

**HIGH LEVELS OF COGNITIVE DIETARY RESTRAINT ARE
ASSOCIATED WITH STRESS FRACTURES IN WOMEN RUNNERS**

by

NANCI S. GUEST

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NANCI S. GUEST

Name of Author (please print)

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Date (dd/mm/yyyy)

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ABSTRACT

Societal emphasis on body image and the 'ideal' body weight drives many women to make conscious *efforts* to limit their food intake in order to achieve or maintain a desired body weight. This attitude and eating behaviour is characterized by a preoccupation with food-related issues, and is referred to as *dietary restraint* or *cognitive dietary restraint (CDR)*. The most commonly used instrument to measure and assess this dietary restraint is the restraint scale of the Three-Factor Eating Questionnaire (TFEQ). Female athletes are faced with body image challenges, as well as trying to achieve a body weight that is optimal for their performance. Many female athletes could therefore be experiencing these restrained eating patterns, to meet the combined pressures of an 'ideal body' and enhanced performance.

Most previous studies have generally found similar physical characteristics and energy intakes among women with differing restraint scores. However, CDR has been associated with subclinical menstrual cycle irregularities (MCI) and increased cortisol levels, both of which can affect bone mineral density (BMD). Preliminary evidence has also reported an association between CDR and BMD or bone mineral content (BMC). Low BMD has been implicated in stress fracture risk, and runners are particularly at risk for lower extremity stress fractures.

The purpose of this investigation was to assess CDR in female runners with a recent stress fracture (SF) and without a history of stress fracture (NSF). We recruited nulliparous normal-weight runners (running ≥ 20 km/wk) who were non-smokers, had regular menstrual cycles, were not currently dieting and had no history of an eating disorder. A sample of 79 runners ($n = 38$ SF, 29 ± 5 yr; $n = 41$ NSF, 29 ± 6 yr) completed a

3-day food record and questionnaire assessing physical activity, menstrual cycle history and perceived stress. The TFEQ was used to assess eating attitudes and behaviours, including CDR.

SF and NSF runners had similar body mass index (21.2 ± 1.8 vs 22.0 ± 2.5 kg/m²), physical activity (35.7 ± 13.5 vs 33.4 ± 1.34 km/wk), perceived stress, and energy and macronutrient intakes. However, CDR was significantly higher in SF runners (11 ± 5.4 vs 8.4 ± 4.3 , $p < 0.05$). We conclude that women runners with a history of recent SF have higher levels of CDR. Subclinical MCI and increased cortisol levels associated with high CDR may contribute to lowered BMD and increased risk for stress fracture.

Prospective studies that include measurements of menstrual cycle characteristics, cortisol levels and BMD are needed to determine if CDR is an independent risk factor for stress fractures, mediated by subclinical MCI and elevated cortisol with subsequent bone loss.

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List of Abbreviations

AA: amenorrheic athlete
ACTH: adrenocorticotrophic hormone
ANCOVA: analysis of covariance
BMC: bone mineral content
BMD: bone mineral density
BMI: body mass index
BQHPA: Baecke Questionnaire of Habitual Physical Activity
CDR: cognitive dietary restraint
CRH: corticotropin-releasing hormone
CT: computerized tomography
DEBQ: Dutch Eating Behaviour Questionnaire
DIT: diet-induced thermogenesis
DXA: dual energy x-ray absorptiometry
EA: eumenorrheic athlete
EAT-40: Eating Attitudes Test
EDI: Eating Disorder Inventory
EE: energy expenditure
FHA: functional hypothalamic amenorrhea
FSH: follicle-stimulating hormone
GnRH: gonadotropin-releasing hormone
HPA: hypothalamic pituitary adrenal
HPO: hypothalamic pituitary ovarian
LPD: luteal phase defects
MCI: menstrual cycle irregularities
MRI: magnetic resonance imaging
NSF: non-stress fracture
PSS: Perceived Stress Scale
PTH: parathyroid hormone
RMR: resting metabolic rate
RS: Restraint Scale
SF: stress fracture
SPSS: Statistical Package for the Social Sciences
TCI: Temperament and Character Inventory
TFEQ: Three-Factor Eating Questionnaire
TFEQ-R: Three-Factor Eating Questionnaire-Restraint
the triad: female athlete triad

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CHAPTER 1:

LITERATURE REVIEW

1.1 Introduction

The female athlete is faced with unique challenges, as she lives in a society that values an “ideal” body shape and competes in a sporting arena where an ideal body weight or lean appearance equates with success. These pressures, surrounding body image, can potentially lead to disordered eating attitudes and behaviours, which are frequently accompanied by detrimental effects to the menstrual cycle and bone health. The “female athlete triad” (Otis et al. 1997) is the combination of disordered eating, menstrual irregularity and osteoporosis/osteopenia, which are interrelated in etiology, pathogenesis and consequences. *Dietary restraint* is one type of disordered eating attitude that has been associated with menstrual cycle disturbances and increased cortisol, both of which can impact bone health. Lower bone mineral density, mediated by the consequences of high levels of dietary restraint, may be a risk for stress fracture. This research study was designed to investigate whether female runners diagnosed with a lower extremity stress fracture will score higher than uninjured runners on the restraint subscale of the Three-Factor Eating Questionnaire (TFEQ).

Accordingly, this review will include early studies on dietary restraint, how it is assessed, characteristics of women with high levels of dietary restraint, and associations between dietary restraint and the menstrual cycle. An overview of the possible mechanisms and implications of dietary restraint on the neuroendocrine system with a particular emphasis on cortisol production and its effect on bone health will follow.

Emphasis will be placed on the potential implications of dietary restraint on bone health through its impact on the menstrual cycle and cortisol production and the subsequent risk for stress fractures.

The multitude of risks for stress fractures in female athletes (and female military recruits) with specific emphasis on bone mineral density as a causative factor will be presented. The described impact on bone health will lead to a discussion of a hypothesis demonstrating a possible association between high levels of dietary restraint and an increased risk for stress fractures in female runners.

1.2 Dietary Restraint

1.2.1 Dietary Restraint Defined

Dietary restraint or cognitive dietary restraint (CDR) refers to the tendency to consciously restrict food intake in order to prevent weight gain or to promote weight loss (Herman and Mack 1975; Herman and Polivy 1975). Dietary restraint, sometimes also known as restrained eating, is a type of eating behaviour that is governed by cognitive processes rather than by physiological mechanisms such as hunger and satiety (Lautenbacher et al. 1992). Those individuals who are consciously aware of constantly monitoring their food intake are known as restrained eaters; predictably, individuals who are not particularly concerned about monitoring their food intake are referred to as “nonrestrained” eaters (Heatherton et al. 1988; Herman and Polivy 1980; Ruderman 1986 for reviews).

The term “dieting”, on the other hand, while similar to dietary restraint, has a slightly different meaning. Dieting generally refers to a purposeful restriction of food

intake for a finite period of time (i.e., people go “on” or “off” a diet) that usually *results* in successful weight loss, at least over the short term. Women who are dieting will inevitably be restraining, however not all *chronically* restrained eaters would necessarily identify themselves as being on a “diet” at any given time. The important distinction between dietary restraint and dieting *per se* for the purposes of this study is reflected in the observation that restrained eaters are constantly making the *effort* to restrict their food intake, but are not necessarily successful in taking in fewer calories than their unrestrained counterparts in the long-term.

Central to the concept of restrained eating is the individual’s intention to control food intake in order to maintain or lose weight. However, the behaviour of restrained eaters in the laboratory has been shown to be variable; under some conditions they eat less than unrestrained eaters, while under others they may show relative overeating. This variability in eating behaviour is often attributed to the “counterregulatory” aspects of restraint, which includes periods of overeating, thereby accounting for the lack of weight-loss experienced by some restrained eaters. In other words, some restrained eaters may “diet” successfully for a period of time but are often foiled by certain disinhibiting events (consumption of forbidden foods/alcohol) that tend to interfere with self-control and result in overeating. Others, however, may not experience these episodes of disinhibition. The research studies that investigate these aspects of restraint will be explained in further detail in following sections.

1.2.2 Early Studies on Eating Behaviour and Dietary Restraint

Over three decades ago, Schachter (1968; 1971) and Nisbett (1972) attempted to isolate the determinants of eating behaviours in obese and normal-weight individuals in a

series of experiments and associated theory developments. For his part, Schachter developed the internal-external theory of obesity; that is, he posited that obese individuals were more sensitive to external food cues such as sight, smell and taste, while normal weight individuals were more responsive to internal physiological cues that signaled hunger, such as gastric motility (state of the stomach) and blood glucose levels (Schachter 1971). Schachter (1968; 1971) conducted a variety of experiments in an attempt to reveal these “external” responses to food cues in obese individuals. In one experiment, Schachter demonstrated that normal weight individuals eat more when they are calm than when they are frightened (Schachter 1968). Schachter had hypothesized that, physiologically, an individual in a “frightened” state, would experience a reduction of gastric motility and a release of glucose from the liver, which would suppress and oppose any internal cues for hunger, thereby resulting in reduced food intake. Normal weight individuals did indeed respond as expected – reducing food intake in a state of fear. Conversely, obese subjects did not eat less under the same experimental conditions. In the same study, Schachter also found that normal-weight subjects ate more when they were food deprived than when sated; similar manipulations had no effect on the amounts eaten by obese subjects. Seemingly, the eating behaviour of obese individuals had little to do with the gastric motility or blood glucose levels. Schachter concluded that the internal state is irrelevant to the eating behaviour of obese individuals; rather food-relevant or external cues trigger eating for this group of individuals.

Nisbett (1972) re-examined Schachter's findings and proposed an alternate model to explain the eating behaviours of obese and “hungry” individuals trying to maintain a certain weight. Nisbett hypothesized that individuals have an internal *set point* that

governs eating behaviour, which could explain the seeming inability of obese persons to regulate their eating by internal cues. This set point is biologically determined and/or is established as a result of early nutritional experience, and is a direct function of the *number* of fat cells in the body. He hypothesized that there was inter-individual variability in body fat stores, and some individuals have a higher base-line level of adipose tissue than others. In essence, he suggested that some individuals have a larger number of fat cells than others. Therefore, due to each individual's biologically fixed number of fat cells, subsequent weight fluctuations would only change the *size* of these cells, not their number. The depletion of these fat cells would influence an individual's eating behaviour, that is; food intake patterns would be modified so as to bring one's weight into line with the set point "demanded" by the adipose tissue (i.e., to restore cell size) [Nisbett 1972]. According to Nisbett, biological deprivation (i.e., being below natural set-point) from dieting produces a number of behavioural consequences, including external responsiveness to food cues (Nisbett 1972).

A few years later, Schachter's and Nisbett's research was further extended by Herman and Mack who determined that there was not a strict division between the eating behaviour characteristics of obese and normal weight individuals. Laying the foundation for the further development of the concept of restraint, Herman and Mack hypothesized that there was variability in eating behaviour characteristics *within* groups of normal weight (and obese) individuals. The variability was such that, in certain situations, for example, the eating behaviours of some normal weight individuals more closely approximated those of their obese counterparts in some respects, and in contrast to other normal-weight individuals. Specifically, some normal weight individuals may respond to

external food cues if they are below their set point and behave as obese individuals.

Congruent with Nisbett's reasoning, Herman and Mack suggested that many "normal weight" eaters who were biologically "underweight" would overeat under normal circumstances, but these individuals restrain their eating because of social and cultural pressures to maintain an "ideal" weight. Therefore, these individuals are attempting to maintain a body weight which is "normal" in absolute terms, but low relative to their set point weight (i.e. biological weight)[Herman and Mack 1975].

Experimentally, this difference in "restrained" and "unrestrained" eating behaviour would be seen in their reaction to an eating situation when restraints were temporarily eliminated, such as by prior administration of a dietary pre-load. In one study, subjects were separated into hypothetically deprived (high restraint) and non-deprived (low restraint) groups. In order to quantify the level of restraint, Herman and Mack developed a 10-item questionnaire, the "Restraint Scale" (RS), to measure individuals' concerns about their weight and the level of restriction of food intake, as well as the extent to which they overate when restraints were temporarily removed (Herman and Mack 1975). The expectation that restrained eaters would consume more food with a pre-load, than without, and unrestrained eaters would consume less food with a pre-load, was confirmed. The researchers substantiated Nisbett's theory and concluded that relative deprivation (i.e., high restraint associated with attempting to maintain a weight below the set point) rather than obesity *per se* would determine individual differences in eating behaviour.

In summary, the concept of "restraint" was seen as an important behavioural mechanism affecting the expression of physiologically-based hunger. That is, restraint

could be exercised either to resist the desire to eat in response to internal cues signalling hunger or to terminate eating prior to satiety; restraint could also be abandoned or disengaged under certain conditions such as by the presence of a pre-load (Herman and Mack 1975). Later studies showed the same “counter-regulation” effect during high anxiety situations (Herman and Polivy 1975), and alcohol consumption (Polivy and Herman 1976), as restrained eaters were found to eat more in these situations. The aspect of eating behaviour, where restraint is temporarily removed and loss of control ensues, became known as *disinhibition* (Herman and Polivy 1980).

1.2.3 More Recent Developments in Restraint Theory

The work of Schachter, Nisbett, Herman, Mack and others laid the groundwork for the further development of “restraint theory” which saw a move away from the set point theory of restrained eating behaviour and toward an explanation that emphasized cognitive processes instead. Herman and Polivy (1980) were among the first to elaborate on the initial construct of restraint; they postulated that eating patterns are influenced by a balancing act between physiological factors prompting the desire for food and countervailing cognitive efforts to resist that desire. For restrained eaters, cognitive processes override physiological hunger and satiety cues. In this formulation, “restraint” was described as the “cognitively mediated effort to combat the urge to eat” (Herman and Polivy 1980) and “was [is] further defined more in terms of effort expended toward weight suppression than in terms of achieved success” (Herman and Polivy 1980; p. 223).

In subsequent years, however, the fact that Herman and Mack’s Restraint Scale reflected both restraint and disinhibition was increasingly seen as a limitation; that is, the two constructs were not likely to be perfectly correlated (e.g., some individuals could

have relatively high restraint and low disinhibition, or vice versa). As a result, Stunkard and Messick (1985) and Van Strien and colleagues (1986) developed their own scales that assessed the two constructs separately.

Recently, restraint theory has evolved even further with the contribution of Westenhoefer (1991) who has shown that dietary restraint is not a homogenous construct. Rather, based on discriminant analytic findings he suggested that dietary restraint could be separated into two restraint strategies that he labeled “rigid control” and “flexible control” of eating behaviour. Rigid control was characterized by a dichotomous “all or nothing” approach to eating, dieting and weight. Restrained eaters who fall under this category are likely to diet frequently, but are not very deliberate about what they eat. Tempting or “forbidden” foods are to be avoided, but if eaten, are unlikely compensated for. Flexible control was characterized by a more graduated approach to dieting, including strategies like the “allowance” of limited amounts of sweets that can be consumed on a “guilt-free” basis.

1.2.4 Assessment of Dietary Restraint

There are three principal self-reporting questionnaires – the Restraint Scale (RS), the Three-Factor Eating Questionnaire (TFEQ) and the Dutch Eating Behaviour Questionnaire (DEBQ) that have been extensively used in the study of eating behaviours and other related issues. While the three scales are conceptually related, there are significant differences, particularly between the RS and the restraint subscales of the TFEQ and DEBQ. The instruments differ in reliability, validity and underlying construct assessed, reflecting various approaches to the assessment of eating behaviours generally, and restraint specifically. All three of the aforementioned scales have in

common a motivational component characterizing restrained eaters, including concerns about shape and weight, and desire for thinness. In this section the three instruments are briefly described and highlights of some of the debate as to which questionnaire is “best” in terms of measuring “dietary restraint” are provided.

1.2.4.1 Herman and Mack's Restraint Scale

Herman and Mack's 10-item Restraint Scale (RS)[1975] was the primary measurement tool utilized in the early studies on eating behaviour to differentiate between restrained and unrestrained eating patterns (See Section 1.2.2). The RS included two subscales: “Weight Fluctuation” and “Concern for Dieting”. The scale has been described as representing a continuum – with restrained individuals who are highly conscious of their dietary consumption and carefully monitor food intake and experience weight fluctuations placed at one end, and unrestrained individuals who exhibit little concern about their dietary intake and do not experience weight fluctuations at the other.

