspects of the triad it became evident that menstrual dysfunction and bone health were occurring in female athletes both with and without an eating disorder or disordered eating behaviours (Beals & Manore, 2002).

An underlying belief of many researchers in the FAT area at this time, which dated back to the 1960s when Erdelyi first stated “...we may find menstrual disorders that may be associated with too much sports activities”, was that strenuous exercise is the factor that can disrupt reproductive function in females (Erdelyi, 1962, Bloomberg, 1977; Loucks et al., 1989, Bullen et al., 1985). Beginning in 1994 American researchers Loucks and colleagues challenged this and proposed the idea that Low Energy Availability (LEA) which can occur with or without an associated eating disorder may be the driving factor in the development of menstrual disruptions and thus the FAT (Loucks & Heath, 1994). Research had already shown that women with FHA display a reduced luteinizing hormone pulsatility in waking hours and increased luteinizing hormone amplitude in sleeping hours when compared to eumenorrhoeic controls (Khoury et al., 1987).

In order to investigate the theory that LEA could have an impact on normal menstrual function, a series of randomised crossover controlled trials were carried out manipulating both dietary intake and exercise energy expenditure. The results of these studies revealed a close association between energy availability and variation of the Gonadotropin-releasing hormone (GnRH) pulses (Loucks & Callister, 1993; Loucks & Heath, 1994; Loucks et al., 1998; Loucks, 2003).

Louckes et al., initially investigated the effect of dietary energy restriction on gonadotropins (Loucks & Heath, 1994). This was then used as the first phase of a study to differentiate the independent effects of energy availability and exercise stress on luteinizing hormone pulsatility in exercising women (Loucks et al., 1998). A sample of seven young, inactive women who had regular menstrual cycles were assigned in random order to two groups: energy balance and

energy restricted. Participants were provided with a liquid dietary supplement (Ensure®), to consume as their sole dietary intake, set at an energy availability of either 45 kcal.kgLBM⁻¹.day⁻¹ (balanced) or 10 kcal.kgLBM⁻¹.day⁻¹ (restricted) for four days beginning on day five, six or seven of their menstrual cycle.

On day eight, nine or ten in the follicular phase of each participant's menstrual cycle, an intravenous catheter was inserted into the forearm. Blood samples were drawn at 10 minute intervals over a 24 hour period, and assayed for LH. Serum cortisol, FSH, oestradiol, plasma glucose and growth hormone were also measured at various time points.

Phase two of the study involved similar methods as phase one, the major difference was as well as consuming the liquid dietary supplement to control energy intake, participants were required to exercise, therefore the LEA was not directly caused by dietary restriction. A total of 9 participants were recruited, they were required to expend 30 kcal.kgLBM⁻¹.day⁻¹ for the four days they consumed the liquid dietary supplement (Ensure®), this time researchers set energy availability at either 45-50 (balanced) or 10-15 (deprived) kcal.kgLBM⁻¹.day⁻¹.

Results from the second phase of the study suggested that being in a state of LEA caused by exercise energy expenditure reduced LH pulse frequency by 11.3% during waking hours, this was 14.9% smaller than the effect of LEA caused by dietary energy restriction. In the exercising women, LEA increased LH amplitude by 36% during waking and sleeping hours, a similar result to the previous study where LEA caused by dietary restriction increased LH amplitude by 40%. Taken together, these results suggest that aside from the impact of the energy cost on energy availability, exercise has no disruptive effect on reproductive function. Energy availability however has a direct effect on ovulatory status (Loucks & Heath, 1994; Loucks et al., 1998).

The most recent in these series of studies, (Loucks, 2003) investigated 29 inactive eumenorrhoeic women, to determine whether LH pulsatility is disrupted at a threshold of energy availability in this population. Participants were randomly allocated to one of three energy availability treatments (10, 20 or 30 kcal.kgLBM⁻¹.day⁻¹). The trial lasted 5 days during the early follicular phase of the menstrual cycle where participants underwent trials in which diet and exercise were controlled. The results indicated energy availability of 30 kcal.kgLBM⁻¹.day⁻¹ had no effect on LH pulsatility. However, at energy availability below 30 kcal.kgLBM⁻¹.day⁻¹, LH frequency decreased and LH amplitude increased. Furthermore, participants with

shorter luteal phases appeared to be most sensitive to decreases in energy availability, showing the largest disruption in LH pulses.

A further training study which employed a dietary intervention produced results consistent with the studies previously outlined. A small sample size of four amenorrhoeic females between the ages of 18 and 34 y were recruited. Each participant performed at least seven hours of exercise per week. Participants completed a questionnaire regarding health, exercise and diet as well as self-reported menstrual and weight history, developed by the study investigators (Kopp-Woodroffe, 1999). Blood samples were drawn for hormone analysis. Participants then began a 20 week diet and exercise intervention. The intervention required participants to decrease energy expenditure by adding one rest day to their current exercise program, and increase energy intake by adding a daily serve (360kcal) of Gatorade Pro® to their current diet. The results showed that not only did this intervention significantly improve energy balance, but menstruation resumed in 3 out of 4 participants during or just after the 20 week intervention programme, again suggesting athletic amenorrhoea is controlled by energy availability (Kopp-Woodroffe, 1999). Collectively, results from the studies outlined above provide evidence that the female athlete triad is more fluid and complex than the original 1997 ACSM model had described.

2.2.6 2000s

In 2002 the Female Athlete Triad Coalition was formed. This not-for-profit coalition consists of representatives from organisations around the world including, the ACSM, International Olympic Committee (IOC) and American Dietetic Association (ADA). The Female Athlete Triad Coalition is dedicated to improving knowledge of the FAT and decreasing the prevalence through education, international leadership, public policy and research.

In response to mounting evidence that the diagnostic criteria of the FAT (namely, disordered eating, amenorrhoea and osteoporosis) were too strict, in 2003 the ACSM assembled a writing team to update the original 1997 position stand. However, conflicting opinions within the scientific community delayed the publication of this new position stand until 2007 (Nattiv et al, 2007). Those opposed to the promotion of the FAT as a major health issue for female athletes argued women should be encouraged to be physically active. They argued that publicising potential negative outcomes from physical activity could contravene global efforts to increase physical activity as part of the fight to combat rising obesity levels (DiPietro et al, 2006). It was thought that this could cause confusion for active females and compromise public

health programs designed to encourage females to lead an active lifestyle in order to derive the associated health benefits. Opponents also stressed the efforts that had gone into allowing females to reach the same level of accessibility in competitive sports as males, and fear that promotion of the FAT would hinder this success. Opponents also suggested that redefining the components of the FAT and describing its components on a continuum would allow a large percentage of females both athletes and non-athletes to be classified as “at risk” of this supposedly “life threatening” syndrome by just having subclinical symptoms of one component.

In response to this challenging opinion, the updated 2007 ACSM Position Stand clearly states that the benefits of exercise far outweigh the risks, and emphasises women should be encouraged to participate in exercise. The position stand outlines that energy availability is reduced by extreme dietary restriction, extreme exercise expenditure or a combination of these factors and because LEA has been shown to have adverse effects on reproductive function and bone health, female athletes need to take care to avoid LEA.

In the updated position stand the definitions of the triad components have been modified to energy availability (with or without an associated eating disorder/disordered eating behaviors), menstrual function and bone mineral density (Nattiv et al, 2007). A new model (Figure 2.) presenting the triad as a three dimensional sliding scale, where the athlete can fall anywhere between the disease state (osteoporosis, disordered eating/ energy deficiency and/or amenorrhoea) and good health (good bone health, healthy eating habits/energy status and normal ovulatory menstrual cycles) is also presented.

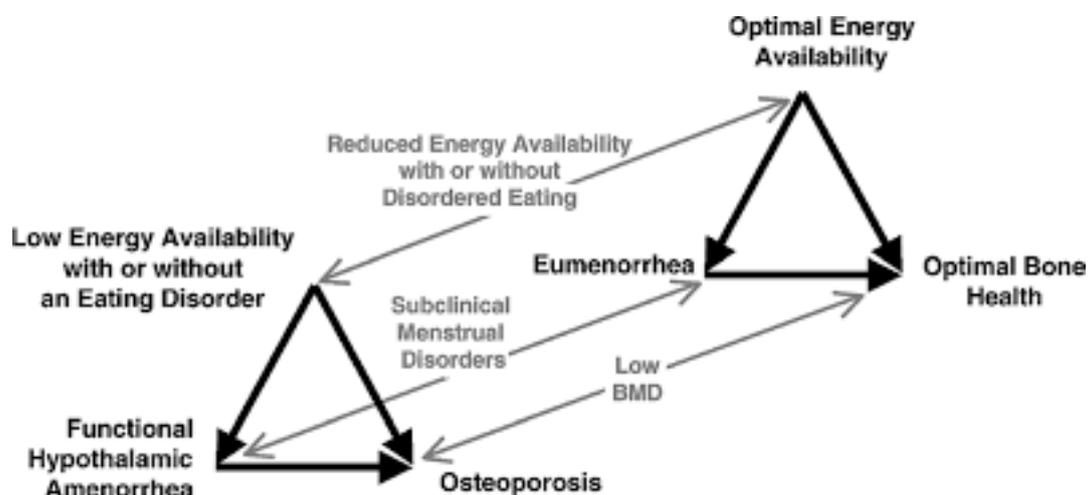


Figure 2 The Female Athlete Triad updated model. Reprinted with permission from “American College of Sports Medicine position stand. The female athlete triad,” by Nattiv et al., 2007 *Med Sci Sports Exerc.* 39(10), 1867-1882.

The new model recognises an important feature of the triad: an athlete can fall anywhere along the continuum for each separate condition at any one time.

In 2014, a comprehensive consensus statement on diagnosis, treatment and return to play on the Female Athlete Triad was published by the Female Athlete Triad Coalition (De Souza et al., 2014) which has been endorsed by the ACSM and the American Bone Health Alliance. Very soon after the IOC published their latest consensus statement (Mountjoy et al., 2014). While both groups aimed to supplement the ACSM 2007 revised position stand on the FAT, they do highlight a divide and difference of opinions that has developed amongst well respected researchers in this area. A major modification included within the updated IOC consensus statement is the proposal of a new name to replace the term FAT (Mountjoy et al., 2014). Relative Energy Deficiency in Sport (RED-S) describes the wide range of adverse effects on various body systems beyond those described by the FAT and recognises males too can suffer the negative health and performance implications of LEA. The authors of the updated IOC consensus statement also proposed a traffic light system for athletes to assess risk and readiness to return to play. The authors of the Female Athlete Triad Coalition statement also proposed a tool for risk assessment and return to play (De Souza, 2014; Mountjoy et al., 2014). However, they refute the concept of RED-S and argue the original model of the FAT should continue as the consequences for females are more severe than for males and because it is clear LEA has a causal relationship with menstrual dysfunction and impaired bone health

Low energy availability (LEA) has attracted considerable attention from the research community over the past 35 years. As a result our knowledge of its complexity has also increased. However, debate remains over the appropriate terminology for this complex clinical syndrome.

2.2.7 The Current Understanding of Low Energy Availability (LEA)

It is evident males can also suffer from LEA (Vogt et al, 2005; Rector et al., 2008; Smathers et al., 2009; Thiel et al., 1993). Vogt et al., (2005) investigated 11 professional, male cyclists to compare their exercise energy expenditure with their energy intake over six days. Results indicated athletes were in negative energy balance as low as -7.9 ± 1.1 MJ on training days, however investigators did not measure any health effects. However despite documentation of LEA existing in males there remains a dearth of evidence documenting the extent of its existence within the male athlete and non-athlete populations.

Unfortunately, the use of the original Female Athlete Triad terminology may have unintentionally affected the progression of research in males and may not actually be appropriate to use when investigating males populations. It therefore remains unclear whether LEA affects males to the same extent and severity as females and whether its prevalence is as high. It may be premature to advocate males suffer from the same syndrome as females, as the term RED-S suggests. One thing is certain, researchers do agree the underlying driving factor resulting in this complex clinical syndrome is LEA. Therefore we decided to use the term LEA to describe this medical state that can lead to a complex sequence of health effects.

Table 2.1 History of the prevalence of low energy availability (LEA) among athletes.

| Reference | Design | Participants | Component of LEA | Instrument used | Definition of LEA component | Prevalence and study outcome | Prevalence of two or more components of LEA |
|------------------------------------|-----------------|--|------------------|---|---|--|---|
| Erdelyi, 1962, Hungary | Cross sectional | 557 females (age not specified) Competitive sport | Amenorrhoea | Menstrual cycle charts | “Unfavorable changes” to menstrual cycle | 11.1% | N/A |
| Foremen, 1977, USA | Cross sectional | 47 females (age not specified) Cross country runners Competing at national championships. | Amenorrhoea | Self-reported questionnaire developed by researchers | “Irregular” - variation of 1-4 weeks in menstrual cycle. “Very irregular” - ≥ 2 menstrual cycles/year | 19.1% 23.4% | N/A |
| Drinkwater et al, 1984, USA | Cross sectional | Amenorrhoeic female athletes (24.9 \pm 1.3y)- runners (n=11), crew members (n=3), Eumenorrhoeic well matched controls (25.5 \pm 1.4y) - runners (n=11), crew members (n=3), | BMD | Dual photon absorptiometry | Comparing scores to determine if there is a significant difference between groups | Radius: didn't differ between groups Lumbar vertebrae significantly lower in amenorrhoeic group (p<0.01). | N/A |
| Cann et al, 1984, USA | Cross sectional | 36 Females (16-49 y) Recruited from a Reproductive Endocrinology Centre when attending clinic for disorders associated with amenorrhoea. (11 had hypothalamic amenorrhoea, 10 of which regularly participated in vigorous exercise) 50 eumenorrhoeic matched controls (according to age, not exercise) | BMD | Trabecular (vertebral); CT Cortical (radius); photon absorptiometry. | Comparing scores to determine if there is a significant difference between groups | Radius: didn't differ between groups Lumbar vertebrae significantly lower in amenorrhoeic group | N/A |

Abbreviations BMD = Bone mineral density, CT = Computed tomography scan

Table 2.1 History of the prevalence of low energy availability (LEA) among athletes (continued).

| Reference | Design | Participants | Component of LEA | Instrument used | Definition of LEA component | Prevalence and study outcome | Prevalence of two or more components of LEA |
|---|-----------------|---|------------------|---|--|---|---|
| Pasman & Thompson, 1988, USA | Cross sectional | 15 males and 15 females in each of the 3 groups (18-60 y) Runners, weightlifters, and inactive controls | Eating disorder | EDI – DT, B, BD | Comparing scores to determine if there is a significant difference between groups. | Runners and weightlifters showed greater eating disturbances than controls; females had greater disordered eating than males | N/A |
| Rosen & Hough 1988, USA | Cross sectional | 42 female gymnasts (17-22 y) College representative | Eating disorder | Michigan State university weight control survey | Disordered eating using pathogenic weight control methods $\geq 2x/week$ for ≥ 3 months over the past year. | 62% | N/A |
| Davis & Cowles 1989, Canada | Cross sectional | Female athletes 64 in thin build sports (mean age 19.31 ± 5.11) and 62 in normal build sports (mean age 21.73 ± 3.05) 64 recreational athlete controls mean age 20.97 ± 3.80 | Eating disorder | EDI | Comparing scores to determine if there is a significant difference between groups. | Thin build sports significantly higher score than normal build sports on drive for thinness scale of EDI but not significantly different to controls. | N/A |
| Warren et al., 1990, USA | Cross sectional | 74 female competitive athletes (gymnasts, runners) Non-competitive college athlete controls 52 non-athletic controls (Mean age 19.3 ± 1.6 y) | Eating disorder | EDI EAT | Comparing scores to determine if there is a significant difference between groups. Score >30 | Gymnasts significantly higher than runners on EDI - DT scale. No difference between athletes & controls EDI-BD – gymnasts & non-athlete controls more dissatisfied than runners No significant difference between groups for EAT score Gymnasts 20% Runners 0% Athlete controls 17% Non-athletic controls 12% | N/A |

Abbreviations USA = United States of America, EAT-26 Eating Attitudes Test, DSM-iii = Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised, EDI = Eating Disorder Inventory

Table 2.1 History of the prevalence of low energy availability (LEA) among athletes (continued).

| Reference | Design | Participants | Component of LEA | Instrument used | Definition of LEA component | Prevalence and study outcome | Prevalence of two or more components of LEA |
|---|-----------------|--|------------------|--|--|---|---|
| Benson et al., 1990 Switzerland | Cross sectional | 12 adolescent female gymnasts (mean age 12.5 ± 1.1 y) and 18 swimmers (mean age 12.8±0.9 y) 34 non-athletic adolescent controls (mean age 13.4±1.2y) | Eating disorder | EDI | Scored “high” on the body dissatisfaction EDI sub-scale | 38% - Swimmers 1% - Gymnasts 9% - Control | N/A |
| Wilkins & Boland, 1991 Country not specified | Cross sectional | Athletic group - 99 males (20.7 ± 2.26y) , 78 females (20.51 ±1.93y) Nonathletic controls - 39 males (21.85 ± 5.34y) and 78 females (19.95 ± 2.77y) | Eating disorder | EAT-26, EDI-DT, the restraint scale, the binge scale Participants rated pressure to maintain a lean body weight and degree they used dieting and exercise to control body weight. | Comparing scores to determine if there is a significant difference between groups | More athletes at risk of an eating disorder than non-athletes Females more likely to be at risk of an eating disorder than males Athletes less likely to diet for weight control and less likely to perceive themselves as overweight. Level of competition or level athletes felt pressure to maintain a lean body weight for their sport not associated with risk. | N/A |
| Petrie & Stoever, 1993, USA | Cross sectional | 218 female college representation gymnasts (Mean age 19.4±1.1 y) | Eating disorder | BULIT-R | Scoring over 104 in BULIT-R to be classified as bulimic At least one pathogenic weight loss behaviour | 4.1% - Bulimic 57.3%-Exercising ≥2 hours/day 28.4% - Fasting or going on strict diets ≥4x in the past year 6% - Vomited ≥2x/month 0.5% - Using diuretics 2.4% - Using laxatives | N/A |
| Thiel et al, 1993, Germany | Cross sectional | 84 male wrestlers and rowers (mean age 21.1± 2.4 y) | Eating disorder | EDI | Disordered eating | 11% | N/A |

Abbreviations EAT-26 = Eating Attitudes Test, EDI = Eating Disorder Inventory, EDI-DT = Eating Disorder Inventory Drive for thinness sub-scale, USA = United States of America, BULIT-R = The Bulimia Test-Revised

Table 2.1 History of the prevalence of low energy availability (LEA) among athletes (continued).

| Reference | Design | Participants | Component of LEA | Instrument used | Definition of LEA component | Prevalence and study outcome | Prevalence of two or more components of LEA |
|-----------------------------|-----------------|--|------------------|-------------------|--|--|---|
| Sundgot-Borgen 1993, Norway | Cross sectional | 522 female elite athletes (representing 35 sports) and 448 controls (age range 12-35 y) | Eating disorder | EDI DSM-iii | Score >40 on EDI If DSM-iii is met classified as Anorexia nervosa or bulimia nervosa | 18% - Athletes 5% - Controls | N/A |
| Sundgot-Borgen, 1994 Norway | Cross sectional | Stage 1 522 elite athletes (representing 35 sports) (age range 12-35 y) Stage 2 -103 (of the 117) female elite athletes that met criteria for at risk of an eating disorder and 30 matched controls (age range 12-35 y) | Eating disorder | EDI-DT and EDI-BD | At risk of an eating disorder - scoring > 15 on EDI DT or, >10 on EDI-BD Diagnostic survey for eating disorders | 22.4% Athletes 26% - Controls Of the 103 interviewed athletes classified as at risk of an eating disorder 89% (n=92) "fulfilled criteria for" anorexia nervosa, bulimia nervosa or anorexia athletica. | N/A |
| Rencken et al., 1996. USA | Cross sectional | 49 female recreational athletes age range 17-39 y) Not currently using OCP | Osteoporosis | DXA | Osteoporosis Osteopenia | 14.3% 34.7% | N/A |
| Johnson et al., 1999 USA | Cross sectional | 1445 male (mean age 20.1y) and female (mean age 19.) university representative athletes | Eating disorder | EDI-2, DSM-IV | At risk anorexia nervosa - BMI $\leq 20 \text{ kg.m}^{-2}$ or amenorrhoea or elevation on either EDI DT or EDI BD At risk Bulimia nervosa - 6 episodes of binge eating or vomiting or laxative or diuretic abuse or use of diet pills or elevation on either EDI DT or EDI BD Anorexia nervosa - BMI $< 18.5 \text{ kg.m}^{-2}$, amenorrhoea present (if female), elevated EDI-2 DT + EDI BD score Bulimia nervosa - binge eat & use a purge method (vomiting, laxative, or diuretics) 2x/week for 3 months & elevated EDI DT & EDI BD score | 25.0% females 9.5% males 58% females 38% males 0% 1.1% females 0% males | N/A |

Abbreviations = EDI = Eating Disorder Inventory, EDI-DT = Eating Disorder Inventory Drive for thinness sub-scale, EDI-BD = Eating Disorder Inventory Body Dissatisfaction sub-scale, DSM-iii = Diagnostic and Statistical Manual of Mental Disorders, Third Edition, Revised, DXA = Dual-energy X-ray absorptiometry, USA = United States of America

Table 2.1 History of the prevalence of low energy availability (LEA) among athletes (continued).

| Reference | Design | Participants | Component of LEA | Instrument used | Definition of LEA component | Prevalence and study outcome | Prevalence of two or more components of LEA | | |
|---|--|--|-------------------------------------|--|--|---|---|---|----------------------------------|
| Beals, 2002, USA | Cross sectional | 425 female Collegiate athletes – 15 sports – aesthetic, endurance, team Mean age 19 ± 2y | Eating disorder | Disordered eating -EAT, EDI-BD | Score >20 EAT | 15.2% | Not reported | | |
| | | | | Eating disorder | Score >12 EDIBD | 32.4% | | | |
| | | | Menstrual health | Self-reported questionnaire developed by researchers | Self-reported | Anorexia nervosa 3.3% Bulimia nervosa 2.3% | | | |
| | | | | | Menstrual Irregularity – cycles occurring > every 28-34 days in past year | 31.0% | | | |
| Bone health | Self-reported questionnaire developed by researchers | Menstrual dysfunction – no period ≥6months in past year Reporting ≥1 musculoskeletal injury over their collegiate sporting career | 12.9% 34.3% | | | | | | |
| Torstveit, & Sundgot-Borgen 2005, Norway | Cross sectional | 597 female elite athletes 865 non-athlete controls 13–39 y | LEA | Self-reported questionnaire developed by researchers | Participants considered at risk of LEA if met criteria for any other component or BMI <18.5 kg.m ⁻² | Controls > risk of LEA (69.2%) than elite athletes (60.4%) (p<0.01). Leanness sports (70.1%) > risk of LEA than non-leanness (55.3%) (p<0.001) | Not reported | | |
| | | | | | Disordered eating | Self-report | | Self-reported eating disorder | Controls 21.1% Athletes 18.4% |
| | | | | | | Pathogenic weight control methods EDI – BD | | Diet pills, laxatives, diuretics, vomiting | Controls 36.7% Athletes 20.2% |
| | | | | | | | | EDI - DT | Score ≥14 Score ≥15 |
| | | | | | Menstrual health | Self-reported | | No onset of menses by 16y or, no period ≥3 consecutive cycles or, menstrual cycles >35 days apart | Controls 24.5% Athletes 31.4% |
| Bone health | Self-reported stress fracture | If answered ‘yes’ or ‘I don’t know’ | Controls 12.2% Athletes – 17.25% | | | | | | |

Abbreviations EAT-26 = Eating Attitudes Test, EDI-DT = Eating Disorder Inventory Drive for Thinness sub-scale, EDI-BD = Eating Disorder Inventory Body Dissatisfaction sub-scale, USA = United States of America

Table 2.1 History of the prevalence of low energy availability (LEA) among athletes (continued).

| Reference | Design | Participants | Component of LEA | Instrument used | Definition of LEA component | Prevalence and study outcome | Prevalence of two or more components of LEA |
|------------------------------------|-----------------|---|--|-----------------|---|---|---|
| Beals & Hill, 2006, USA | Cross sectional | 65 Female athletes, university representatives Lean build - diving, cross-country, swimming, and track (sprinting events) mean age (19.5 ± 1.1y) 47 Non-Lean build sports included field hockey, softball, tennis, track (field events) (mean age 19.5 ± 1.4) | Disordered eating | EDI-SC EDE-Q | Clinical eating disorder - Diagnosis (in the past or currently) for anorexia nervosa and/or bulimia nervosa | Lean build 4.6% Non lean build 0.0% | Not reported |
| | | | | | Self-diagnosed eating disorder | Lean build 6.2% Non lean build 0.0% | |
| | | | | | Disordered eating - dissatisfied with body weight & engaging in at ≥ pathogenic weight control behaviour in the past year | Lean build 15.4% Non lean build 25.5% | |
| | | | Menstrual health | Self-reported | Absence of ≥3 consecutive menstrual cycles since menarche No onset of menses by age 16 | Lean build 32.3% Non lean build 17.0% (p = 0.053) | |
| | | | | | ≥1 of the following: 1) <12 cycles in the past 12 months, 2) <6 cycles in the past 6 months, 3) >10-d variation in cycle. | Lean build 12.3% Non lean build 4.3% (p=0.115) | |
| | | | Bone health | DXA | Low BMD, z score ≤ -2 | Lean build 27.7% Non lean build 12.8% (p=0.057) Lean build 3.1% Non lean build 0.0% (p= 0.225) | |
| | | At risk of low BMD, z score ≤ -1 | Lean build 15.4% Non lean build 2.1% (p=0.020) | | | | |

Abbreviations - EDI-BD = Eating Disorder Inventory Body Dissatisfaction sub-scale, EDI-DT = Eating Disorder Inventory Drive for Thinness sub-scale, BMD = Bone mineral density EDI SC Eating Disorder Inventory Symptom Checklist, EDE-Q = Eating Disorder Examination Questionnaire

Table 2.1 History of the prevalence of low energy availability (LEA) among athletes (continued).