While utilized a great deal over the years, there are a number of drawbacks to the RS that have been revealed across a number of studies and over the course of time. Perhaps most importantly, the RS was developed on rational rather than psychometric grounds, with the somewhat predictable result that the construct validity of the instrument has been criticized extensively. For example, Drewnoski and colleagues (1982) found that the RS seemed unable to distinguish between dieting and weight fluctuation. Indeed, the validity of the RS has been questioned due to conflicting results being reported for the weight fluctuation and concern for dieting subscales (Bond, McDowell and Wilkinson 2001). Further, the RS was thought to overestimate restraint in normal weight individuals with a history of being overweight (Lowe 1984). Similarly, in obesity research, the

studies suggested that the RS's properties differed in normal and overweight samples (Ruderman 1986). It appeared that the greater the proportion of overweight people in the sample, "the lower the internal consistency of the scale, the more factors emerge in the analyses, and the greater the proportion of variance accounted for by the items dealing with weight fluctuation" (Ruderman 1986; p. 259). Therefore, the RS may be less reliable and differentially valid for obese compared with normal-weight subjects (Ruderman 1986). Heatherton and colleagues (1988) noted several shortcomings of the scale; namely, deficiencies in its applicability to obese individuals, problems with its factor structure and difficulties completing the scale, due to cultural differences or an individual's apparent lack of concern about weight. Such factor instability across populations was a concern and possibly indicative of differential validity (Allison, Kalinsky and Gorman 1992). Moreover, the irrelevance of some items for different target groups (i.e., general vs. clinical samples) has also been raised as a major concern.

A decade later, in response to concerns about the psychometric adequacy of the RS, two scales were developed for the study of eating behaviours; namely, the Three-Factor Eating Questionnaire (TFEQ) and the Dutch Eating Behaviour Questionnaire (DEBQ). Both questionnaires were developed almost contemporaneously and included a measure of restrained eating.

1.2.4.2 The Dutch Eating Behaviour Questionnaire

Van Strien and colleagues (1986) developed the DEBQ for Assessment of Restrained, Emotional, and External Eating Behaviour. Initially, the item pool for the DEBQ consisted of 100 items drawn from a number of sources including previous research by Van Strien, Frijters, Bergers, and Defares (1986) and Pudel's Latent Obesity

Scale (Pudel 1975). After eliminating items that were factorially complex or having unusual content, the final 33-item questionnaire was developed; it contained three subscales, which as its name implies, measured restrained, emotional and external eating. The restraint subscale of the DEBQ (DEBQ-R) was composed of 10-items.

In terms of reliability, the DEBQ-R performs very well. The scale's internal consistency is quite high, usually $\geq .90$ according to Gorman and Allison (1995). Moreover, the test-retest reliability was found to be .92 over a 2-week span (Allison, Gorman and Kalinsky 1992). Studies by Van Strien et al. (1986) and Wardle (1987) assessing the factor structure of the DEBQ have found an apparently stable factor solution with a highly simple structure of the total DEBQ (three scales, 33 items). Moreover, in both studies, the existence of a restraint scale factor was clearly confirmed and their derived solutions appeared stable across gender and relative weight categories (Van Strien et al. 1986). However, there has been some debate as to whether the DEBQ-R is unidimensional, or whether, it actually measures two aspects of restrained eating: intention to diet and actual dieting success (Ogden 1993).

1.2.4.3 The Three-Factor Eating Questionnaire (TFEQ)

By far the most utilized and studied of the three questionnaires is the TFEQ formulated by Stunkard and Messick (1985). The TFEQ is the measurement instrument of choice for the present study as well. The formal or full name of this questionnaire is the Three-Factor Eating Questionnaire to Measure Dietary Restraint, Disinhibition, and External Eating Behaviour. Stunkard and Messick (1985) utilized questions from Herman and Mack's Restraint Scale and Pudel's Latent Obesity Questionnaire (Meyer and Pudel, 1977) as well as incorporating questions of their own.

The initial 67-item pool was reduced to a final 51 items and three factors. The three factors of eating behaviour measured by the TFEQ questionnaire are cognitive control of eating (factor I – 21 items), disinhibition (factor II – 16 items) and susceptibility to hunger (factor III – 14 items) (Stunkard and Messick 1985; Bond, McDowell and Wilkinson 2001). Stunkard and Messick (1985) further elaborated on the content of the three factors by stipulating: (a) Factor I is the conscious restriction of food intake known as restrained eating; (b) Factor II refers to the disinhibition of cognitive control of eating, or in other words, losing control of dietary restraint; and (c) Factor III is the feeling of hunger and its behavioural consequences.

Stunkard and Messick (1985) in finalizing the questionnaire tested it for reliability and inter-correlation in a study involving 98 subjects. The scale was found to be able to discriminate between groups previously defined as different in restrained eating behaviour. Indeed, the researchers performed several factor analyses of the TFEQ; and although some variation in factor structure was found across samples, the first factor (Cognitive Restraint) was found to be quite robust. These results were virtually replicated in a later study by Hyland and colleagues (1989) when a confirmatory factor analysis was performed on the TFEQ. Hyland and colleagues found that “Factor I” was clearly one of restraint. Ganley (1988) factored the TFEQ responses of 442 women with the same result; that is, a clear restraint factor emerged that was remarkably close to the findings of Stunkard and Messick (1985).

While various factor analytic studies have found the TFEQ-R to have a unidimensional factor structure (Stunkard and Messick 1985; Hyland et al. 1989; Ganley 1988), later studies have shown otherwise. Allison and others (1992) conducted a factor

analysis that showed that the TFEQ-R contained two factors labelled Cognitive Restraint and Behavioural Restraint – a finding that was later rejected on statistical grounds. However, as discussed earlier, Westenhoefer (1991) identified “rigid control” and “flexible control” as two restrained eating/dieting categories within the construct of dietary restraint. In Westenhoefer’s study (1991) the TFEQ was administered to a large number of subjects ($n = 54,525$; $46,132 = \text{female}$; $8,393 = \text{male}$) in a German weight reduction program. He found evidence that rigid control was associated with higher BMI, more frequent and more severe binge eating or overeating, and higher scores for disinhibition or overeating. Flexible control, on the other hand, was associated with lower BMI, less frequent and less severe binge eating or overeating, and a higher probability of successful weight loss during a one-year weight loss program. Follow-up research (McGuire et al. 2001; Shearin et al. 1994; Smith et al. 1999; Williamson et al. 2000) supports Westenhoefer’s findings in part but also introduces a key difference in that some, not all, of these studies found that flexible control was associated with lower BMI. Westenhoefer, Stunkard and Pudel (1999) added items to the original scales and validated two revised scales called the Flexible and Rigid Control dimensions of dietary restraint; their findings support Westenhoefer’s earlier research findings in 1991. Most recently, Stewart, Williamson and White (2002) partially replicated and extended the findings of Westenhoefer and others (1999) that rigid, but not flexible, dieting strategies are associated with eating disorder symptoms such as binge eating. Stewart and colleagues (2002) found a positive relationship between Rigid Control and eating disorder symptoms and BMI, a finding consistent with the results of other studies. However, these researchers, as well as McGuire et al.’s earlier study (2001), failed to find

a negative correlation between Flexible Control and eating disorder symptoms and BMI as originally reported by Westenhoefer (1991) and supported in some subsequent studies (Westenhoefer, Stunkard and Pudel 1999; Smith et al. 1999). Therefore the matter is left open to further investigations.

1.2.4.4 Further Comparative Analyses

As already alluded to, there has been some debate over which measure of dietary restraint is the “best” in any given situation. Given that each measurement tool has its strengths and limitations and, consequently, an assessment as to which is “best” is partly dependent upon the proposed research questions of any given study.

There is consensus that the restraint scales of the TFEQ and DEBQ measure intentions to restrict food intake and actual restraint of food intake. In contrast to the RS, both scales have been shown to have good validity with respect to various measures of food intake (Van Strien 1999; Wardle and Beales 1987; Laessle et al. 1989b). Wardle (1980) also found the RS to have high relationships with measures of binge eating, meaning that the RS indeed tends to select dieters having a high tendency toward disinhibition of restraint (Van Strien 1999).

The construct validity of the RS and the restraint components of the TFEQ and the DEBQ were investigated by Laessle and others (1989b), by relating these scales to self-reported mean daily caloric intake and to other measures associated with disordered eating and weight consciousness. A high score on the RS was closely related to consequences of unsuccessful dieting such as weight fluctuations and disinhibited eating, but not to successful restriction in everyday life. High scores on the restraint components of the TFEQ and DEBQ were more representative of successful dieting behaviour and

best reflect restraint, without necessarily including restraint as it occurs with alternating periods of overeating (Laessle et al. 1989b).

In an investigation involving 901 undergraduate students (69% female; 31% male) designed to compare a variety of parameters in the RS, TFEQ, and DEBQ, Allison and colleagues (1992) found that, compared to the RS and DEBQ-R, the TFEQ-R had good internal consistency (.904) and good test-re-test reliability ($n = 34$; .91). Further, of the three scales, the TFEQ-R had the best discriminant validity with respect to social desirability and was least susceptible to dissimulation; that is, the responses were unlikely to be “faked” in order to appear more acceptable. In the final analysis, these researchers suggested that if there was any concern that study participants might attempt to “fake” their answers or appear in a more socially desirable light, then the TFEQ was the appropriate measurement instrument as it was psychometrically sound, yet less reactive than the alternatives.

More recently, Van Strien (1999) proposed that individuals exhibiting high restraint are not a homogenous group; rather, high restraint individuals could be placed in one of two sub-groups – successful dieters or unsuccessful dieters. Successful dieters have high restraint and a low susceptibility toward failure (i.e., episodes of disinhibition), whereas unsuccessful dieters are prone to failure and periods of overeating. The TFEQ is able to distinguish between these two subgroups by including measurements of disinhibition and hunger subscales, whereas the RS, on the other hand, lacks this added capability.

It is important for researchers to be cognizant of both the differing capabilities and the strengths and limitations of each of the measurement tools – the evaluation and

refinement of which is constantly evolving. This baseline understanding of the various instruments is necessary in order to ascertain which tool will best assist in addressing the research questions of any proposed study. The focus of this research study involves the *effects* of attempting to restrict food intake (i.e. physiological effects to the neuroendocrine system); the measurement of which is only possible with the TFEQ or DBEQ. For this reason, as well as research findings as to the “robustness” of the restraint subscale of the TFEQ, its non-susceptibility to dissimulation, its relatively good internal consistency and test-re-test scores, its best overall discriminant validity with respect to social desirability – all of which have been canvassed in the above – the restraint subscale of the TFEQ was selected to quantify restraint in this study.

1.3 Characteristics of Women with High Restraint

Over the years, women who are considered restrained eaters have been studied in an attempt to reveal any psychological, physiological or additional behavioural (aside from restraint) traits that may further characterize them. Studies assessing energy intake and expenditure, macronutrient distribution, body mass index (BMI), and psychosocial characteristics are described below, with a view to identifying those factors that may require consideration in the analysis of present and future investigations.

1.3.1. Energy Intake and Expenditure

Laessle and colleagues (1989a) attempted to study restrained eaters beyond the laboratory setting, and found restrained eaters to have lower reported energy intakes in comparison to unrestrained eaters – a finding consistent with those of many later studies

(Tuschl et al. 1990a; Schweiger et al. 1992; Klesges, Isbell and Klesges et al. 1992; Janelle and Barr 1995; McLean and Barr 2003), but not all (Barr, Prior and Vigna 1994; Barr, Janelle and Prior 1994). Laessle et al. (1989a) used the restraint scale of the TFEQ (TFEQ-R) to assess dietary restraint in 60 young women, and to divide them into restrained and unrestrained eaters. Seven-day food records showed that the high restraint group ate approximately 400 kcal per day fewer than the low-restraint group (1956 kcal vs. 2338 kcal respectively). Similarly, Klesges Isbell and Klesges (1992) found that high-restrained eaters ingested fewer calories per pound of bodyweight compared to low-restrained individuals (10.27 kcal/lb vs. 12.82 kcal/lb respectively). This study, however, included overweight individuals who may be more likely to be restrained eaters (Poehlman, Viers & Detzer 1990) and to underreport (Braam et al. 1998) compared to normal-weight individuals.

Although underreporting nutrient intake is common (Asbeck et al. 2002), and there is speculation that restrained eaters may be even more likely to underreport intake, this has not been confirmed. Poehlman, Viers & Detzer (1990) assessed dietary restraint, using the TFEQ-R, in 44 non-obese females ranging in age from 18-39 years, and found an association between high levels of dietary restraint and lower resting metabolic rate (RMR), however, this relationship did not reach significance ($r = -0.29$; $p < 0.07$).

Although the researchers reported that dietary restraint was not significantly related to total energy intake (EI), a comparison of energy intake to expenditure in restrained vs. unrestrained individuals was not reported. It is therefore difficult, from the given data, to determine if underreporting occurred or if restrained eaters were in fact meeting a lower energy requirement.

Tuschl et al. (1990a) used the doubly-labelled water method to measure average daily energy expenditure (EE) in 23 normal-weight women, aged 18-30 years, who were classified as restrained or unrestrained eaters as assessed by the TFEQ-R. The researchers attempted to clarify whether or not restrained eaters underestimate their reported intake, and found that EE, along with intake, was in fact lower than that of unrestrained eaters. In the restrained eaters, self-reported average energy intake was approximately 250 kcal/day less, and EE was approximately 300 kcal/day less, in comparison to the unrestrained eaters. The authors suggested that the lower reported intake of restrained eaters was meeting their lower energy requirements, and underreporting was no more common in this group than in unrestrained individuals. However, there are possibly some methodological flaws in Tuschl and colleagues' study (1990a) that may have lead to such results. Firstly, a 1-tailed t-test was used to show significant differences in energy intake between the restrained and unrestrained group. However, if a 2-tailed t-test had been used to analyze the data, significance would not have been achieved. Thus, the study lacked adequate power to detect a difference between groups. Secondly, the study did not report at what phase of the menstrual cycle the energy expenditure measurements were taken in each of the groups. This last concern could potentially be a confounding factor due to numerous reports (Schweiger et al. 1992; Barr, Prior and Vigna; Barr, Janelle and Prior 1994; Lebenstedt, Platte and Pirke 1999; McLean and Barr 2003) of an association between restrained eaters and sub-clinical menstrual disturbances (discussed later). In restrained eaters, these menstrual cycle disturbances may include anovulation and shortened or disrupted luteal phases. Such luteal phase defects could inhibit naturally occurring increases in body temperature, and therefore energy expenditure, that occur

during the luteal phase of the menstrual cycle (Barr, Janelle and Prior, 1995). If EE is higher in the luteal phase, and this variable was neither considered nor controlled for in this study, the potential does exist that more subjects in the unrestrained group were in this “higher energy expenditure” phase of their cycle and therefore expending more energy as reported from the doubly-labelled water technique results. Further, due to shortened luteal phases reported in restrained eaters, individuals in this group would theoretically be more likely to be in the follicular phase (i.e., the phase of lower energy expenditure), which would automatically be extended in those with a shortened luteal phase. This would result in lower average EE across the whole menstrual cycle, compared to those with normal luteal phase lengths (i.e., unrestrained eaters). Alternatively, this could support Tuschl and colleagues’ (1990a) findings of lower EE in restrained eaters, if measurements were taken across one entire menstrual cycle in both groups.

Platte et al. (1996) also hypothesized that restrained eaters may have reduced RMR and/or diet induced thermogenesis (DIT), which may or may not be caused by weight cycling. The researchers compared energy expenditure (RMR and DIT), by indirect calorimetry using a ventilated hood system, in 12 women with high vs. 12 women with low restraint scores who were similar in age, height, lean body mass and body weight. The researchers found a significantly lower RMR in the high restraint group. In a second study, 12 weight cycling and 12 weight stable restrained eaters, who were well-matched for age, height, lean body mass and weight, were compared, and did not differ with regard to RMR and DIT. Interestingly, the measured RMR in unrestrained eaters was indistinguishable from that predicted on the basis of height and weight using the

Harris Benedict equation (Harris and Benedict 1919), but not so for the restrained group. The authors suggest that lowered RMR in restrained eaters may be a predisposing condition, to which restrained eating is a behavioural adaptation to prevent weight gain or becoming overweight. However, Platte et al.'s study contains similar limitations to that of Tuschl and colleagues' (1990a) as the small sample size in this study, as well as the absence of data indicating what phase of the menstrual cycle that RMR measurements were taken, are a concern.

A study with a larger sample size to duplicate the findings of Platte et al. (1996) and Tuschl et al. (1990a) appears warranted, to further investigate the energy requirements of restrained vs. unrestrained eaters. Future investigations should use a larger sample, compare groups of individuals in the same phase of their menstrual cycle and control for or assess underreporting, to provide more compelling evidence of a lower energy requirement in restrained eaters.