| Reference | Design | Participants | Component of LEA | Instrument used | Definition of LEA component | Prevalence and study outcome | Prevalence of two or more components of LEA |
|----------------------------------|-----------------|--|---------------------|--|---|------------------------------|---|
| Barrack et al., 2008, USA | Cross sectional | 93 female high school cross country representatives 13-18y (if menstruating) 15-18 y (if menstruation has not started) | Disordered Eating | EDE-Q | Score ≥ 3 on any of the 4 subscales | Not reported | Not reported |
| | | | Menstrual health | Self-reported questionnaire developed by researchers | no onset of menses by age 15 | 3.2% | |
| | | | | | no period ≥ 3 consecutive months in past year | 17.2% | |
| | | | Bone health | DXA | Interval between menses <21 days and >31 days in past year | 5.4% | |
| Osteoporosis – z score ≤ -2 | 11.8% | | | | | | |
| | | Osteopenia - z score ≤ -1 | 39.8% | | | | |
| Rector et al., 2008 USA | Cross sectional | 43 male recreational cyclists (n=27) + runners (n=16) 20-59 y | Reproduction health | Serum testosterone and estradiol | Total testosterone - < 260ng/dL or > 1500ng/dL | All in reference range | Not reported |
| | | | Bone health | DXA | Osteoporosis – z score ≤ -2 Osteopenia - z score ≤ -1 | Not reported | Runners – 19% Cyclists – 63% |
| Smathers et al., 2009 USA | Cross sectional | 43 male cyclists and non-athlete controls 20-59 y | Reproduction health | Serum testosterone | Total ≥ 0.28 nmol/L ⁻¹ Free ≥ 0.62 pmol/L ⁻¹ | All in reference range | No association found between testosterone and bone health |
| | | | Bone health | DXA | Osteoporosis – z score ≤ -2 | Cyclists – 9% | |
| Hoch et al., 2009 USA | Cross sectional | 80 female University sports representatives (110 sports) 13-18 y | LEA | EI - 3 day diet record EE - calculated using, Ainsworth compendium of physical activity | Osteopenia - z score ≤ -1 ≤ 45 kcal/kg/LBM | Cyclists - 25% 36% | N/A |

Abbreviations - EDI-DT = Eating Disorder Inventory, EDE-Q = Eating Disorder Examination Questionnaire, DXA = Dual-energy X-ray absorptiometry, EI= Energy intake, EEE= Exercise energy expenditure, USA = United States of America

Table 2.1 History of the prevalence of low energy availability (LEA) among athletes (continued).

| Reference | Design | Participants | Component of LEA | Instrument used | Definition of LEA component | Prevalence and study outcome | Prevalence of two or more components of LEA |
|-----------------------------|-----------------|---|---|---|---|--|---|
| Quah, 2009 Malaysia | Cross sectional | 67 female Elite national representatives Leanness (30) – gymnastics, karate, taekwondo & pencak silat (13-28y) Non-leanness (37) – fencing, archery, shooting, hockey, squash (16-28y) | Disordered eating | EDI-2 | Scoring ≥ 12.1 on Drive for thinness scale Or scoring ≥ 8.5 on Bulimia scale Or scoring ≥ 6.8 on Perfectionism scale | Leanness – 89.2% Non-leanness – 89.2% | All 3 components 1.9% |
| | | | Menstrual health | Self-reported questionnaire developed by researchers | Primary amenorrhoea – no onset of menses by age 16 with the presence of sex characteristics or absence of sex characteristics at age 14 | Leanness – 9.5% Non-leanness 0% | Disordered eating + poor bone health 9.4% |
| | | | | | Secondary amenorrhoea - no period ≥ 6 months in past year | Leanness – 14.3% Non-leanness 0% | menstrual irregularities eating + poor bone health 1.9% |
| | | | | | Oligomenorrhoea – 4-6 menstrual cycles in past year Z score < -2.0 | Leanness – 23.8% Non-leanness – 14.3% Leanness – 0% Non-leanness 0% | |
| | | | | | $-2.0 < z \text{ score} < -1.0$ | Leanness – 13.3% Non-leanness – 8.3% | |
| | | Bone health | DXA | | | | |
| Thein-Nissenbaum, 2011, USA | Cross sectional | 311 female High school sport representatives Aesthetic ($16.1 \pm 1.3y$) Endurance ($15.4 \pm 1.1y$) Team/aerobic ($15.3 \pm 1.1y$) | Eating disorder | EDE-Q | Average score ≥ 4 across subscales Or score ≥ 4 on any of the 4 subscales | 35.4% - Total 37.1% - Endurance 33.1% - Team/aerobic | Not reported |
| | | Menstrual health | Healthy Wisconsin High School Female Athlete Survey | Menstrual dysfunction ≤ 9 menstrual cycles in the past year or no menstruation by 15 years | 41.5% - Aesthetic 18.8% - Total 19% - Endurance 16.5% - Team/aerobic 18.8% - Aesthetic | | |
| | | Bone health | Self-perceived injury history | Self-perceived injury | 65.6% - Total 69.7% - Endurance 60.8% - Team/aerobic 78.0% - Aesthetic | | |

Abbreviations = EDI = Eating Disorder Inventory, EDE-Q = Eating Disorder Examination Questionnaire, DXA = Dual-energy X-ray absorptiometry, USA = United States of America

Table 2.1 History of the prevalence of low energy availability (LEA) among athletes (continued).

| Reference | Design | Participants | Component of LEA | Instrument used | Definition of LEA component | Prevalence and study outcome | Prevalence of two or more components of LEA |
|---------------------------------------|-----------------|---|-----------------------|---|--|--|---|
| Hoch et al., 2011, USA | Cross sectional | 22 Female professional ballet dancers 18-35 y | Eating disorder | EDE-Q (fairburn) | Score ≥ 4 on any of the 4 subscales | 32% | 14% all 4 components |
| | | | LEA | EA = EI - EE EI - 3 day diet record EE - Accelerometer worn for 72 hours | Negative energy balance | 77% | 23% 3 out of the 4 |
| | | | Menstrual health | Self-reported questionnaire developed by researchers | Amenorrhoea - no period ≥ 3 consecutive cycles Oligomenorrhoea - menstrual cycles >35 days apart | 9% | 14% 2 out of 4 components |
| | | | Bone health | DXA | Z score < -1.0 Z score < -2.0 | 27% 23% | 36% ≥ 1 component |
| | | | Cardiovascular health | Flow mediated dilation | $< 5\%$ | 0% | 14% no components |
| Reed, 2013, USA | Longitudinal | 19 female Division 1 soccer players (18-21 y) | LEA | EI - 3 day diet record EE - Calculated using Ainsworth compendium of physical activity + heart rate monitor + OwnCal feature of the Polar Team2 software | $< 30 \text{ kcal.kg}^{-1}.\text{LBM}^{-1}$ | 26.3% pre-season 33.3% mid-season 11.8% post-season. | N/A |
| Gabriela Morgado, 2013. Brazil | Cross sectional | 24 female adolescent tennis players 14.77 \pm 2.16y | LEA | EI - 3 day diet record EE - Calculated using Ainsworth compendium of energy expenditures for youth. | $< 45 \text{ kcal.kg}^{-1}.\text{LBM}^{-1}$ | 87.5% | N/A |
| | | | | | $< 30 \text{ kcal.kg}^{-1}.\text{LBM}^{-1}$ | 33.3% | |

Abbreviations - LEA = Low energy availability, EA = Energy availability, EI = Energy intake, EE = Exercise energy expenditure, EDE-Q = Eating Disorder Examination Questionnaire, DXA = Dual-energy X-ray absorptiometry, USA = United States of America

Table 2.1 History of the prevalence of low energy availability (LEA) among athletes (continued).

| Reference | Design | Participants | Component of LEA | Instrument used | Definition of LEA component | Prevalence and study outcome | Prevalence of two or more components of LEA |
|--|-----------------|---|--|--|--|------------------------------|--|
| Melin, 2014b Denmark and Sweden | Cross sectional | 40 female competitive club or national representative endurance athletes 18-38 y | LEA | EA = EI - EE EI – 7 day diet record EE – heart rate monitor and training logs | Reduced EA 30 – 44. kcal.kg ⁻¹ .LBM ⁻¹ | 4.3% | Did not include LEA as a component when combining At least one component 50% 2 components 15% All three components 2.5% |
| | | | Disordered eating | EDE (Cooper 1989) and EDI-3 | Low EA ≤30 kcal.kg ⁻¹ .LBM ⁻¹ Eating disorder – if DSM-111-R is met | 2.0% 25% | |
| | | | Menstrual health | Self-reported questionnaire developed by researchers | Disordered eating - Scoring ≥ 14 on Drive for thinness scale Or scoring ≥19 on Body dissatisfaction scale | 15% | |
| | | | | | Primary amenorrhoea - no onset of menses by age 15 Secondary amenorrhoea – absence of ≥ 3 consecutive menstrual cycles Oligomenorrhoea - menstrual cycles >35 days apart | 10% 35% 15% | |
| | | | | | Low bone density - z score <- 1.0 Osteoporosis - z score <-2.0 | 37.5% 7.5% | |
| Barrack et al., 2014, USA | Cross sectional | 259 female competitive athletes Leanness and non-leanness Mean age 18.1±0.3 y | Disordered eating | TFEQ OR EDE-Q | ≥3 on dietary restraint scale ≥9 on dietary restraint scale Reporting ≥1 prior pathogenic weight control behaviour | 12.5% | Not reported |
| Menstrual health | | | Self-reported questionnaire developed by researchers | No onset of menses by ≥ 15 years or missing ≥3 consecutive menstrual cycles OR <4 cycles in the past year or 4-9 cycles in the past year or menstrual cycle length ≥36 days in the past year. | 10.9% | | |
| Bone health | | | DXA | Osteopenia - Z score < -1 Osteoporosis - Z score ≤ -2 | 21.0% 7.6% | | |
| | | | | Incurring a bone stress injury in past year | Self-reported | 10.8% | |

Abbreviations – LEA = Low energy availability, EDI = Eating Disorder Inventory, EDE-Q = Eating Disorder Examination Questionnaire, TFEQ = Three-Factor Eating Questionnaire, DXA = Dual-energy X-ray absorptiometry, USA = United States of America, LBM = Lean body mass, EA = Energy availability

2.3 Common Identifiers of Low Energy Availability (LEA) and Their Associated Health and Performance Consequences

LEA is effectively a state of starvation. This means the body uses energy primarily for the most important physiological requirements of survival, such as cellular maintenance, thermoregulation and locomotion, while body systems less important for survival reduce their functionality or shut off. The most commonly discussed in the literature include the reproductive, skeletal, cardiovascular, gastrointestinal and immune systems (Sundgot-Borgen & Garthe, 2011). Not only does LEA have the ability to seriously compromise health, consequences of LEA can lead to decreased sporting performance, and for an athlete's career, this may be devastating (Johansson et al., 1990; Vanheest et al., 2014).

2.3.1 Biochemical and Clinical Indicators

2.3.1.1 *Reproduction Hormones*

One of the main signs of LEA amongst females is the change in reproductive function. Low energy availability is a proven causal factor in the development of menstrual disorders ranging from FHA to subclinical menstrual disturbances such as anovulation and luteal phase deficiency (Loucks, 2007; Loucks & Thuma, 2003). Due to a lack of research, it is currently unclear whether these acute disturbances in menstrual function lead to long term fertility issues.

The underlying pathophysiology in FHA is suppression of gonadotropin secretion, due to a reduction in pulse frequency of GnRH and either no differences in pulse amplitude (Laughlin & Yen, 1997; Rickenlund et al., 2004) or higher pulse amplitude in eumenorrhoeic athletes compared to non-athletes (Loucks et al., 1989). As GnRH pulse frequency drops, there is a reduction in the number of LH and FSH pulses. Without frequent (every 60 to 90 minutes) ovarian stimulation by the gonadotropins (LH and FSH), oestrogen and progesterone levels decrease and ovulation will not occur, initiating the range of menstrual disturbances described above.

Over the past two decades researchers have investigated the possible underlying signalling system that initiates the GnRH pulse drop. Leptin, the hormone secreted by fat in proportion to body fat stores may be the critical link between energy availability and the integrity of hypothalamic-pituitary gonadal axis. Leptin levels have been shown to decrease in periods of short term fasting, even before marked decreases in body fat occur (Boden et al., 1996; Steiner et al., 1997). Some researchers have shown that leptin levels are on average lower in female

athletes with amenorrhoea compared with their eumenorrhoeic counterparts and speculate this may mediate the changes in the neuroendocrine axes associated with LEA. However A full description of this potential mechanism is beyond the scope of this literature review (Thong et al., 2000; Laughlin & Yen, 1997; Wauters et al., 2000; Corr et al., 2011).

Although the functionality of the hypothalamic pituitary gonadal axis in female athletes is dependent on their level of energy availability, recent research suggests genetic factors are likely to also play a role (Caronia et al., 2011). This may help explain the variable susceptibility of women to the changes in GnRH secretion that characterise FHA (Guebels et al, 2014; Schaal et al, 2011). More research is needed to clarify the relationship among genotype, phenotype and the environment in determining an athlete's susceptibility to disruptions to the hypothalamic pituitary gonadal axis.

Clinical menstrual disorders are indicated symptomatically by delayed onset of menarche (primary amenorrhoea), absence of menses (secondary amenorrhoea) or irregular menses, (oligomenorrhoea). Functional hypothalamic amenorrhoea is diagnosed if amenorrhoea is present in pre-menopausal females in the absence of pregnancy or any medical condition (e.g thyroid disease, polycystic ovarian syndrome, Kallmann syndrome, Turner syndrome, hyperprolactinemia, pituitary tumors) that could explain it.

The definition of these disorders continues to vary between studies. Most studies define primary amenorrhoea as, no onset of menses by age 15 years (Barrack et al, 2008; Melin et al., 2014b), however, other definitions have been used such as no onset of menses by age 16 years with the presence of sex characteristics or absence of sex characteristics at age 14 years (Quah et al, 2009). The definition of secondary amenorrhoea ranges from absence of at least three consecutive menstrual cycles to no period for at least six months in past year. Oligomenorrhoea has the largest variation in definitions ranging from cycles occurring every 28-34 days in past year (Beals & Manore, 2002), cycles occurring every 21 to 31 days in past year (Barrack et al., 2008), four to six menstrual cycles in past year (Quah et al., 2009) and menstrual cycles greater than 35 days apart (Hoch et al., 2011; Melin et al., 2014b).

Due to inconsistent definitions it is not surprising recent research has shown a wide range in prevalence of menstrual disorders among female athletes. As shown in Table 2.1 the prevalence ranges from 3.2% to 10.0% for primary amenorrhoea (Melin et al, 2014b; Barrack et al, 2008), 5.3% to 35.0% for secondary amenorrhoea (Melin et al., 2014; Quah et al., 2009) and from 5.4% to 31.0% for oligomenorrhoea (Barrack et al, 2008; Beals & Manore, 2002). In order to

accurately compare these studies standardised definitions of these clinical menstrual disorders are needed.

There has been minimal exploration into the effects of LEA on male reproductive hormones. Several studies have reported significantly lower testosterone levels among male endurance runners, cyclists and jockeys compared with non-athlete controls (Hackney, 1996; Hackney et al, 1997; Hackney, 1989; De Souza et al, 1994; Dolan et al., 2012). One cross sectional study compared testosterone levels of untrained controls to those of elite male cyclists. Both groups total testosterone levels were within the reference range ($\geq 0.28 \text{ nmol.L}^{-1}$) for healthy males but 12.5% cyclists versus 6.7% controls had free testosterone levels below the reference range ($\geq 0.62 \text{ pmol.L}^{-1}$).

Therefore, like females, males may also experience the metabolic, hormonal, and physiological effects of LEA. Further research is warranted, as existing studies are limited (Hackney, 1996; Hackney et al., 1997; Hackney, 1989), and the sample size is often small (around 10–15 in each group). In addition, the long term effects of LEA on reproduction in both males and females is still yet to be explored.

2.3.1.2 Bone health

The relationship of bone turnover is a complex interplay between bone formation and bone resorption. Two cell types control bone turn over, osteoblasts (promote bone growth) and osteoclasts (promote bone resorption), both of which are continually active. It is the relationship between the two that determines the overall turnover and metabolism of bone (Dempster & Raisz, 2015). It is difficult to determine if the reduction in bone mineral density during LEA is due primarily to the hormonal responses of LEA or coupled with a reduction in bone promoting nutrient intake (e.g. magnesium, calcium, vitamin D), because the two are so closely related (Dempster & Raisz, 2015).

Table 2.1 includes a number of studies where the prevalence of impaired bone health has been investigated in various athletic groups. The estimated prevalence of osteoporosis in female athletes ranges from 0% in American professional ballet dancers (Hoch et al., 2011) and Malaysian national representatives (Quah et al., 2009) to 7.5% in European club or national representative endurance athletes (Melin et al., 2014b) and 11.8% in American high school cross country runners (Barrack et al., 2008). The prevalence of osteopenia estimations are even broader ranging from 8.3% to 39.8% (Barrack et al., 2008). Furthermore, female athletes competing in sports that emphasis leanness have reported more musculoskeletal injuries and

appear to have a higher prevalence of low bone mineral density when compared to athletes where leanness is less important (Quah et al, 2009; Thein-Nissenbaum et al, 2011).

Initially poor bone health does not have obvious symptoms and is therefore known as the silent component of LEA. Unfortunately many athletes are not aware they have low bone mineral density until they develop a stress fracture. Bone stress fractures occur most frequently in the tibia and metatarsals, but are also reported in the fibular, femur, ribs, and navicular (Bennell et al., 1996; Johnson et al., 1994; Matheson et al, 1987; Barrack et al., 2014).

Recently a prospective cohort study was published that aimed to evaluate the effect of single and combined risk factors for the FAT on the incidence of bone stress injuries (Barrack et al., 2014). Researchers combined databases from three prospective cohort studies across the USA, which included a total of 259 female competitive and recreational athletes. Variables that measured the same construct were merged and subsequently defined identically. Participants were categorised based on physical activity (competitive athlete or recreational athlete) and sport/activity (leanness or non-leanness). Dietary restraint was measured using The Three Factor Eating Questionnaire (TFEQ) or the Eating Disorder Examination and participants were also required to report any pathogenic weight control behaviours such as self-induced vomiting, diuretic and laxative use to determine disordered eating behaviours. Participants were required to have DXA (dual-energy x-ray absorptiometry) scans to determine bone mineral density and to complete self-reported questionnaires developed by researchers to determine menstrual history and incidence of bone stress injuries. The results indicated the percentage of athletes developing bone stress injuries increased from 15-21% in athletes meeting the criteria for one FAT component to 21-30% for those meeting the criteria for two components and 29-50% for those meeting the criteria for all three components of the FAT. These results indicate the more FAT risk factors a female athlete has, the more likely they are to develop a bone stress injury.

Although most literature has focused on the bone health of female athletes, low bone mineral density levels have been found in male recreational and elite cyclists and long distance runners (Rector et al., 2008; Smathers et al., 2009). Rector et al., undertook a cross sectional study in the USA, investigating male recreational cyclists and runners, bone health. DXA scans were used to measure bone mineral density. Results showed as many as 63% were diagnosed as having osteopenia and male cyclists were three times more likely to have osteopenia of the spine than long distance runners (Rector et al., 2008). This finding follows our understanding that weight bearing exercise promotes bone growth and maintenance, which may explain the difference in bone mineral density found between the two sports (Whitney & Rolfes, 2007).

While another cross sectional study carried out in the USA estimated the prevalence of osteopenia and osteoporosis in competitive male cyclists to be 25% and 9% respectively (Table 2.1) (Smathers et al., 2009).

It is well documented that oestrogen deficiency leads to increased osteoclast formation, which in turn causes an up regulation in bone resorption (Audi et al., 2002). Several studies have been published supporting the mechanistic role of oestrogen in bone turnover (Manolagas et al., 2013).

In addition to oestrogen, there is strong evidence from prospective randomised crossover controlled trials as well as observational studies of amenorrhoeic athletes to suggest exercise associated menstrual disturbances in females are associated with abnormalities of other neuroendocrine systems that consequently produce a hypometabolic, energy conserving state and compromise bone health. These include a reduction in resting metabolic rate, thyroid hormones, leptin, insulin, glucose, insulin-like growth factor and insulin like growth factor-3 as well as elevations in, ghrelin, growth hormone and cortisol (Loucks & Heath, 1994; Hilton & Loucks, 2000; Loucks, 2004; Loucks, 2003).

Although the exact mechanism is not completely understood it is well established that thyroid hormones triiodothyronine (T3), and its prohormone thyroxine (T4) play an integral role in bone formation (Williams, 2009). One study investigated the status of the hypothalamic-pituitary-thyroid axis in eumenorrhoeic female athletes (n=9) and non-athletes (n=9) and compared it to that of amenorrhoeic female athletes (n=9). Serum levels of thyroid hormones, T4, T3, free T4, free T3 and Reverse T3 (rT3) an inactive form of T3, were considerably reduced ($P < 0.01$) in amenorrhoeic athletes, whereas only serum T4 levels were significantly decreased in eumenorrhoeic athletes (Loucks et al., 1992).

Another study investigated 29 regularly menstruating inactive females to determine the dose-response relationship between energy availability and several markers of bone turnover (Ihle & Loucks, 2004). It was found that in exercising women with energy availability less than 30 kcal.kgLBM⁻¹.day⁻¹, the hormones that promote bone growth as well as the rate of bone growth were suppressed within five days. Whereas an increase in bone resorption markers was only observed when a severe degree of energy restriction (10 kcal.kgLBM⁻¹.day⁻¹), which was associated with a reduction of serum oestrogen by 18%, was imposed. These findings may explain why some exercising eumenorrhoeic females with subclinical menstrual disorders (luteal phase deficiency or anovulation) and small alterations in metabolic hormones may

experience reductions in bone mineral density without severe reductions in oestrogen. This hypothesis has not been explored in male athletes and further studies over a longer period of time are required to determine the dose response relationship between LEA and bone turnover.

Alongside dietary factors, research shows that exposure to oestrogen in a female's adolescent years determines peak bone mass (Soyka et al, 2000). Thus it has been suggested that primary amenorrhoea or even sub clinical menstrual disturbances in adolescent years may prevent young females from reaching their genetic potential for peak bone mass (Ihle & Loucks, 2004). Unfortunately this is an area where there is limited research, and due to the severity of long term health consequences it is an area that requires further investigation.

2.3.1.3 Cardiovascular Health

LEA may increase an athlete's risk of developing premature cardiovascular disease (De Souza & Williams, 2004; Jáuregui-Garrido et al, 2012). Endothelial dysfunction, an accepted marker of future cardiovascular risk, has been reported in amenorrhoeic athletes with LEA (Vanheest et al., 2014; Soleimany et al., 2012; Rickenlund et al., 2005).

Oestrogen plays an integral role in the recruitment of the vascular endothelial nitric oxide signaling system. This system is directly involved in controlling the dilation and proliferation of vascular smooth muscle. Research has shown that in a state of LEA when oestrogen levels are decreased, endothelium dependent arterial vasodilation may occur (Hoch et al., 2003) leading to reduced blood flow to working muscles, impaired skeletal muscle oxidative metabolism and a rise in blood lipids (De Souza & Williams, 2004; Friday et al, 1993; Rickenlund et al, 2005).

The aetiology behind this rise in blood lipids has been widely discussed (Jáuregui-Garrido et al., 2012). Some researchers have suggested elevated cortisol levels may be a contributing factor whilst the known down regulatory effect of total T3 and oestrogen levels on cellular hepatic low density lipoprotein receptors has also been thought to play a role (Misra et al., 2006). Other studies have suggested the increase in lipoprotein levels could be explained by a higher rate of synthesis of cholesterol-rich lipoprotein in people with LEA. Following an investigation of fasting serum lipid profiles and cholesterol ester transfer protein activity in 24 Japanese females with anorexia and five matched controls, Ohwada et al., concluded that the mean low density lipoprotein and high density lipoprotein values were elevated in anorexia nervosa and this was related to a significantly higher activity of cholesterol ester transfer protein than in controls (Ohwada et al., 2006).

It is currently unclear whether this rise in blood lipids is associated with increased risk of cardiovascular disease and is an area that warrants further research.

2.3.1.4 Nutritional Intake

When an athlete's energy availability is low, it is likely energy intake, as well as macronutrient and micronutrient intakes are also low, putting them at risk of all nutrient deficiencies (Beals & Manore, 1998; Manore, 1999). Low energy intake, especially when coupled with excessive exercise can lead to low body fat levels. Although LEA does not mean low body fat per se, it can also be an important clinical indicator of disordered eating and poor energy availability (Thong et al., 2000).

The site of nutrient absorption occurs in the gut with the majority of carbohydrates and nutrients absorbed in the small intestine, the duodenum, jejunum and ileum. Water is mostly absorbed in the large intestine and the rate of absorption is determined by the nature of the transporter molecules on the intestinal wall (Patton & Thibodeau, 2014). When the body is in a state of starvation, and is lacking specific nutrients some of these transporter molecules are down regulated, and thus less nutrients are absorbed, possibly worsening the situation (Monteleone et al., 2004).

Adequate nutrient intake is essential for maintaining health, growth, repair and utilising energy from the diet. As well as their role in bone maintenance, nutrients play an important role in the development and functionality of immunocompetent cells, thus nutrient deficiencies can lead to immunosuppression and dysregulation of immune responses. Specifically, micronutrients such as vitamin A, C, B6, B12 and folic acid play an important role in white blood cell production (Fairfield, & Fletcher, 2002; Murata et al, 1994). Thus, these deficiencies can impair phagocyte function and cytokine production as well as adversely affect certain aspects of humoral and cell-mediated immunity, thereby causing athletes to become more susceptible to infection and illness, in particular upper respiratory tract infection (Marcos, 1997; Nattiv et al., 2007; Nieman, 1997; Nieman, 1998).

Low energy intake can put athletes at risk of inadequate carbohydrate intake. Insufficient carbohydrate intake can result in glycogen depletion and low blood glucose levels (Burke et al, 2004). Hilton and Loucks suggest LH pulsatility may not be dependent on energy availability per se but rather on glucose availability, meriting further investigation as it may play a fundamental role in the treatment of LEA in the future (Hilton & Loucks, 2000).

Nattiv et al., (2007) reported athletes practicing disordered eating behaviours and weight control methods often report irregular meal patterns and a high dietary fiber intake (Nattiv et al., 2007). This may cause gastrointestinal discomfort and symptoms such as bloating and constipation (Black et al, 2003; Bonci et al., 2008). Sufferers also face an increased risk of dehydration, electrolyte imbalances and dental enamel loss cause by chronic vomiting and/or reflux (Mountjoy et al., 2014).

2.3.1.5 *Sporting Performance*

A cross sectional study has recently been published determining the relationship between ovarian suppression and swimming performance related to LEA (VanHeest et al., 2014). A small sample of ten elite female swimmers aged 15-17 years were followed for 12 weeks. Every two weeks participants were evaluated for measures of ovarian (estradiol and progesterone) and metabolic hormones (T_3 , IGF-1), as well as energy availability. The four-hundred-meter freestyle swim velocity was used as the measure of sporting performance. Participants were retrospectively classified as having either ovarian suppressed or cyclic menstrual function. Ovarian suppression was defined as serum progesterone concentrations $<15.9\text{nmol.L}^{-1}$ at weeks zero and two as well as the absence of cyclical increases in estradiol. Cyclic menstrual function was defined as serum progesterone concentrations at the luteal phase $<15.9\text{nmol.L}^{-1}$ or greater at weeks 0 or 2. If estradiol concentration increased above 25pg. ml^{-1} during a four week period this was also deemed cyclic.

The average menstrual cycle length was significantly longer in the ovarian suppressed group (86 SD = 2 days) compared with the cyclic group (29 SD = 1 days). At baseline, participants were required to record the timing of their last two periods. Prior to the study commencing no ovarian suppressed athletes were amenorrhoeic, however all but one were oligomenorrhoeic (inconsistent cycle lengths >36 days), all cyclic athletes reported current eumenorrhoeic menstrual patterns (cycle lengths were between 28 and 31 days). Energy intake was determined via 3-day diet record every two weeks, whilst exercise energy expenditure was calculated taking a daily average of participant's 7-day training records. Energy availability was defined as energy intake minus exercise energy expenditure and calculated relative to fat free mass.

Throughout the study, energy availability was significantly lower in the ovarian suppressed group compared to the cyclic group ($p<0.001$). At week 2, 6 and 10 the ovarian suppressed group had significantly lower oestrogen levels than the cyclic group ($p=0.006$, $p=0.003$ and $p=0.002$, respectively). At baseline there was no significant difference ($p=0.98$) in swim

velocity between the groups. The cyclic groups showed a positive response to the training throughout the 12-week period improving their 400m swim time by 8.2%, whereas the ovarian suppressed groups performance decreased by 9.8% ($p < 0.001$). The results of this study reinforces findings from earlier cross sectional investigations and indicates that female athletes suffering from even the most subtle menstrual disturbances are at risk of compromising their sporting performance (De Souza et al., 2003; Loucks et al., 2003).

Johansson and colleagues (1990) published a case series documenting 23 Swedish athletes (7 elite, 16 recreational) with stress fractures followed for an average of 6.5 years (Johansson et al., 1990). At follow-up all seven elite athletes reported ending their sporting career as a result of the stress fracture, decreasing their level of competition to recreational. In addition, a case control study reported that cardiovascular function was negatively affected after two months of LEA, which resulted in lower running speeds amongst elite female endurance athletes (cross country skiers, middle and long distance runners) (Ingier & Sundgot-Borgen., 1991). These studies provide additional evidence that components of LEA (impaired bone health and cardiovascular function) have a negative impact on athletic performance.

2.3.1.6 Mortality

To suffer from an eating disorder, such as anorexia nervosa or bulimia nervosa, is to have a serious mental health illness and the associated mortality rates are high (Smink et al., 2012). In 1997 the World Health Organisation (WHO) estimated the mortality rate for bulimia nervosa to be 19%, whilst the risk of premature death from an eating disorder is 6-12 times higher than the general population (World Health Organisation, 1997; The National Eating Disorders Collaboration, 2012).

Although the mortality rate of athletes due to LEA complications is yet to be investigated extreme cases of medal hopefuls dying due to inappropriate weight loss practices have been reported (Sundgot-Borgen & Garthe, 2011), indicating an this is an important area to investigate.

Despite the many benefits engaging in regular exercise promotes, it is clear when coupled with inadequate dietary intake the health and performance consequences, in female and potentially male athletes can be devastating. It is necessary action is taken to identify athletes at risk of LEA to reduce the prevalence of LEA and its co-morbidities. In order to do this it is important standardised screening tools that are validated in both male and female athletes are developed and used, to ensure prevalence data is accurate and comparable.

2.4 Current Prevalence of Low Energy Availability (LEA)

Although a large body of research exists wherein the prevalence of LEA and its associated health complications is estimated, it is difficult to determine the current and past prevalence of LEA. As shown in Table 2.1 early studies estimating the prevalence of LEA were limited to females and carried out in high risk populations such as competitive long distance runners and dancers (Frusztajer et al., 1990; Petrie & Stoeber, 1993; Rosen & Hough, 1988; Warren et al., 1990), however recent research includes a much larger range of sports and level of competition as well as male athletes. Differences continue to lie in the experimental design, methodology and most importantly the definitions of each component of LEA, making studies very difficult to compare accurately.

An obvious gap in the literature exists in the estimation of the prevalence of LEA. The few studies that have estimated its prevalence in female athletes have found a large variation in results. A cross sectional European study estimated the prevalence of reduced and low energy availability amongst club level endurance athletes. Energy availability was calculated by subtracting mean exercise energy expenditure (measured with training logs and heart rate monitors) from the mean energy intake (measured with 7 day diet records) and calculated relative to fat free mass. Results indicated 4.3% of participants were in a state of reduced energy availability ($\leq 45 \text{ kcal.kgLBM}^{-1}.\text{day}^{-1}$), and 2% in a state of LEA which was defined as $\leq 30 \text{ kcal.kgLBM}^{-1}.\text{day}^{-1}$ (Melin et al., 2014b). Another cross sectional study comparing American high school athletes with matched controls defined LEA as $\leq 45 \text{ kcal.kgLBM}^{-1}.\text{day}^{-1}$. Similar to Melin et al., (2014b) energy availability was measured by subtracting exercise energy expenditure from energy intake, however different methods were used to measure these. Energy intake was calculated via prospective 3-day food diary and exercise energy expenditure was calculated using duration of sports participation, intensity of exercise, weight, age and sex from the Ainsworth compendium of physical activity (Ainsworth et al., 2011). Results indicated 36% of athletes were in a state of LEA (Hoch et al., 2009). Further an American cross sectional study on professional ballet dancers aged 18-35 years, defined LEA as negative energy balance and estimated an alarming 77% of participants were in a state of LEA (Hoch et al., 2011).

The variation in methods used to estimate the prevalence of LEA along with inconsistent definitions of the level of energy availability LEA occurs makes it difficult to compare these studies. There is an obvious need to collect prevalence data on LEA using appropriate tools and a standard definition.

2.5 Tools and Techniques To Identify Athletes With and At Risk Of Low Energy Availability (LEA)

Prevention of FAT/RED-S is most preferable, followed by early detection. As previously described in section (section, 2.2.5) the main driving factor for the development of FAT/RED-S is LEA. Thus diagnosis tends to focus on identifying the presence, causes and risk factors associated with LEA. A major limitation to its diagnosis is the absence of standardised guidelines to determine energy availability.

The Female Athlete Triad Coalition recently published a “Triad-specific self-report questionnaire” however they admitted at that time there was limited evidence related to the effectiveness of the screening questions (De Souza et al., 2014). However, even more recently, Melin et al, (2014a), published the Low Energy Availability in Females Questionnaire (LEAF-Q). This is a validated screening tool used to detect LEA and Triad conditions, with or without disordered eating/eating disorders. The questionnaire consists of questions relating to reproductive and gastrointestinal function as well as injury history (Melin et al., 2014a). To date no study has been published using the full version of the LEAF-Q, however with a 90% specificity and 78% sensitivity it shows a lot of promise and is likely to be widely used as the initial screening tool to detect female athletes at risk of LEA. At present no such tool is available for male athletes.

2.5.1 Techniques To Detect Low Energy Availability

Energy availability is defined as energy intake minus the cost of energy expenditure during exercise relative to fat free mass, however the threshold for what is considered LEA is less clear (Nattiv et al., 2007). As shown in Table 2.1 the definition of LEA ranges from ≤ 30 kcal.kgLBM⁻¹.day⁻¹ and ≤ 45 kcal.kgLBM⁻¹.day⁻¹ to a negative value when calculating energy intake minus exercise energy expenditure (Hoch et al., 2009; Hoch et al., 2011; Melin et al., 2014b).

Current methods used to measure energy availability generally require a skilled interviewer or clinician, are timely, and often imprecise. Through retrospective or prospective diet records/recalls we can estimate energy intake. It has been found that traditional paper-based methods used to determine dietary intakes are imprecise in an athletic population (De Souza et al., 2014). Thus the Female Athlete Triad Coalition recommends guidance for assessment of energy intake and dietary patterns should be provided by an experienced sports dietitian (De Souza et al., 2014). Exercise energy expenditure is most commonly estimated through

physical activity logs/diaries and is often supplemented with data from modern sports technology (e.g. heart rate monitors and accelerometers) and/or scored for exercise intensity according to the Ainsworth Compendium of physical activity (Ainsworth et al., 2011).

2.5.2 Tools To Detect Eating Disorders

In contrast to LEA, there are a number of validated eating disorder tools which are commonly used to screen athletes for disordered eating behaviors (Garner, 2004; Secord & Jourard, 1953; Martinsen et al., 2014). In 2014 the IOC stated the Eating Disorder Examination Interview was the “Gold Standard” in determining athletes with disordered eating behaviors and eating disorders (Mountjoy et al., 2014). Unfortunately, as this assessment requires a skilled interviewer and the athlete to be present it is rarely used (Table 2.1) (Fairburn et al., 2008).

The EAT-26, Eating Disorder Examination Questionnaire (EDE-Q) and EDI are frequently used, self-reported, eating disorder screening tools to determine athletes at risk of an eating disorder (Garner et al., 1982; Garner, 2004; Hilbert et al., 2007). Although these three screening tools are well-validated amongst the general population unfortunately they have not been validated in an athletic population.

In early 2014, the Brief Eating Disorder in Athletes Questionnaire (BEDA-Q) was published (unfortunately after the decision regarding the eating disorder screening tool for this study was made) (Martinsen et al., 2014). Like the EDI-3 this tool complements the LEAF-Q as it is used to discriminate between female athletes with and without eating disorders. Although the LEAF-Q and BEDA-Q are athlete specific they still exclude men, athletes with a disability and are validated in European populations without diverse ethnicity. These are all important components to be considered when revised editions of these questionnaires or new screening tools are being developed. This will help to ensure that particular athlete groups are not left out of the screening process. It will also allow health professionals as well as coaches, parents and athletes themselves can be consistent in identifying the signs and symptoms of LEA and seek the appropriate treatment before serious health consequences can occur.

Following screening, accurate diagnosis of LEA and its health consequences depends on the multidisciplinary team including a physician, sports dietitian and mental health professional.

2.5.3 Techniques To Detect Poor Bone Health

There are various techniques used to measure bone mineral density such as Computed Tomography (CT), DXA and quantitative ultrasound (QUS). In the most recent literature the majority of researchers use the gold standard DXA scans to estimate the prevalence of athletes suffering from low bone mineral density (BMD) and use the WHO definitions to categorise participants as having normal BMD ($N-1.0$ SD), osteopenia (≤ -1.0 SD, $N-2.5$ SD), or osteoporosis (≤ -2.5 SD) (Barrack et al., 2008; Hoch et al., 2011; Melin et al., 2014b; Quah et al., 2009) (Table 2.1). Due to its high accuracy, DXA can be used both to diagnose osteoporosis and osteopenia as well as a prognostic tool to predict the risk of fractures. However, it is expensive, requires specialist equipment and usually requires hospital/clinic visits, making it impractical for many.

2.5.4 Techniques To Detect Other Health Consequences Of Low Energy Availability

The most common technique to diagnose clinical menstrual disorders is via self-reported menstrual history (Table 2.1). However this method alone does not provide appropriate information to determine the presence of subclinical menstrual disorders such as luteal phase deficiency (inadequate progesterone secretion) and anovulation (absence of ovulation). The only means of detecting subclinical menstrual disorders is by measuring sex steroid hormones during the luteal phase of the menstrual cycle, which is both costly and time consuming.

Cardiovascular risk can be assessed by measuring flow mediated dilation, which is an indirect measure of endothelial cell function (Anderson et al., 1995). Total cholesterol is known to be positively associated with cardiovascular disease (Collaboration, 2007). As previously described, in a state of LEA athletes are at risk of a rise in blood lipids (section 2.3.1.3.) thus another commonly used measure of cardiovascular risk is analysis of blood lipid profiles (Heart Foundation, 2015). Indirect calorimetry is used to measure resting metabolic rate which may assist in confirming a suppressed metabolism resulting from LEA.

2.6 Treatment Options for Low Energy Availability (LEA)

Unfortunately there are no official guidelines available for the treatment of LEA in athletes. However the ACSM and IOC both clearly state the treatment of LEA should constitute non-pharmacological measures such as increasing energy intake or reducing exercise energy expenditure, to increase energy availability (De Souza et al., 2014; Mountjoy et al., 2014). In extreme cases where non-pharmacological treatment is unsuccessful after at least a year, the ACSM recommends pharmacological treatment to be considered (De Souza et al., 2014).

The health professionals involved in the treatment of LEA may vary depending on its underlying cause. If the cause of LEA is simply under eating or weight loss, whether it is intentional or inadvertent, referral to a sports dietitians and in some cases an exercise physiologist is sufficient. If LEA is due to intentional disordered eating a physician should also be included in the treatment, and if the underlying cause is a clinical eating disorder, this requires more intensive treatment and referral to a mental health practitioner for psychological treatment is necessary (De Souza et al., 2014).

2.6.1 Non-pharmacological

The strongest evidence for successful treatment of LEA, regardless of its origin, is through increasing energy availability, via an increase in energy intake and/or reducing exercise energy expenditure, in order to initially promote resumption of menstrual function and in some cases weight gain.

Dueck performed a small case-control study in the USA (Dueck et al, 1996), where data was collected on one amenorrhoeic female athlete (track runner) while 3 eumenorrhoeic athletes acted as controls. The amenorrhoeic athlete was given a diet and training intervention program to follow for 15 weeks. The intervention program involved reducing training by one day per week and consuming one sport nutrition beverage providing 360 kcal each day. The intervention produced positive results, with the amenorrhoeic runner's serum levels of LH increasing from 3.9 mIU.ml⁻¹ to 7.3 mIU.ml⁻¹ whereas the controls had a decrease from 7.9 mIU.ml⁻¹ to 5.7 mIU.ml⁻¹. Although at the end of the 15 week intervention the subject experienced no changes in her menstrual cycle, resumption of menstruation did occur after following the programme for an additional 3 months (Dueck et al., 1996). This is only a small observational study, however these findings are consistent with later research from the aforementioned study by Kopp-Woodroffe (Section 2.2.5) (Kopp-Woodroffe et al., 1999).

The statement from Dr Erdelyi in the first documented study on menstrual disorders in female athletes states that when we “*find menstrual disorders that may be associated with too much sports activities*” “*we should decrease the sports activities and include a temporary rest in the training period*”. This remains current practice, however in many less severe cases of LEA the rationale for reducing training and exercise may be an overly conservative approach that may impact sporting performance, therefore many athletes and coaches may be reluctant to include additional rest days in their training programme (De Souza et al., 2014).

A recently published study from the USA investigated physically active women with FHA to determine whether an increase in energy intake alone would improve energy balance and restore reproductive function as well as improve bone health and muscle strength (Cialdella-Kam et al, 2014). Based on self-reported menstrual history and ovulation status, 18 endurance trained female athletes were assigned to either the exercise associated menstrual disorder group (n = 8) or the eumenorrhoeic group (n = 10). Following collection of baseline data, participants in the menstrual disorder group participated in a 6 month carbohydrate-protein dietary supplement intervention (360 kcal/day, 54g carbohydrate/day, 20g protein/day). Results showed all participants in the intervention group resumed menses post intervention, 88% were ovulating and 75% had gained weight. Although not statistically significant 3 of the 4 participants who had LEA at baseline and were in the intervention group showed a positive increase in energy availability. At baseline three of the intervention group were considered to have LEA ($<30 \text{ kcal.kgLBM}^{-1}.\text{day}^{-1}$) whereas only one was post intervention. This study indicates dietary intervention to increase energy availability without a reduction in exercise energy expenditure may be a suitable treatment option. This may be of particular value to athletes who are not prepared to reduce their training hours as fear performance will be adversely effected.

A common finding amongst these and other studies is that the strongest predictor of amenorrhoeic athletes restoring normal menstrual function, is weight gain (Dueck et al., 1996; Fredericson & Kent, 2005; Kopp-Woodroffe et al., 1999; Cialdella-Kam et al., 2014). Further, among case studies of female, amenorrhoeic athletes, improvements in bone mineral density have been shown with weight gain and resumption of menses (Fredericson & Kent., 2005; Zanker et al., 2004) as well as favourable changes in bone metabolism without a further decline in bone health (Kopp-Woodroffe et al., 1999; Mallinson et al., 2013). Due to the lack of long term intervention studies it is difficult to draw a conclusion

on the effect dietary intervention has on bone health however these results suggests a non-pharmacological approach may be effective.

2.6.2 Pharmacological

The most common pharmaceutical intervention involves treating LEA with the oral contraceptive pill or other forms of oestrogen therapies which have been shown to restore menses in females with FHA (Cumming, 1996). However evidence is equivocal in regards to the effects of this strategy on BMD in the athletic population. Researchers have found vertebral and femoral neck bone density significantly increased by 8.0% and 4.1% respectively with oestrogen replacement therapy whilst the control group (n=5) had no significant change in bone mineral density at either site (Cumming, 1996). On the other hand, others have shown a detrimental effect on bone mass in young females following oestrogen therapy (Hartard et al., 2007).

It is well recognised that oestrogen therapy is also unlikely to normalise other metabolic factors associated with LEA that impair bone formation, leading researchers to investigate other avenues of hormone replacement therapy. Androgen therapy has been shown to result in favourable changes in bone turnover markers however no significant improvements in bone mineral density were detected (Gordon et al., 2002). Insulin-like growth factor 1 therapy has been shown to increase bone mineral density in anorexic women (Grinspoon et al, 2002) however limited research has been carried out in this area to determine its effectiveness. Similarly although leptin replacement therapy has been shown to result in significant increases in BMD this was coupled with a decrease in body mass (Sienkiewicz et al., 2011). As this population most likely needs to gain weight this is a potentially dangerous treatment that is currently advised against (De Souza et al, 2014; Mountjoy et al, 2014).

Correcting menstrual status in female athletes with the use of oestrogen or other hormonal replacement therapies may lead to a false reassurance while the underlying problem being LEA continues to exist and adversely impact on health (Bergström et al., 2013). These results suggest that non-pharmacological options for the treatment of LEA should prevail over pharmacological therapies as their effectiveness has not been proven.

Further research is urgently needed as there is a dearth of evidence supporting the proposed long-term benefits of non-pharmacological treatment with a real shortage of prospective longitudinal studies to determine the specific nutritional intervention that is the most

effective in treating LEA and its associated health consequences and the approximate time frame. Also although difficult to implement due to problems with compliance and ethical issues, it is clear prospective research is also needed on the long term effects of pharmacological treatment in this population.

In order to determine the most appropriate treatment options for New Zealand athletes, it is important to determine athletic populations at greatest risk of LEA, along with possible reasons for why these athletes are exposed to higher risk. This would allow treatment to be appropriately targeted to allow for the most predictable positive outcome.

2.7 Summary

Low energy availability has attracted a good deal of attention from the research community over the past 35 years. As more research is published our understanding of the complexity of LEA increases. It is evident that this issue is not simply a triad of conditions, but a highly complex syndrome driven by energy availability with adverse effects not only on menstrual function and bone health but many aspects of an athletes physiological and psychological health, as well as sporting performance. It is recognised that males can suffer from such a syndrome and it is important they are not excluded from future research.

Effort needs to focus on appropriately informing not only physicians, but also athletes, coaches, officials and parents about LEA and its associated health consequences. Furthermore, in order to improve prevention and early detection strategies of LEA, more research is needed. Efforts should be continually undertaken to develop standardised, validated, diagnostic and screening tools appropriate for use in both male and female athletic populations around the world. This would permit the prevalence of LEA to be accurately measured and compared, allowing identification of high risk groups, as well as reduce its prevalence by encouraging appropriate treatment to occur before health and performance are severely compromised. Future investigations are required to determine the prevalence of subclinical conditions associated with LEA as these athletes are at very high risk for developing the associated clinical conditions.

As prevalence appears to vary from one country to another, it is important individual countries carry out their own prevalence studies, using internationally accepted protocols and do not rely solely on international data. To date there is no information on the prevalence of LEA in New Zealand, therefore it is timely to undertake cross sectional studies in this population. In addition it is important to identify easy to administer screening tools that can be used to identify individuals at risk of LEA. Prevention of LEA, or at least early detection of it, is undoubtedly better than a cure.

Therefore the aim of the present study is to describe the estimated prevalence of New Zealand recreational athletes at risk of low energy availability and disordered eating.

3 Methods

3.1 Study Design

The LEANZ study used a cross-sectional design, where recreational athletes were recruited to complete an online, self-administered, forced-choice survey (Appendix A). It was created and hosted by the online survey program So Go Survey© (2014 SoGoSurvey, Inc, Herndon, VA, USA) (Appendix A). It consisted of 98 questions, both short answer and multi-choice, and was designed to take 20-30 minutes to complete.

3.2 Ethical Approval and Informed Consent

This study was approved via Departmental Ethical approval at the University of Otago Human Ethics Committee (Appendix B).

Before beginning the questionnaire, participants logged onto the survey webpage with the password provided, and were required to give informed consent by clicking a box. Participants were informed that participation in the study was voluntary. They were made aware the study was anonymous and they could withdraw at any time.

3.3 Participants

3.3.1 Recruitment

Fifty-eight gym and fitness centre managers throughout New Zealand were approached via telephone or in person and provided with a brief description of the study, including the aims and the inclusion criteria for participants.

Twenty-four gyms and fitness centres expressed interest (Appendix C) and were sent an A4 poster via email for advertising purposes along with a detailed study information sheet, letter to email to members informing them of the LEANZ study, web link and password to access the questionnaire website (Appendix A and Appendix B).

Gyms and fitness centres advertised the study questionnaire by displaying the provided poster, sending an email to members, posting the survey link on Facebook and Twitter or spreading the word verbally.

3.3.2 Inclusion Criteria

Inclusion criteria were recreational athletes who were current New Zealand residents, over the age of 18 years. For the purposes of this study, a recreational athlete was defined as an individual with a current training schedule that involved the following:

- 1) ≥ 150 to 300 minutes (2 ½ to 5 hours) of moderate intensity physical activity (able to talk but not sing during exercise)
- or 2) 75 to 150 minutes (1 ¼ to 2 ½ hours) of vigorous intensity physical activity (unable to say a few words without pausing for a breath)
- or 3) an equivalent combination of both moderate and vigorous activities, each week.

People who met these criteria and expressed interest in the study were provided with the contact details of the study researchers in order for them to ask questions and receive the information sheet.

3.3.3 Incentives

Participants were informed in the information letter that upon completion of the study they were eligible to receive a fact sheet on food and exercise (Appendix C), as well as entry into a monthly prize draw for a \$50 grocery voucher. Participants were invited to provide their email address to receive the fact sheet and to receive notification regarding the monthly prize draw.

3.4 Questionnaire Sections

The LEANZ questionnaire consisted of several sections (Appendix A) including demographics, number, types and level of sports played, training and physical activity patterns, dietary habits, weight history, injury history, gastrointestinal symptoms and two female-specific sections regarding menstrual cycle history and oral contraceptive use. The LEANZ questionnaire also included the Low Energy Availability in Females Questionnaire (LEAF-Q) (Melin et al, 2014a) and sections of the Eating Disorder Inventory-3 (EDI-3) (Garner, 2004). Additional questions on topics associated with low energy availability that were not covered by these two validated questionnaires were also incorporated. These included “have you gained any weight in the past 3 months”, if participants responded “yes” they were asked “was this weight gain intentional” and “how did you achieve this weight gain”. Other added questions included “other people think that I am too thin”, “have you ever been told that if you were to decrease your weight your athletic performance would improve” and “do you have a family history of osteoporosis”. We added the question “have you had any exercise related injuries in the last year”, participants answering “yes” were later asked “when did you first experience these injuries”. Participants were asked “do you avoid certain types of foods” if participants responded “yes” they were asked “what foods do you avoid” and “why do you avoid these foods”. Another added question was “are you on any special diet”. Diet was not defined in the questionnaire. However, if participants responded “yes” to this question they were asked “what special diet are you on” and “why are you on this diet”.

A question in the EDI-3 asks “in the past 3 months, how often have you exercised 60 minutes or more to lose or control your weight”. To clarify why participants were exercising an additional question was added to the LEANZ questionnaire, where participants who stated they had used exercise to lose or control their weight were asked to specify whether they were exercising to lose weight or exercising to control their weight (Appendix A, questions 41, 42, 43, 45, 50, 51, 52, 67, 68, 69, 80, 82, 83, 88, 89).

3.4.1 The Low Energy Availability in Females Questionnaire (LEAF-Q)

The LEAF-Q is a validated tool used for screening female athletes at risk of the female athlete triad and thus at risk of LEA (Melin et al., 2014a). It consists of 25 questions regarding gastrointestinal symptoms, injuries and menstrual dysfunction, from 20 of these questions scores of 0-49 are derived. When a score of at least 8 was obtained, participants were considered at risk of LEA.

It is important to be mindful that the LEAF-Q has been designed for use on females, with 14 of the 20 scored questions being female specific (menstrual history and hormonal contraceptive use Appendix A, questions 21-33, 38). Whilst females can score up to 49, males only have the opportunity to score 19. Thus the cut-off score of 8 is likely to underestimate the prevalence of male participant's at risk of LEA. To adjust for this a new scoring system was developed to allow a more accurate comparison between males and females at risk of LEA.

Initially, the score needed to be classified as at risk of LEA (8) was calculated as a percentage of the total possible score for a female (49). This was found to be 16.3%. From this we determined the score males would need to obtain 16.3% of their total possible score (19) to be three. It is possible to score three after answering one question, thus this method was likely to result in an overestimation of the prevalence of LEA. Therefore we developed a new method and calculated the average score of the non-menstrual questions (gastrointestinal symptoms and injuries, Appendix A, questions 56-56, 83, 84) for females that were classed as at risk of LEA. This was used to determine the cut-off score to compare males and females risk of LEA when eliminating the female specific questions.

It is important to note this is only a proposed method. Further research is needed to validate this method and provide scientific evidence of its merit.