Other studies assessing differences in energy intake and/or underreporting in restrained vs. unrestrained eaters have had mixed results. Poppitt et al. (1997) investigated the degree of underreporting in 33 women (18 obese and 15 non-obese) recruited to a long-stay metabolic facility where *ad libitum* food intake was covertly measured for 24 hours. The study found similar levels of underreporting in obese and non-obese participants, as well as restrained and unrestrained eaters as measured by the TFEQ-R. Similarly, a number of studies have observed no association between TFEQ-R score and energy intake in normal-weight women; that is, lower energy intakes were not associated with restraint in these women (Lindroos et al. 1997; Barr, Prior and Vigna 1994; Barr, Janelle and Prior 1994). Researchers Lafay and colleagues (1997) studying

the determinants and nature of underreporting in a free-living population in France found an overall high prevalence of dietary underreporting, in 1030 weight-stable subjects (501 women and 529 men), with an even higher prevalence in obese individuals.

Underreporting was also associated with cognitive dietary restraint and attempting to diet, independent of weight status. Although restraint was only assessed by one question: “do you have to reduce food intake in order to maintain your bodyweight”, and not a multi-question validated scale, the findings do support underreporting in those concerned with food intake and bodyweight maintenance.

More recently, Asbeck and colleagues (2002) studied resting energy expenditure (REE; indirect calorimetry) in 83 young adults (20-38 years, 55 women, 28 men), who were assessed under weight-stable conditions with a 7-day dietary record and the TFEQ-R. A high prevalence (37%) of severe underreporting was seen in all subjects, with a higher prevalence in women than men (49% vs. 14.3%, respectively). Underreporting subjects had a reduced EI but there were no significant differences in nutritional status (BMI, fat mass and fat-free mass), EE and the proportion of energy from macronutrients between normal and underreporting subjects. However, high restraint was associated with a higher degree of underreporting in both men and women.

In summary, there does not appear to be unequivocal evidence that restrained eaters have a lower energy expenditure or are more likely to underreport, compared to unrestrained eaters. However, if chronic lower energy intakes of restrained eaters are reported in association with menstrual cycle disturbances, this may be of significance to those studies investigating the association of such disturbances with bone health, such as in the current research study. A more detailed discussion of the consequences of energy

intakes inadequate in meeting the exercise energy expenditure (referred to as “the energy drain” hypothesis - see section 1.2. of literature review) and its association with menstrual irregularities will appear in following sections (Warren 1980; Loucks, Verdun and Heath 1998).

1.3.2. Macronutrient Intake

Qualitatively, differences in macronutrient distribution have been reported in some, but not all studies investigating dietary habits and food choices in restrained eaters. In Laessle and colleagues’ study (1989a) previously mentioned, researchers also found that restrained eaters consumed a higher proportion of protein, and had a tendency to avoid high fat/carbohydrate calorie dense foods compared to unrestrained eaters. Other studies have reported lower fat intakes or avoidance of high fat foods in those with higher levels of restraint (Tuschl et al. 1990a,b; McLean, Barr and Prior 2001a). In 24-hour food records, McLean, Barr and Prior (2001a) reported similar intakes of carbohydrates between high and low restraint groups, and higher protein and lower fat intakes in the high restraint group. However, data collected from the same group, using three-day food records, revealed trends toward higher protein and lower fat, but differences in macronutrient intakes between groups did not reach significance.

The implication of higher protein intakes with regard to bone health may be of some relevance if detected, as some animal (Brand et al. 1999) and human (Metz, Anderson and Gallagher 1993) studies have associated high protein intakes with adverse effects on bone, due to increases in urinary calcium excretion. In contrast however, several studies demonstrate reduced bone density and increased rates of bone loss in individuals habitually consuming low protein diets (Freudenheim, Johnson and Smith

1986; Hannan et al. 2000), and a positive relationship between protein intake and bone mass (Cooper et al. 1996). In addition, Kerstetter, O'Brien and Insogna (2003) reported that secondary hyperparathyroidism was induced by low protein diets, which was attributed to a reduction in intestinal calcium absorption (as assessed by dual stable calcium isotopes). Therefore, although the long-term consequences of low protein-induced changes in calcium metabolism are not known, they could be detrimental to skeletal health. In short, it appears that maintaining adequate protein is essential to bone health, and bone health may only be compromised if protein intake is excessively high over long periods, which did not appear to be the case in McLean, Barr and Prior's (2001a) or Laessle et al.'s (1989a) findings.

Although there appears to be minor differences in nutrient intakes between individuals with high and low restraint, many other studies (Barr, Prior and Vigna 1994; Barr, Janelle and Prior 1994; Janelle and Barr 1995) have not detected any differences in macronutrient composition between these groups, although high activity levels in one of these studies (Barr, Janelle and Prior 1994), the authors noted, may have increased carbohydrate intake in all subjects, which may have overridden any potential associations between macronutrient intake and restraint. In summary, although there have been some differences reported in nutrient intakes between individuals with high and low restraint, these do not appear to be significant with regard to their effects to bone.

1.3.3. Body Mass Index

Some studies have also found higher BMI values in normal-weight women with high restraint (Tuschl et al. 1990a,b). However, most studies report similar values for BMI between high and low restraint groups or slight non-significant elevations in the

high restraint group (Laessle et al. 1989a; Schweiger et al. 1992; Lautenbacher et al. 1992; Barr, Prior and Vigna 1994; Platte et al. 1996; McLean, Barr and Prior 2001a). When dietary restraint is separated into two dietary strategies namely “rigid control” and “flexible control” (Westenhoefer 1991; see assessment section), it has been reported that higher BMI may be more associated with rigid control (Westenhoefer, Stunkard and Pudel 1999; Smith et al. 1999), however more recently this association has not been supported (McGuire et al. 2001; Stewart, Williamson and White 2002). Although there appears to be some reports of higher BMI values in restrained eaters, this fact would only be of concern if the opposite findings were found (i.e. low BMI in restrained eaters) as *low* BMI has been associated with lower BMD values. For example, Teegarden et al. (1998) found that weight, height, and lean mass were correlated with bone mineral measures at every site in a study of 215 women aged 18-31 years. Similarly, Sahin et al. (2003) found that lean mass correlated with BMD at all sites measured in post-menopausal women. In short, if higher BMI is detected in restrained eaters, this would not be implicated in negative impacts to bone; moreover, it may be protective.

1.3.4. Other Behavioural and Psychological Characteristics

Other behavioural and psychological characteristics that have been the focus of studies of restrained eaters include: body size perception, body dissatisfaction, and other personality traits. King, Polivy and Herman (1991) investigated the cognitive aspects of restraint in female restrained and unrestrained college students and in female obese and eating disorder (ED) patients (all obese and ED subjects were restrained eaters). Subjects read an essay that included various descriptions of another person and were later asked to recall the essay as completely as possible. As predicted, restrained eaters recalled more

weight- and food-related items than other appearance items compared to the unrestrained eaters. The authors suggest that a focus on weight and food is a basic organizing principle for restrained eaters, and takes precedence over other equally valid appearance-related information. In addition, individuals with high restraint scores were more likely to mention food- and weight-related words when describing themselves or as part of their lists of favourite activities and major concerns.

Lautenbacher et al. (1992) reported that restrained eaters were more dissatisfied with their bodies and were more uncertain about their body size in two of three perceptual tasks measuring body size perception. Restrained eaters did not show any systematic under- or overestimation, but did show less perceptual accuracy. Results of a study by Davis et al. (1993) also indicated that certain psychological variables such as emotional reactivity, body dissatisfaction and a greater focus on their bodies were strongly and positively correlated with restraint. This study also found that weight concerns and dieting behaviour in young women were influenced more by the size of their skeletal structure, which cannot be altered by diet or exercise, than the degree of adiposity (Davis et al. 1993). However, more recently, Jansen, Huygens and Tenney (1998) conducted an experiment where restrained eaters were presented with body shape and weight words both supraliminally and subliminally during a computerized Stroop task (word association test). Contrary to the hypothesis that restrained subjects would show an attentional bias for body shape and weight words (stimuli) during the automatic stage of information processing, they did not in fact show distortions in the processing of these stimuli. The authors point out that the absence of cognitive distortions in the processing of body shape and weight information may demonstrate a qualitative

difference between normal restrained eaters and subjects with eating disorders of clinical severity (Jansen, Huygens and Tenney 1998). However, parallels have been noted between counter-regulation of restrained eaters and bingeing associated with eating disorders (Polivy and Herman 1985). These similarities include a predominantly female population, preoccupation with weight, appearance and eating, as well as with perfectionism.

In a study (Gendall et al. 1998) investigating personality traits and restraint, researchers measured eating behaviour with the TFEQ and character traits with the Temperament and Character Inventory (TCI). They found that self-transcendence was positively correlated with restraint and self-directedness was negatively correlated with total TFEQ score, disinhibition, and susceptibility to hunger. Their findings suggest that individuals with low self-directedness and high self-transcendence may be more reactive and susceptible to societal pressures and the ideology of slenderness. This may have implications to bone health if this “slenderness” ideology actually translates into a measurable physical characteristic (i.e. a slimmer body type or lower BMI), which has been associated with lower BMD as described previously. In short, this fact reveals that certain personality characteristics may make some women more susceptible to engage in restrained eating patterns in order to fulfill the slenderness ideology imposed by a society, with possible subsequent negative impacts to bone.

1.3.5 Summary

Although there may be some reported differences in reported energy intake, energy expenditure, macronutrient composition and various personality characteristics, BMI may be the most critical variable to control for in the current research study. There

is a well-known association between BMI and BMD, and it is the possible effect of restraint on BMD, and therefore stress fracture risk, which is central to the present research investigation. However, menstrual cycle disturbances and cortisol excretion levels are fundamental differences between restrained and unrestrained eaters surrounding the present investigation, and will be discussed in detail in following sections.

1.4 Reproductive and Menstrual Cycle Disturbances and the Neuroendocrine System

Early studies in animals and humans have established an association between stress and reproduction. Cumulative data over recent years have also provided consistent biochemical evidence that reproductive and menstrual function are disrupted by psychogenic or physiologic stress, which activates central neuroregulatory networks, and results in disruption of hormonal patterns required for normal reproduction and menstrual cyclicity. Disordered eating behaviours may act as a stressor and thus play a role in corresponding reproductive and menstrual cycle disturbances.

In the following sections an overview of the physiology of the neuroendocrine system and its relationship to reproduction and menstrual cycle function is provided. Investigations describing the proposed mechanisms by which stress may disrupt reproduction and menstrual function will then be presented.

1.4.1 The Hypothalamic-Pituitary-Ovarian Axis

The neuroendocrine system pertains to the anatomical and functional relationships between the nervous system and the neuroendocrine apparatus (Berne and Levy 1988).

The hypothalamic-pituitary-ovarian (HPO) axis (Figure 1) is a component of the neuroendocrine system that neurally and hormonally controls reproductive function. *Luteinizing hormone (LH)* and *follicle-stimulating hormone (FSH)* are gonadotropins synthesized in, and secreted from the anterior pituitary, whose function, in part, is to regulate reproductive processes and sex steroid secretion of the gonads (ovaries in women). The secretion of FSH and LH by the anterior pituitary is stimulated primarily by a single hypothalamic hormone; that is, *gonadotropin-releasing hormone (GnRH)*. In turn, FSH and LH stimulate the production of *estrogen* and *progesterone* and the monthly release of the egg (ovum) from the ovaries (Berne and Levy 1988). Disruption of the HPO axis has been implicated in a multitude of reproductive and menstrual cycle disturbances (Berga 1996), generally stemming from the suppression of GnRH release. Research investigating such disruptions will be discussed in greater detail in following sections.

1.4.2 The Hypothalamic-Pituitary-Adrenal Axis

The hypothalamic-pituitary-adrenal (HPA) axis is another component of the neuroendocrine system that can be activated by physiological or psychological stress (Berga 1996). The presence of stressors can stimulate the release of *corticotropin-releasing hormone (CRH)* from the hypothalamus, which in turn stimulates the pituitary to release *adrenocorticotrophic hormone (ACTH)* (Berne and Levy 1988). The release of ACTH then stimulates the adrenal gland to produce cortisol. Urinary and serum cortisol levels act as peripheral markers of stress, and when they are elevated, are indicative of the activation of the HPA axis. Activation of the HPA axis can negatively impact the functioning of the HPO axis (Figure 2) [Berga 1996].

Fig 1. Hypothalamic-pituitary-ovarian axis.

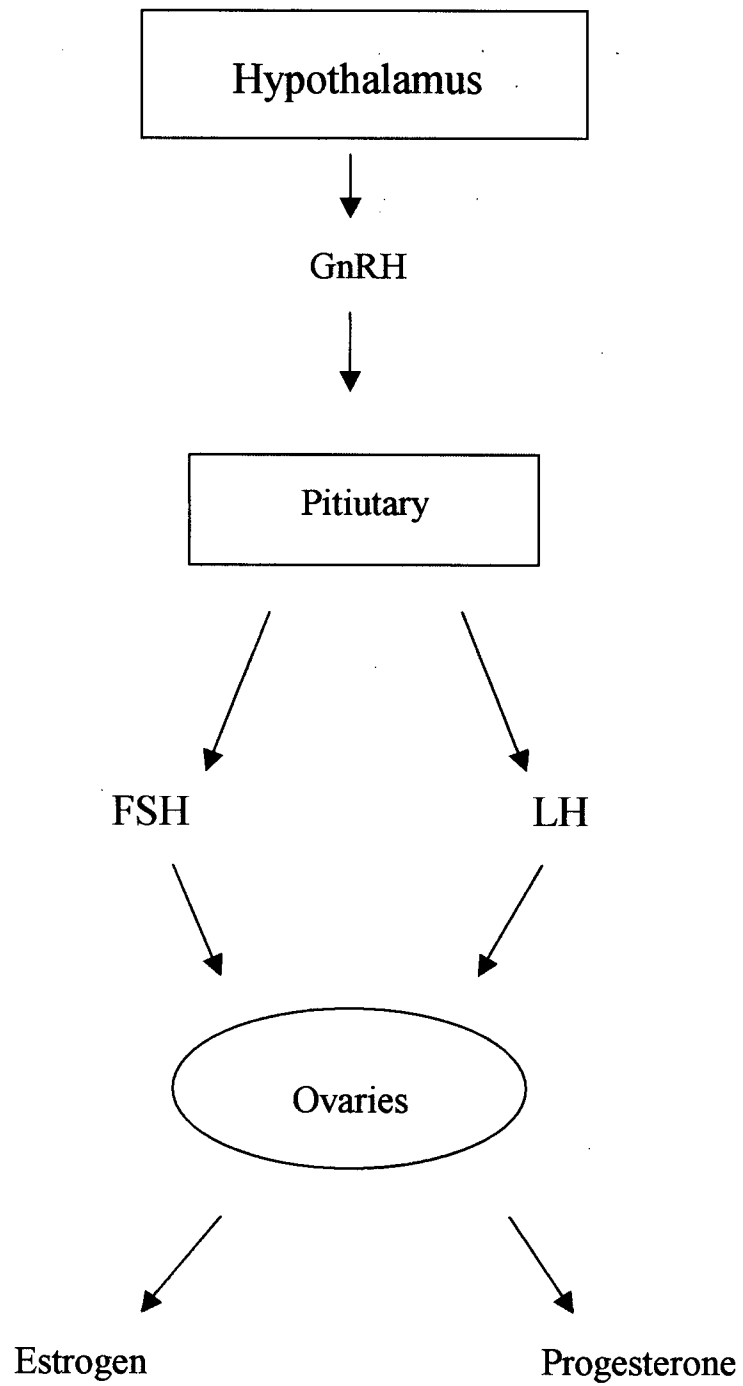
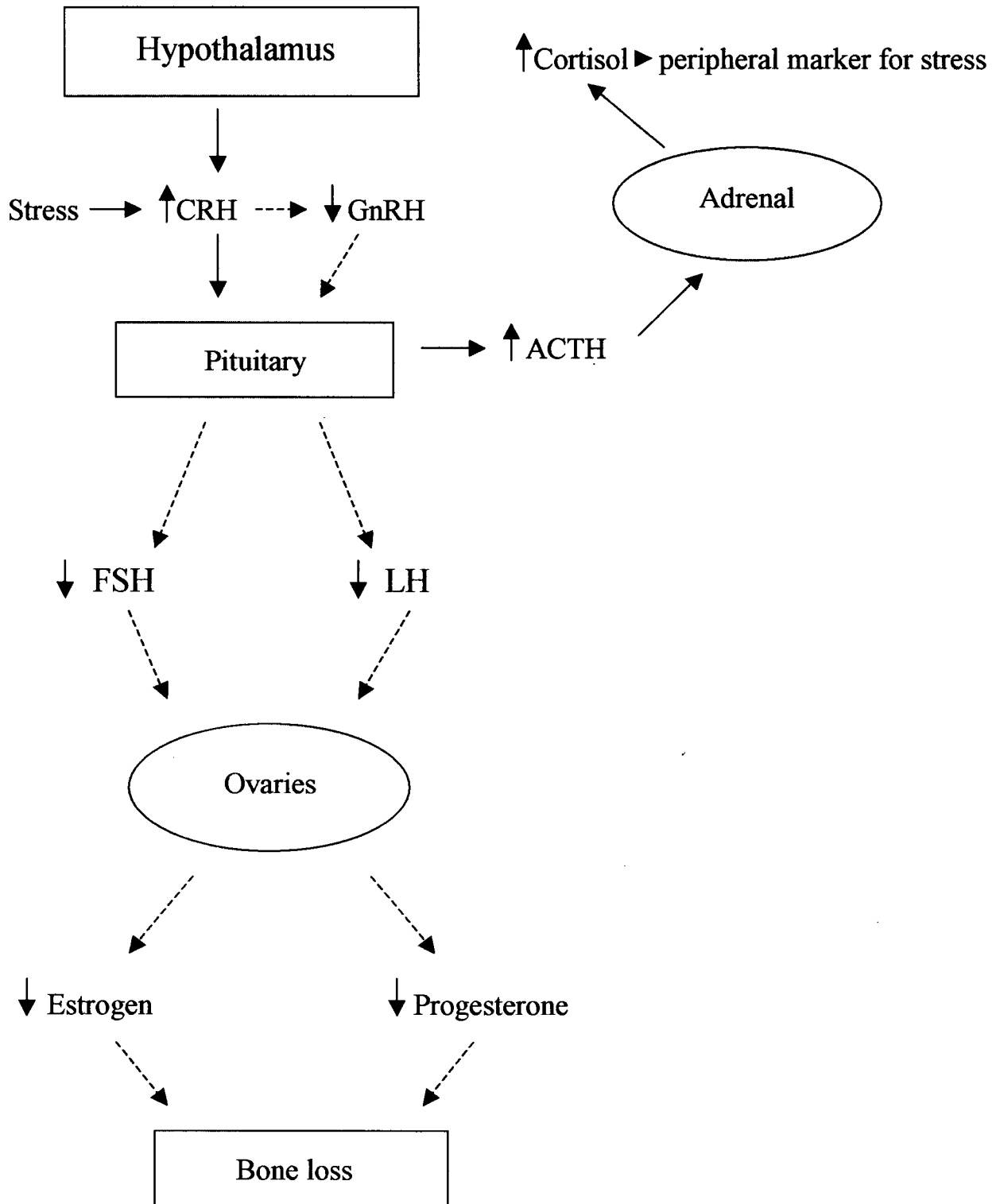