3.4.2 The Eating Disorder Inventory 3 (EDI-3)

The EDI-3 is a validated tool commonly used to measure the level of psychological traits or symptoms clinically relevant to the development and maintenance of an eating disorder. It does not provide a diagnosis of an eating disorder. The EDI-3 consists of 12 eating disorder risk scales, 6 composite scales and various eating disorder checklists. In this study we only used three of the subscales, the drive for thinness, bulimia and body dissatisfaction subscales, as these scales take into account the major risk factors for eating disorders that we felt were relevant to athletes. This has been verified in previous literature (Beverly et al, 1990; Sundgot-Borgen et al. 1993; Pasmán & Thomson, 1988). These risk factors include high levels of exercise, restrictive dieting, body dissatisfaction and weight concerns (Garner, 2004).

When answering the subscale questions there are six possible answers with which participants could respond. These answers were in the format of a Likert scale ranging from always to never and were scored from zero to four with the high scores reflecting extreme

behavioural or psychological traits associated with eating disorders. Scores at the non-symptomatic end received a score of zero.

A total score was calculated for each subscale. From this total raw score participants were categorised for each subscale as having a low (low clinical), medium (typical clinical) or high (elevated clinical) risk of suffering from an eating disorder that is driven by these traits. Criteria for categorisation as low, medium or high risk varied for each subscale and are described below.

3.4.2.1 The Drive For Thinness Scale

The drive for thinness scale was used to determine each participant's preoccupation with restrictive dieting, concern about dieting and fears of weight gain. It consists of seven questions relating to these traits that are answered by selecting one of the six possible answers as described above. In clinical samples the drive for thinness scale has been described as one of the core features of an eating disorder (Garner, 2004).

Participants who scored between 25 and 28 were categorised in the high risk category, those who scored between 17 and 24 were categorised in the medium risk category and participants with a score of ≤ 16 were considered low risk.

3.4.2.2 The Bulimia Scale

The bulimia scale was used to determine participant's tendency to think about or engage in uncontrollable overeating or purging to lose weight, and being emotionally upset in response to these behaviours. This scale is composed of eight questions relating to these traits that are answered by selecting one of the six possible answers as described above. Participants who scored between 19 and 32 were classed in the high risk category signifying a high level of psychopathology which is likely to indicate the presence of a clinical eating disorder. Participants scoring in this range were advised by the study researchers to see their General Practitioner to get a referral to get evaluated by a specialist familiar with eating disorders (Appendix E).

Those who scored between 5 and 18 were categorised in the medium risk category indicating they often have thoughts and behaviours consistent with binge eating and there is concern about the presence of clinically relevant overeating tendencies.

Participants with a score of ≤ 4 were considered at low risk of possessing these traits.

3.4.2.3 *The Body Dissatisfaction Scale*

The body dissatisfaction scale was used to assess participants overall feelings regarding the shape and size of certain body areas including hips, buttocks, thighs and stomach. This scale has ten questions relating to these traits that are answered by selecting one of the six possible answers as described above (section 3.4.2).

Previous research shows females put more emphasis on these body parts than males (Chatterton & Petrie, 2013; Silberstein et al., 1988). Further studies have shown drive for muscularity is a risk factor for development of eating disorders in males (Rodgers et al., 2012). Therefore, two male specific questions were added to the LEANZ questionnaire that both males and females answered. However when scoring the body dissatisfaction sub-scale these two questions replaced two questions on the original body dissatisfaction scale for the male participants in this study. Specifically the statement “I like the shape of my buttocks” was changed to “I like the shape of my upper body,” and “I think that my buttocks are too large” was changed to “I think that my torso is too small” (Appendix A, Males answered question 74 and 96 instead of question 61 and 76). These questions were adopted from previous research in this area (Martinsen et al., 2010).

Participants who scored between 36 and 40 were categorised in the high risk category, meaning they showed extreme discontent with their current body shape or size, as well as body mass. Those who scored between 22 and 35 were categorised in the medium risk category reflecting high discontent with their current body shape or size, as well as body mass and participants with a score of ≤ 21 were considered low risk.

3.4.2.4 *The Eating Disorder Inventory Risk Composite*

To calculate the EDI-3 Risk Composite, individual subscale scores were converted into a standard form. From the total raw score of each scale participants were assigned a corresponding T-score derived from comparing their scores to the international non-clinical sample used to validate the EDI-3 (Garner., 2004).

By combining the summed T-scores for the drive for thinness, bulimia and body dissatisfaction scales the EDI risk composite was calculated. The EDI risk composite provides an overall measure of disordered eating attitudes and behaviours (restricting, bingeing, purging) from these three subscales (drive for thinness, bulimia, and drive for thinness), with equal weighting for each scale.

Like the individual subscales, scores for the EDI-3 risk composite were broken down into three levels representing clinical ranges. An EDI-3 risk composite score in the high (elevated clinical) range was over the 95th percentile (T-score ≥ 56) for the international non-clinical sample used to validate the EDI-3. Scoring in this range indicated the individual has extreme eating and weight concerns that involve a fear of weight gain, desire to be thinner, binge eating tendencies, and body dissatisfaction.

3.4.2.5 The Eating Disorder Inventory Symptom Checklist

The Eating Disorder Inventory Symptom Checklist (EDI-SC) was used to determine participant's use of pathogenic weight control methods and behavioural disordered eating symptoms. It comprised five questions that provide data on the frequency of symptoms of an eating disorder including binge eating, self-induced vomiting, exercise patterns, laxative use and weight history. Each question has six possible answers ranging from "never" to "once a day or more". Each question was scored independently, and if one or more of the behavioural criteria were met the participant met the criteria for referral to an eating disorder specialist.

3.4.3 Questionnaire Adjustments

As the questionnaires were developed and validated in North American and European populations, it was decided that some questions were not applicable to New Zealand and were therefore adapted and re-worded for the New Zealand population. These questions were "in the past 6 months have you, lost 20 pounds or more" which was changed to "in the past 6 months have you, lost 9 kg or more" and "if I gain a pound, I worry that I will keep gaining" which was change to "if I gain a kilogram, I worry that I will keep gaining" (Appendix A, questions 47 and 58).

Some questions were also reworded to better suit New Zealand English using common local terminology.

These questions were "have you used oral contraceptives earlier" was changed to "have you used oral contraceptives in the past", "do you feel gaseous or bloated in the abdomen, also when you do not have your period" was changed to "do you feel gaseous or bloated? (If you are a female please only answer for times when you do not have your period)", "do you get cramps or stomach ache which cannot be related to your menstruation" was changed to "do you get stomach aches? (If you are a female please only answer for times when you do not have your period)", "how often do you have bowel movements, on average" was changed

to “on average how often do you have bowel movements”, “I have the thought of trying to vomit in order to lose weight” was changed to “I think about trying to vomit in order to lose weight” (Appendix A, Questions 35, 53, 54, 55, 62).

The question “do you have normal menstruation” was changed to “at present do you have normal menstruation” and to the possible answers “no, I’m pregnant” was added.

In the LEAF-Q the question “what kind of hormonal contraceptives do you use” offers five hormonal contraception types for participants to respond with. In New Zealand these are often referred to by their generic or brand names thus to each of the five hormonal contraceptive types we added examples of common generic or brand names offered in New Zealand. These included Ortho Evra for hormonal patch, Nuva Ring for hormonal ring, Mirena and Copper IUD for hormonal implants and Depot medroxyprogesterone acetate (DMPA) for hormonal injection (Appendix A, question 40).

3.4.4 Demographics

Demographic questions were asked regarding gender, age, occupation and highest education level attained. Participants were also asked about their current smoking status and current medication use.

Body mass index (BMI) was calculated using self-reported height and body weight. Typically those who scored high on the drive for thinness scale of the EDI-3 have a perceived discrepancy between their actual body weight and their ideal body weight (Garner, 2004). Therefore participants were also asked to state their desired body weight. Actual BMI and desired BMI were classified into four categories (underweight, normal weight, overweight, and obese) according to the New Zealand Ministry of Health guidelines (Ministry of Health, 2014).

3.4.5 Menstrual Status

Female’s current menstrual status was determined by considering the individuals response to two of three questions (Appendix A, question 24, 25 and 30). Only women who reported not currently using any form of hormonal contraception were classified with eumenorrhoea (menstrual cycles occurring at between 21 and 35 days), oligomenorrhoea (menstrual cycle length >45 days) or amenorrhoea (either primary; no menarche by age 15 or secondary; established menstruation has ceased for three or more consecutive menstrual cycles outside of pregnancy) (Mountjoy et al., 2014). Again only females who reported not currently using

any form of hormonal contraception were considered for analysis of any relationship between menstrual status and LEA.

3.4.6 Physical Activity

The recreational athletes represented 47 different sports. To simplify this analysis these individual sports were grouped into the following categories: i. as either team or individual; ii. sport that requires body revealing clothing (netball, swimming and gymnastics) or one that does not; iii. sport that emphasises low body weight or one that does not; iv. sport that is subjectively scored or one that is not; and v. sport that has weight restrictions or one that does not. Classification of each sport was decided by all four members of the research team and following discussions the final groupings were made (Appendix D).

Additionally each sport was scored for exercise intensity according to the Ainsworth Compendium of physical activity (Ainsworth et al., 2011). The Ainsworth Compendium of physical activity is a resource commonly used to assist with the coding and comparison of the intensity of physical activity data provided from questionnaires and records. A mean sport intensity score for each participant was calculated. This was the mean intensity of all sports each participant stated they participated in.

3.4.7 Injury History

Participants were divided into two groups; those who had experienced a sports related injury in the past year and those who had not. Participants classified into the injury group were further divided into four groups depending on the type of injury they have had experienced over the past year (fracture, musculoskeletal, ligament and other).

3.5 Statistical Analysis

3.5.1 Sample Size Calculation

A sample size estimate was calculated for the primary aim of estimating the prevalence of LEA in this population. An assumption that the prevalence of LEA would be in the region of 20%, a sample of 256 was calculated to provide an estimate of LEA prevalence with 95% confidence interval of approximately 5% (Burrows et al, 2007). As the population of interest is of limited size and with the project's time restraints, it was decided to recruit until a population of 170 was reached.

3.5.2 Data Handling Approach

Raw data was cleaned and coded on Excel (version 15.0.4667.1000, Microsoft Excel, 2013). All statistical analysis was carried out using Stata 13.0 (StataCorp, Texas). Mean and standard deviation were used to represent all continuous data in the sample. The estimated prevalence of participants scoring in clinical ranges and meeting referral criteria for each validated questionnaire were presented as a number, percent and 95% confidence interval (CI). A two-sided test of two proportions was used to determine if there was a statistically significant difference between the proportions of participants at risk of LEA for different variables, including sport category and sex. Differences between groups are presented as mean and standard error of the mean. Logistic regression was used to determine the odds of being at risk of LEA for every one unit increase in a participant's characteristic. Results were expressed as odds ratios and confidence intervals. A level of <0.05 was selected to detect significant differences.

4 Results

4.1 Respondent Characteristics

One hundred and seventy participants (61 males and 109 females) completed the LEANZ questionnaire. A further 17 people showed interest in the study however did not complete the questionnaire and were not included in the analysis. The descriptive characteristics of the participants are presented in Table 4.1.

Participants were aged from 18 to 56 years (females 18 to 54 years, males 18 to 56 years). The mean height was 1.72 m (SD= 0.09 m), and mean weight 71.2 kg (females 64.8 kg (SD= 9.6 kg) and males 82.5 kg (SD= 11.5 kg)).

Participants reported being physically active, with a mean number of hours exercised per week greater than the physical activity guidelines. Time engaged in exercise ranged from 1.3 to 18 hours per week for males and 2 to 24 hours per week for females.

The majority of participants (73.5%) were University educated.

4.1.1 Actual and Desired Body Mass

Although the majority of participants were of healthy body mass (as classified by BMI), five participants, all of whom were female, reported a height and body mass which would class them as underweight. All five of these participants desired a weight that categorised them in this underweight BMI category.

Of the 83 female participants who were classed as having a healthy weight for their height, 77 desired a weight which would class them as healthy. Of the remaining six, two wished to be underweight and four overweight. On the other hand all of the 26 males who were classed as having a healthy BMI, desired to stay within this category.

Only two of the 16 females categorised with an overweight BMI desired a weight that classed them within this BMI category. The remaining 14 desired a weight that would place them in the “healthy” BMI range. Seventeen of the 26 males with an overweight BMI desired a weight that classed them in their current BMI category, seven reported a desired weight classing them as “healthy” and the remaining two desired a weight where they would be classified as “obese”.

Six participants, three males and three females were categorised as “obese” according to their current BMI. Of the females, only one desired a weight that would keep her in this

category. This participant did not play a sport but attended gym classes or walked/jogged four times a week. The other two females desired a weight that would classify them as “healthy.” All three males however desired a weight that would classify them as “overweight”.

Five participants did not state a desired BMI, they were all classed as having a BMI in the “healthy” range.

Table 4-1 Respondent anthropometric and demographic characteristics.

| Characteristic | Total (n=170) | Male (n=61) | Female (n=109) |
|---|--------------------------|-------------|----------------|
| Weight(kg) ^{1,2} | 71.2 (13.4) ² | 82.5 (11.5) | 64.8 (9.6) |
| Height (m) ^{1,2} | 1.72 (0.09) | 1.81 (0.07) | 1.68 (0.1) |
| Body mass index (kg.m²) ^{2,3} | 23.8 (3.2) | 25.2 (3.0) | 23.0 (3.0) |
| Age (y) ¹ | 24.7 (7.1) | 26.2 (7.3) | 23.8 (6.9) |
| Exercise per week (h) ^{1,2} | 8.5 (5.0) | 9.1 (4.2) | 8.4 (5.4) |
| Body mass index (kg.m²) ^{3,4} | | | |
| Underweight (<18.5) ^{3,4} | 4 (2.4) | 0 (0.0) | 4 (3.7) |
| Healthy range (18.5 – 24.9) ^{3,4} | 114 (67.1) | 31 (50.8) | 83 (76.2) |
| Overweight (25.0 – 29.9) ^{3,4} | 42 (38.5) | 26 (42.6) | 16 (14.7) |
| Obese (≥30.0) ^{3,4} | 9 (5.3) | 4 (6.6) | 5 (4.6) |
| Highest level of education attained | | | |
| Primary School ^{1,4} | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| Some Secondary School ^{1,4} | 10 (5.9) | 4 (6.6) | 6 (5.5) |
| Completed Secondary School(7th form or NCEA Level 3) ^{1,4} | 21 (12.4) | 6 (9.8) | 15 (13.8) |
| Technical /trade school ^{1,4} | 4 (2.4) | 1 (1.6) | 3 (2.8) |
| Polytechnic ^{1,4} | 10 (5.9) | 2 (3.3) | 8 (7.3) |
| University ^{1,4} | 125 (73.5) | 48 (78.7) | 77(70.6) |

¹ Self-reported

² Mean (Standard deviation)

³ Calculated from self-reported height and weight

⁴ Number (percentage)

Note: Percentages may not add up to 100% due to rounding.

4.2 The Low Energy Availability in Females Questionnaire (LEAF-Q)

Scoring at least eight on the LEAF-Q classifies respondents as being at risk of LEA. Table 4.2 describes the percentage of participants at risk of LEA. A total of 57 (33.5%, 95% CI 26.5, 41.2%) participants met this criteria, with a significant sex difference for the percent classed as at risk of LEA (males 13.1%, 95% CI, 5.8%, 24.2%, females 45.0%, 95% CI, 35.4%, 54.8%; $p < 0.001$). Logistic regression indicated females have approximately 5.4 times greater probability (OR 5.4, 95% CI 2.4, 12.5, $p < 0.001$) of being at risk of LEA when compared to males.

The mean score for males on the LEAF-Q was 3.8 whereas the mean score for females was 7.3. However because the LEAF-Q is designed for females and includes questions on menstruation, we proposed several new scoring systems to enable generation of a comparable score for males. This is outlined in section 3.4.2. When calculating the percentage of males classified as at risk of LEA using our new proposed scoring systems we found:

1. Converting the risk cut-off for females to a percentage and then using this percent for males, resulted in, forty three of the 61 males (70.5%, 95% CI 57.4%, 81.5%) scoring above the cut off (14.9% or at least 3), thus as predicted this method was likely to be an overestimation.
2. Females who were classed as being at risk of LEA scored an average of (5) for the non-menstrual/non-female specific questions (gastrointestinal symptoms and injuries). The number of males who also scored more than 5 on these questions increased the number classed as at risk from LEA from 8 (13.1%, 95% CI, 5.8%, 24.2%) (using the standard scoring point system) to 20 (32.8%, 95% CI 21.3%, 46.0%). 38 females that were also classed as at risk of LEA on the original scoring system (77.6% 95% CI, 63.4%, 88.2%) met this criteria.

Table 4-2 Estimated prevalence of participants at risk of low energy availability and at risk of an eating disorder.

| | At risk LEA | At risk LEA + DT | | | At risk LEA + BD | | | At risk LEA + B | | | At risk LEA + RC | | | |
|---------------------------|-------------|------------------|------------|-----------|------------------|-----------|-----------|-----------------|-----------|-----------|------------------|------------|---------|----------|
| | | L | M | H | L | M | H | L | M | H | L | M | H | |
| Total (n=170) | Yes | 57 (33.5) | 44 (77.2) | 7 (12.3) | 6 (10.5) | 44 (77.2) | 11 (19.3) | 2 (3.5) | 36 (63.7) | 17 (29.8) | 4 (7.0) | 51 (89.5) | 3 (5.3) | 3 (5.3) |
| | No | 113 (66.5) | 100 (88.5) | 12 (10.6) | 1 (0.9) | 91 (80.5) | 19 (16.8) | 3 (2.7) | 93 (82.3) | 18 (15.9) | 2 (1.8) | 106 (93.8) | 6 (5.3) | 1 (0.9) |
| Male (n=61) | Yes | 8 (13.1) | 7 (87.5) | 1 (12.5) | 0 (0.0) | 7 (87.5) | 1 (12.5) | 0 (0.0) | 6 (12.5) | 1 (12.5) | 1 (12.5) | 7 (87.5) | 0 (0.0) | 1 (12.5) |
| | No | 53 (86.9) | 53 (100.0) | 0 (0.0) | 0 (0.0) | 50 (94.3) | 3 (5.7) | 0 (0.0) | 52 (98.1) | 0 (0.0) | 1 (1.9) | 52 (98.1) | 1 (1.9) | 0 (0.0) |
| Female (n=109) | Yes | 49 (45.0) | 37 (75.5) | 7 (14.3) | 5 (10.2) | 37 (75.5) | 10 (20.4) | 2 (4.1) | 30 (61.2) | 16 (32.7) | 3 (6.1) | 44 (89.8) | 3 (6.1) | 2 (4.1) |
| | No | 60 (55.1) | 47 (78.3) | 12 (20.0) | 1 (1.7) | 41 (68.3) | 16 (5.0) | 3 (26.7) | 41 (68.3) | 18 (30.0) | 1 (1.7) | 54 (90.0) | 5 (8.3) | 1 (1.7) |

Abbreviations: DT = Scoring in the range that categorises participants in the low (L) medium (M) or high (H) risk of an eating disorder based on score from the Eating Disorder Inventory-3 drive for thinness sub-scale, BD= Scoring in the range that categorises participants in the medium or high risk of an eating disorder based on score on the Eating Disorder Inventory-3 body dissatisfaction sub-scale, B= Scoring in the range that categorises participants in the medium or high risk of an eating disorder based on score on the Eating Disorder Inventory-3 bulimia sub-scale, RC = Scoring in the range that categorises participants in the medium or high risk of an eating disorder based on score on the Eating Disorder Inventory-3 risk composite scale.

Note: Percentages may not add up to 100% due to rounding. Data are presented as n(%)

4.3 The Eating Disorder Inventory (EDI-3) Questionnaire

Figure 3 and Figure 4 show the estimated prevalence of females and males scoring in each clinical range (low, medium or high risk of an eating disorder) for each Eating Disorder Inventory-3 (EDI-3) sub-scale.

Scores from the drive for thinness, body dissatisfaction and bulimia sub-scales showed the majority of participants were considered to have a low risk of an eating disorder with 98.4% (95% CI, 91.2%, 100%), 93.4% (95% CI, 84.1%, 98.2%) and 95.1% (95% CI, 86.3%, 99.0%) of males and 77.1% (95% CI, 68.0%, 84.6%), 71.6% (95% CI, 62.1%, 79.8) and 65.1% (95% CI, 55.4%, 74.9%) and of females scoring in the low clinical range for these sub-scales respectively. A higher percentage of males than females scored in this range for all three sub-scales.

Nineteen participants (11.2%, 95% CI 6.9%, 16.9% all female) scored within the medium clinical range on the EDI-3 drive for thinness sub-scale. One male (1.6%, 95% CI 0.0%, 8.8%) and six females (5.5%, 95% CI 2.0%, 11.6%) scored within the high clinical range on this sub-scale. These participants are considered at high risk of an eating disorder.

Table 4.2 shows that of the participants who were classified as at risk of LEA, 12.5% (95% CI, 0.3%, 52.5%) of these males and 24.5% (95% CI, 13.3%, 38.9%) of these females were also considered at risk of an eating disorder due to their score on the EDI-3 drive for thinness sub-scale (medium or high risk category). All the males who were not at risk of LEA were also not at risk of an eating disorder (low risk category) on this sub-scale, however 21.7% (95% CI, 12.1%, 34.2%, n= 13) of females were at risk of an eating disorder but not LEA.

Just over one fifth of participants (20.5%, 95% CI 14.8%, 27.5%, n=35) scored within the medium clinical range on the EDI bulimia sub-scale. This was predominately females with only one male meeting this criteria. Two male participants (3.3%, 95% CI, 0.4%, 11.3%) however scored within the high clinical range on the EDI-3 bulimia-scale. Four females (3.7%, 95% CI, 1.0%, 9.1%) also met this criteria.

A quarter of males (25%, 95% CI, 3.1%, 65.1%) and nearly 40% (38.8%, 95% CI, 25.2%, 53.8%) of females who were classified as at risk of LEA were also classified as at risk of an eating disorder from their score on the EDI-3 bulimia sub-scale (medium or high risk category). Of the participants not classified as at risk of LEA, 1.9% (95% CI, 0.0%, 10.1%) of males and 31.7% (95% CI, 20.3%, 45.0%) of females also scored in this range on the EDI-3 bulimia sub-scale.

Four males (6.6%, 95% CI, 1.8%, 15.9%) and 26 females (23.9%, 95% CI, 16.2%, 33.0%) scored within the medium clinical range for the EDI-3 body dissatisfaction sub-scale. A total of five participants, all of whom were females, scored within the high clinical range on the EDI-3 body dissatisfaction sub-scale.

Of female participants who were classified as at risk of LEA 24.5% (95% CI, 13.3%, 38.9%) scored in the range that classifies them as at risk of an eating disorder due to their score on the EDI-3 body dissatisfaction sub-scale (medium or high risk category). A similar proportion (31.7%, 95% CI, 20.3%, 45.0%) of females who were not considered at risk of LEA also scored in this range. From the males who were classified as at risk of LEA 12.5% (95% CI, 0.3%, 52.7%) were also classified as at risk of an eating disorder based on their EDI-3 body dissatisfaction score (medium or high risk category) and 5.7% (95% CI, 1.2%, 15.7%) of males who were not classified as at risk of LEA were at risk of an eating disorder (medium risk category) on this sub-scale.

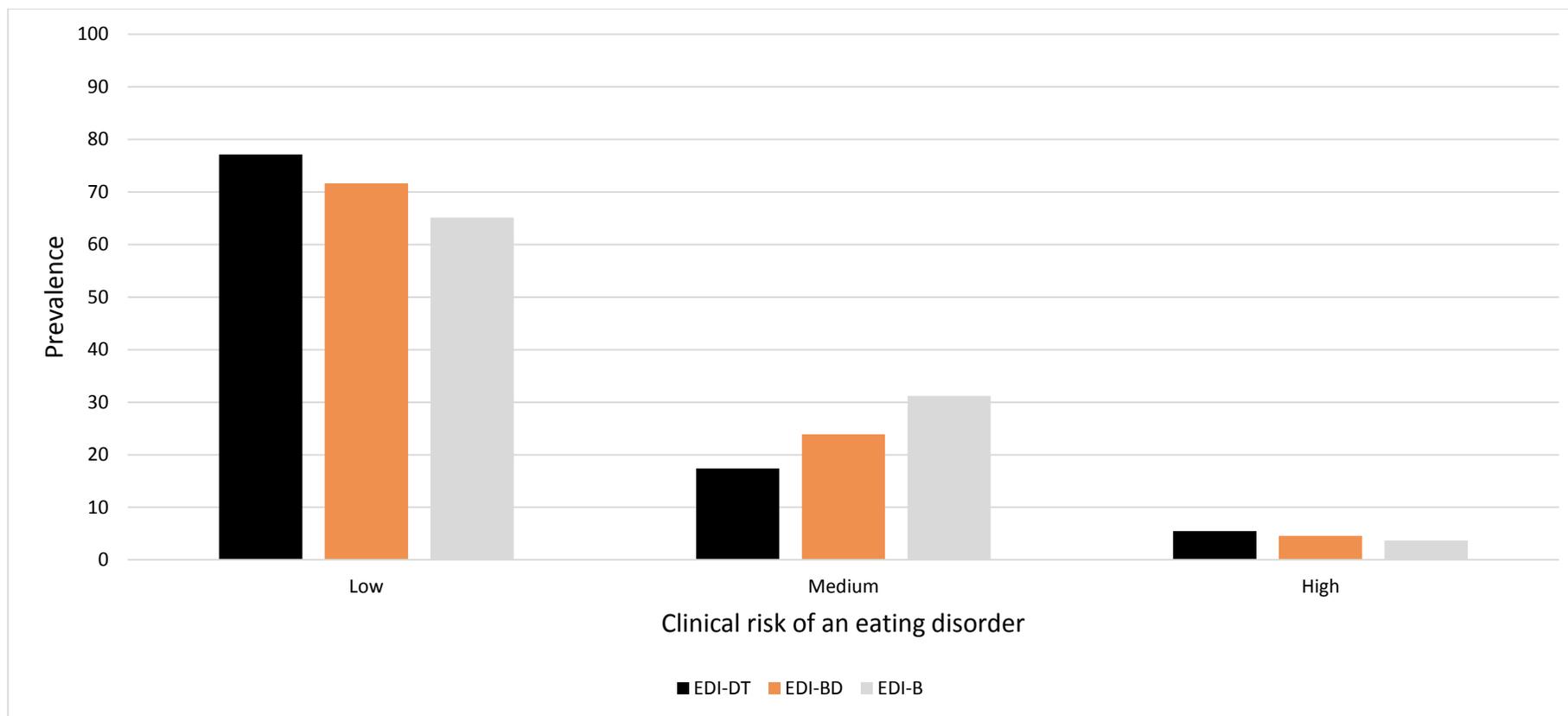


Figure 3. Estimated prevalence (%) of female participants scoring in the clinical range for each Eating Disorder Inventory sub-scale

Abbreviations - DT = Eating Disorder Inventory drive for thinness sub-scale, EDI-B = Eating Disorder Inventory bulimia sub-scale, EDI-BD = Eating Disorder Inventory body dissatisfaction sub-scale.