Fig 2. Hypothalamic-pituitary-ovarian and - adrenal axes. *Dashed line* represents suppression of HPO axis as a result of *solid line* HPA axis activation.



1.4.3 The HPO Axis and Stress-induced Activation of the HPA Axis

Animal (Rivier and Vale 1984; Rivier, Rivier and Vale 1986; Petraglia, Vale and Rivier 1986) and human studies (Barbarino et al. 1989; Loucks et al. 1989; Berga, Daniels and Giles 1997) strongly support the concept that activation of the HPA axis by psychogenic or metabolic challenge plays a causal role in reducing GnRH drive resulting in reproductive and menstrual cycle disturbances. Gonadal function relies on the intricately interdependent relationship between the sex organs and the hypothalamic-pituitary axis. The GnRH pulse generator is modulated by many factors, including the concomitant release of CRH from the hypothalamus during stress, which in turn suppresses GnRH secretion. CRH can also activate the hypothalamic release of beta-endorphin, an opioid peptide that has also been shown to inhibit GnRH pulsatility (Petraglia, Vale and Rivier 1986; Barbarino et al. 1989). Although the exact mechanisms of these pathways are not fully understood, it appears that stress can disrupt the GnRH pulse generator by CRH and beta-endorphin release via HPA axis activation (Petraglia, Vale and Rivier 1986; Barbarino et al. 1989). Suppressed GnRH will in turn reduce the pituitary production of LH and FSH resulting in lower levels of estrogen and progesterone production in the ovaries. Serum and urinary cortisol levels are commonly used as biomarkers of HPA axis activation, as their elevation frequently indicates the presence of stress in an individual (Berga et al. 2000). Therefore, an individual with elevated cortisol levels may be experiencing stress that has activated the HPA axis, with a concomitant suppression of GnRH resulting in reproductive or menstrual cycle disturbances.

1.4.4 Studies Investigating Reproduction, the Menstrual Cycle and the HPA Axis

Both animal and human studies, as referenced above, show that exposure to stress is accompanied by the disruption of reproductive function. In early animal experiments stress was shown to increase reproductive disturbances (Krulich et al. 1974; Blake 1975). Researchers hypothesized that these disturbances were possibly due to the inhibitory effect of CRH on the hypothalamic (GnRH release), and hence, pituitary secretions (LH release) required for normal reproductive function (Rivier and Vale 1984; Rivier, Rivier and Vale 1986).

Rivier and Vale (1984) found that CRH injected intracerebroventricularly into intact and estrogen-treated ovariectomized rats caused a rapid and prolonged dose-related inhibition of LH secretion. Although the response to peripheral injections was absent in lower doses, CRH blocked the proestrous LH surge in 50% of the rats in higher doses (Rivier and Vale 1984). The same group (Rivier, Rivier and Vale 1986) also exposed castrated male rats to stress through intermittent electroshocks, and again found inhibition of LH release. In addition, this study found that the administration of a CRH antagonist reversed the inhibitory effect of stress on LH. The authors concluded that endogenous CRH at least partially mediates stress-induced inhibition of LH release in the rat, and further suggested that the most probable hypothesis is that CRH acts within the brain to inhibit GnRH secretion into portal circulation (Rivier, Rivier and Vale 1986).

Researchers have also hypothesized that CRH can also activate the hypothalamic release of beta-endorphin, an opioid peptide that has also been shown to inhibit GnRH pulsatility (Petraglia, Vale and Rivier 1986; Barbarino et al. 1989). Petraglia and colleagues (Petraglia, Vale and Rivier 1986) found that a beta-endorphin antagonist

reversed the CRH-induced decrease in LH concentrations in castrated male rats.

Therefore, stress can possibly disrupt the GnRH pulse generator by CRH release and/or as well as subsequent beta-endorphin release via HPA activation (Petraglia, Vale and Rivier 1986).

Olster and Ferin (1987) also demonstrated that exogenous CRH administration results in inhibition of LH and FSH secretion in ovariectomized rhesus monkeys. These results are consistent with the hypothesis that elevated CRH levels could contribute to decreased LH and FSH secretion and, thus, disruption of reproduction function under conditions of stress in non-human primates.

Although animal studies have shown that CRH decreases plasma LH, possibly by inhibiting hypothalamic release of GnRH (Rivier and Vale, 1984; Petraglia, Vale and Rivier 1986; Oster and Ferin 1987), Barbarino and colleagues (1989) were among the first researchers to investigate whether CRH and opioid pathways are involved in suppressed gonadotropin secretion in humans. Fifteen normal-weight women, aged 19-30 years, were studied during the midluteal phase of their menstrual cycle. CRH was infused into all 15 women, with five women also receiving GnRH stimulation. CRH induced a significant decrease in plasma LH (62%) and FSH (36%) in all women. CRH infusion did not alter the gonadotropin response to GnRH, suggesting that inhibition may be occurring at a higher level, presumably inhibiting GnRH secretion. The researchers also infused naloxone, an opioid antagonist, plus CRH in the 10 women who had received CRH alone during the midluteal phase of a different cycle. The addition of naloxone to CRH (5 women) reversed the LH and FSH inhibition when naloxone was started one hour after the start of the CRH infusion. However, when naloxone was started one hour

before CRH infusion (5 women), plasma LH and FSH concentrations did not change. Therefore, it appears that there is a putative CRH-opioid interaction on GnRH secretion supported by the ability of naloxone to reverse LH inhibition when the antagonist is infused after CRH has stimulated beta-endorphin release. These results demonstrate that in normal-menstruating women during the midluteal phase of the menstrual cycle, CRH inhibits the secretion of both LH and FSH. The authors suggested that the disruptive effect of stress on reproductive function in the women could be, at least in part, dependent on decreased gonadotropin secretion induced by elevated endogenous CRH levels. Further, plasma cortisol increased similarly in both the CRH and CRH plus naloxone infusions. The authors also suggested that CRH-induced inhibition of gonadotropin secretion is primarily mediated by endogenous opioid peptides, and this effect is not dependent on glucocorticoid (cortisol) levels. Explanation for this last finding, however, was addressed in subsequent studies (Berga et al. 1989; Berga, Daniels and Giles 1997) who reported that secretory patterns of cortisol are less robust than the profound inhibition of GnRH and LH, and this has been attributed to the feedback mechanisms (of cortisol) that blunt the pituitary-adrenal response to sustained elevations in CRH drive (Jacobson and Sapolsky 1991). Theoretically, inhibitory mechanisms restraining cortisol secretion would be important from a homeostatic perspective, as cortisol is a potent metabolic and neurotoxic hormone (Jacobson and Sapolsky 1991). Although modest increases in cortisol may underestimate CRH drive due to these inhibitory mechanisms, cortisol levels may still be the best peripheral marker of stress (Berga et al. 2000). Therefore, increases in cortisol may not be implicated in the initial

activation of the HPA axis, and resultant HPO axis dysfunction, but are a by-product of the stress response, functioning as negative feedback to CRH release.

The data surrounding stress and reproductive dysfunction appears to be clear with regard to decreased FSH and LH as being a consequence of GnRH suppression, however there has been no consensus on the exact mechanisms by which GnRH pulsatility is disrupted. Further investigations into the HPO and HPA axes have been conducted primarily on women with infertility as well as athletes with menstrual disorders. The following sections focus on menstrual cycle irregularities (MCI), mainly in athletes, and set out additional research implicating the activation of the HPA as the etiologic pathway causing varying degrees of MCI.

1.5 Menstrual Cycle Irregularities

1.5.1 The Menstrual Cycle

In order to fully appreciate the continuum of menstrual dysfunction, an understanding of hormonal events and normal menstrual physiology is necessary. The menstrual cycle is regulated by the complex interaction of pituitary hormones (luteinizing hormone and follicle-stimulating hormone), and ovarian sex hormones (estradiol and progesterone). The menstrual cycle is divided into three sequential phases. The *follicular phase* begins with the onset of menstrual bleeding and is of variable length; the *ovulatory phase* lasts 1-3 days, and culminates in ovulation; the *luteal phase* usually has a constant length of 13-14 days and terminates with the onset of menstrual bleeding. The overall menstrual cycle has an average duration of 21-35 days – the variability in length generally dependent upon the length of the follicular phase (Berne and Levy 1988).

Ovarian cyclicity depends directly on the appropriate level of secretion of hypothalamic GnRH, which, when reduced, will cause a significant decline in LH and FSH, thereby compromising ovulation (Berga 1996). Decrements in the GnRH-LH/FSH drive exist along a continuum and may have daily as well as inter-person variability. Due to this variability in GnRH secretion, ovarian compromise exists as a spectrum and may manifest as MCI. These MCI occur along a continuum of severity from normal ovulatory cycles to luteal insufficiency and short luteal phases in asymptomatic cycles of regular length, to menstrual irregularity (oligomenorrhea), anovulation, and in the most extreme disturbance, amenorrhea (Broocks et al. 1990; DeSouza et al. 1998). Amenorrhea has been variably defined as the absence of three or more consecutive menstrual cycles (Shangold 1990), or less than three menstrual periods per year, no more than one menstrual period in the last 10 months (Loucks and Horvath 1985) or no menstrual period for six months (National Institute of Health 2002). Irregular cycling or *oligomenorrhea*, has been defined as menstruation every 45-90 days (National Institute of Health 2002). *Luteal phase defects (LPD)* include shortened luteal phases and disruption of LH pulses (De Souza 2003). The health consequences of MCI, whether overtly absent or irregular, or nonsymptomatic as seen in LPD, are of significant concern, particularly to bone (for a review, see Bennell et al. 1997).

1.5.2 Hypothalamic Amenorrhea

The most common cause of reduced GnRH drive is functional, i.e. not due to organic causes, and theoretically reversible. When the disruption of GnRH drive is sufficient to cause anovulation and amenorrhea, and is associated with environmental variables such as excessive exercise and/or subsequent energy availability, weight loss, or

psychogenic/emotional stressors, this condition is often referred to as functional hypothalamic amenorrhea (FHA). FHA is therefore a common and potentially reversible form of ovary quiescence in which psychophysiologic and behavioural responses to life stressors can activate central neuroregulatory networks that disrupt pulsatile hypothalamic release of GnRH (Berga 1996). FHA presents as a clinical syndrome characterized by hypoestrogenism, low LH and low or normal FSH levels (Warren and Fried 2001; Berga et al. 2003). Generally there are two origins associated with FHA – eating disorders and athletics. FHA frequently develops in women with low body weight caused by excessive exercise or disordered eating. In the case of many female athletes, a combination of both disordered eating and intense athletic training is occurring.

Psychogenic amenorrhea has also been reported as lack of menses that occurs from a psychologic origin and usually excludes intense exercise or eating disorders as possible causes for anovulation (Facchinetti et al. 1993). This observation is significant to the current research study, as there is a possibility of an association between MCI and psychological stress that originate from the cognitive processes in highly restrained eaters.

1.5.3 Luteal Phase Defects

As previously discussed MCI occur along a continuum of severity, and generally affect the luteal phase first. Luteal phase defects (LPD) include shortened luteal phases (10 d or less) and/or and disruption of amplitude and/or frequency of LH pulses (De Souza 2003). LPD are defined by low-peak progesterone levels and are often, but not always, a precursor to anovulation. LPD and anovulation can be present in a menstrual cycle of normal total length and pattern, and therefore go unnoticed by the individual.

The most important feature of the luteal phase of a menstrual cycle is the formation of a *corpus luteum* that develops from the cellular wall of a postovulatory follicle in response to a surge of LH (McNeely and Soules 1988). In turn, the most important function of the formed *corpus luteum* is production of the ovarian steroid progesterone, which is essential for the secretory transformation of the endometrium and maintenance of early pregnancy (McNeely and Soules, 1988). As will be further discussed, progesterone is also an important sex steroid required for the maintenance of bone health (Prior 1990).

Similar to FHA, LPD are presumably a result of disrupted GnRH release from the hypothalamus, with subsequent suppression of LH (De Souza 2003). Progression from the initial LPD may result in anovulation despite normal estrogen levels, and eventually amenorrhea with hypoestrogenism (Shangold and Levine 1982). Further, in a recent non-human primate study Williams et al. (2001) observed LPD in the cycles immediately preceding a transition from ovulatory cycles to amenorrhea, in the eight monkeys studied. In the cycle before the transition to amenorrhea, there was a significant reduction in serum progesterone (34%) and LH, consistent with menstrual cycles classified as LPD; three monkeys were also anovulatory. The authors suggested that these findings demonstrate a progression of LPD to amenorrhea, which also appeared to occur during the transition back to ovulatory cycles from amenorrhea when supplemental calories were introduced (Williams et al. 2001).

1.5.4 Studies Investigating FHA and LPD

Although research defining MCI (Sherman and Korenman 1974) and studying exercise and MCI (Shangold et al. 1979) had been occurring for over a decade, Bullen and colleagues (1985) were among the first researchers to prospectively investigate

whether strenuous exercise would induce menstrual disorders. Twenty-eight initially untrained college women with documented ovulation and luteal adequacy were introduced to daily strenuous exercise over a period of two menstrual cycles (8 weeks). To ascertain the influence, if any, that weight loss might exert, the researchers randomly assigned the subjects to weight-loss and weight-maintenance groups. The normalcy of the menstrual cycles during the period of exercise was judged independently according to clinical and hormonal criteria, the latter comprising serial measurements of gonadotropin and sex-steroid excretion. Only four subjects (three in the weight-maintenance group) had a normal menstrual cycle during training. In the weight-loss group, the number of women who had luteal abnormalities, as compared with those who lost the surge in luteinizing hormone, altered significantly over time, the latter occurring more frequently ($P < 0.01$) as training progressed. Within six months of termination of the study, all subjects were again experiencing normal menstrual cycles. An important feature of this study protocol was that no allowance was made for adaptation to exercise load – the subjects went from almost no exercise directly into an intense exercise program. Presumably, this dramatic change would have been psychologically stressful as well, however this was not measured. The authors did suggest that vigorous exercise, particularly if compounded by weight loss, can disturb reproductive function in women and that this disturbance in reproductive function is reversible with the cessation of intense exercise (Bullen et al. 1985).