Note: Percentages may not add up to 100% due to rounding.

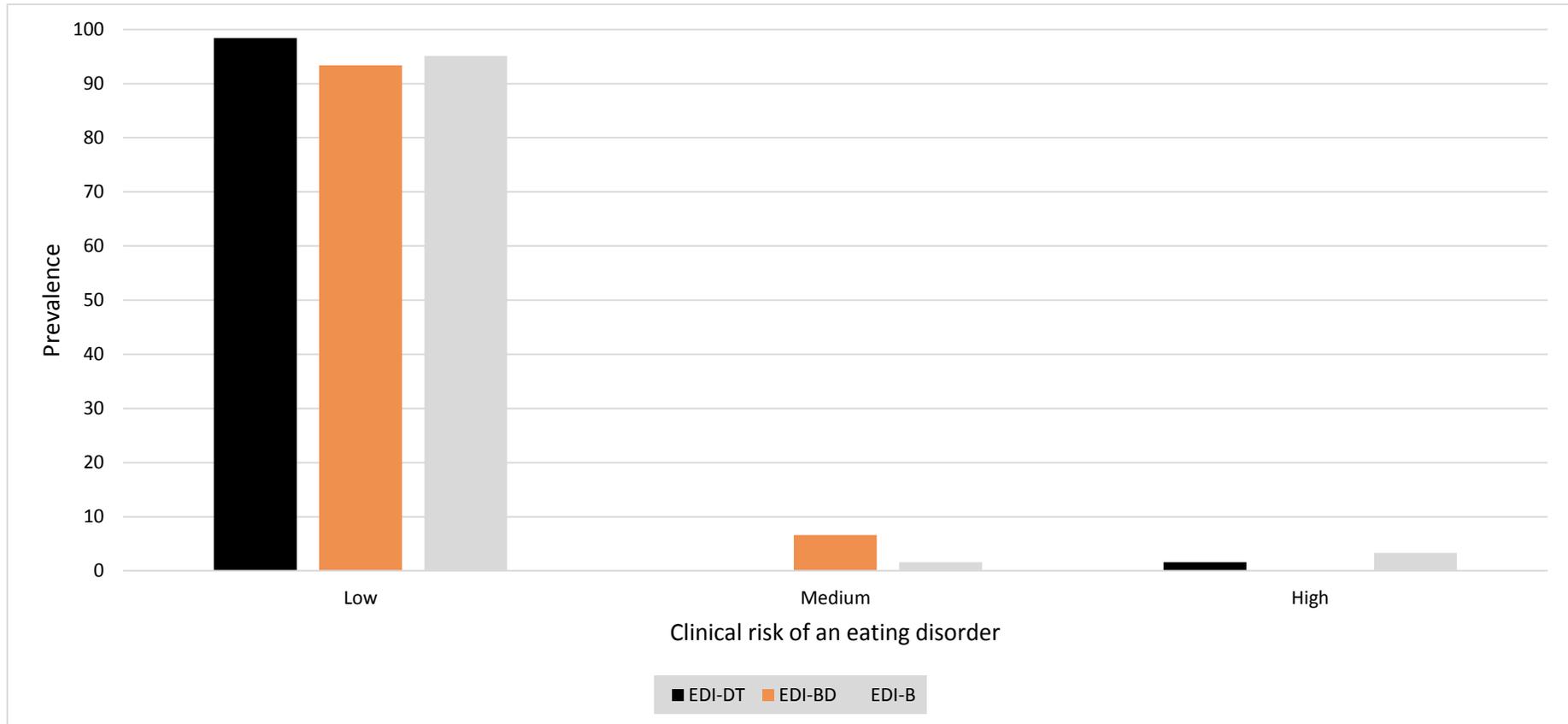


Figure 4 Estimated prevalence (%) of male participants scoring in the clinical range for each Eating Disorder Inventory sub-scale

Abbreviations – EDI-DT = Eating Disorder Inventory drive for thinness sub-scale, EDI-B = Eating Disorder Inventory bulimia sub-scale, EDI-BD = Eating Disorder Inventory body dissatisfaction sub-scale.

Note: Percentages may not add up to 100% due to rounding.

4.3.1 Multiple Questionnaires

The majority of participants (n=118, 69.4%, 95% CI, 61.9%, 76.2%) scored in the range which classified them as having a low risk of an eating disorder for all three EDI-3 sub-scales (drive for thinness, bulimia and body dissatisfaction), this was 91.8% of males (95% CI, 81.9%, 97.3%, n=56) and 56.9% of females (95% CI, 47.0%, 66.3%, n=62). Seven female (6.4%, 95% CI, 2.6%, 12.8%) and no male participants scored within the medium risk range for all three EDI-3 sub-scales. Only two of these seven females were also classified as at risk of LEA. No participants scored within the high risk range for all three EDI-3 sub-scales.

4.3.2 Eating Disorder Inventory Risk Composite (EDIRC)

Figure 3. describes the estimated prevalence of participants scoring in the low, medium and high range for risk of an eating disorder on the Eating Disorder Inventory Risk Composite (EDIRC).

The majority of participants (n= 157, 92.4%, 95% CI, 87.3%, 95.9%) scored in the low risk range, with a higher percentage of males (n= 59, 96.7%, 95% CI, 88.7%, 99.6%) classified as having a low risk of an eating disorder when compared to females (n= 98, 89.9%, 95% CI, 82.7%, 94.9%). About 45% (44.9%, 95% CI, 34.8%, 55.3%) of females and 11.9% (CI, 4.9%, 22.9%) of males scoring in this range were classified as at risk of LEA.

Only one male scored in the medium risk range for the EDIRC, this individual did not meet the criteria for being at risk of LEA. Eight females (7.3%, 95% CI, 3.2%, 14.0%) scored in this range with three of them also meeting the criteria for being at risk of LEA.

Of the participants who scored in the high risk range for the EDIRC one male, who was also considered at risk of LEA met this criteria along with three females, two of who met the criteria for being at risk of LEA. The male participant that fell within this range had a body mass of 63.8 kg (BMI 21.1 kg.m²) and the three females 64 kg, 82 kg and 90 kg (BMI 20.2 kg.m², 26.2 kg.m² and 29.1 kg.m² respectively).

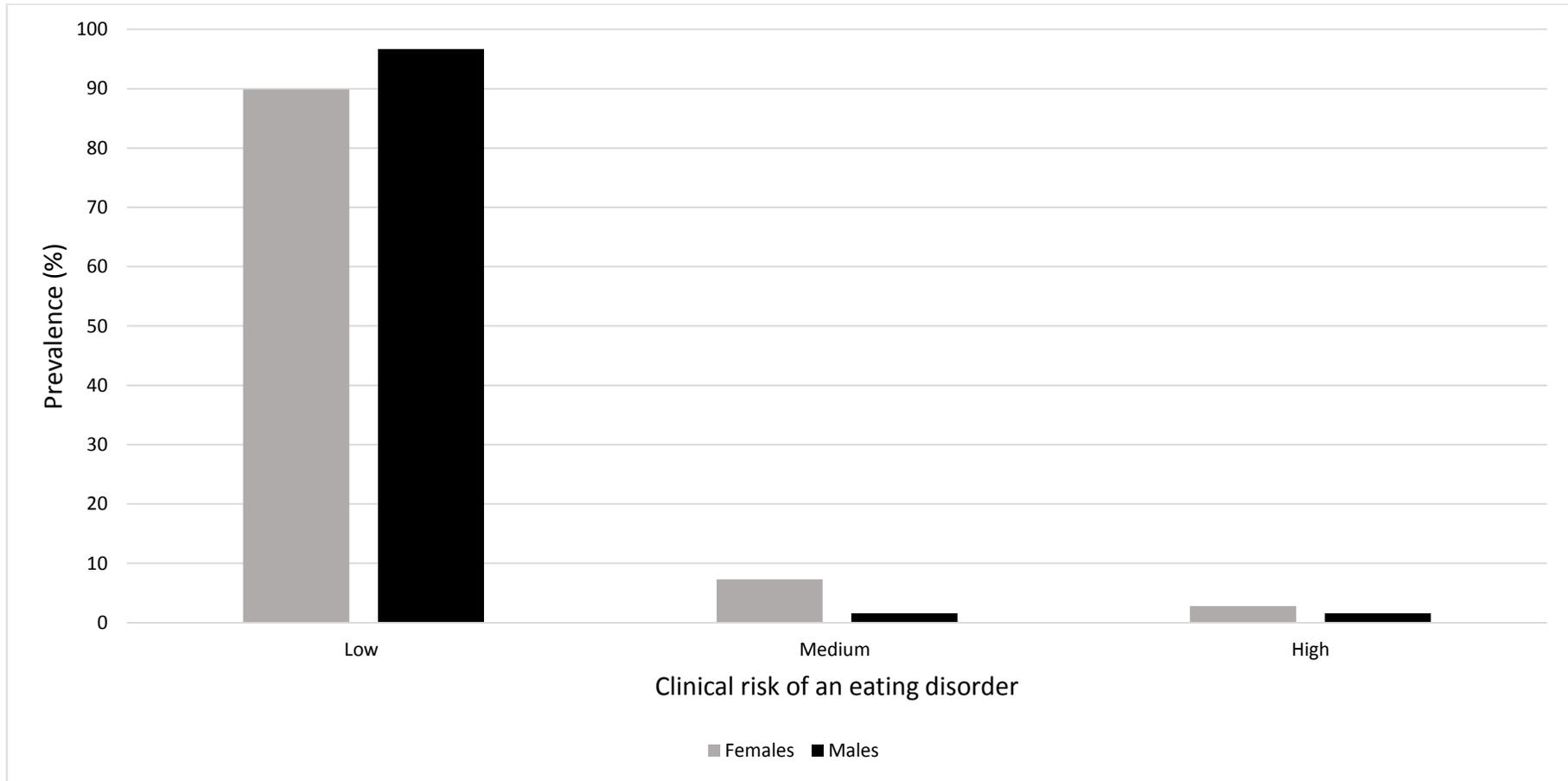


Figure 5 Estimated prevalence (%) of participants scoring in each clinical range for the Eating Disorder Inventory Risk Composite

Note: Percentages may not add up to 100% due to rounding

4.4 Predictors for Being at Risk of Low Energy Availability (LEA).

Table 4.3. describes an estimate of the prevalence of being at risk of LEA by sport category. Participants had the opportunity to list the five main sports they engage in. For the purpose of this analysis only the first sport each participant listed was taken into consideration, making the assumption it is the main sport they play.

Twenty seven percent (95% CI 16.2%, 37.3%) of participants competing in team sports were considered at risk of LEA. This was significantly lower ($p=0.045$) than participants competing in individual sports, where 45.2% (95% CI 29.5%, 60.9%) met the criteria. There was no association between participating in a team or individual sport and risk of an eating disorder ($p= 0.639$).

There was no statistically significant difference ($p=0.290$) between the estimated prevalence of participants at risk of LEA among participants that play sports that emphasise low body weight (42.3%) when compared to those that do not (31.0%). There was also no association between participating in a team or individual sport and risk of an eating disorder ($p=0.448$)

Due to difficulty in determining whether running is considered a sport requiring body revealing clothing or not, separate analyses were carried out with running in both categories. Tendencies for a difference were observed when running was categorised as requiring revealing clothing ($p=0.065$) and when running was categorised as not requiring revealing clothing ($p=0.098$). In both instances a higher percentage of participants were considered at risk of LEA if they participated in a sport that requires body revealing clothing to be worn (39.7% and 40.3%) compared to participants that compete in sports that do not (22.5% and 25.5%).

Insufficient numbers of participants reported participating in sports that are subjectively scored and sports with weight restrictions to perform statistical tests.

Table 4-3 Estimated prevalence of participants at risk of low energy availability by sport category.

| Sport category | | At risk LEA (n) | Percent | 95% CI | Difference (SD) % | P value |
|---|------------|------------------------|----------------|---------------|--------------------------|----------------|
| Team vs individual | T (n=71) | 19 | 26.8 | 16.2, 37.3 | 18.4 (9.1) | 0.045 |
| | I (n=42) | 19 | 45.2 | 29.5, 60.9 | | |
| Emphasising low body weight (leanness) | Yes (n=26) | 11 | 42.3 | 22.0, 62.7 | 11.3 (10.6) | 0.290 |
| | No (n=87) | 27 | 31.0 | 21.1, 41.0 | | |
| Requiring body revealing clothing including running | Yes (n=73) | 29 | 39.7 | 28.2, 51.2 | 17.2 (9.2) | 0.065 |
| | No (n=40) | 9 | 22.5 | 9.0, 36.0 | | |
| Requiring body revealing clothing not including running | Yes (n=62) | 25 | 40.3 | 27.8, 52.9 | 14.8 (8.9) | 0.098 |
| | No (n=51) | 13 | 25.5 | 13.1, 37.9 | | |

Note – Participants were considered at risk of LEA if they scored ≥ 8 on the LEAF questionnaire.

Abbreviations: T= participation in a team sport, I = participation in an individual sport, LEA = Low energy availability

The odds of being at risk of LEA based on participant characteristics are presented in Table 4.4.

There was a statistically significant negative association between BMI and risk of LEA. For every one unit decrease in BMI the odds of being at risk of LEA are 11% higher (OR 0.89, 95% CI 0.80, 1.00, $p=0.045$).

There was a significant positive association with risk of LEA and number of hours of exercise per week. For every extra hour of exercise per week participants were 1.08 (OR 1.08, 95% CI 1.00, 1.15, $p=0.039$) times more likely to be at risk of LEA. There was no statistically significant association between level of education, age and number of sports played and being at risk LEA.

Males and females odds of being at risk of LEA by participant characteristics were then looked at separately. Table 4.5. describes the odds of females being at risk of LEA by participant characteristics. For every extra hour exercise female participants performed per week the odds of being at risk of LEA were 1.13 times greater (OR 1.13, 95% CI 1.03, 1.25, $p=0.016$). There was no association between BMI, level of education, age and number of sports played and female participants being at risk LEA.

As shown in Table 4.6. there was no significant association between BMI, number of hours exercise per week, level of education, age and number of sports played and male participants being at risk LEA.

Table 4-4 Odds ratio of being at risk of low energy availability¹, for every one unit increase in characteristic, n= 170.

| Characteristic | Odds Ratio | 95% Conf. Interval | P value |
|--|------------|--------------------|---------|
| BMI (kg.m²) | 0.89 | 0.80, 1.00 | 0.045 |
| Exercise per week (hours) | 1.08 | 1.00, 1.15 | 0.039 |
| Education ² | 0.92 | 0.73, 1.17 | 0.499 |
| Age (years) | 0.98 | 0.93, 1.03 | 0.346 |
| Intensity of sport ³ | 1.00 | 0.79, 1.27 | 0.988 |
| Sports played (number) | 0.87 | 0.68, 1.11 | 0.257 |

¹ Participants were considered at risk of LEA if they scored ≥ 8 on the LEAF questionnaire.

² From lowest education level to highest - primary school, some secondary school, completed secondary school (7th form Bursary or NCEA Level 3), technical /trade school, polytechnic, university.

³ Measured using the Ainsworth compendium of physical activity, score was an average of all sports each participant listed (up to 5).

Table 4-5 Odds ratio of being at risk of low energy availability¹ for every one unit increase in characteristic among females, n=109

| Characteristic | Odds Ratio | 95% Conf. Interval | P value |
|--|------------|--------------------|---------|
| BMI (kg.m²) | 0.91 | 0.79, 1.04 | 0.160 |
| Exercise per week (h) | 1.13 | 1.03, 1.25 | 0.016 |
| Education ² | 1.03 | 0.77, 1.37 | 0.847 |
| Age (y) | 1.0 | 0.94, 1.05 | 0.909 |
| Intensity of sport ³ | 1.27 | 0.83, 1.93 | 0.273 |
| Sports played (number) | 0.92 | 0.70, 1.22 | 0.582 |

¹ Participants were considered at risk of LEA if they scored ≥ 8 on the LEAF questionnaire

² From lowest education level to highest - primary school, some secondary school, completed secondary school (7th form Bursary or NCEA Level 3), technical /trade school, polytechnic, university

³ Measured using the Ainsworth compendium of physical activity, score was an average of all sports each participant listed (up to 5).

Table 4-6 Odds ratio of being at risk of low energy availability¹ for every one unit increase in characteristic among males, n=61.

| Characteristic | Odds Ratio | 95% Confidence Interval | P value |
|--|------------|-------------------------|---------|
| BMI (kg.m²) | 1.24 | 0.93, 1.66 | 0.142 |
| Exercise per week (h) | 0.99 | 0.81, 1.20 | 0.893 |
| Education ² | 0.70 | 0.44, 1.14 | 0.155 |
| Age (y) | 0.98 | 0.87, 1.10 | 0.714 |
| Intensity of sport ³ | 0.99 | 0.67, 1.47 | 0.972 |
| Sports played (number) | 0.50 | 0.21, 1.19 | 0.116 |

8

¹ Participants were considered at risk of LEA if they scored ≥ 8 on the LEAF questionnaire

² From lowest education level to highest - primary school, some secondary school, completed secondary school (7th form or NCEA Level 3), technical /trade school, polytechnic, university.

³ Measured using the Ainsworth compendium of physical activity

4.4.1 Menstrual Cycle Status

Every female participant reported having experienced their first menstruation (menarche) and also reported it had occurred naturally. Ten females reported being 15 years or older when they experience menarche. Half (n=5) of these females were classified as at risk of LEA.

Of the 109 female participants, 71 (65.1%) were currently using the OCP. Of those not using the OCP 21.1% (n=8) were currently using another form of hormonal contraception, including a hormonal coil (n=4), hormonal injection (n=3) and hormonal implant (n=3).

Of the females not currently taking the OCP just over half (n=21, 55.3%) had used them in the past (between 1 and 20 years ago). The time frame these participants reported using the OCP for ranged from 1 to 20 years.

Those who were currently using the OCP or had used it in the past (n=92) were asked why they use/used it. The majority (n=66) used the OCP for contraception, 12 used it to regulate their menstrual cycle in relation to performance, six used the OCP for reduction of menstrual pains, four for reduction of bleeding, and two for other reasons not specified. The remaining two females stated they used the OCP to maintain menstruation. Both of these participants were classified as at risk of LEA. There was no significant association between current use of OCP and risk of LEA (OR, 1.37, 95% CI, 0.62, 3.02, p=0.439).

Female participants were asked how many periods they have had during the last year. For every three month decrease in number of periods in the last year females had experienced they were 1.61 times more likely to be at risk of LEA (OR 1.61, 95% CI 1.00, 2.59, p=0.049). There was no significant association between the number of periods females who were currently not using the OCP or another form of hormonal contraception and risk of LEA (OR 1.50, 95% CI 0.91, 2.46, p= 0.109).

Female participants were asked if they felt they currently have normal menstruation, 79 (72.5%) stated they did, 29 (26.6%) felt they did not and one (0.9%) participant did not know.

Menstrual status of female participants is presented in Figure 4. In total 30 (27.5%) of the 109 female participants were not currently taking the OCP or any other form of hormonal contraception. Of these women, 21 (70%) felt they had normal menstruation

with 20 (66.7%) reporting having their last period in the past month, classifying these participants as eumenorrhoeic. Five of the 20 eumenorrhoeic athletes were classified as at risk of LEA. The one remaining female reported having her last period in the past one to two months. Due to difficulty determining whether she met the criteria for eumenorrhoea or oligomenorrhoea she was not classified in a menstrual status group however was classed as at risk of LEA.

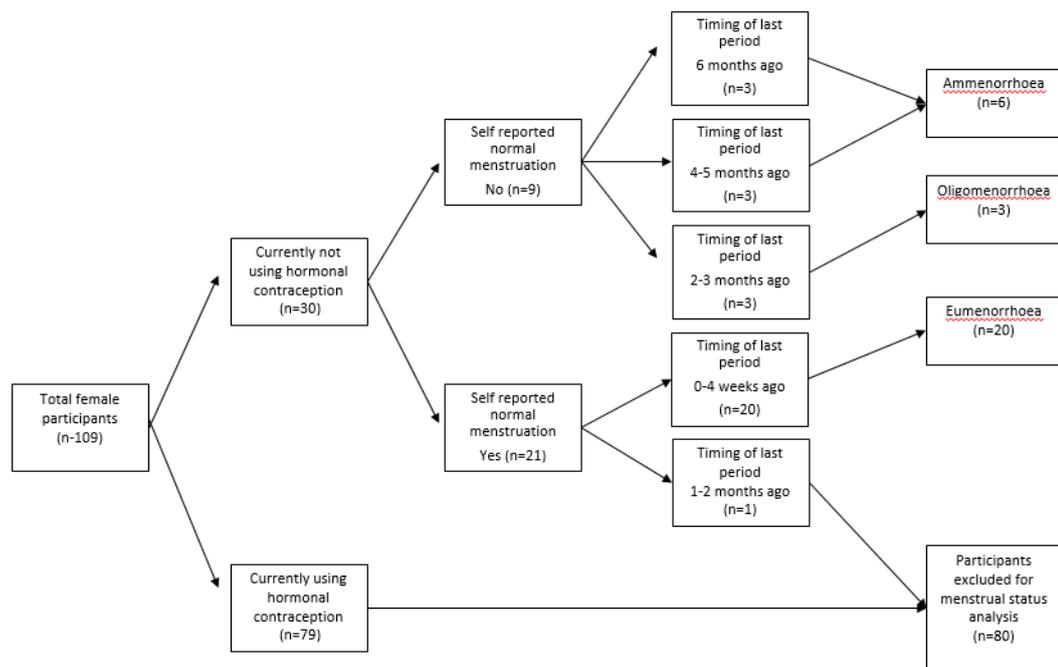


Figure 6 Female participants classification of menstrual status.

Of the nine participants who felt they did not have normal menstruation three had their last period 2-3 months ago classifying them as oligomenorrhoeic. Two of these oligomenorrhoeic females were also classified as at risk LEA

The final six had not had a period for at least four consecutive months classifying them as amenorrhoeic. Four of which were also classified as at risk of LEA, and one had a BMI <18.5 kg.m²

Of the 79 females currently taking an oral contraceptive pill or using any other hormonal contraceptive 20 (25.3%) stated they did not currently have normal menstruation, seven of which had not had a period for at least six months and the remaining 13 had their last period 2-3 months ago. Forty seven reported having their last period within the past month, eight, one to two months ago, four, three to four months ago and one participant that had not had a period in the past 5 months.

The participant that did not know if she currently has normal menstruation was on the OCP and reported having her last period over six months ago.

4.4.2 Injury History and Bone Health

Males had a higher incidence of exercise related injury with 73.8% (n=45) reporting an injury in the past year compared to 58.7% of females. Six (9.8%) males reported at least one fracture, none of which were stress fractures. On the other hand a similar proportion of females (8.3%, n= 9) reported at least one fracture but four out of nine of these were stress fractures. Three of the females described their stress fracture as occurring in the “foot” and the other in the “femur”, and all four of these females were at risk of LEA.

Although there was no significant association between days off training due to sporting injury and risk of LEA for males (p=0.731), for every extra day off training due to injuries females were two times more likely to be at risk of LEA (OR, 2.04, 95% CI, 1.26, 3.31, p=0.004)

There was a significant positive association between the total number of exercise related injuries both male and female participants had suffered in the past year and risk of LEA. For every extra injury males had suffered in the past year they were 4.10 times more likely to be at risk of LEA (OR, 4.10, 95% CI, 1.31, 12.85, p=0.015). For every extra injury females had suffered in the past year they were 2.04 times more likely to be at risk of LEA (OR 2.04, 95% CI 1.22, 4.75, p=0.011).

All the males that were at risk of LEA (n=8) had sustained at least one exercise related injury in the past year compared to 75.5% (n=37) of females that were at risk of LEA.

A total of ten participants reported a family history of osteoporosis, of these participants one male and one female had suffered from at least two exercise related fractures in the past year.

4.4.3 Diet and Exercise Habits and Risk of Low Energy Availability (LEA)

To the question ‘are you on any special diet?’ nineteen participants (11.2%) responded ‘yes’. Diets participants listed included, but were not limited to, dairy-free, gluten-free, vegetarian, various weight loss diets and the low FODMAPS diet. The acronym FODMAPS refers to a range of carbohydrates commonly found in the diet - fermentable oligosaccharides, disaccharides, monosaccharides and polyols. These carbohydrates can be poorly absorbed in the intestine and restricting their intake has been found to reduce symptoms of irritable bowel syndrome (Halmos et al., 2014).

Participants following the FODMAPS diet, as well as two of the four participants following gluten-free diets, were undertaking these diets for medical reasons. The remaining 14 participants had decided to commence following a special diet due to personal preference.

Eight of these participants (42.1%) were considered at risk of LEA. From the different diets participants listed, two out of eight participants following weight loss diets, two out of three participants following the FODMAPS diet and three out of four participants following the gluten-free diets were considered at risk of LEA.

One hundred and six participants (62.4%) stated they avoid certain types of food, including 65.1% (n=71) of females and 57.4% (n=35) of males. Common foods that were avoided were high-sugar and high-fat, junk food, processed foods and carbohydrates. There was no association between avoiding foods and risk of LEA (OR 1.49, 95% CI 0.76, 2.92, p=0.247).

When separated into the different food types participants were avoiding, there was still no association with risk of LEA (carbohydrates p=0.852, high-fat p=0.325, junk food (including fizzy drink, takeaways, lollies, chocolate, p=0.534), and high-sugar, p=0.915).

There was no association between risk of LEA and avoiding certain foods for weight loss ($p=0.882$) or risk of LEA and avoiding foods with the perception it would make them more “healthy” ($p=0.926$).