The most convincing evidence to support the concept that disruption in GnRH drive is stress-induced is found in later research that consistently reports findings of elevated cortisol levels, reflecting HPA axis activation, in women with FHA (Suh et al.

1988; Berga et al. 1989; Biller et al. 1990; Loucks et al. 1989). Suh and colleagues (1988) found lowered LH pulse frequency and higher cortisol levels in 10 women with FHA compared with normal-menstruating women. The same group of researchers (Berga et al. 1989) in a later study, compared hypothalamic function by measuring a number of parameters, including FSH, LH and cortisol in 15 women with FHA and 16 women without FHA. LH and FSH were significantly lower, and 24-h cortisol secretion was significantly higher in FHA women compared to non-FHA women (Berga et al. 1989). Loucks and colleagues (1989) also reported lower LH secretion in amenorrheic athletes (AA) compared to cycling athletes and sedentary controls. The AA also had an augmented LH release when administered an exogenous dose of GnRH, suggesting their HPO axis abnormalities (amenorrhea) were caused by decreased endogenous GnRH due to activation of the HPA axis (Loucks et al. 1989).

Biller and colleagues (1990) also investigated the role of the HPA axis in FHA, during their study involving 10 women who had amenorrhea related to weight loss or psychological stress. In this study, FHA women were found to exhibit mild hypercortisolism, as compared to controls, which was demonstrated by elevated 24-h mean serum cortisol levels and urinary-free cortisol values in the FHA group.

Berga and colleagues (Berga, Daniels and Giles 1997) compared cortisol and LH in FHA women ($n = 19$), women with other causes of anovulation ($n = 19$) and eumenorrheic women ($n = 19$) and found higher cortisol excretion levels in women with FHA, while the other two groups had similar and lower values. Although the differences in cortisol levels between the groups appeared small, the authors noted that chronic activation of the HPA axis induces compensatory mechanisms designed to curtail cortisol

secretion, and the amplification of cortisol in FHA likely indicates chronic HPA activation and greater HPA reactivity in women with this condition. Women with FHA also had a marked reduction in GnRH input as evidenced by a LH pulse frequency more than 50% lower than seen in eumenorrheic women (LH was not measured in women with other causes of anovulation)[Berga, Daniels and Giles 1997].

In summary, it appears that women with FHA are anovulatory because of reduced GnRH input, as LH pulse frequency, a reliable indicator of GnRH input, is decreased. Further, evidence that disruption of GnRH drive is stress-induced is supported by consistent reporting of elevated cortisol levels also occurring in women with FHA and, when measured, women with decreased LH levels (Suh et al. 1988; Berga et al. 1989; Biller et al. 1990; Berga, Daniels and Giles 1997).

Less severe menstrual disturbances in active women and athletes include LPD, previously described. The incidence of LPD in non-active women is controversial, but estimates vary from 2% to 5% in normal ovulatory women and 3% to 20% in women with infertility (De Souza 2003). However, the prevalence of LPD in athletes is much greater; indeed, some reports estimate as high as 79% of exercising women have LPD (DeSouza et al. 1998). As such, LPD is the most common MCI associated with exercise. In the presence of a "normal" menstrual cycle length, short luteal phases (< 10 days), and reduced progesterone production by as much as 50% were reported in runners, as early as 1979 by Shangold et al. (1979). Many studies have found an association between exercise and LPD, and it was presumed that exercise itself produced disturbances in the HPO axis resulting in shortened luteal phases, impaired progesterone production and/or

anovulation. However, researchers have recently challenged this hypothesis (Loucks, Verdun and Heath 1998; Williams et al. 1995, 2001).

Loucks et al. (Loucks, Verdun and Heath 1998) studied regularly menstruating women, and defined and controlled energy availability as dietary energy intake minus exercise energy expenditure, and independently defined and controlled exercise stress as everything associated with exercise except its energy cost. The researchers studied the LH pulsatility in all women in the mid-follicular stage of the menstrual cycle. They controlled for inter-individual differences in LH pulsatility, by repeating the controlled diet and exercise treatments of four days, once with balanced and once with low energy availability, in random order, and compared the results to determine the effect of energy availability. To determine the effects of exercise stress, they compared results in two groups of women, one receiving exercise treatments and one not. The results revealed that energy availability disrupted LH pulsatility and that exercise stress did not. LH pulsatility was disrupted regardless of whether energy availability was reduced by dietary restriction alone or exercise expenditure alone. Furthermore, supplementing the diet to replace the cost of exercise prevented the disruption of LH pulsatility in exercising women (Loucks, Verdun and Heath 1998).

As previously discussed, Williams et al. (2001) also supported the “energy availability” hypothesis, by inducing amenorrhea in eight monkeys by training them to run voluntarily on a motorized treadmill for longer and longer periods while their food intake remained constant. The amenorrhea was successfully reversed when four monkeys had their energy intake supplemented by 58%, while maintaining the volume and intensity of exercise (Williams et al. 2001).

These studies may explain the most common cause of exercise-related menstrual disturbances seen in athletes, however there are MCI occurring in non-exercising and lightly exercising individuals, that cannot be explained by the energy availability hypothesis, and this must be considered when studying other non-athletic populations. As well, although these studies provide evidence that low energy availability during exercise can induce menstrual disturbances that are reversed with increased energy intake, studies over a longer period of time using larger sample sizes are needed to confirm these results. Further research is also needed to elucidate the exact mechanism by which low energy availability disrupts LH pulsatility, as well as the need to understand the inter-individual variability in women who may experience more or less extreme disruptions in reproductive function. Lastly, there is still an abundance of research supporting the psychogenic causes of reproductive dysfunction, which is difficult to control due to the inter-individual variability in personality characteristics and response to stressors, and difficulty in measuring and controlling for all sources of stress. One such stressor may be certain forms of disordered eating, such as cognitive dietary restraint. Cognitive dietary restraint has been implicated in menstrual disturbances, and possibly HPA activation, perhaps independently of energy availability.

1.6 Disordered Eating and Menstrual Cycle Irregularities

It is well established that menstrual cycle irregularities (MCI) are widely reported in individuals with clinical eating disorders such as anorexia nervosa and bulimia nervosa (Marshall and Kelch 1979). Amenorrhea, in fact, is one of the DSM-IV diagnostic criteria for anorexia nervosa (American Psychiatric Association 1994).

Further, oligomenorrhea and amenorrhea have been reported in over half of bulimic patients (Pirke et al. 1987). MCI can also be present in less overt forms such as LPD and anovulation as previously described, which have been associated with subclinical disordered eating behaviours, such as dietary restraint (Schweiger et al. 1992; Barr, Prior and Virga 1994; Barr, Janelle and Prior 1994; Lebenstedt, Platte and Pirke 1999; McLean and Barr 2003).

The prevalence of MCI in female athletes varies considerably depending on the sport, age, activity level, parity and nutritional status, however the prevalence in athletes (1-44%) is greater than that in the general population (2-5%) [Loucks and Horvath 1985]. In a questionnaire survey of 226 athletes, the prevalence of MCI was higher in gymnastics (100%) followed by lightweight rowing (67%), distance running (65%), and ballet (52%), with lower levels found in swimming (31%) and team sports (17%) [Wolman and Harries 1989]. Further, female athletes have been reported to exhibit disordered eating patterns more frequently than the general female population (Otis et al. 1997; Rosen, McKeag and Hough 1986).

Eating disorders and amenorrhea are also two components of the Female Athlete Triad ("the triad") [Otis et al. 1997], where osteoporosis/osteopenia is a possible consequence of these conditions and the third component of the triad (bone status of female athletes will be discussed in greater detail in section 1.10). Disordered eating is central to the triad, referring to a wide spectrum of eating attitudes and behaviours used in an attempt to lose weight in order to achieve a low bodyweight and/or lean appearance. The spectrum of disordered eating behaviours ranges in severity from unhealthy attitudes about food and dieting, to restricting intake and/or bingeing and purging, to the most

severe clinical cases of bulimia and anorexia nervosa (American Psychiatric Association 1994; Garner, Olmstead and Garfinkel 1985; Rosen, McKeag and Hough 1986). The existence of the triad is implicit in studies that have established a relationship between disordered eating behaviours and MCI (Marcus et al. 1985; Beals and Manore 2000; Snead et al. 1992; Zanker and Swaine 1998) as well as MCI and low BMD as discussed later.

Although individuals with anorexia and athletes without clinical eating disorders share some common features such as low body weight and menstrual disturbances, there are many distinguishing features. Generally, athletes without eating disorders have purposeful training, an accurate body image, and good muscular development whereas individuals with anorexia often have aimless physical activity, a flawed body image and poor muscular development (McSherry 1984). The disturbances in the menstrual cycles in the two groups appear to be similar with regards to endocrinological phenomena, such as activation of the HPA as previously described, supporting the view that eating disorders and energy deficiency associated with exercise may have a shared etiology (Pirke et al. 1989).

It has been suggested that in athletes, low calorie intakes and high training loads cause a 'energy drain', which is analogous to the inadequate energy intake observed in anorexics (Warren 1980). Warren (1980) studied dancers who had developed amenorrhea, and subsequently regained menses during prolonged periods of rest. Resumption of menses occurred without changes in body weight or percent body fat, and the author concluded that amenorrhea was modulated more by exercise than body weight

or body fat. It should be noted however, that these findings can also be explained by lower levels of psychogenic stress the dancers underwent the prolonged rest periods.

A study by Loucks and colleagues (Loucks, Verdun and Heath 1998), as previously mentioned, expanded on the energy drain hypothesis, suggesting that some women may fail to adequately compensate the diet for the additional cost of exercise, leading to the impairment of reproductive function. Their data suggested that if the energy costs of exercise are balanced by energy intake, hypothalamic release of LH is maintained at normal levels (Loucks et al. 1998). Further, the increased cortisol levels reported, may be attributed to activation of the HPA axis due to energy deficiency, as cortisol is a glucoregulatory hormone (Loucks 2003).

Nevertheless, female athletes have an increased awareness of the influence of body composition on athletic performance, and pressures to decrease body weight or body fat to unrealistic levels can contribute to the development of disordered eating practices (Wilson et al. 1992). Alternatively, some athletes may inadvertently be consuming inadequate calories to meet their needs if training loads are excessive, and although disordering eating behaviours may not be present, menstrual cycle disturbances may still exist.

1.7 Dietary Restraint and Menstrual Cycle Irregularities

There are a number of differences in behavioural, physical and personality characteristics between groups with high and low restraint scores that have been highlighted in a previous section (see section 1.3). However, the most significant difference and possibly the greatest threat to health posed by restrained eating may in fact

reside in its association with certain characteristics of the menstrual cycle, as well as cortisol excretion levels. Restraint has been associated with menstrual cycle disturbances (Schweiger et al. 1992; Barr, Prior and Vigna 1994; Barr, Janelle and Prior 1994; Lebenstedt, Platte and Pirke 1999; McLean and Barr 2003) and increased cortisol levels (McLean, Barr and Prior 2001a; Anderson et al. 2002) which may result in negative effects on bone (Van Loan and Kiem 2000; McLean, Barr and Prior 2001b) and thereby stress fracture risk, the focus of this research study.

Although disordered eating practices do not always fit the strict criteria of anorexia and bulimia, many women, both non-athletes and athletes, have significant weight, eating and body image concerns that result in abnormal eating attitudes and behaviours. As discussed previously, dietary restraint is one such attitude/behaviour, where women who are highly restrained eaters, have significant weight, eating and body image concerns and make conscious *efforts* to try and limit food intake in order to achieve or maintain a desired body weight (Herman and Mack 1975; Lautenbacher et al. 1992). This form of abnormal eating behaviour, in contrast to clinical eating disorders, is not frequently associated with extreme menstrual cycle disturbances such as amenorrhea, possibly because energy intake and body weight are within the normal range. However, mounting evidence reveals that highly restrained eaters may experience less obvious menstrual cycle disturbances (Schweiger et al. 1992; Barr, Prior and Vigna 1994; Barr, Janelle and Prior 1994; Lebenstedt, Platte and Pirke 1999; McLean and Barr 2003). Therefore, dietary restraint may also prove to be an additional risk factor for menstrual cycle disturbances.

One of the initial studies demonstrating an association between dietary restraint and the menstrual cycle was conducted by Schweiger et al. (1992) who examined the association of different types of “everyday eating behaviour” with disturbances in menstrual function. The researchers prospectively compared women separated into two groups: low dietary restraint ($n = 13$) and high dietary restraint ($n = 9$), as identified by the TFEQ restraint subscale (TFEQ-R). The two groups were similar with respect to age, age at menarche, absolute weight and height, BMI and activity level. Eleven of the 13 women with low dietary restraint had menstrual cycles that fulfilled the criteria for normal serum estrogen and progesterone, and luteal phase length of nine days or more. Only two of the nine women with high dietary restraint had cycles that satisfied these criteria. Of the remaining seven women, one had an anovulatory cycle and six had decreased progesterone concentrations and/or a shortened luteal phase. The authors concluded that high cognitive restraint in “everyday eating behaviour” might be a risk factor for the development of menstrual disturbance in young women.

Shortly thereafter, Barr, Prior and Vigna (1994) assessed the relationship between dietary restraint and menstrual cycle characteristics in 27 ovulatory women, who were previous participants in a longitudinal study of spinal cancellous BMD (Prior et al. 1990). Physical characteristics, reported energy intake and menstrual cycle length were similar between high and low restraint groups, however, luteal phase length was shorter in the high restraint group. This study is of considerable relevance to the present study, as many of the subjects were highly active runners, and high restraint as opposed to high activity, was found to be associated with a shortened luteal phase. Around the same time, Barr, Janelle and Prior (1994) also prospectively assessed ovulatory function in 23 vegetarians

and 22 non-vegetarians with clinically normal menstrual cycles. Women with high restraint had significantly fewer ovulatory cycles, a shorter luteal phase length, and a lower luteal phase index (luteal phase length/cycle length) compared to those with low restraint.

Lebenstedt, Platte and Pirke (1999) also found that athletes with menstrual disturbances scored significantly higher on the TFEQ-R, compared to athletes with normal menstrual function. Thirty-three normal-weight, female endurance athletes aged 18-35 yr with a body mass index (BMI) of 18-25 kg·m⁻² were recruited from regional field and track clubs. Athletes were categorized according to their salivary progesterone concentrations over the studied menstrual cycle into two groups: athletes with normal menstrual function (n = 21) and athletes with menstrual disorders (n = 12). Menstrual cycles were classified as disturbed if progesterone values failed to meet the criteria of a luteal length longer or equal to nine days and an increase of the progesterone concentration in the luteal phase. There were no significant differences in age, gynecological age, BMI, and absolute and relative fat mass between the athletes with and without menstrual disorders. Weight, height, and lean body mass were significantly higher in women with menstrual irregularities. The reported daily exercise time did not differ between groups and was not related to luteal phase length ($r = 0.01$, $P = 0.49$) or progesterone in the luteal phase ($r = -0.02$, $P = 0.48$). Women who scored ≤ 3 were categorized as unrestrained eaters and those who scored ≥ 10 as restrained eaters. The researchers reported that disturbed menstrual function, in particular, decreased and shortened progesterone production in the luteal phase, was associated with a higher level of dietary restraint (Lebenstedt, Platte and Pirke 1999).

Other evidence of an association between restraint and the menstrual cycle is provided by the study of McLean and Barr (2003), who found that women with high restraint scores were more likely to report irregular cycles. This study investigated associations of restraint with selected physical, lifestyle, personality and menstrual cycle characteristics in 596 female university students. Women were assessed by the TFEQ-R, and categorized as having high ($n = 145$), medium ($n = 262$) or low ($n = 189$) restraint. The three groups had similar ages, heights and weights. The proportions of women reporting irregular cycles were compared by restraint group; results indicated that the prevalence of self-reported menstrual cycle irregularity in women with high restraint (34.7%) was double that of women with low and medium scores (17.1 and 17.0%, respectively). The researchers then re-analyzed the data after excluding women who reported a history of an eating disorder and found that the association between dietary restraint and MCI was maintained: the prevalence of menstrual irregularity among women with high, medium and low restraint was 32.5%, 14.6% and 16.1%, respectively.

The relationship between high levels of restraint and menstrual cycle disturbances in normal-weight women is well documented, however, the exact mechanism is still under investigation, but almost certainly involves the neuroendocrine system (Prior, Vigna and McKay 1992; McLean and Barr 2003). As outlined in previous sections, LPD have been attributed to disruption of GnRH pulsatility, mediated by stress and activation of the HPA axis. Chronic stress, as hypothesized to be present in restrained eaters, may manifest as subclinical MCI and increased cortisol levels.

1.8 Dietary Restraint and the Neuroendocrine System

While the relationship between clinical eating disorders and high cortisol levels is well known (Monteleone et al. 1999), the relationship between dietary restraint and cortisol has only been recently explored. The specific mechanisms and relationship between dietary restraint and ovulatory disturbances in normal-weight women are not fully understood, but they may be mediated by disturbances in the neuroendocrine system, specifically cortisol excretion levels (Prior, Vigna and McKay 1992; McLean, Barr and Prior 2001a). Women with high scores for restraint may experience more psychological stress related to the constant monitoring and control of food intake than women with lower scores (McLean, Barr and Prior 2001a). Stress, whether psychological or physical can increase the release of cortisol, via the HPA axis activation outlined earlier. Previous studies have found inconsistent relationships between restrained eating and cortisol.

1.8.1 Studies Investigating Restraint and Cortisol

The first study to investigate endocrine characteristics in restrained eaters was conducted by Pirke and colleagues (Pirke et al. 1990) who studied 22 healthy young women aged 18-24 years with a BMI between 18-24 kg·m⁻². Women who scored above the 75th percentile on the TFEQ restraint subscale (TFEQ-R) were classified as restrained (n = 9) and those who scored below the 50th percentile as unrestrained (n = 13). Age, weight and BMI were similar between the two groups. Blood was sampled at 30 min intervals using an overnight protocol, and similar concentrations for serum cortisol were found in the restrained and unrestrained groups. However, this finding does not

necessarily provide evidence that cortisol excretion levels are similar in restrained and unrestrained eaters. Presumably, stress in association with food-related decisions and/or intake would *not* be occurring during an overnight protocol when food was not being consumed (McLean, Barr and Prior 2001a). The implications of this methodological flaw warranted additional research exploring endocrine responses in restrained eaters.

More recently, McLean, Barr and Prior (2001a) measured 24-hour urinary cortisol excretion levels in females with low and high scores for dietary restraint. Participants (aged 21.6 ± 2.5 y, $n = 62$) with normal-length menstrual cycles were recruited into low ($n = 29$) and high ($n = 33$) restraint groups, as assessed by the TFEQ-R. Age, height, weight, BMI and menstrual cycle length were similar between the groups. Twenty-four hour urinary free cortisol excretions were significantly higher in the high- compared to the low restraint group (418.8 ± 134.6 vs. 354.7 ± 83.7 nmol), as well as ratios of urinary cortisol (nmol) to creatinine (mmol) (42.9 ± 12.9 vs. 36.9 ± 8.9 ; $p < 0.05$). The researchers controlled for other physiologic stressors such as fasting and intense exercise, which may have confounded their results. Although women with high restraint scores did report higher levels of exercise than those with low restraint scores, hours of weekly exercise was not correlated with either urinary cortisol or cortisol-creatinine ratios. The authors therefore dismissed the likelihood that exercise was responsible for higher cortisol concentrations in women with high restraint scores.

This study was also the first to use the Perceived Stress Scale (PSS)[Cohen, Kamarck and Mermelstein 1983] in relation to restraint scores. Women with high restraint scores scored higher on the PSS. The authors noted the possibility that those who perceive events in their lives as more stressful experience more negative feelings

with regard to their weight and attempt to limit food intake to reduce the stress they attribute to body dissatisfaction (McLean and Barr 2003). Alternately, higher restraint may lead to a general feeling of stress, which is subsequently extrapolated into other situations, and thus increase their perceived stress in other life events (McLean and Barr 2003). Potential relationships between PSS score and cortisol was not reported in McLean and Barr's study, however, only one study was found in the literature (van Eck et al. 1996) that investigated this relationship, and they did not find an association. Although stress is known to be associated with menstrual cycle disturbances (Harlow and Matanoski 1991), PSS scores were not independently associated with restraint scores in McLean and Barr's study, nor did PSS differ between women with regular and irregular cycles. Therefore, the authors concluded that perceived stress does not appear to explain the relationship between restraint and menstrual irregularity (McLean and Barr 2003).

Finally, food intake was also carefully monitored on the day of urine collection, to ensure that cortisol excretion was not increased due to very low energy intake and/or severely altered macronutrient intakes. The finding that urinary cortisol excretion, a biological marker of stress, was higher in women with high restraint scores compared with those with low restraint scores supports the hypothesis that dietary restraint is a stressor with corresponding physiological responses from the neuroendocrine system (McLean, Barr and Prior 2001a).

Anderson and colleagues (2002) also investigated the relationship between self-reported restrained eating and cortisol using multiple measures of dietary restraint. Eighty-five college-age women completed the Restraint Scale (RS) and the TFEQ-R and provided a saliva sample for analysis of cortisol. Both measures of restraint were

positively associated with elevated levels of salivary cortisol, although the TFEQ-R was more strongly associated than the RS. These findings concur with those of McLean, Barr and Prior (2001a), where high restraint was also found to be associated with elevated cortisol levels.

In summary, it appears that the constant effort to monitor and control food intake, that characterizes highly restrained eaters, may act as a psychogenic stressor that causes subclinical menstrual disturbances and increased cortisol excretion. Both MCI and increased cortisol levels have been shown to be a result of HPA axis activation. MCI and increased cortisol levels may also negatively impact bone. Therefore the stress caused by restraint may be implicated in lowered bone mineral content and/or bone mineral density, as will be discussed in following sections.

1.9 Cortisol and Bone Health

Elevated cortisol levels have been associated with menstrual and reproductive disturbances, as they represent a peripheral marker for HPA axis activation, as discussed in previous sections (sections 1.4-1.7). However, the physiological consequences associated with higher circulating levels of cortisol are not restricted to menstrual disturbances. Cortisol is the primary glucocorticoid produced in the human adrenal gland, and excessive glucocorticoid levels, both from endogenous as well as therapeutic exogenous sources, have been implicated in bone loss. Therefore, higher cortisol levels are of concern as they are associated with menstrual cycle disturbances that indirectly affect bone, in addition to the direct effects they exert on bone metabolism. Further, these two scenarios may have additive affects.

1.9.1 Bone Characteristics and Metabolism

Bone is described as either cortical or trabecular (also known as cancellous). Cortical bone is made up of dense, calcified tissue, and forms the external part of long bones. The density and strength of cortical bone provides structure and protection. Trabecular bone is characterized by an inner network of thin, calcified trabeculae, is primarily found in the vertebrae and femoral neck, and to a lesser extent in the wrist and femoral shaft. The less dense and more open weave in trabecular bone allows for greater metabolic activity, as well as higher bone turnover (Khan et al. 2001).

Bone remodelling is regulated by the interdependence of systemic hormones and locally produced factors that act in concert to maintain bone mass and density (Canalis 1983). Bone density is regulated by a classical negative feedback loop, which functions by controlling the local balance between bone formation and bone resorption; together known as bone remodelling. Variations in genetically determined bone mass (in the absence of disease) are influenced by exercise, hormones, nutrition and lifestyle factors (Heaney 1996).

Specialized bone cells regulate bone metabolism by responding to various environmental signals including chemical, electrical, mechanical and magnetic stimuli (Einhorn 1996). There are three types of cells in bone: *osteoblasts*, *osteocytes* and *osteoclasts*. The osteoblast is the bone cell responsible for bone formation; it produces bone matrix, both collagen and ground substance. Osteoblasts express receptors for estrogen and 1,25-dihydroxy-vitamin D in their nuclei. The function of osteoblasts is controlled by endocrine, paracrine and autocrine factors. Hormones such as parathyroid hormone (PTH), vitamin D₃, glucocorticoid hormones, growth hormone (GH) and

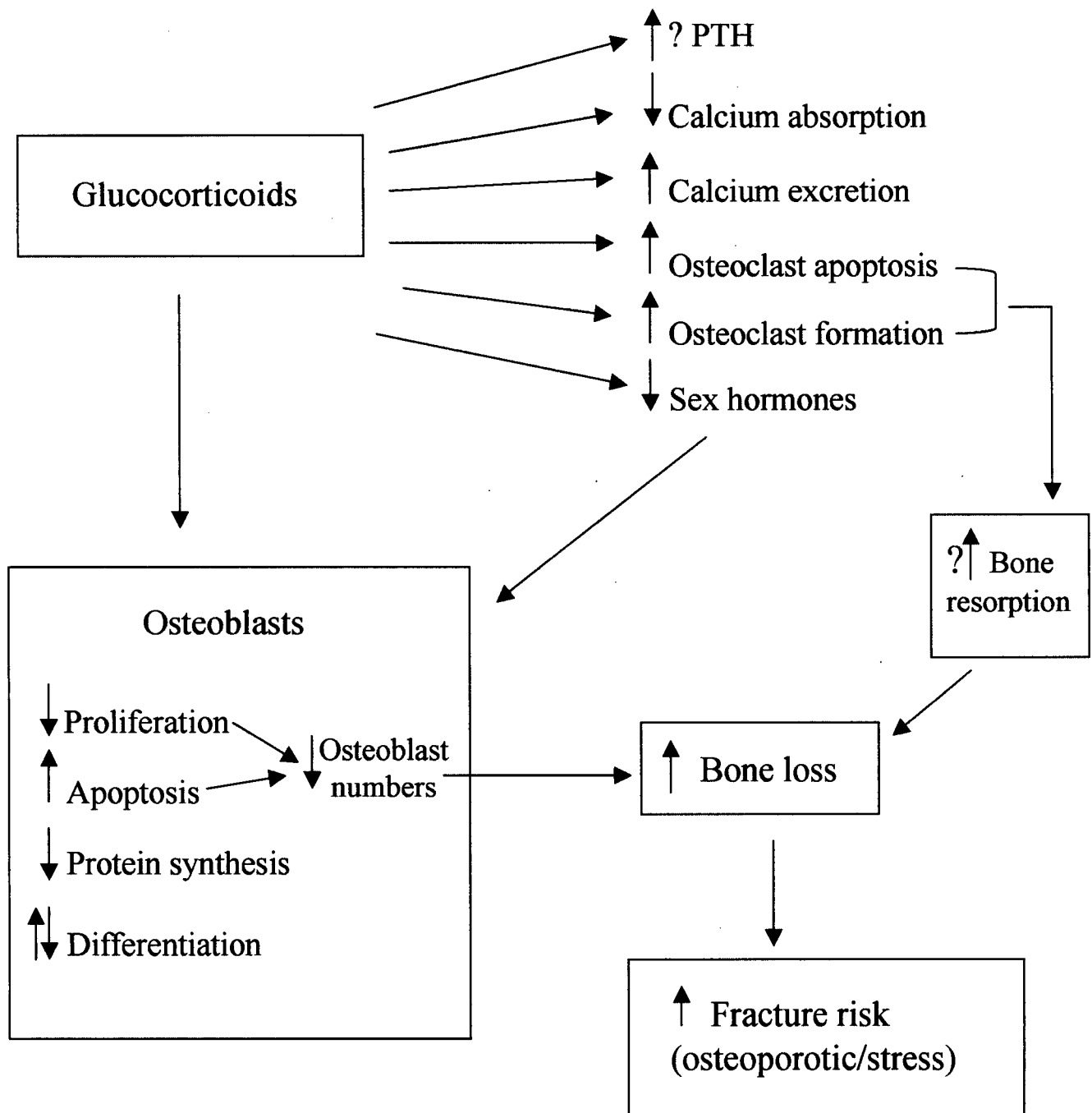
gonadal steroids all act on the osteoblast (Puzas 1996). Osteocytes are mature bone cells that form a complex network throughout bone matrix, which makes them ideal for communication surrounding mechanotransduction (the physiological process where bone is regulated according to certain strain thresholds). The osteoclast is the bone cell responsible for removal of old bone, which is called bone resorption (Khan et al. 2001).

Bone is a dynamic tissue, and bone remodelling is coupled, where the process of bone breakdown is followed by new bone formation. The bone resorption phase, with a resulting “erosion” pit, occurs over a few weeks and is followed by bone formation. Bone formation occurs as osteoblasts cover the pit and secrete osteoid, which upon calcification turns into new bone; this process takes several months (Rehman and Lane 2003).

1.9.2 Glucocorticoid-induced Bone Loss

Glucocorticoids have marked effects on bone metabolism, with their predominant effect being a loss of trabecular bone induced by several mechanisms (Figure 3). The negative impacts of glucocorticoids on bone were established long ago - in 1932 Harvey Cushing described the syndrome of endogenous cortisol excess that was later named after him (Cushing’s syndrome). He noted that pituitary tumours producing ACTH, also resulted in excessive production of glucocorticoids that eventually led to osteoporosis (Cushing 1932). Due to the rarity of Cushing’s disease, most studies in the literature on glucocorticoid-induced bone loss or osteoporosis refer to exogenous over-exposure to cortisone and its synthetic derivatives. The adverse effects to bone health became apparent as glucocorticoids became more frequently used therapeutically for a variety of

Figure 3. Proposed mechanisms: glucocorticoid-induced bone loss



conditions and diseases. Although few studies have focused on endogenous hypercortisolism, the resultant bone damage appears to be qualitatively the same.

Glucocorticoids are commonly used in the treatment of inflammatory and autoimmune diseases, certain malignancies and to prevent transplant rejection (Rehman and Lane 2003). However, chronic glucocorticoid therapy results in bone loss or osteoporosis, and is in fact the leading cause of secondary osteoporosis (Reid 2000). Glucocorticoid-induced bone loss is multifactorial, with the most important facets resulting from: decreased bone formation, altered calcium homeostasis (Canalis 1996) and possibly enhanced resorption, although the occurrence of this last factor has recently been questioned (Reid 1998).

1.9.2.1 Effects on Sex Hormones

The relationship between reproductive hormones and bone has been recognized for many years. Estrogens, progesterone and androgens are important in the development and maintenance of the skeleton (Prior et al. 1994). Increased rates of spinal bone loss have been shown to be associated with low serum levels of estrogen (Klibanski et al. 1980) as well as low progesterone levels (Prior et al. 1990). Estrogen withdrawal, as seen in menopause, is associated with an increased number of osteoclast precursor cells in bone marrow (Jilka et al. 1992), and is causally associated with increased bone resorption and bone turnover leading to increased fractures in this well-studied population. Although estrogen levels can be variable, from normal to low in MCI, the universal characteristic of ovulatory disturbances includes some degree of progesterone deficiency (Prior et al. 1990).

Elevated glucocorticoid (cortisol) levels are indicative of HPA axis activation, as described in previous sections, which suppresses LH secretion and consequently sex steroid production. Animal studies have reported glucocorticoid inhibition of FSH-induced estrogen production in cultured rat granulosa cells (Hsueh and Erickson 1978). Further, another study in rats showed that glucocorticoids and estrogen deficiency were additive in increasing bone loss (Goulding and Gold 1988). A similar additive effect has been seen in post-menopausal women (who are estrogen deficient), who have greater susceptibility to bone loss while receiving glucocorticoid therapy (Als, Gotfredsen and Christiansen 1985). In men, a reduction of testosterone has been reported in those receiving glucocorticoid therapy (Schaison, Durand and Mowszowicz 1987; Doerr and Pirke 1976). A hypothesized mechanism for the associated hypogonadism during administration of synthetic glucocorticoids, is the suppression of ACTH, resulting in the absence of adrenal androgen production and subsequent suppression of the gonadal axis. In such circumstances sex hormone deprivation will induce high bone turnover and subsequent bone loss (Ziegler and Kasperk 1998).

However in Cushing's syndrome, where excess glucocorticoids originate endogenously, ACTH is also in excess and adrenal androgens are still present (Ziegler and Kasperk 1998). Therefore, it is difficult to ascertain from these data the specific effects to bone that may occur in alternative causes of hypercortisolism, such as that caused by stress. Nevertheless, estrogen deficiency (and progesterone to a lesser extent) has been well established as a cause of bone loss, and any degree of hypogonadism has the potential to negatively impact bone.