In the past 3 months the majority (70.6%) of participants had exercised greater than 60 minutes to lose weight, 93% ($n=112$) of who were also at risk of LEA. Over a third of females (36.7%) and 27.9% males reported doing so between once a month and once a week, 35.8% females and 23% of males 2-6 times per week and 6% of females and 7% of males reported at least once a day. About two fifths (43%) of males and one fifth (22%) of females reported never exercising to lose weight. There was no significant association between exercising to lose weight and risk of LEA ($p=0.081$)

Table 4.7 describes the estimated prevalence and frequency of participants using pathogenic weight control methods in the past 3 months. A total of 10 (5.9%) participants, 1 (1.6%) male and 9 (8.3%) females, had used at least one pathogenic weight control method, in the past 3 months. All 10 participants using pathogenic weight loss methods reported vomiting to lose weight, 7 of which were also at risk of LEA (1 male, 6 female). The male who reported vomiting to lose weight, stated he did so 2-6 times per week and was classified as at risk of LEA. A total of two females also reported vomiting to lose weight 2-6 times per week in the past 3 months, one of these females was also classified as at risk of LEA. No male and one female (0.9%) participant reported vomiting once a day to lose weight, this participant was also at risk of LEA.

No participant had used laxatives to lose weight in the past three months.

Participants were no more likely to be at risk of LEA or an eating disorder if they had been told their performance would increase if they were to lose weight ($p=0.334$ and 0.913 respectively).

Table 4-7 The estimated prevalence and frequency of participants using pathogenic weight control methods¹ in the past 3 months

| | Pathogenic weight loss method | Once a month or less | 2-3x/ month | Once a week | 2-6 times a week | Once a day or more |
|---------------------|--------------------------------------|-----------------------------|--------------------|--------------------|-------------------------|---------------------------|
| Total n=170 | Laxatives | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| | Vomiting | 6 (3.5) | 0 (0.0) | 0 (0.0) | 3 (1.7) | 1(0.6) |
| Male n=61 | Laxatives | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| | Vomiting | 0 (0.0) | 0 (0.0) | 0 (0.0) | 1 (1.6) | 0 (0.0) |
| Female n=109 | Laxatives | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) |
| | Vomiting | 6 (5.5) | 0 (0.0) | 0 (0.0) | 2 (1.8) | 1 (0.9) |

Note: Percentages may not add up to 100% due to rounding. Data are presented as n(%)

5 Discussion

5.1 The Low Energy Availability in Females Questionnaire (LEAF-Q)

This is the first and only study to report the estimated prevalence of recreational New Zealand athletes at risk of low energy availability (LEA). The main finding from this study was 33.5% (95% CI, 26.5, 41.2%, n=57) of participants were classified as at risk of LEA, including 44.9% (95% CI 35.4%, 55.3%. n=49) of females and 13.1% (95% CI, 5.8%, 24.2%, n=8) of males. There are a number of studies estimating the prevalence of LEA in various athletic populations, however, very few studies have investigated the prevalence of recreational athletes at risk of LEA. It is important to first identify athletes at risk as this will determine if there is a need to carry out more costly, time consuming prevalence studies that have a much higher participant burden. At risk individuals can be identified from a simple questionnaire however when measuring the true prevalence, a randomly selected group of participants would be required to provide biochemical samples, food and exercise diaries as well as have costly clinical body composition measures such as DXA scans. Therefore, the present study is useful in aiding health professionals and researchers to specifically target screening and treatment programs to high-risk populations, maximising prevention and early detection interventions.

The majority of previous research has focused on an athlete's risk of eating disorders and disordered eating, poor nutrition, and weight loss, and did not include questions on menstrual status and bone health (Johnson et al., 1999; Sundgot-Borgen & Larsen., 1993). It is now commonly accepted that LEA can occur with or without an associated eating disorder or disordered eating behaviours, thus these studies are likely to have underestimated the prevalence of athletes at risk. Indeed we found that only 22.8% (95% CI, 12.7%, 35.8%, n=13) of those at risk of LEA were also at risk of an eating disorder (scored in the medium or high range on the EDIRC).

Currently the only published study using the full LEAF-Q was conducted by Melin et al., (2014a) in order to validate it as a screening tool. To ensure they were validating the screening tool on a likely high risk population, only female athletes competing in sports that emphasised leanness were included. They found that 62% of athletes were at risk of LEA, which is higher than reported in our study. However to make a more direct comparison, the estimated prevalence of those at risk of LEA among female athletes in

the current study that competed in sports that emphasise leanness was 69.2% which is comparable to the findings of Melin et al (2014a).

We identified only one previous study that used menstrual dysfunction and bone health to identify athletes at risk of LEA (Torstveit & Sundgot-Borgen, 2005). In this study, the risk of LEA in elite Norwegian athletes competing in a range of sports was compared to recreational athlete controls. The investigators reported 69.2% of recreational female athletes were at risk of the FAT, which is higher than our finding where 44.9% (95% CI 35.4%-55.3%. n=49) of recreational female athletes were at risk of LEA.

There is a lack of evidence investigating the risk for LEA with increasing level of competition. Although Torstveit et al., reported a lower prevalence (60.4%) of elite female athletes at risk of LEA when compared to recreational athletes in general, research has shown risk factors for LEA are more commonly seen in athletes as the level of competition increases. Starting sport-specific training at a young age, dieting, a sudden increase in training load, and sports-related injury are risk factors for LEA more commonly seen in higher-level athletes (Sundgot-Borgen, 1994). This variation in the estimation of athletes at risk of LEA may be explained by the fact that elite athletes may have higher pressures from coaches, and themselves to maintain or lose weight than recreational athletes', in saying this, it would be surprising that an athlete could compete at an elite level in such an energy deficient state.

This is an area that would benefit from further research as the risk factors for an elite athlete may be different from those for a recreational athlete especially those not participating in a formal sport. Pressure to win from the athlete themselves, coaches, team mates etc may be increased and may place athletes engaging in higher levels of competition at increased risk of behaviours leading to LEA. Another likely explanation is the difference in screening tool used to define those "at risk". Whilst we used the validated LEAF-Q screening tool, Torstviet et al, used a non-validated screening tool the researchers developed themselves. This lack of a consistent screening tool is a frequent issue that arises when comparing any prevalence research on LEA (FAT or RED-s) which makes it very difficult to confidently and accurately compare studies.

Hoch et al., carried out more comparable research to the present study. They estimated the prevalence of LEA amongst university representative athletes and found the percentage of female athletes with LEA (≤ 45 kcal.kgLBM⁻¹.day⁻¹) was 36% (Hoch et

al., 2009). As they were measuring biochemical markers of LEA, a more specific and objective measure of LEA than self-reported clinical signs, it is not surprising they found slightly less females met the criteria than we did in our study.

Unfortunately no studies have reported the prevalence of male athletes at risk of LEA, thus we unable to make any comparisons with our results. The existence of LEA in male athletes has been alluded to since the Female Athlete Triad was first termed in 1993 (Yeager et al., 1993). However over two decades later this gap in the literature remains.

Several studies have documented the existence of risk factors for LEA in male athletes. Vogt et al. (2005), found that during a pre-season training camp, elite male cyclists were in negative energy balance as low as -7.9 ± 1.1 MJ on training days. Also Rector et al. (2008), & Smathers et al. (2009), found a high prevalence of osteopenia amongst male cyclists (63% and 25% respectively) and Thiel et al. (1993), found 11% of male wrestlers and rowers were at risk of an eating disorder.

The lack of prevalence studies investigating male athletes is concerning especially in light of the results of the present study. We found 13.1% (95% CI, 5.8%, 24.2%, n=8) of males were classified as at risk of LEA using the female validated LEAF-Q scoring system, and 32.8% when the scoring system was adjusted to exclude the female specific questions, indicating a large proportion of male athletes are potentially at risk of LEA.

However, it is important to note there are a number of practical difficulties associated with assessing the risk of LEA in males, mainly due to the difficulty in detecting reproductive effects in men compared to women. Further the exact clinical implications in males are yet to be determined and it is unknown what level of energy availability these clinical implications start to occur. It is also possible there is a degree of ignorance on the part of coaches, managers and team mates with regard to the existence of LEA in males and the implication it may have on their health and performance.

Therefore further research investigating LEA in male athletes is needed to improve understanding of the prevalence. Of utmost importance is development of a validated screening tool for male athletes that will aid in the early identification of male athletes at risk of LEA.

5.2 The Eating Disorder Inventory-3 (EDI-3)

An Eating Disorder Inventory Risk Composite (EDIRC) score in the low risk (low clinical) range (1st-75th percentile) is common among non-clinical samples and does not indicate risk of an eating disorder (Garner, 2004). In the present study the vast majority of participants (males 87.5%, 95% CI, 47.3%, 99.7%, n=7, females, 89.8%, 95% CI, 77.8%, 96.6%, n=44) who were classified as at risk of LEA were considered not at risk of an eating disorder. Thus, it appears many participants at risk of LEA do not have a formal eating disorder or disordered eating behaviour. This is an important finding in terms of developing screening tools and education programmes so researchers and health professionals can target their intervention largely to reach the general population of recreational athletes not solely those with eating disorders. Similarly Melin et al. (2014a), observed only 28% of participants with reduced energy availability or LEA also had disordered eating or an eating disorder. Our findings add merit to their statement that diagnosis of disordered eating or eating disorders is not sensitive enough to use as the sole clinical indicator of current LEA (Melin et al., 2014a).

An EDI-3 risk composite in the medium (typical clinical) range (from the 76th-94th percentile) is common for those diagnosed with eating disorders, however is relatively rare among non-clinical samples and they are considered at risk of an eating disorder (Garner, 2004).

With 7.6% (95% CI, 4.1%, 12.7%, n=13) of participants (3.3%, 95% CI, 0.4%, 11.3% of males, n=2 and 10.1%, 95% CI, 5.1%, 17.3%, females, n=11) at risk of an eating disorder (meeting the criteria for medium or high risk of an eating disorder on the EDIRC) our rates are lower than previous research where the prevalence has been found to be between 12.5% among university and school representative athletes in the USA and 89.5% among elite Malaysian athletes (Barrack et al., 2014; Hoch et al., 2011; Melin et al., 2014b; Thein-Nissenbaum et al., 2011). Again this could be explained by the use of different screening tools, different athletic populations in terms of the type of sports played, level of competition as well as age groups studied.

In our study, one male and two females who were classified as at risk of LEA scored in the high risk range for the EDRC. In studies of non-clinical samples it is rare for an individual to score within this range and they are considered very high risk of an eating disorder (Garner, 2004). The three participants who scored within the high risk range

were advised to see their General Practitioner to get a referral to get evaluated by a specialist health professional familiar with eating disorders (Appendix E).

When validating the EDIRC Garner found it to be positively correlated with body weight, meaning those who score within the high range could be expected to have a high body mass (Garner, 2004). Whilst one of the females in the high risk range did have a BMI in the “overweight” category (29.05 kg.m²) the other had a body mass of 64 kg (BMI 20.2 kg.m²) and did not play a sport. The male that fell within this range had a body mass of 63.8 kg (BMI 21.1 kg.m²) and is a regional representative runner. Scores in this range may be even more concerning for these individuals in the healthy body mass index category. For example a representative runner is likely to have the aim of becoming a national representative however being at high risk of an eating disorder and being at risk of LEA may compromise the likelihood of him achieving this.

Although the EDI is a frequently used tool among athletic populations, researchers do not use standardised cut off scores for classifying athletes as at risk of LEA (Beals & Manore, 2002; Melin et al., 2014b; Quah et al., 2009). Definitions vary from scoring at least 12.1 on the Drive For Thinness scale or at least 8.5 on the Bulimia scale or ≥ 6.8 on the Perfectionism scale (Quah et al., 2009) to scoring greater than 12 on the Body Dissatisfaction scale (Beals & Manore, 2002). Therefore it is very difficult to compare the prevalence of athletes with eating disorders or disordered eating behaviour, even between studies that have used the same screening tool.

If researchers are going to continue to use the EDI, it is important future research validates its use in an athletic population and defines a scoring system for classifying athletes at risk of an eating disorder. Any future eating disorder screening tools should also be validated in an athletic population.

5.3 Risk factors and Predictors of Low Energy Availability (LEA)

5.3.1 The Association Between Low Energy Availability (LEA) and The Menstrual Cycle

In the present study two of the three amenorrhoeic and four of the six oligomenorrhoeic athletes were classified as at risk of LEA. Thus, the LEAF-Q did not classify all athletes with menstrual disorders to be at risk of LEA. This is because they did not report the presence of enough other physiological symptoms of LEA to meet the criteria for being at risk of LEA.

In contrast to our study, Torstveit et al., had a criteria where every athlete with menstrual dysfunction ranging from a short luteal phase right through to amenorrhoea (irrespective of its origin), were considered at risk of LEA (the FAT) (Torstveit & Sundgot-Borgen, 2005). It is important when screening for athletes at risk of LEA, that those with functional hypothalamic menstrual dysfunction, a known clinical identifier of LEA, are not overlooked as this could engender these athletes with a false sense of health and exclude them from possible treatment plans. However it is also important these females are differentiated from females with menstrual dysfunction not related to energy deficiency such as poly-cystic ovary syndrome (PCOS), thyroid disease, polycystic ovarian syndrome, Kallmann syndrome, Turner syndrome, hyperprolactinemia and pituitary tumors, among others.

When validating the LEAF-Q, Melin et al. (2014a) found four of the five participants that had been diagnosed with PCOS or menstrual disorders other than FHA/oligomenorrhoea had 'lower current' energy availability and three also had low bone mineral density. Whilst Melin et al. (2014a) reiterated the importance of differential diagnosis of the origin of menstrual dysfunction they also identified the importance of assessing the coexistence of LEA conditions despite the type of menstrual dysfunction. We did not include an exclusion criteria for females with menstrual disorders other than FHA, such as polycystic ovaries, as these females may also be at risk of LEA. This may explain why all of the participants reporting menstrual dysfunction were not classified as at risk of LEA.

Five of the 20 eumenorrhoeic females in our study were classified as at risk of LEA. This indicates some of these athletes were showing other signs of LEA such as gastrointestinal or injury issues. These individuals may have subclinical menstrual dysfunction. Thus further validation studies in this potential sub clinical population are warranted where

biochemical testing could be used to confirm a short luteal phase or anovulation in these participants.

In summary not all female athletes with menstrual disturbances are necessarily in a state of LEA, further not all female athletes at risk of LEA will show clinical signs of menstrual dysfunction. Thus it is important when investigating the true prevalence of LEA that future research employs biochemical testing to firstly determine if the athletes are showing signs of sub-clinical menstrual dysfunction as well as determine the origin of menstrual dysfunction in female athletes to ensure appropriate treatment is pursued.

5.3.2 The Association Between Low Energy Availability (LEA) and Participation in Various Sports Categories

The results from our study suggest participants taking part in team sports were at less risk of LEA (27%, 95% CI 16.2%, 37.3%) than participants competing in individual sports (45.2%, 95% CI 29.5%, 60.9%) ($p=0.045$). Previous research has also reported a lower prevalence of predictors of LEA amongst athletes participating in team sports compared to other sports (Beals, 2004; Thein-Nissenbaum et al., 2011).

Torstviet and Sundgot Borgen (2005), found a higher percentage of athletes competing in sports that emphasise leanness (70.1%) were considered at risk of LEA when compared to athletes that did not compete in sports that emphasise leanness (55.3%). Results of the present study were not consistent with Torstviet and Sundgot-Borgen's findings. Participants taking part in sports which emphasise leanness were no more at risk of LEA when compared to participants who take part in sports which did not emphasise leanness. ($p=0.290$). It is possible our sample of participants who reported their primary sport to be a sport that emphasises leanness, was too small to have the power to detect a significant difference between the two groups.

5.3.3 The Association Between Low Energy Availability (LEA) and Body Mass Index (BMI).

In the present study we found a statistically significant negative association between BMI and risk of LEA. For every one unit decrease in BMI participants were 11% more likely to be at risk of LEA (OR 0.89, 95% CI 0.80, 1.00, $p=0.045$). Melin et al. (2014a) also reported a trend towards participants classified as at risk of LEA having a lower BMI compared to participants classified as low risk of LEA.

We identified 4 participants with a BMI in the underweight category (<18.5). All 4 participants took part in a sport that emphasised leanness, including running, gymnastics, and road cycling. Whilst 2 (out of 4) of these athletes were at risk of LEA one of which had oligomenorrhoea, none were at risk of an eating disorder. In comparison, Torstveit et al., used a broader definition of “at risk” where any athlete with a BMI of <18.5 kg.m² was considered at risk for LEA (Torstveit & Sundgot-Borgen, 2005). This resulted in a higher prevalence of females being classified as at risk of LEA (64%) in comparison to our study (45%, 95% CI 35.4%-54.8%. n=49) and they did not report the BMI of participants at risk of an eating disorder.

Further, using the LEAF-Q, Melin et al. (2014a), found that of the 6 participants with a BMI in the underweight (BMI <18.5) category, 5 were at risk of LEA (Melin et al., 2014). Due to the small numbers of participants classified as underweight in the present study and the study by Melin et al. (2014a) the impact on prevalence is unlikely to be statistically significant.

The finding that no participants with a BMI in the underweight category were considered at risk of an eating disorder and only half were at risk of LEA, may be explained by Western societies perception of a low body mass being ‘attractive’. Therefore people in the underweight BMI category may feel less pressure to reduce weight further. Further, athletes with eating disorders have been found to be not only underweight, but of healthy weight or even overweight thus indicating underweight athletes are at no higher risk than athletes who fall within the other BMI categories (Klungland & Sundgot-Borgen, 2012). It is worth noting that the two participants who were classified as underweight and were at risk of LEA reported competing in sport at a “regional representative” level whereas the two who were not classified as at risk of LEA only reported participating in sport at a lower “competitive” level. The external pressure, from coaches, parents and peers, and internal pressure, from the athlete themselves, to maintain this low body weight may be greater as the competition level increases. This was not measured in the present study but future work should investigate the effect of competition level and external influences on LEA.

5.3.4 The Association Between Low Energy Availability (LEA) and Exercise-Related Injuries

Another possible predictor of LEA that warrants further investigation is the number of exercise-related injuries and days of missed training due to sporting injury. In the present study 58.7% of females and 73.8% of males reported having an injury in the past year. Further there was a significant positive association between the total number of exercise-related injuries both male and female participants had suffered in the past year and risk of LEA (males OR, 4.10, 95% CI, 1.31, 12.85, $p=0.015$, females OR 2.04, 95% CI 1.22, 4.75, $p=0.011$). The finding that every male who was classified as at risk of LEA reported at least one exercise-related injury in the past year warrants further investigation. This is an association future research needs to investigate. If screening tools can be validated in male athletes to detect those at risk of LEA this may assist in reducing sporting injuries that can have a serious impact on an athletes health and sporting performance.

When validating the LEAF-Q, Melin et al. (2014a) found that a high LEAF-Q injury score was associated with impaired bone health in female athletes, thus the significant associations between number of sporting injuries, days of missed training due to injury and risk of LEA in females in the present study are not surprising. It is known stress fractures and other musculoskeletal injuries are a sign of LEA (Burrows et al., 2003). However the finding of a possible association between days off training due to exercise-related injuries and risk of LEA suggests future research should investigate the aetiology behind all exercise-related injuries and risk of LEA.

The association between LEA and injury risk is highlighted by the finding that three of the four participants who had experienced a stress fracture in the past year were also at risk of LEA. This finding is in agreement with Barrack et al., who reported a significant relationship between female athletes meeting the criteria for increasing number of female athlete triad risk factors and the development of bone stress injuries (Barrack et al., 2014). No male participants reported the occurrence of a stress fracture in the past year thus we cannot comment on male athletes with stress fractures, but, it appears New Zealand female recreational athletes incurring stress fractures, are a high risk population for LEA. Further, previous research has reported the most common areas of stress fractures to be in the tibia and metatarsal (Bennell et al., 1996; Johnson et al., 1994; Matheson et al., 1987; Barrack et al., 2014). Consistent with these findings is that in the present study, the most commonly reported (3 out of 4) stress fractures occurred in the foot.

In summary the possible association of LEA and exercise related injuries is an important area for future research to investigate in both male and female athletes to allow for possible improvements in health, performance and duration of sporting career due to less training days lost due to injury.

5.3.5 The Association Between Low Energy Availability (LEA) and Training Volume

The dose response relationship between training volume (hours per week) and risk of LEA was found to be non-significant among males ($p=0.893$) but significant among female athletes ($p=0.016$). Our results suggest for every extra hour of exercise female participants undertook per week, the odds of being at risk of LEA were 1.13 times greater (95% CI 1.02, 1.25, $p=0.016$).

The fact that there was no significant relationship between training volume and risk of an eating disorder, nor was there a significant relationship between risk of LEA and frequency of exercising to lose weight ($p=0.200$), complements our finding that the majority of participants at risk of LEA were not at risk of an eating disorder. Individuals may be more likely to exercise to lose weight if they had an eating disorder or disordered eating behaviours. These findings indicate increasing training volume may be a risk factor for developing unintentional LEA, possibly as a result of lack of knowledge of specific increased energy needs. Further a commonly proposed treatment option for LEA is to reduce exercise load, therefore this finding that increased hours of exercise per week can lead to increased risk of LEA is of importance in recognising this is an appropriate treatment option.

The lack of relationship between LEA and training volume in males may be due to the questionnaire used to determine risk of LEA (The LEAF-Q) not being validated in males. Further the sample size of males in the current study ($n=61$) may have been too small to detect any significant difference.

5.3.6 The Association Between Low Energy Availability (LEA) and Eating and Weight Loss Habits

Although a high proportion (37.1%) of participants had been told that if they were to decrease their weight, their athletic performance would improve, it was interesting to find that these participants (both male and female) were no more likely to be at risk of LEA or an eating disorder when compared to those who had not been. These results

were contrary to Muscat et al., who found that athletes and sport participants who recalled critical comments regarding their weight or body size, reported greater disordered eating behaviours (Muscat & Long, 2008). Again this difference may be explained by the difference in wording of the questions, being advised to lose weight and receiving critical comments regarding weight or body size may be interpreted and/or acted upon differently. However it is also possible the timing of when athletes are criticised about their weight may have an influence on their risk of LEA. For example athletes may reduce their dietary intake or increase exercise energy expenditure around the time they were criticised about their weight however it may not have a long term effect in most people. Further the present studies population is recreational athletes, therefore sporting “performance” may not be as important to them as elite athletes.

It was concerning to note 5.9% (n=10) of participants (n=1, 1.6% males and n=9, 8.3% females) in the present study had used at least one pathogenic weight loss method in the past three months and that 70% of these participants were currently at risk of LEA.

This was a lower prevalence than a Norwegian cross sectional study where 36.7% of recreational female athletes reported using pathogenic weight loss methods (Torstveit, & Sundgot-Borgen 2005). Torstveit, & Sundgot-Borgen (2005) did not have a specific time frame in which the use of pathogenic weight loss methods were reported, whereas the current study specifically asked participants to report their use in the past 3 months. This could be an explanation for the difference in prevalence between the two studies. Another possible explanation, is that Torstveit & Sundgot-Borgen (2005), undertook their study a decade ago, therefore it is possible the prevalence of athletes using pathogenic weight loss methods has changed in this time. They also included the use of diet pills and diuretics as pathogenic weight loss methods as well as the methods the current study included (use of laxatives and vomiting). It is possible including these additional pathogenic weight loss methods may explain the higher prevalence in Torstveit, & Sundgot-Borgen’s (2005) study. Therefore these are two other pathogenic weight loss methods to be considered when undertaking further research.

Extreme exercise has been described as a precipitant for the development and maintenance of anorexia nervosa (Epling et al, 1983; Holtkamp et al, 2004). Petrie and Stoeber (1993), found 57.3% of gymnasts competing at a university representative level

were exercising at least two hours a day to lose weight whereas we found 6% of females and 7% of males reported exercising at least once a day to lose weight. However gymnastics is a sport where athletes are known to have a high drive for thinness (Warren et al., 1990). Further the study by Petrie and Stoever (1993), was carried out in the USA in the early 1990's when there were limited regulations on weight control methods for athletes. Considering participants in the current study were recreational athletes and 28.3% (n=48) were classified in the "overweight" or "obese" BMI category they may have interpreted exercising to "lose weight" differently to Petrie and Stoever's (1993) population of gymnasts, and may have been trying to lose weight for health reasons. Our finding that there was no significant association between exercising to lose weight and risk of LEA ($p=0.081$) indicates this is possibly the situation. Thus it is difficult to compare our results to Petrie and Stoever's (1993) findings from such an extremely high risk population, who are more likely to be exercising excessively due to disordered eating behaviours or an exercise addiction (Petrie & Stoever, 1993).

However Petrie & Stoever (1993), found only 6% of participants had vomited at least twice in the past month which is consistent with our study where 5.9% participants had vomited at least once in the past 3 months to lose weight (Petrie & Stoever, 1993). Considering gymnasts are a high risk population for disordered eating and eating disorders it is of concern that our study in New Zealand recreational athletes showed a similar prevalence of participants regularly vomiting to control their weight (Petrie & Stoever, 1993). This pathogenic weight control technique is unhealthy and can lead to clinical eating disorders. Future research could investigate why these athletes choose vomiting as their weight loss method to assist in the development of education programmes as well as develop tools and techniques to reduce this prevalence.

Overall it appears there is a need for longitudinal data to be collected to investigate the risk of LEA in athletes with regard to the timing of receiving criticism about their weight and sporting performance. It would be valuable to determine if the athlete's risk of LEA changes before, directly after, and months to years after receiving negative feedback about their weight. This yet again highlights the need for standardised screening tools to be developed so consistent, comparable data can be collected.

Further with such a high percentage of participants using pathogenic weight loss methods this is an important area to focus on when developing prevention and education interventions.

6 Strengths and Limitations

The strengths of this study include the use of validated screening tools for estimating the risk of LEA and eating disorders. The study questionnaire was also designed to have a low respondent burden. It only took a short time to complete, participants could access and complete the survey from any internet capable device and it was relatively inexpensive to administer, this allowed a large sample size to be collected in a short time frame. Also the LEANZ questionnaire included questions of all three aspects of the Female Athlete Triad.

Another strength to the current study was the method of data handling. Participants responses to each question on the LEANZ questionnaire were electronically downloaded to an excel spreadsheet. This eliminated the human error that can occur when manually inputting data onto a spreadsheet.

This study must be interpreted with certain limitations in mind. Our participants were not recruited from a random sample so we cannot be sure they are representative of our target population, therefore we have reported the estimated prevalence of New Zealand recreational athletes at risk of LEA.