1.9.2.2 Effects on Calcium Homeostasis and Parathyroid Hormone

The increased bone resorption occurring in individuals receiving glucocorticoid therapy (or endogenous excesses) may be in part due to direct effects of glucocorticoids on bone, but is primarily due to the result of decreased intestinal calcium absorption and increased urinary calcium excretion (Canalis 1996). The exact mechanism of inhibition of intestinal calcium absorption by corticosteroids is unknown. Initially, the inhibition of calcium absorption was thought to be related to vitamin D metabolism, but several studies (Hahn, Halstead and Haddad 1977; Hahn et al. 1979; Hahn, Halstead and Baran 1981) have provided evidence of normal 25-hydroxyvitamin D in patients who were given corticosteroids, when compared to matched controls. Therefore, it appears that since calcium absorption inhibition is not mediated by vitamin D metabolites, this is likely to represent a direct effect on the calcium transport system in the small intestine (Reid 2000). Although no disturbance of renal vitamin D metabolism in the kidney has been demonstrated, this organ plays an important role in glucocorticoid-induced bone loss in that calcium excretion is increased, reflecting a decrease in tubular calcium reabsorption as well as increased glomerular filtration rate (Ziegler and Kasperk 1998). Further, hypercalciuria occurs in spite of increased parathyroid hormone (PTH) levels; decreased calcium absorption and increased urinary excretion add to the hypo-calcemic stimulus to the parathyroid glands resulting in secondary hyperparathyroidism (Ziegler and Kasperk 1998). However, PTH levels are not always elevated in patients receiving corticosteroid therapy, which may depend on when measurements were taken (Hahn et al. 1979). For example, elevated PTH may only be detected after increases in calcium excretion and reduced absorption have been induced directly by glucocorticoids, thus

resulting in secondary hyperparathyroidism. This has been demonstrated by Suzuki et al. (1983), who found significant increases in urinary calcium excretion that were important in the development of secondary hyperparathyroidism in corticosteroid treated patients. The authors suggested that the combination of increased urinary calcium excretion and reduced intestinal absorption may have produced secondary hyperparathyroidism, and therefore may have increased bone resorption (Suzuki et al. 1983). However, Chiodini et al. (1998) did not observe any correlation between bone resorption markers and PTH levels in patients with Cushing syndrome, and they suggest that in these patients, the excess of glucocorticoid, and not that of PTH, plays a predominant role in loss of bone mass. In short, glucocorticoids may directly stimulate PTH secretion, as seen in some patients, although calcium malabsorption in both the gut and renal tubule also contributes (Reid 2000).

In summary, although the exact mechanisms are not known, the net effect of inhibition of intestinal calcium absorption and increased renal excretion, along with secondary hyperparathyroidism, will result in negative calcium balance, and subsequent bone loss.

1.9.2.3 Direct effects on bone metabolism

The most significant effect of glucocorticoids in bone is inhibition of bone formation; glucocorticoid therapy has a suppressive effect on osteoblast formation, survival and activity. Suppression of osteoblast *formation* is caused by a shift in differentiation of mesenchymal cells away from osteoblastic lineage (Canalis 2003), and decreased osteoblastic *function* includes reduced protein synthesis (i.e. for bone collagen), which is probably mediated by direct glucocorticoid receptor regulation of a

number of important osteoblast genes, including type 1 collagen, osteocalcin and others (Reid 1998). This effect may be mediated in part by the reduced production of local growth factors, such as insulin-like growth factor 1 (Manolagas and Weinstein 1999). In addition, it appears that glucocorticoids hasten the apoptotic demise of both osteoblasts and osteocytes further contributing to reduced bone formation (Weinstein et al. 1998). Evidence of these impairments is seen in both animal and human histomorphometric studies, where rate of bone production within each bone remodelling unit and duration of activity in each unit are reduced (Reid 2000). Further, clinical assessments of circulating osteoblastic markers, particularly osteocalcin, consistently show evidence of reduced bone formation (Dempster 1989; Prummel et al. 1991).

Data on the effects of glucocorticoids on osteoclasts are contradictory. There is evidence that glucocorticoids increase osteoclast formation from precursor cells in bone marrow, however they also increase apoptosis of mature osteoclasts (Dempster et al. 1997). The opposing effects may account for findings in organ culture, where glucocorticoids can either increase or decrease bone resorption, depending on the culture conditions (Reid 2000). Animal and human studies remain inconclusive and difficult to interpret, as there are increases in eroded surfaces but a decrease in osteoclast numbers. This may be due to a reduced rate of osteoblastic recruitment where osteoclastic erosion pits have formed, yet remain unfilled for an extended period of time, rather than as an acceleration of bone resorption itself (Reid 1998). It has therefore been suggested that there may not be an increased rate of bone resorption, and many human studies for biochemical markers of bone resorption would support this conclusion (Prummel et al. 1991; Reid 1998, 2000).

In summary, high levels of glucocorticoids, mainly cortisol and its synthetic derivatives, have significant impacts to bone both directly through alterations in bone metabolism and remodelling, and indirectly through altered calcium homeostasis and reduced sex hormones.

1.10 Restraint, Cortisol and Bone

As previously mentioned, most studies investigating the detrimental effects of glucocorticoids on bone have involved patients who were receiving corticosteroid therapy. Aside from studies investigating Cushing's syndrome and the associated endogenous excesses in cortisol, few studies have addressed the negative impacts of cortisol in bone in the absence of disease. However, there have been reports of an association between depression and hypercortisolism, and subsequent bone loss. It has been suggested that depression might be a significant, but ignored, risk factor for osteoporosis, and this may be mediated by elevated cortisol levels (Cizza et al. 2001). Hypercortisolism is a well-known biological correlate of depression (Cizza et al. 2001; Parker, Schatzberg and Lyons 2003) and several studies have reported that low BMD is more frequent in depressed subjects than in the general population (Schweiger et al. 1994; Michelson et al. 1996; Schweiger et al. 2000). Greendale et al. (1999) also showed that in healthy men and women, aged 70-79 years, urinary free cortisol was an independent risk factor for future fracture, when depression (and other variables) were used as covariates. In short, hypercortisolism and lower BMD, frequent findings in depressed patients, provide some evidence that relatively modest elevations in endogenous cortisol are associated with detrimental effects on bone. Therefore it is

plausible that other psychogenic causes of hypercortisolism, such as that proposed to be associated with cognitive dietary restraint (McLean, Barr and Prior 2001a), may also contribute to bone loss. Due to the severe implications surrounding bone and cortisol excess, investigations directed at the hormonal consequences of alternative psychological stressors are warranted.

As previously mentioned, McLean, Barr and Prior (2001a) set out to establish a possible link between dietary restraint and urinary cortisol, and did in fact report elevated cortisol in highly restrained eaters. The data supported the hypothesis that cortisol, a biological marker for stress, was elevated due to the stress of chronic preoccupation with food, characteristic of highly restrained eaters. This finding, along with the association of cortisol to menstrual cycle disturbances would plausibly attribute lowered bone mineral density (BMD) or content (BMC) to the mediating effects of excess cortisol.

McLean, Barr and Prior (2001b) investigated the possibility of an association between restraint and BMD and/or BMC during their study involving 62 regularly menstruating women aged 21.7 ± 2.5 years, who were separated into high ($n = 33$) and low ($n = 29$) restraint groups as assessed by the TFEQ-R. Initial analysis did not reveal a significant difference in body composition (an independent predictor of BMD) or bone characteristics of women with high compared to low restraint scores. However, after the researchers identified that exercise level was associated with bone characteristics in this group of women, additional analyses were conducted to further explore the possibility of a relationship between dietary restraint and bone. When hours of exercise was included as a covariate in comparisons of bone parameters between women with high and low restraint, tendencies toward lower values for BMD and BMC in women with high

restraint scores were observed. In these analyses, the difference in total body BMC was significant; as well, there were nonsignificant tendencies for lower spinal BMD and BMC, and total body BMD. Further, a possible association between dietary restraint and bone was also observed in regression analysis: restraint score entered the equations for total body BMD and BMC, and was narrowly excluded from the equation for spinal BMD.

In addition to the associations between restraint and bone described above, McLean, Barr and Prior (2001a) had also observed higher 24-h urinary cortisol/creatinine excretion among women with high restraint in the aforementioned study. Correlation analysis revealed that urinary cortisol/creatinine excretion was negatively associated with total body BMC in all women. As discussed in previous sections, higher levels of cortisol have been associated with increased reproductive disturbances due to HPA axis activation, and lower levels of reproductive hormones have been consistently reported to be associated with lower bone mass, regardless of the cause (Carmichael and Carmichael 1995; Emans et al. 1990). Further, cortisol negatively affects bone through its influence on bone formation, bone resorption, and intestinal calcium absorption and renal calcium excretion (Canalis 2000). McLean, Barr and Prior (2001b) also reported similar calcium intakes between groups for high and low restraint, yet calcium excretion was lower in the high restraint group. The authors suggested that this may reflect a cortisol-mediated decrease in calcium absorption through the intestine. In summary, the authors concluded that although mean cortisol values were within the laboratory reference range in both the high and low restraint groups, it is possible that long term exposure to even moderately higher circulating cortisol levels seen in highly restrained eaters may affect the attainment

or maintenance of peak bone mass (McLean, Barr and Prior 2001b). In addition, although exercise has been shown to have osteogenic properties, dietary restraint, or factors associated with dietary restraint, may offset some of the positive benefits of moderate exercise for bone health. This attenuation of the positive effects of exercise on bone may be mediated by higher prevailing cortisol levels in women with high dietary restraint causing subclinical ovulatory disturbances which in turn have been associated with loss of spinal trabecular bone over one year (Prior et al. 1990), as well as the direct effects of cortisol on bone.

Around the same time, Van Loan and Keim (2000) set out to determine if women who were restrained eaters ($n = 96$; score ≥ 9) as assessed by the TFEQ-R had lower BMC or BMD compared to non-restrained eaters ($n = 89$; score < 9). Mean age, weight, height, fat-free mass (FFM) and percentage body fat were similar between the two groups. The initial analysis did not reveal any differences in BMD or BMC when women with restraint scores < 9 and ≥ 9 were compared. However, subsequent covariance analysis of women grouped into weight categories revealed significantly lower BMC (but not BMD) in women scoring above the median for restraint, in three out of four weight categories. When examined by quartile of body weight, only women in the highest quartile (≥ 71 kg) showed no effect on BMC of high restraint scores. The researchers also observed a significant negative relation between restraint score and BMC. ANCOVA (analysis of covariance) showed significant differences in pre-planned comparisons for BMC, but not BMD, when adjusted for body weight.

The authors pointed out other factors that may influence BMC including: physical activity, hormonal changes as a result of childbearing, and possibly the time of

year at which the dual energy x-ray absorptiometry (DXA) measurements were done. However, the researchers observed no significant differences between the groups in physical activity, number of children, or the time of year at which DXA measurements were made. The authors suggested that a possible explanation for the lower BMC in the lowest weight quartile are hormonal, as menstrual cycle disturbances have been reported in women with high restraint (Schweiger et al. 1992; Barr, Prior and Vigna 1994; Barr, Janelle and Prior 1994; Lebenstedt, Platte and Pirke 1999; McLean and Barr 2003). Further, as previously mentioned, results from Prior et al. (1990) showed that even without symptoms of ovulatory disturbances, women may have luteal phase defects (LPD) that affect BMD. The effect of luteal cycle changes may be explained, at least partially, by changes in progesterone concentration. Progesterone facilitates bone formation but it also increases bone turnover. The observed lower levels of progesterone in LPD, may therefore result in decreased bone formation and a loss of bone mineral, even with normal production of estradiol.

The aforementioned studies (Maclean, Barr and Prior 2001b; Van Loan and Kiem 2000) linking restraint to bone loss, may be explained by lower progesterone and possibly lower estrogen concentrations due to a shorter luteal phase in addition to direct damage to bone homeostasis, both of which are mediated by some degree of hypercortisolism. In summary, women with high restraint, elevated cortisol levels, and associated subclinical MCI, such as LPD, may be experiencing bone loss. The bone loss that may be evident in individuals with high levels of cognitive dietary restraint, and possible subsequent hypercortisolism and MCI, may then put them at increased risk for stress fracture.

1.11 Stress Fractures and the Female Athlete

Athletic activity results in repetitive strains that are essential for the maintenance of bone health and strength, and often result in increased bone mass. Intense or excessive activity may however overwhelm normal repair processes, and cause an accumulation of microdamage that may lead to stress fracture.

Stress fractures were first reported in military recruits in the 19th century and have become increasingly prevalent in athletes in the last two decades. Stress fractures are a common overuse injury in the athletic population with a multiplicity of potential risk factors including low bone mineral density (BMD), low reproductive hormones, dietary insufficiency, body composition, biomechanical variants, training surface/footwear and excessive training. Although physical fitness and training, nutrition and environmental factors also play a role in stress fracture risk, this review will focus on the role of BMD, menstrual cycle irregularities (MCI), and on potential associations with certain disordered eating behaviours, such as dietary restraint.

1.11.1 Stress Fracture Pathophysiology

A stress fracture is a partial or complete fracture of bone that results from a repeated stress applied to the bone that is lower than that required to fracture the bone in a single loading situation (Martin and McCulloch 1987). Increased metabolic activity of bone is a manifestation of remodelling, or adaptation to mechanical properties, in response to changes in loading patterns. This is also known as mechanotransduction, as previously described (Kahn et al. 2001). Microscopically, remodelling occurs in a well-defined sequence. When bone encounters a new, sustained mechanical *stress*, osteoclasts

begin to remove the old bone matrix, thus creating tunnels in the framework of the bone. Subsequently, osteoblasts fill the tunnels with new bone matrix. Ideally, the coupled sequence occurs rapidly enough that the bone is able to support continuing loads of the same nature, despite the increased porosity of the bone. In pathologic situations, the porous bone inadequately accommodates continued loading. This is due either to excessive microdamage that cannot be repaired by normal remodelling processes, because depressed remodelling processes cannot adequately repair microdamage that occurs at a physiological rate, or because of a combination of these factors (Kahn et al. 2001). When a fracture occurs in the former situation, this has been referred to as a “fatigue fracture”, and in the latter, an “insufficiency fracture” (Callahan 2000). In these situations bone stress injury will occur along a continuum. The least degree of injury representing bone remodelling at a subclinical level is termed *bone strain* or silent stress reaction because it is pain free. The next level of injury results in a *stress reaction*, characterized by bony tenderness clinically, and mildly increased uptake of radioisotope on bone-scanning, but without evidence of damage to the bone itself. Continued loading may then eventually lead to *stress fracture*, which is defined by significant focal uptake of radioisotope on bone-scanning, and evidence of a fracture on computerized tomography (CT) scan, magnetic resonance imaging (MRI), or plain X-ray (Kahn et al. 2001).

1.11.2 Menstrual Disturbances and Bone Health in Female Athletes

As previously mentioned, the prevalence of menstrual disturbances in female athletes varies considerably depending on the sport, age, activity level, parity and nutritional status, however the prevalence in athletes (1-44%) is greater than that in the

general population (2-5%)[Loucks and Horvath 1985]. Further, younger, nulliparous women of excessive leanness who train intensely appear to be at the highest risk for MCI (Bennell et al. 1997). Females who participate in sports such as ballet, gymnastics and running that value leanness and/or low body weight for appearance or performance tend to have the highest incidence of MCI. Retrospective studies based on questionnaire data report higher rates for runners, compared with other sports – with one survey reporting menstrual disturbances in 65% of distance runners (Bennell et al. 1997).

Estrogen deficiency occurring in female athletes is, for the most part, attributable to hypothalamic amenorrhea, a state of GnRH dysregulation. As previously discussed the effects of estrogen deficiency on bone are characterized by an acceleration of bone turnover with a disproportionate augmentation of resorption compared with formation, although the exact mechanisms are not fully understood at this time. Functional hypothalamic amenorrhea (FHA) due to excessive exercise (often accompanied by energy deficiency) or stress has been demonstrated to be associated with a reduction of trabecular, and to a lesser degree cortical bone (Verma and Sherman 2001).

It has been known for two decades that low concentrations of ovarian hormones in amenorrheic and oligomenorrheic athletes are associated with reduced bone mass and increased rates of bone loss (Marcus et al. 1985; Bennell et al. 1997). It was not until the mid-1980s, that researchers linked a decrease in BMD in the lumbar vertebrae to athletes with amenorrhea. One of the earliest investigations into MCI and bone status was conducted by Drinkwater et al. (1984) who studied 14 amenorrheic athletes (AA) to determine whether the hypoestrogenic state of amenorrhea was associated with a decrease in regional bone mass relative to that of 14 of their eumenorrheic peers (EA).

The two groups of athletes were matched for age, weight, height, sport and training regimens. Vertebral BMD was significantly lower (approx. 14%) in the AA compared with EA. In addition, mean estrogen concentrations and progesterone peak were lower in AA compared with EA.