The cross sectional design of this study has limitations in itself. In this study prevalence was estimated at one point in time, therefore trends over time cannot be reported. This is important because pressure on an athlete to lose weight may be acute and have an impact on health at the time but may not have been captured in the current questionnaire.

Further we investigated the associations between possible risk factors for LEA and prevalence of athletes at risk of LEA, however due to the cross sectional design we have no way of determining the sequence of events that lead to our finding. Therefore we cannot imply causality and rely on previous research to draw these conclusions.

However, the current cross sectional study has indicated a number of associations that may exist. Therefore combined with findings from previous research the results of this study are useful in generating hypotheses and topics to investigate in future research.

Further the current study could be used as the first of a series of repeated cross sectional studies to gather data on any changes in prevalence of LEA that may occur in this population over time.

Although we used two previously validated questionnaires, these questionnaires had been validated in USA and European populations. Although effort was made to ensure the wording was appropriate for New Zealanders, interpretation may have been different. Ideally, questionnaires used for this type of study would have been validated for use in NZ.

Further the LEAF-Q has only been validated in female endurance athletes. Our study included males as well as athletes from a much wider range of sports including team sports, aesthetic sports and power sports. The LEAF-Q needs to be tested in these other populations to ensure its validity. The scoring system for males risk of LEA using the LEAF-Q proposed in the results section also needs validation as this could be a useful tool if deemed specific enough.

Using a questionnaire as our method of data collection may also result in bias. Firstly those who participate in surveys may be more likely to be thinking about the topic, therefore, bias the sample towards those with LEA. Conversely, previous literature shows athletes with eating disorders may not respond truthfully to questionnaires due to the fear that coaches, peers and parents may react negatively (Beals, 2004; Wilmore et al., 1992). Therefore our results may be underestimating the prevalence of disordered eating. However in the current study we did take steps to encourage participants to respond truthfully. This included making participation in our study anonymous to guarantee confidentiality as well as allowing athletes to carry out the study in their own time where coaches etc. were not present. Further, by having our study completed online participants submitted their answers electronically, thus participants did not have to worry that researchers could identify their answers.

We recognise the limitations of self-reporting and the ability of adolescents and adults to accurately recall past events, however due to funding and time constraints it was not possible to have experts individually interview participants. Again validated questionnaires were used to ensure questions were worded in the best way possible. For example both the LEAF-Q and EDI-3 rarely offer just a 'yes' or 'no' answer option thus reducing the likelihood participants answer "no" if the option "sometimes" is not available or "yes" if the answer "often" is not available. To reduce data errors due to non-response questions we created a forced answer questionnaire where participants had to answer the question before moving onto the next question, this was a strength of this study. However there may have been some circumstances where participants did

not feel any of the offered responses were accurate, and this may have caused participants to select a random answer to move them onto the next question which may have caused data errors. Further a lot of answer options in the LEANZ questionnaire were close-ended which may have again caused data error for example one participants interpretation of “usually” could be very different to another participant.

With self-report surveys there is the chance of reporter bias. In this study athletes may have found some questions sensitive topics, in particular the eating disorder questions, and may have feared their answers could be tracked to back to them. This could have lead participants to modify their response.

As our data collection was an online survey, it was limited to those who have access to a computer and the internet. Additionally we predominantly advertised in gyms, where people could afford a gym membership, so our participants were not likely to represent those with a low socioeconomic status, therefore this population of New Zealanders may have been missed from our study.

Although including all sports in our study means results are generalisable to a wide population it did mean that the sample size for each sport was limited, this meant we could not compare each sports risk of LEA but had to subjectively divide the sports into categories. Previous literature has shown subjective grouping of sports may not be reliable (Sundgot-Borgen, 1993). Sundgot-Borgen (1993), estimated the prevalence of pathogenic weight control methods and self-reported eating disorders among elite female athletes and non-athlete controls. Athlete participants were categorised into six groups based on the type of sport they competed in these included; technical sports, endurance sports, aesthetic sports, weight-dependent sports, ballgames and power sports. Results found a significant difference in prevalence of eating disorders between the sports categorised in the endurance group, indicating a variation of risk of an eating disorder within the endurance group.

Similarly the low number of male and female participants assigned to each sporting category, especially classified as at risk of LEA means that the study may not have enough power to detect some significant differences. To overcome this we would have had to increase the sample size, and target specific sports to allow for comparison across all sports. Unfortunately this was not possible due to the time constraints of data

collection. Additionally ethnicity data was not collected in the present study so we do not know how risk of LEA may differ based on ethnicity.

In the current study a recreational athlete was described as participating in 2.5 -5 hours of moderate intensity exercise or the equivalent in vigorous intensity physical activity (1 ¼ to 2 ½ hours) exercise per week. The results however, did indicate some participants reported higher or lower levels of exercise. Therefore it is possible some participants were misclassified as recreational athletes which may have affected the interpretation of the results.

A limitation of this study was that self-reported height and weight was used to calculate BMI. A recent study investigating the accuracy of web-based self-reported height, weight, and body mass index found there was moderate to high agreement between self-reported and measured anthropometric data, indicating online self-reported height and weight can be a valid method of collecting anthropometric data (Pursey et al, 2014).

Finally the use of BMI for classifying overweight in athletes has its limitations, since the influence of large muscle mass on the BMI in athletes may misclassify individuals as overweight or obese (Nevill et al, 2006). Thus this data must be interpreted with caution.

Due to the aforementioned limitations and the sensitivity and specificity of the LEAF-Q being 78% and 90% respectively, there is potential for misclassification bias, where some athletes at risk of LEA and some at no risk of LEA may have been misclassified (Melin et al., 2014a). Further random errors occurring in the data may also have caused this to occur, for example if a participant misread a question and provided an inaccurate answer.

7 Conclusion and Future Research

A substantial percentage of New Zealand recreational athletes *may* currently be in a state of LEA or at *increased risk* of becoming so over time. This puts these athletes at risk of long term health and performance consequences associated with LEA. It is clear that participant's risk of LEA is not necessarily due to the presence of an eating disorder. This confirms the importance of ensuring screening tools do not include disordered eating as essential criteria for classification of at risk of LEA, rather as a risk factor and separate measure.

Based on the results of this investigation, several areas of future research are warranted. The purpose of this study was to investigate the prevalence of New Zealand recreational athletes *at risk* of LEA not the prevalence of LEA *per se*. Thus, no clinical interviews, blood samples, or measures of bone mineral density were carried out. The next step is to determine the actual prevalence of LEA to ensure the use of the LEAF-Q in the New Zealand population is appropriate for use as a screening tool, so it can then be used to estimate risk in other New Zealand sporting populations such as elite athletes, and individual sports. Additionally having comparison groups of non-athletes would assist in determining if New Zealand athletes are at greater risk than the general population.

It is important we encourage New Zealand recreational athletes to continue taking part in physical activity as the health benefits are well established. However as a preventative approach to reducing the risk of LEA pre-participation screening to identify high risk athletes should take place. However as some recreational athletes do not participate in a formal sport, educational public health initiatives that reach all recreational athletes should be implemented in New Zealand. Ideally we would aim to carry out a multimedia campaign such as the 5+ a day and 30 minutes a day push play public health initiatives. Here we would inform the public on the possible health consequences of LEA whilst encouraging healthy participation in physical activity.

Additionally education in the formative schooling years could help to prevent the occurrence of LEA later in life and therefore avoiding long and short term health complications. Education initiatives could be carried out in sports clubs and gyms as well as schools where it could be integrated into the health and physical education curriculum.

This would help to create awareness and especially knowledge around LEA, its associated health and performance implications as well as high risk populations.

Further, although the EDI-3 Risk composite and the LEAF-Q may be useful as preliminary screening tools for identifying New Zealand athletes at risk of LEA, the sensitivity of the EDI-3 risk composite for athletes must be investigated as well as a screening tool to identify male athletes at risk of LEA. Furthermore the diagnosis of LEA and eating disorders must be confirmed by clinical examinations.

Future prospective longitudinal studies are needed to increase our understanding of risk factors of LEA, and develop appropriate prevention and treatment options to minimise the health and performance implications for our athletes. This would also allow us to compare risk of LEA without the possible memory bias of retrospective studies, and help identify temporal relationships. This study has provided valuable normative data that can act as a baseline, to measure the effectiveness of any education program and preventative interventions for LEA.

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9 Appendices

Appendix A

*** Required Information**

page 1

1. By completing this questionnaire you are providing informed consent to participate in this research. Please email leanz@otago.ac.nz if you would like to be sent the information sheet on this study. (Select one option)

Yes, I provide informed consent to participate in this research.

Go to Page No. 2

If Did Not Answer Then Stop, you have finished the survey

page 2

2. What is your email address? (providing your email is only for the purpose of sending you the nutrition fact sheet and entering you in the prize draw)

*** 3. What is your highest level of education? (Select one option)**

- Primary School Some Secondary School Completed Secondary School (7th form or NCEA Level 3) Technical /trade school Polytechnic
 University

4. What is your occupation? N.B. If you are a student please specify the course you are doing.

*** 5. What is your age (years)?**

*** 6. What is your height (cm)?**

*** 7. What is your current weight (kg)?**

*** 8. What is your highest weight with your present height: (kg) (excluding pregnancy)?**

*** 9. What is your lowest weight with your present height: (kg)?**

*** 10. What is your desired weight (kg)?**

*** 11. Do you smoke?** (Select one option)

- Yes
 No

*** 12. Do you use any medication (excluding oral contraceptives)?** (Select one option)

- Yes
 No

Go to Page No. 3

Go to Page No. 4

If Did Not Answer Then Go to Page No. 3

page 3

*** 13. What kind of medication?**

page 4

14. Do you currently play any sports? (Select one option)

- Yes No

Yes:Go to Page No. 5

No:Go to Page No. 6

If Did Not Answer Then Go to Page No. 5

page 5

Please fill in the following table regarding the sports you play.

15. Please list all the sports you currently play.

* (a) 1

(b) 2

(c) 3

(d) 4

(e) 5

16. What level do you compete in this sport at?

| | Social | Competitive | Regional representative |
|----------------------------|-----------------------|-----------------------|-------------------------|
| *(a) 1 (Select one option) | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| (b) 2 (Select one option) | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| (c) 3 (Select one option) | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| (d) 4 (Select one option) | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| (e) 5 (Select one option) | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |

Please fill in the following table regarding your average week of exercise.

17. Number of exercise sessions.

| | 0 | 1 | 2 | 3+ |
|-------------------------------------|-----------------------|-----------------------|-----------------------|-----------------------|
| * (a) Monday (Select one option) | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| * (b) Tuesday (Select one option) | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| * (c) Wednesday (Select one option) | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| * (d) Thursday (Select one option) | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| * (e) Friday (Select one option) | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| * (f) Saturday (Select one option) | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| * (g) Sunday (Select one option) | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |

18. Total hours of exercise e.g. 6.5 hours.

(a) Monday

(b) Tuesday

(c) Wednesday

(d) Thursday

(e) Friday

(f) Saturday

(g) Sunday

19. Type of exercise e.g. weights at gym, light jog, Cross Fit session, bike 5km to work, rugby training.

(a) Monday

(b) Tuesday

(c) Wednesday

(d) Thursday

(e) Friday

(f) Saturday

(g) Sunday

page 7

*** 20. What sex are you?** (Select one option)

Male

Go to Page No. 22

Female

Go to Page No. 8

page 8

*** 21. How old were you when you had your first period?** (Select one option)

11 years or younger

Go to Page No. 9

12-14 years

Go to Page No. 9

15 years or older

Go to Page No. 9

I don't remember

Go to Page No. 9

I have never menstruated

Go to Page No. 16

page 9

*** 22. Did your first menstruation come naturally (by itself)?** (Select one option)

Yes

Go to Page No. 11

No

Go to Page No. 10

I don't remember

Go to Page No. 11

page 10

*** 23. What kind of treatment was used to start your menstrual cycle?** (Select one option)

Hormonal treatment

Weight gain

Reduced amount of exercise

Other

page 11

*** 24. At present do you have normal menstruation - (regular monthly period)?** (Select one option)

Yes

Go to Page No. 12

No I'm pregnant

Go to Page No. 12

No

Go to Page No. 13

I don't know

Go to Page No. 12

page 12

*** 25. When was your last period?** (Select one option)

0-4 weeks ago

1-2 months ago

3-4 months ago

5 months ago or more

*** 26. Are your periods regular? (A 28 to 34 day cycle) (Select one option)**

- Yes, most of the time
- No, mostly not

*** 27. For how many days do you normally bleed? (Select one option)**

- 1-2
- 3-4
- 5-6
- 7-8
- 9 days or more

*** 28. Have you ever had problems with heavy menstrual bleeding? (Select one option)**

- Yes
- No

*** 29. How many periods have you had during the last year? (Select one option)**

- >11 Go to Page No. 14
- 9-11 Go to Page No. 14
- 6-8 Go to Page No. 14
- 3-5 Go to Page No. 14
- 0-2 Go to Page No. 13

page 13

*** 30. When did you have your last period? (Select one option)**

- 2-3 months ago Go to Page No. 14
- 4-5 months ago Go to Page No. 14
- 6 months ago or more Go to Page No. 14

page 14

*** 31. Have your periods ever stopped for 3 consecutive months or longer (besides pregnancy)? (Select one option)**

- No, never
- Yes, it has happened before
- Yes, that's the situation now

*** 32. Do you experience changes in menstruation when you increase your exercise intensity, frequency or duration? (Select one option)**

- Yes Go to Page No. 15
- No Go to Page No. 16

page 15

*** 33. How does your menstruation change? (Check one or more options)**

- I bleed less
- I bleed fewer days
- My menstruations stops
- I bleed more
- I bleed more days

page 16

*** 34. Do you use oral contraceptives? (Select one option)**

- Yes
- No

Go to Page No. 19

Go to Page No. 17

page 17

*** 35. Have you used oral contraceptives in the past? (Select one option)**

- Yes
- No

Go to Page No. 18

Go to Page No. 20

page 18

*** 36. When did you use oral contraceptives?**

*** 37. How long did you use oral contraceptives for?**

page 19

*** 38. Why do/did you use oral contraceptives? (Select one option)**

- Contraception
- Reduction of menstruation pains
- Reduction of bleeding
- To regulate the menstrual cycle in relation to performances etc..
- Otherwise menstruation stops
- Other

page 20

*** 39. Do you use any other kind of hormonal contraceptives? (e.g. hormonal implant or coil) (Select one option)**

- Yes No

Yes:Go to Page No. 21

No:Go to Page No. 22

page 21

*** 40. What kind of hormonal contraceptives do you use? (Select one option)**

- Hormonal patches - e.g. Ortho Evra
 Hormonal ring - e.g. Nuva Ring
 Hormonal coil - e.g. Mirena, copper IUD
 Hormonal implant - e.g. Jadelle, Implanon
 Hormonal injection -e.g. depot medroxyprogesterone acetate (DMPA)

page 22

*** 41. Have you gained any weight in the past 3 months? (Select one option)**

- Yes Go to Page No. 23
 No Go to Page No. 25

page 23

*** 42. Was this weight gain intentional? (Select one option)**

- Yes Go to Page No. 24
 No Go to Page No. 25

page 24

*** 43. How did you achieve this weight gain?**

page 25

*** 44. I think about dieting? (Select one option)**

- Always Usually Often Sometimes Rarely
 Never

*** 45. Other people think that I am too thin? (Select one option)**

- Yes
 No

*** 46. I think that my thighs are too large? (Select one option)**

- Always Usually Often Sometimes Rarely
 Never

*** 47. In the past 6 months, have you lost 9kg or more? (Select one option)**

- Yes No

*** 48. I am terrified of gaining weight. (Select one option)**

- Always Usually Often Sometimes Rarely
 Never

*** 49. I think my hips are too big. (Select one option)**

- Always Usually Often Sometimes Rarely
 Never

*** 50. Are you on any special diet? (Select one option)**

- Yes
 No

Go to Page No. 26

Go to Page No. 27

page 26

*** 51. What special diet are you on?**

52. Why are you on this diet?

page 27

*** 53. Do you feel gaseous or bloated? (If you are a female please only answer for times when you do not have your period) (Select one option)**

- Yes, several times a day Yes, several times a week Yes, once or twice a week or more seldom Rarely or never

*** 54. Do you get stomach aches? (If you are a female please only answer for times when you do not have your period) (Select one option)**

- Yes, several times a day Yes, several times a week Yes, once or twice a week or more seldom Rarely or never

*** 55. On average how often do you have bowel movements? (Select one option)**

- Several times a day Once a day Every second day Twice a week Once a week or more rarely

*** 56. How would you describe your normal stool? (Select one option)**

- Normal (soft) Diarrhoea-like (watery) Hard and dry

57. Comments regarding gastrointestinal function.

page 28

*** 58. If I gain a kilogram, I worry that I will keep gaining weight. (Select one option)**

- Always Usually Often Sometimes Rarely
 Never

*** 59. I feel extremely guilty after overeating. (Select one option)**

- Always Usually Often Sometimes Rarely
 Never

*** 60. I eat moderately in front of others and stuff myself when they are gone. (Select one option)**

- Always Usually Often Sometimes Rarely
 Never

*** 61. I think my buttocks are too large.** (Select one option)

- Always Usually Often Sometimes Rarely
 Never

*** 62. I think about trying to vomit in order to lose weight.** (Select one option)

- Always Usually Often Sometimes Rarely
 Never

*** 63. I think that my hips are just the right size.** (Select one option)

- Always Usually Often Sometimes Rarely
 Never

*** 64. I am preoccupied with the desire to be thinner.** (Select one option)

- Always Usually Often Sometimes Rarely
 Never

*** 65. I feel satisfied with the shape of my body.** (Select one option)

- Always Usually Often Sometimes Rarely
 Never

*** 66. In the past 3 months, how often have you gone on eating binges (eating a large amount of food while feeling out of control)?** (Select one option)

- Never Once a month or less 2-3 times per month Once a week 2-6 times per week
 Once a day or more

*** 67. Do you avoid certain types of foods?** (Select one option)

- Yes Go to Page No. 29
 No Go to Page No. 30

page 29

*** 68. What foods do you avoid?**

69. Why do you avoid these foods?

page 30

*** 70. I have gone on eating binges where I felt that I could not stop.** (Select one option)

- Always Usually Often Sometimes Rarely
 Never

*** 71. I eat or drink in secrecy.** (Select one option)

- Always Usually Often Sometimes Rarely
 Never

*** 72. I think that my stomach is too big.** (Select one option)

- Always Usually Often Sometimes Rarely
 Never

*** 73. I eat when I'm upset.** (Select one option)

- Always Usually Often Sometimes Rarely
 Never

*** 74. I think my torso is too small.** (Select one option)

- Always Usually Often Sometimes Rarely
 Never

*** 75. I think about bingeing (overeating).** (Select one option)

- Always Usually Often Sometimes Rarely
 Never

*** 76. I like the shape of my buttocks.** (Select one option)

- Always Usually Often Sometimes Rarely
 Never

*** 77. In the past 3 months, how often have you used laxatives to control your weight or shape?** (Select one option)

- Never Once a month or less 2-3 times per month Once a week 2-6 times per week
 Once a day or more

*** 78. I eat sweets and carbohydrates without feeling nervous.** (Select one option)

- Always Usually Often Sometimes Rarely
 Never

*** 79. I think that my stomach is just the right size.** (Select one option)

- Always Usually Often Sometimes Rarely
 Never

*** 80. Have you ever been told that if you were to decrease your weight your athletic performance would improve?** (Select one option)

- Yes
 No

*** 81. In the past 3 months, how often have you exercised 60 minutes or more to lose or control your weight? (Select one option)**

- Never Once a month or less 2-3 times per month Once a week 2-6 times per week
 Once a day or more

Never:Go to Page No. 32
 Once a month or less:Go to Page No. 31
 2-3 times per month:Go to Page No. 31
 Once a week:Go to Page No. 31
 2-6 times per week:Go to Page No. 31
 Once a day or more:Go to Page No. 32

page 31

*** 82. Was this to lose weight or control your weight? (Select one option)**

- Lose weight
 Control weight

page 32

*** 83. Have you had any exercise related injuries in the last year? (Select one option)**

- No, not at all Yes, one or two Yes, three or four Yes, five or more

No, not at all:Go to Page No. 34
 Yes, one or two:Go to Page No. 33
 Yes, three or four:Go to Page No. 33
 Yes, five or more:Go to Page No. 33

page 33

84. How many days absence from exercise due to injuries have you had in the last year? (Select one option)

- None 1-7 days 8-14 days 15-21 days 22 days or more

Please fill in the following table regarding your exercise related injuries in the last year.

85. Have you experienced this type of injury in the past year?

| | Yes | No |
|--|-----------------------|-----------------------|
| *(a) Muscular (Select one option) | <input type="radio"/> | <input type="radio"/> |
| *(b) Ligament (Select one option) | <input type="radio"/> | <input type="radio"/> |
| *(c) Fracture (including stress fractures) (Select one option) | <input type="radio"/> | <input type="radio"/> |
| *(d) Other (Select one option) | <input type="radio"/> | <input type="radio"/> |

86. If yes, how many injuries of this type have you had?

| | 1 | 2 | 3 | 4+ |
|---|-----------------------|-----------------------|-----------------------|-----------------------|
| (a) Muscular (Select one option) | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| (b) Ligament (Select one option) | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| (c) Fracture (including stress fractures) (Select one option) | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |
| (d) Other (Select one option) | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> | <input type="radio"/> |

87. If yes, please describe your injury in more detail e.g left foot stress fracture or right hamstring strain

(a) Muscular

(b) Ligament

(c) Fracture (including stress fractures)

(d) Other

88. If yes, when did you first experience these injuries? (If you have experienced multiple injuries please state multiple dates e.g. hamstring strain May 2013, calf strain Dec 2013

(a) Muscular

(b) Ligament

(c) Fracture (including stress fractures)

(d) Other

page 34

*** 89. Do you have a family history of osteoporosis (poor bone health) ? (Select one option)**

- Yes
 No

page 35

*** 90. In the past 3 months, how often have you made yourself sick (vomited) to control your weight? (Select one option)**

- Never Once a month or less 2-3 times per month Once a week 2-6 times per week
 Once a day or more

*** 91. I feel bloated after eating a normal meal. (Select one option)**

- Always Usually Often Sometimes Rarely
 Never

*** 92. I think that my thighs are just the right size. (Select one option)**

- Always Usually Often Sometimes Rarely
 Never

*** 93. I exaggerate or magnify the importance of weight. (Select one option)**

- Always Usually Often Sometimes Rarely

Never

*** 94. I stuff myself with food. (Select one option)**

Always Usually Often Sometimes Rarely
 Never

*** 95. When I am upset, I worry that I will start eating. (Select one option)**

Always Usually Often Sometimes Rarely
 Never

*** 96. I like the shape of my upper body. (Select one option)**

Always Usually Often Sometimes Rarely
 Never

page 36

97. If you have any extra comments you would like to add please write them in the text box below.

page 37

Thank you for filling out our questionnaire. If you would like to receive a nutrition tip sheet and go into the monthly draw to win a \$50 grocery voucher please tick the 'yes' box below. We will send the nutrition sheet to the email address provided at the start of the survey. Thanks again we value your thoughts and appreciate your time.

98. I would like to receive a nutrition tip sheet and go into the monthly draw to win a \$50 grocery voucher (Select one option)

Yes No

Appendix B



Form Updated: November 2013

UNIVERSITY OF OTAGO HUMAN ETHICS COMMITTEE APPLICATION FORM: CATEGORY B

(Departmental Approval)

Please ensure you are using the latest application form available from:
<http://www.otago.ac.nz/administration/committees/otago000864.html>

1. University of Otago staff member responsible for project:
Black Katherine Dr

2. Department/School:

Human Nutrition

3. Contact details of staff member responsible:

katherine.black@otago.ac.nz

4. Title of project:

Low Energy Availability questionnaire (LEA questionnaire)

5. Indicate type of project and names of other investigators and students:

Staff Research

Names

Dr Rachel Brown, Mrs Rebecca Cooke

Student Research

Names

Jo slater

Level of Study (e.g. PhD, Masters, Hons)

MSc

External Research/

Names

Collaboration

Institute/Company

6. **When will recruitment and data collection commence?**

May 2014

When will data collection be completed?

May 2015

7. **Brief description in lay terms of the aim of the project, and outline of the research questions that will be answered** (approx. 200 words):

Inadequate energy intake in relation to training load and daily activities amongst athletes results in insufficient energy being available for normal physiological functions. Low energy availability can occur with or without an associated eating disorder or disordered eating. It is thought to be common amongst athletes and can have severe health implications particularly for reproductive function and bone health (1,2). The prevalence of this problem amongst recreational athletes in New Zealand is unknown.

Therefore, the aim of this study is to describe the prevalence of low energy availability, disordered eating and eating disorders amongst recreational athletes in New Zealand.

References

- 1) De Souza MJ, Nattiv A, Joy E, et al, (2014) 2014 Female Athlete Triad Coalition Consensus Statement on Treatment and Return to Play of the Female Athlete Triad. Br J Sports Med 48:289. doi:10.1136/bjsports-2013-093218
- 2) Loucks AB, Kiens B, Wright HH, (2011) Energy availability in athletes. J Sport Sci 29, 7-15

8. **Brief description of the method.** Include a description of who the participants are, how the participants will be recruited, and what they will be asked to do:-

Participants

Individuals will be asked to complete a training schedule at the start of the survey and will need to accumulate at least 150 to 300 minutes (2 ½ to 5 hours) of moderate intensity physical activity or 75 to 150 minutes (1 ¼ to 2 ½ hours) of vigorous intensity physical activity, or an equivalent combination of both moderate and vigorous activities, each week. Participants must be older than 16 years to participate in the study.

Recruitment

We will approach local gyms and sports clubs to ask if they could advertise our study via a poster, or post on their Facebook or Twitter account. A link to the survey will be provided on the poster.

Methods

Those wishing to participate can log in to the survey webpage and complete an online survey which will contain both short answer and multi-choice questions. The questionnaire is based on previously validated questionnaires on eating habits and symptoms associated with low energy availability. Participants will also be asked to recall their training programs, and questions regarding their body mass. It is likely that the survey will take 20-30 minutes to complete (see attached).

Upon completion of the study participants will be provided with a factsheet on food and exercise. They will also be entered into a monthly draw to win a \$50 voucher. In order for both of these to be sent to them, they will be asked to provide their email address if they wish to receive this.

Reporting Sheet for use ONLY for proposals considered at departmental level

9. **Disclose and discuss any potential problems:** (For example: medical/legal problems, issues with disclosure, conflict of interest, safety of the researcher, etc)

There are no foreseen problems with this study. None of the researchers have a conflict of interest.

***Applicant's Signature:**

Name (please print):

Date:

**The signatory should be the staff member detailed at Question 1.*

ACTION TAKEN

Approved by HOD

Approved by Departmental Ethics Committee

Referred to UO Human Ethics Committee

Signature of **Head of Department:

Name of HOD (please print):

Date:

****Where the Head of Department is also the Applicant, then an appropriate senior staff member must sign on behalf of the Department or School.**

Departmental approval: *I have read this application and believe it to be valid research and ethically sound. I approve the research design. The research proposed in this application is compatible with the University of Otago policies and I give my approval and consent for the application to be forwarded to the University of Otago Human Ethics Committee (to be reported to the next meeting).*

IMPORTANT NOTE: As soon as this proposal has been considered and approved at departmental level, the completed form, together with copies of any Information Sheet, Consent Form, recruitment advertisement for participants, and survey or questionnaire should be forwarded to the Manager, Academic Committees or the Academic Committees Administrator, Academic Committees, Rooms G22, G23 or G24, Ground Floor, Clocktower Building, or scanned and emailed to either gary.witte@otago.ac.nz, or jane.hinkley@otago.ac.nz

Reporting Sheet for use ONLY for proposals considered at departmental level

INFORMATION SHEET TEMPLATE: NOTES FOR APPLICANTS
(Delete all notes and prompts before providing to Human Ethics Committee)

The template on the following pages is a guide for providing information to potential participants before they agree to take part in the research project. Not all of the suggestions or headings on this template will necessarily apply to all projects. Delete those that do not apply and/or make the necessary amendments. An Information Sheet is written in the form of a customised letter of invitation to each target group of research participants. It must contain all the information they need in order to make an informed decision about whether or not they wish to participate in your research. What are they asked to do? What will they experience?

An Information Sheet is expected to be submitted with the application for ethical approval in all Category A applications and most Category B Reporting Sheets. The Information Sheet template can be used as a prompt for a cover letter introducing the research even in cases where a formal written Consent Form is not used, e.g. an anonymous survey.

The Information Sheet should be written in simple, clear language (free from jargon and technical terms) that is age and culture appropriate for your participants, so that they can fully understand what they will be doing and experiencing. This is the principle of Informed Consent.

The Information Sheet you submit with your application should be the final version you intend to provide to your participants. All traces of the prompts in italics from the Human Ethics Committee to the researcher should be removed and it should be carefully proof-read for spelling, grammar and formatting.

[Date]



Low Energy Availability questionnaire (LEA questionnaire)
**INFORMATION SHEET FOR
PARTICIPANTS.**

Thank you for showing an interest in this project. Please read this information sheet carefully before deciding whether or not to participate. If you decide to participate we thank you. If you decide not to take part there will be no disadvantage to you and we thank you for considering our request.

What is the Aim of the Project?

Inadequate energy intake in relation to training load and daily activities amongst athletes results in insufficient energy being available for normal physiological functions. Low energy availability can occur with or without an associated eating disorder or disordered eating. It is thought to be common amongst athletes and can have severe health implications particularly for reproductive function and bone health. The prevalence of this problem amongst recreational athletes in New Zealand is unknown.

Therefore, the aim of this study is to describe the prevalence of low energy availability, disordered eating and eating disorders amongst recreational athletes in New Zealand.

This project is being undertaken as part of the requirements for a Masters in Science.

What Types of Participants are being sought?

We are seeking participants whose current training schedule involves at least 150 to 300 minutes (2 ½ to 5 hours) of moderate intensity physical activity or 75 to 150 minutes (1 ¼ to 2 ½ hours) of vigorous intensity physical activity, or an equivalent combination of both moderate and vigorous activities, each week. Participants must also be older than 16 years.

We will be advertising in local gyms and sports clubs as well as on their Facebook or Twitter accounts.

Upon completion of the study you will be provided with a factsheet on food and exercise. You will also be entered into a monthly draw to win a \$50 voucher.

Number of participants?????

What will Participants be asked to do?

Should you agree to take part in this project, you will be asked to log in to the survey webpage and complete an online survey which will contain both short answer and multi-choice questions. It is likely that the survey will take 20-30 minutes to complete.

Reporting Sheet for use ONLY for proposals considered at departmental level

Upon completion of the study you will be provided with a factsheet on food and exercise. You will also be entered into a monthly draw to win a \$50 voucher. You will need to provide your email address to receive the factsheet and to be entered into the draw.

Please be aware that you may decide not to take part in the project without any disadvantage to yourself.

What Data or Information will be collected and what use will be made of it?

In the questionnaire you will be asked to recall your training program, as well as answer questions regarding your body mass, history of injuries, eating patterns and for females the history of your menstrual cycle. This data will be used to determine your risk of having low energy availability, disordered eating or an eating disorder.

All written records will be stored securely in Dr Katherine Black's office, as will the list connecting the ID numbers present on the raw data to the email (where provided) of individual participants. Working data files containing non-identifiable information only, will be stored on the Department of Human Nutrition computer server which is password protected. Access to the files will be restricted to the study investigators. The material will be shredded ten years after study completion, as per University requirements.

Should we find any anomalies in your data we will seek to advise you to contact an appropriate health professional, which may include Medical Doctors, Psychologists and Dietitians.

The results of the project may be published and will be available in the University of Otago Library (Dunedin, New Zealand) but every attempt will be made to preserve your anonymity.

- *Will the participants have the opportunity to correct or withdraw the data/information?*
If you believe any data is incorrect you may ask to see your data and correct or withdraw it from the study at any time.
- *Will participants be given the opportunity to view the data or information that relates to them either before or after the completion of the research? At what stage will this opportunity be given to them?*
If you wish to view your data then you should contact either Jo Slater or Dr Katherine Black, you can do this at any time during the study.

Can Participants change their mind and withdraw from the project?

You may withdraw from participation in the project at any time without any disadvantage to yourself.

What if Participants have any Questions?

If you have any questions about our project, either now or in the future, please feel free to contact either:-

Reporting Sheet for use ONLY for proposals considered at departmental level

Name of Student: Jo Slater

and

Name of Supervisor: Dr Katherine Black

Department of Human Nutrition

Department of Human Nutrition

University Telephone Number: +643479 8358

Email Address: slajo001@student.otago.ac.nz

Email Address: katherine.black@otago.ac.nz

This study has been approved by the Department of Human Nutrition. However, if you have any concerns about the ethical conduct of the research you may contact the University of Otago Human Ethics Committee through the Human Ethics Committee Administrator (ph 03 479-8256). Any issues you raise will be treated in confidence and investigated and you will be informed of the outcome.

Low Energy Availability questionnaire (LEA questionnaire)

**CONSENT FORM FOR
PARTICIPANTS**

I have read the Information Sheet concerning this project and understand what it is about. All my questions have been answered to my satisfaction. I understand that I am free to request further information at any stage.

I know that:-

1. My participation in the project is entirely voluntary;
2. I am free to withdraw from the project at any time without any disadvantage;
3. Personal identifying information such as e-mail addresses will be destroyed at the conclusion of the project but any raw data on which the results of the project depend will be retained in secure storage for at least five years;
4. There are no foreseeable risks for completing this study, however if you do feel uncomfortable whilst participating you may withdraw at any point without any consequences.
5. Once you have completed the questionnaire you will be eligible to receive a factsheet on nutrition and exercise as well as entry into a monthly draw for \$50. For these you will need to provide your email address.
6. The results of the project may be published and will be available in the University of Otago Library (Dunedin, New Zealand) but every attempt will be made to preserve my anonymity.

I agree to take part in this project.

.....
(Signature of participant)

.....
(Date)

.....
(Printed Name)





Low Energy Availability questionnaire (LEA questionnaire)

This research study aims to describe the prevalence of low energy availability, disordered eating and eating disorders amongst recreational athletes in New Zealand.

Inclusion criteria: Recreational New Zealand athletes, over the age of 16 years, whose current training schedule involves at least 150 to 300 minutes (2 ½ to 5 hours) of moderate intensity physical activity or 75 to 150 minutes (1 ¼ to 2 ½ hours) of vigorous intensity physical activity, or an equivalent combination of both moderate and vigorous activities, each week.

Exclusion criteria: Elite athletes and those who do not meet the inclusion criteria.

This study is the first of its kind in New Zealand. It will therefore provide us with new knowledge of the prevalence of low energy availability amongst New Zealand's recreational athletes. Upon completion of this study participants will be provided with a factsheet on food and exercise. They will also be entered into a monthly draw to win a \$50 voucher

The time commitment that will be required is 20-30 minutes to complete the questionnaire.

Dr Katherine Black, University of Otago, Science II 7c4, ph:+64 3 479 8358, katherine.black@otago.ac.nz

This project has been reviewed and approved by the Department of Human Nutrition, University of Otago

| | |
|---|--|
| Low Energy Availability questionnaire (LEA questionnaire) | katherine.black@otago.ac.nz |
| Low Energy Availability questionnaire (LEA questionnaire) | katherine.black@otago.ac.nz |
| Low Energy Availability questionnaire (LEA questionnaire) | katherine.black@otago.ac.nz |
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| Low Energy Availability questionnaire (LEA questionnaire) | katherine.black@otago.ac.nz |

IMPORTANT NOTES/CHECK LIST: Category B Reporting Sheets

Detach this page of notes *before* making the copies to be forwarded to the University of Otago Human Ethics Committee.

1. This form should only be used for proposals which are **Category B**, as defined in the policy document "Policy on ethical practices in research and teaching involving human participants", and which may therefore be properly considered and approved at departmental level. These proposals should not be health research.
2. A proposal can only be classified as Category B if **NONE** of the following is involved:-
 - Personal information - any information about an individual (e.g. name, contact details, position etc.) who may be identifiable from the data once it has been recorded in some lasting and usable format, or from any completed research;
(Note: this does not include contact details needed for a limited time for practical purposes but which are unlinked to research data and destroyed once the details are no longer needed.)
 - Any form of physical or psychological stress;
 - Situations which might place the safety of participants or researchers at any risk;
 - A potential conflict between the applicant's activities as a researcher, clinician or teacher and their interests as a professional or private individual;
 - The participation of minors or any other vulnerable individuals;
 - Any form of deception which might threaten an individual's emotional or psychological well-being.
 - The research is being undertaken overseas by students.

If any of the above is involved, then the proposal is **Category A**, and must be submitted to the **University of Otago Human Ethics Committee** using the standard Category A application form, before the teaching or research commences.

If the research involves the following, the proposal should be submitted to the **University of Otago Human Ethics Committee (Health)** on the **health** application form:

- Participants are recruited from health services (patients)/patient data is to be examined;
 - The taking or handling of any form of tissue or fluid sample from humans or cadavers;
 - Any form of physical or psychological stress;
 - The administration or restriction of food, fluid or a drug to a participant;
3. Ensure the application is in the name of a University of Otago staff member (and not, for example, the student researcher).
 4. Ensure the Consent Form, Information Sheet, Advertisement and any survey or questionnaire have been carefully proofread for spelling, grammar and formatting, as the institution as a whole is likely to be judged by them.
 4. A Category B proposal may commence as soon as departmental approval has been obtained; however it is best practice to plan ahead to ensure the Committee has time to audit your proposal and respond to you (audit happens the week prior to the Human Ethics Committee meeting). Should the Committee have any concerns about the proposal, you will be notified in writing.

Reporting Sheet for use ONLY for proposals considered at departmental level

5. Please submit a Category B Reporting Sheet together with copies of any Information Sheet, Consent Form, recruitment advertisement for participants, and survey or questionnaire immediately after it has been signed by the Head of Department to the Human Ethics Committee, via:

Gary Witte (Manager, Academic Committees), or Jane Hinkley (Academic Committees Administrator), Academic Committees Office, Rooms G22, G23 or G24, Ground Floor, Clocktower Building. Forms can be emailed to either gary.witte@otago.ac.nz or jane.hinkley@otago.ac.nz.

Appendix C

Gyms and fitness centres that agreed to advertise the LEANZA study to their clients

| Gym/fitness centre | Contact person | Phone number | Email | Address |
|---------------------------------------|-----------------|----------------|--------------------------------------|-----------------------------|
| World Fitness Centre | Phil Shaw | 4792385 | reception@worldfitness.co.nz | 266 Hanover St, Dunedin |
| Body Synergy Gym | Rowan | (03) 4777226 | rowan.ellis@bodysynergy.co.nz | Dunedin |
| Les Mills Dunedin | Karen Lee | (03) 4772295 | karenlee@lesmills.co.nz | 12 Dowling St, Dunedin |
| Moana Pool | Pauline Leijnse | (03) 4719780 | paulien.leijnse@dcc.govt.nz | Littlebourne Rd, Dunedin |
| Configure Express - The Gym for Women | Tanya | (03) 4774750 | dunedin@configureexpress.co.nz | 15 Filleul St Dunedin 9016 |
| Anytime Fitness | Michael Sanders | (03) 4550013 | michael.sanders@anytimefitness.co.nz | 92 MacAndrew Rd, Dunedin |
| Snap Fitness | Glen | (03) 4778286 | dunedin@snapfitness.com | 122 Cumberland St, Dunedin |
| Elleslie YMCA | Joshua Slessor | | Joshua.slessor@ymcauckland.org.nz | Auckland |
| City Fitness | Rachel | 0800 348 637 | customercare@cityfitness.co.nz | Auckland |
| Hamilton YMCA | Strimi Naidoo | | Strimi.naidoo@ymcauckland.org.nz | 83 Penbrooke place Hamilton |
| Jetts Whangarei | Sharleen | 0800 JETTS 247 | whangarei@jetts.co.nz | Whangarei |
| Jetts Botany | Dayna | 0800 JETTS 247 | botany@jetts.co.nz | Botany, Auckland |
| Jetts Northland | Tracey | 0800 JETTS 247 | marketing@jetts.co.nz | Northland |
| Body Therapeutics | Paula | 04 4733466 | info@bodytherapeutics.co.nz | Wellington |
| Temple Fitness | Renee | 06 863 0244 | info@templefitness.co.nz | Gisborne |
| Human movements fitness centre | Mary-Anne | 06 356 1570 | rabba@inspire.net.nz | Palmerston North |

| | | | | |
|-------------------------------|--------------------|-------------|---------------------------------------|--------------|
| Crossfit Christchurch | Chris and Jo | 03 928 2489 | jo@crossfitchristchurch.com | Christchurch |
| Marlborough line stadium 2000 | Anna | 03 577 8300 | healthandfitness@stadium2000.co.nz | Blenheim |
| Advance fitness club | Stephanie | 03 218 8624 | invercargill@advancefitnessclub.co.nz | Invercargill |
| City Fitness | Rachel | 04 478 6228 | jbillereception@cityfitness.co.nz | Wellington |
| Les Mills New Lynn | Janenne | 09 826 0404 | newlynn@lesmills.co.nz | Auckland |
| Mountain Gym | Renee and Michelle | 03 302 8889 | michelle.carson@mountaingym.co.nz | Methven |
| Nfinite fitness and health | Jonno | 06 835 5565 | nfinitefitness@windowslive.com | Napier |
| Empower Fitness Limited | Jackie and Fiona | 07 376 9557 | fiona@empowerfitness.co.nz | Taupo |

Dear World Gym member,

The Department of Human Nutrition at the University of Otago is currently investigating the health and dietary practices of gym goers in New Zealand. In particular we are looking at energy intake in relation to training load and daily activities and the effects on health.

I am writing to request your participation in this study, if you are interested please find attached the information sheet that gives a detailed description of the study and its aims. The answers you provide will be anonymous and after reading the attached information sheet should you chose to participate you are free to withdraw at anytime without any implications to yourself.

After reading the information sheet, should you agree to take part in this project, we ask you to log in to the survey webpage (via the link below) and complete an online survey which will contain both short answer and multi-choice questions. It is likely that the survey will take 20-30 minutes to complete.

Upon completion of the study you will be provided with a factsheet on food and exercise. You will also be entered into a draw to win a \$50 grocery voucher. Please note you will need to provide your email address to receive the factsheet and to be entered into the draw.

For more information please contact me or Jo Slater via leanz@otago.ac.nz Thank you for your time.

Kind regards

Dr Katherine Black.

Senior Lecturer
Sport and Exercise Nutrition Research Group
Department of Human Nutrition
University of Otago
PO Box 56
Dunedin
9054
e-mail: katherine.black@otago.ac.nz
Telephone: +64 (3) 479 8358



PERFORMANCE NUTRITION

Performance Nutrition

Sports Nutrition

A healthy diet helps keep our energy levels high, immune system happy and also assists in keeping our weight within the healthy range. A healthy diet involves enjoying a **range of foods** from all food groups, including lean meat and meat alternatives (legumes, nuts, eggs), grainy breads and cereals, low fat dairy products, small amounts of good fats like avocado, and always remembering to have a colorful mix of fruit and vegetables (5+ a day).

A healthy diet is paramount in preparing our bodies to get the most out of our workout.

Once we have the basics right we can work on **timing our meals** and snacks around training to maximise the benefits of exercise.

What Should I Eat Before Exercise?

Carbohydrates are the energy source that fuel our body during exercise. They help maintain normal blood glucose levels which is important for both our mental and physical performance. **2-4 hours prior** to exercising aim to have a meal or snack that contains at least **50 g of carbohydrates**. See over page for pre exercise meal and snack ideas.

Do I Need To Eat During Exercise?

When exercising for long periods of time our body often needs a fuel top up. As a general rule of thumb, when training for 1.5-3 hours aim to consume **30-60 g of carbohydrates** per hour of exercise or **up to 90 g** of carbohydrates for longer training days. See over the page for snack ideas to try during exercise.



What Should I Eat After Exercise?

Good recovery post exercise is vital for optimising muscle gains and repair and replenishing glycogen stores. After exercising if you are heading straight home for your next meal, make the most of this timing and include a **serve of high quality protein** (e.g. lean meat, chicken or fish, low fat dairy products or tofu) as well as a **serve of carbohydrates** (grainy bread, brown rice, quinoa, wholegrain cereal, kumara). If your next meal is more than an hour after your training enjoy a healthy snack that contain both protein and carbohydrates.

Sports Nutrition

Meal Time



And What About Hydration?

Dehydration can cause drowsiness, lethargy and early fatigue, all of which decrease our exercise performance. We don't always know when we are dehydrated but a fluid loss as **small as 1-2 %** can cause a decrease in performance. It is important to be well hydrated before, during and after working out. There is no need to drink excessive amounts as this can have adverse effects. **Aim to have 2L** of water spread across each day.

Depending on the temperature of the day and the intensity of the workout the amount may need to be adjusted. We can gauge our hydration status by looking at our urine, which should be a pale yellow colour.

Where Do I Find Out More?

All nutrient requirements are specific to you and your training load. For more detailed information refer to the **Sports Dietitians Australia** website or make an appointment to see your local **Registered Nutritionist or Dietitian**.



NOTE: Portions of meals and snacks will vary depending on the total energy expenditure of the individual and will need to be increased/decreased accordingly.

PRE EXERCISE MEAL IDEAS

Breakfast

- 1 cup cooked porridge with 1/2 cup low fat milk + 1/3 cup tinned fruit in natural juice or a drizzle of honey

or

- 2 slices of wholegrain toast with honey or jam + 1 piece of fruit

Lunch

- Tomato, onion + cheese (30g Edam) toasted sandwich on wholegrain bread

or

- 1/2 cup baked beans on 2 slices of wholegrain bread

Dinner

- Chickpea + kumara Thai red curry made with light coconut milk + packed full of seasonal vegetables, served with 1 cup of brown or basmati rice.

POST EXERCISE MEAL IDEAS

Breakfast

- 1/2 cup of natural muesli (with nuts), 1/2 cup of low fat milk, 1/3 cup yoghurt + 1/3 cup tinned fruit in natural juice

or

- 2 eggs on 2 slices of wholegrain toast with 1/4 avocado

Lunch

- Grainy bread sandwich, 30g Edam cheese, 50-80g lean meat + garden salad

or

- 1 medium slice frittata packed with vegetables including potato or kumara

Dinner

- Stir-fry 120-150g lean meat chicken or tofu 1/2-2 cups of vegetables + 1 cup of brown or basmati rice

or

- 120-150g grilled fish, 1 cup couscous + 1/2 plate garden salad – lettuce, tomato, capsicum, cucumber

Something To Snack On

PRE EXERCISE SNACK IDEAS

- 1 slice of raisin toast with a scraping of margarine
- 1 slice wholegrain toast or bread with honey or jam
- 1 hot cross bun
- 1 wholemeal crumpet with honey
- Piece of fruit – bananas are great

DURING EXERCISE SNACK IDEAS

- Jam or honey sandwich on white bread
- A banana
- Fruit bread with jam
- A fruit based muesli bar
- Sports drinks, bars and gels
- 1 handful of dried fruit
- New potatoes
- Lollies

POST EXERCISE SNACK IDEAS

- Banana + berry smoothie
- Yoghurt + natural muesli
- 1 slice wholegrain toast with melted Edam cheese
- 1 handful of almonds + dried fruit
- 1 slice of wholegrain toast with peanut butter
- A glass of low fat Milo, hot or cold

Appendix D

Categorisation of Sports

| Sport | Intensity level ¹ | Requiring body revealing clothing | Emphasising low body weight | Subjectively scored | Weight restrictions | Team or individual |
|-------------------|------------------------------|-----------------------------------|-----------------------------|---------------------|---------------------|--------------------|
| Hockey | 8.0 | Yes | No | No | No | Team |
| Netball | 8.0 | Yes | No | No | No | Team |
| Handball | 8.0 | No | No | No | No | Team |
| Basketball | 8.0 | No | No | No | No | Team |
| Touch rugby | 8.0 | No | No | No | No | Team |
| Ice hockey | 8.0 | No | No | No | No | Team |
| Dodgeball | 8.0 | No | No | No | No | Team |
| Rugby union | 10.0 | No | No | No | No | Team |
| Soccer/football | 10.0 | No | No | No | No | Team |
| Volleyball indoor | 8.0 | No | No | No | No | Team |
| Volleyball beach | 8.0 | Yes | No | No | No | Team |
| Cricket | 5.0 | No | No | No | No | Team |
| Cross country | 9.0 | No/Yes | Yes | No | No | Individual |
| Ultra running | 9.8 | No/Yes | Yes | No | No | Individual |
| Cross Fit | 7.8 | No | No | No | No | Individual |
| Powerlifting | 6.0 | Yes | No | No | No | Individual |

¹ As scored by the Ainsworth Compendium of physical activity

Categorisation of Sports

| Sport | Intensity level² | Requiring body revealing clothing | Emphasising low body weight | Subjectively scored | Weight restrictions | Team or individual |
|-----------------|------------------------------------|--|------------------------------------|----------------------------|----------------------------|---------------------------|
| Swimming | 10.0 | Yes | No | No | No | Individual |
| Water polo | 10.0 | Yes | No | No | No | Team |
| Free diving | 7.0 | No | No | No | No | Individual |
| Mountain biking | 12.5 | No | No | No | No | Individual |
| Road cycling | 10.0 | Yes | Yes | No | No | Individual |
| Biking leisure | 4.0 | No | No | No | No | Individual |
| Track cycling | 16.0 | Yes | Yes | No | No | Individual |
| Rowing | 8.5 | Yes | Yes | No | No | Team |
| Triathlon | 10 | Yes | Yes | No | No | Individual |
| Group exercise | 5.5 | No | No | No | No | Individual |
| Pump class | 5.5 | No | No | No | No | Individual |
| Dance | 6.5 | Yes | No | Yes | No | Individual |
| Gymnastics | 3.8 | Yes | Yes | Yes | No | Individual |
| Marital arts | 10.0 | No | No | No | Yes | Individual |
| Walking | 3.3 | No | No | No | No | Individual |

² As scored by the Ainsworth Compendium of physical activity

Categorisation of Sports

| Sport | Intensity level ³ | Requiring body revealing clothing | Emphasising low body weight | Subjectively scored | Weight restrictions | Team or individual |
|--------------------------|------------------------------|-----------------------------------|-----------------------------|---------------------|---------------------|--------------------|
| Tennis | 7.0 | No | No | No | No | Individual |
| Badminton | 7.0 | No | No | No | No | Individual |
| Squash | 12.0 | No | No | No | No | Individual |
| Climbing – rock/mountain | 8.0 | No | No | No | No | Individual |
| Equestrian | 4.0 | No | No | No | No | Individual |
| Kayaking | 5.0 | No | No | No | No | Individual |
| White water kayaking | 5.0 | No | No | No | No | Individual |
| Canoe Polo | 5.0 | No | No | No | No | Team |
| Softball | 5.0 | No | No | No | No | Team |
| Curling | 4.0 | No | No | No | No | Individual |
| Multi-sport | 10 | Yes | Yes | No | No | Individual |
| Golf | 4.5 | No | No | No | No | Individual |
| Snowboarding | 7.0 | No | No | No | No | Individual |
| Skiing | 7.0 | No | No | No | No | Individual |
| Surfing | 3.0 | No | No | No | No | Individual |

³ As scored by the Ainsworth Compendium of physical activity

Appendix E



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07/10/2014

Dear LEANZ study participant,

Thank you once again for taking part in the LEANZ study. We really appreciated your time and effort in completing our survey.

As part of the LEANZ questionnaire a validated screening tool (the Eating Disorder Inventory-3, Risk Composite) was used to assess the presence of disordered eating behaviors amongst participants. Disordered eating can occur for several reasons, but can have some health implications.

We are writing to inform you that after analysing your results, we that you scored particularly high in these questions. Based on these results we strongly suggest that you make an appointment with General Practitioner. You may want to take this letter to inform them of your results on the Eating Disorder Inventory-3. We can provide you with further score details, but your GP will be able to help in terms of your health.

If you do not wish to visit your GP then other help services include:

Healthline – 0800 611 116 nurses can refer to local health services.

If yourself or your General Practitioner have any questions please contact us on leanz@otago.ac.nz.

Kind regards,

The LEANZ study team
Dr Katherine Black, Jo Slater.

Appendix F

**WOLTERS KLUWER HEALTH, INC. LICENSE
TERMS AND CONDITIONS**

May 19, 2015

This Agreement between Jo Slater ("You") and Wolters Kluwer Health, Inc. ("Wolters Kluwer Health, Inc.") consists of your license details and the terms and conditions provided by Wolters Kluwer Health, Inc. and Copyright Clearance Center.

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