Since then, several other cross-sectional studies in which athletes were currently experiencing amenorrhea or oligomenorrhea have reported lower lumbar spine BMD when compared to eumenorrheic or sedentary controls (Marcus et al. 1985; Cook et al. 1987; Myburgh, Bachrach and Lewis 1993; Mickelsfield et al. 1995; Tomten et al. 1998; Cobb et al. 2003). Drinkwater, Bruemmer and Chestnut (1990) also reported that the vertebral BMD was significantly lower in athletes with a history of irregular menses, even after menses had resumed, and that a linear relationship existed between the duration and/or degree of irregularity and vertebral BMD. Other studies have also shown a linear relationship between severity of menstrual irregularities and declining BMD (Miller and Klibanski 1999; Tomtem et al. 1998).

Beginning in the 1990's many studies further demonstrated that bone loss was not only limited to the lumbar spine in the amenorrheic or oligomenorrheic athlete, but was also found to be lower in whole body and appendicular sites. Myburgh, Bruemmer and Chestnut (1993) found lower femoral neck and total body BMD in athletes with amenorrhea compared with eumenorrheic controls. Later, Rencken, Chestnut and Drinkwater (1996) measured BMD at multiple skeletal sites in 49 athletes aged 17 to 39 years. In this study, athletes with amenorrhea were shown to have significantly lower BMD at the lumbar spine, femoral neck, greater trochanter, Ward triangle, intertrochanteric region, femoral shaft and tibia compared to the eumenorrheic athletes.

These findings have been corroborated in many other studies of amenorrheic runners (Mickelsfield et al. 1995; Tomten et al. 1998; Pettersson et al. 1999). More recently, Cobb et al. (2003) also reported that oligo/amenorrheic runners had lower BMD than eumenorrheic runners at the spine (-5%), hip (-6%), and whole body (-3%), even after accounting for weight, percent body fat, EDI score, and age at menarche.

Although most researchers agree that the hormonal culprit contributing to loss of BMD in athletes with MCI is primarily a low estrogen environment, others propose that low progesterone levels are also critically involved in bone loss. As previously mentioned, short luteal phases and anovulation, together with a decrease in progesterone can be present in women with outwardly normal cycling patterns. Prior et al. (1990) postulated that many young athletic women with ovulatory disturbances have low progesterone levels, and due to progesterone's trophic role in bone metabolism (Prior 1990), these abnormalities may have a detrimental effect on bone mass, particularly at the lumbar spine. In a prospective study involving eumenorrheic women, mostly runners, Prior et al. (1990) found that recurrent short luteal phase cycles and anovulation were associated with spinal trabecular bone loss of approximately 2-4% per year. They also found that therapy with medroxyprogesterone (vs. placebo) led to an increase in BMD in women with a range of menstrual disturbances (Prior et al. 1994). In addition, Cobb et al. (2003)[discussed in greater detail in section 1.12] reported that disordered eating as assessed by the Eating Disorder Inventory (EDI)[which measures attitudes about food and body size] was associated with low BMD in women reporting regular menstrual cycles. The authors noted that it is possible that runners who reported regular menstrual cycles may have been experiencing subclinical MCI (and therefore may have lowered

progesterone and/or estrogen), which may have contributed to bone loss, as this has been reported (see above – Prior et al. 1990). However, this could not be determined, as luteal phase characteristics and ovulatory function were not measured.

In summary, the mechanism for the effects of menstrual disturbances on BMD is most likely multifactorial, however the main research findings support low circulating reproductive hormones as the main cause, as estrogen has indirect and possibly direct effects on bone (Jilka et al. 1992), and progesterone may have bone trophic properties (Prior 1990). This hypogonadism is most likely due to suppression of GnRH by HPA axis activation, (see discussion in section 1.4.4). As mentioned, athletes with MCI may also manifest other risk factors such as stress, low body weight and dietary deficiencies, since many studies measuring bone mass do not control for all variables, it is therefore difficult to evaluate the relative importance of each potential cause (Constantini 1994). In short, prolonged menstrual disturbance and hypogonadism appear to promote bone loss, and subclinical MCI may go unnoticed by the athlete but are also harmful to bone.

1.11.3 Menstrual Disturbances, Bone Mineral Density and Stress Fracture Risk

Although stress fractures are commonly seen in many sports, repetitive weight-bearing activities such as running (and marching) are the most frequently reported causes of stress fracture (Matheson et al. 1987; Ha et al. 1991). In general, the lower extremity is the most common site of stress fracture, and among runners, the tibia is the bone most commonly injured (Matheson et al. 1987).

Most of the data linking hormonal and nutritional (including disordered eating) abnormalities with stress fractures in female athletes (evidence of the female athlete triad) have been derived from retrospective studies. Menstrual cycle factors may have an effect

on stress fracture etiology through the influence of lowered levels of reproductive hormones on bone remodelling and BMD (Brukner and Bennell 1996; Bennell et al. 1996a). Many studies have reported that stress fractures are more common in athletes with current or a history of MCI (Barrow and Saha 1988; Myburgh et al. 1990). As well, excessive exercise, stress, undernutrition, and eating disorders are all causes of hypothalamic hormone deficiencies and are also associated with osteopenia (BMD between 1 and 2.5 SD below the mean for young adults)[Miller and Klibanski 1999], and potentially stress fractures. Theoretically, low BMD could contribute to the development of a stress fracture by decreasing the fatigue resistance of bone to loading and increasing the microdamage (Carter et al. 1981). Stress injury to the bone as a result of *excessive* bone strain with an accumulation of microdamage is likely the cause for stress fracture in most athletes and military recruits. However, in the case of female athletes, specifically those experiencing conditions inherent to the female athlete triad, depressed bone remodelling (due to metabolic dysregulation) in response to *normal* bone strain (or in combination with excessive strain) may be occurring; in these cases energy deficiency and/or stress causing hypothalamic dysfunction and GnRH suppression may exist (Zeni et al. 2000).

Many experts propose that low BMD may contribute to the development of stress fractures by reducing bone strength and allowing the microdamage from repetitive loading to accumulate. For example, Myburgh and colleagues (1990) found that BMD in the spine and hip was significantly lower in athletes with stress fractures than in controls. In addition, females with stress fracture were also more likely to have a menstrual irregularity and less likely to be using oral contraceptives. Bennell and colleagues

(1996a) also found that female track and field athletes who developed stress fractures had lower bone densities in both the axial and appendicular skeletons. Although fractures did not occur in the lumbar spine where lower BMD was seen, which would provide a causal relationship, the authors suggested that this lower BMD may be an indicator of other factors associated with stress fracture risk, such as ovarian dysfunction and dietary insufficiency. However, the support for a causal relationship was provided by the significantly lower BMD in the foot and tibia, where stress fractures did occur (Bennell et al. 1996b). In two more recent prospective studies, BMD was implicated in stress fracture risk. Lauder and colleagues (2000) found that femoral neck BMD was significantly associated with the probability of stress fracture in active-duty army women. Another prospective study of 693 female Marine recruits found that mean BMD and cortical bone thickness of the tibia were significantly lower among the 37 women (5.3%) who incurred stress fractures compared to those who did not (Beck et al. 2000). However, not all studies have shown an association between BMD and stress fracture risk (Carbon et al. 1990), although the failure to detect an association may have been due to small sample size.

In summary it appears that there is a higher prevalence of current and past menstrual disturbances in females with stress fractures compared to those without, and lower BMD may also contribute to an increase in this overuse injury. Again however, more prospective studies that control for a wide variety of variables in female athletes are necessary to elucidate the complexity of stress fracture risk.

1.12 Dietary Restraint and Stress Fracture Risk

The association between subclinical MCI and lower values for spinal BMD has been reported. As previously mentioned, in a prospective study involving women (mostly runners) pre-screened as having normal menstrual cycles, Prior et al. (1990) found that recurrent short luteal phase cycles and anovulation were associated with spinal bone loss of approximately 2-4% per year. This evidence combined with the subclinical menstrual disorders seen in highly restrained eaters (Schweiger et al. 1992, Barr, Prior and Vigna 1994; Barr, Janelle and Prior 1994; Lebenstedt, Platte and Pirke 1999; McLean and Barr 2003) then suggests that women with high restraint scores may be more susceptible to lower BMD. As well, higher urinary cortisol associated with high restraint and its effect on bone health may also link dietary restraint to BMD (McLean, Barr and Prior 2001a; Anderson et al. 2001). And in fact, preliminary evidence has linked restraint to lowered BMD/BMC (McLean, Barr and Prior 2001b, Van Loan and Kiem 2000). As described earlier, MCI and low BMD have been implicated in stress fracture risk. Therefore, an association between high scores for dietary restraint and risk for stress fracture may exist, even in the absence of clinical disturbances in menstrual function. In a review of the literature to date, there are no studies that have investigated the possible association between high levels of dietary restraint and stress fractures. Therefore, there appears to be sufficient justification for the investigation of restraint scores in female runners diagnosed with a stress fracture and comparing those to injury-free female runners.

In addition, a review of the literature located only one study that examined the prevalence of stress fractures in a group of female athletes, and included dietary behaviour as a possible risk factor. Bennell et al. (1995) investigated the prevalence and

nature of stress fractures retrospectively in a group of 53 female competitive track and field athletes and related these fractures to selected bone density, menstrual, body composition and dietary risk factors. A total of 45 fractures were reported in 22 women, with the most common sites of fracture being the tibia, tarsal navicular and the metatarsals. There were no significant differences between the groups with and without a stress fracture history in terms of age, weekly hours of training, age entering competition, or body composition. Bone density at various sites was lower in the stress fracture group but these differences were not statistically significant. Women in the stress fracture group had reached menarche later, and had a greater history of menstrual disturbances.

However, the most interesting finding in Bennell's study, linking disordered eating behaviours and stress fractures, was the significantly higher score on the Eating Attitudes Test (EAT-40) in the stress fracture group. The EAT-40 is a 40-question test relating to three factors- dieting, bulimia and food preoccupation, and oral control – and is useful for identifying women with abnormal weight and eating concerns (Garner and Garfinkel 1979). The results showing a significantly higher score (higher scores reflect higher levels of weight and eating concerns) on the EAT-40 in the stress fracture group compared to the non-stress fracture group were supported by other questions related to dieting and 'carefulness about weight'. In a multivariate analysis, the questions regarding "carefulness about weight" were an independent predictor of a history of stress fracture (Bennell et al. 1995). In this study, Bennell et al. (1995) examined competitive track and field athletes, and used a variety of outcome measures to determine possible risk factors for stress fracture. Although Bennell et al.'s study did not directly assess dietary restraint,

the results suggest that additional assessment of eating attitudes and behaviours between runners with and without stress fractures is warranted.

Cobb et al. (2003) conducted an additional study of interest in investigating all three components of the female athlete triad in 91 competitive female distance runners, aged 18-26 years. Disordered eating was measured by the eating disorder inventory (EDI), and MCI was defined as oligo/amenorrhea (0-9 menses per year). In this study, women with 10 or more menses per year were classified as being eumenorrheic. An elevated score on the EDI (highest quartile) was associated with oligo/amenorrhea, after adjusting for percent body fat, age, miles run per week, age at menarche, and dietary fat. Oligo/amenorrheic runners had lower BMD than eumenorrheic runners at the spine (-5%), hip (-6%), and whole body (-3%), even after accounting for weight, percent body fat, EDI score, and age at menarche. Eumenorrheic runners with elevated EDI scores had lower BMD than eumenorrheic runners with normal EDI scores at the spine (-11%), with trends at the hip (-5%), and whole body (-5%), after adjusting for differences in weight and percent body fat.

A key finding in this study was the association of disordered eating to lowered BMD, in the absence of overt menstrual dysfunction. The EDI measured attitudes about food and body size, and the researchers verified that elevated scores on the EDI translated to reported eating practices; women with elevated EDI scores reported lower total energy intakes (by approximately 19% d⁻¹) and lower percent fat intakes (by approximately 25% d⁻¹) than women with normal EDI scores. In restrained eaters, lower energy (Tuschl et al. 1990a; Schweiger et al. 1992; Klesges, Isbell and Klesges 1992; Janelle and Barr 1995; McLean, Barr and Prior 2001b) and lower percent fat intakes (Tuschl et al. 1990a,b) have

also been reported. In addition, athletes with elevated EDI scores were heavier, which the authors suggested may be due to dissatisfaction with their natural body type. Again, in some studies, restrained eaters have been found to have higher BMI's than unrestrained eaters (Tuschl et al. 1990a,b), possibly predisposing them to restraint in an attempt to achieve their perceived ideal body weight. Further, none of the 91 women in Cobb et al.'s study (2003) indicated that she was dieting to lose weight, suggesting that the observed dietary restriction represents long-term, chronic restriction, rather than temporary attempts to lose weight. Again these observations may perhaps be indicative of chronic monitoring of food intake, as seen in restrained eaters. Therefore, there is the possibility that female runners in this study with high EDI scores may also be highly restrained. Further, as previously mentioned, although these athletes were having "normal" menstrual cycles, the authors noted that some athletes with subclinical MCI's such as LPD and anovulation, may have been missed as menstrual status was self-reported as opposed to being assessed by laboratory testing. This observation may point to the possible missing link of an association between high EDI scores and lowered BMD in the absence of clinical MCI, supporting the hypothesis that subclinical MCI and cortisol may be the mediating factors in the loss of bone.

Cobb et al.'s study (2003) confirmed the existence and significance of the "female athlete triad," in a group of female runners, as disordered eating, MCI, and osteopenia/osteoporosis were present in many of the athletes. Disordered eating in female runners was correlated with oligo/amenorrhea, and the association between oligo/amenorrhea and low BMD in female runners was independent of body weight and

body composition. Lastly, the study provided novel evidence that disordered eating is associated with low BMD in women runners with regular menstrual cycles.

In summary, stress fractures are common overuse injuries in female runners and are associated with low BMD, disordered eating and MCI. These interrelated conditions have been termed the female athlete triad, usually when the symptoms of each component reach a clinically recognizable pathological state. Therefore, less obvious subclinical levels in the triad components may be overlooked due to their non-symptomatic nature. However, less severe forms of disordered eating and MCI may in fact predispose female athletes to an increased risk for stress fracture and/or sub-optimal long-term bone health, including premature osteoporosis.

Dietary restraint is one type of disordered eating that has been associated with subclinical MCI, such as LPD and anovulation, increased cortisol excretion as well as lowered BMD/BMC. Elevated cortisol and subclinical MCI associated with restraint may be the mediating factors in reports showing lowered BMD/BMC in restrained eaters. Lower levels of reproductive hormones and lower BMD have been implicated in stress fracture risk. Therefore, restrained eaters may have an increased risk for stress fracture, and the recognition of this plausible link warrants investigation.

1.13 Research Questions, Hypotheses and Objectives

Research Questions

Data obtained from this research study will provide information pertaining to the following two research questions:

i) Primary Question:

Do female runners diagnosed with lower extremity stress fractures have higher scores on the dietary restraint scale compared with uninjured female runners with similar physical and lifestyle characteristics, and level of physical activity?

ii) Secondary Question:

Do energy and specific nutrient intakes (carbohydrate, fat, protein, fiber, calcium, vitamin D, iron), obtained from 3-day food records, differ between the two groups?

Null Hypotheses (Null)

To address the research questions, three null hypotheses are tested by this study.

- 1) Scores on the Three-Factor Eating Questionnaire subscales, Baecke Questionnaire of Habitual Physical Activity and Perceived Stress Scale will not differ between groups.
- 2) The age, physical and lifestyle characteristics will not differ between the stress fracture group and the injury-free group.
- 3) There will be no difference in energy, macronutrient, fiber, calcium, vitamin D, or iron intake between groups.

Specific Aims or Objectives

The specific objectives of this study are to investigate whether a group of female runners with lower extremity stress fractures have a significantly higher score on the restraint subscale of the TFEQ compared to injury-free female runners with similar physical characteristics and level of physical activity. Due to the fact that there are a number of variables other than restraint score that may affect susceptibility to stress

fracture (i.e. age, body mass index {BMI}, training regimen), these variables will be monitored and, if necessary, used as covariates in the comparison between groups.

The main objectives of this study are:

- 1) To assess levels of dietary restraint through the TFEQ questionnaire for the stress fracture group and the control group without any injuries.
- 2) To compare energy and nutrient intakes (including calcium) from 3-day food records between the two groups.
- 3) To compare perceived stress between the two groups.
- 4) To compare lifestyle variables between the two groups (special diets, smoking, alcohol, caffeine, supplement use etc.).
- 5) To compare the type and amount of physical activity between the two groups.

1.14 References

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Chapter 2:

High Levels of Cognitive Dietary Restraint Are Associated with Stress Fractures in Women Runners

2.1 Introduction

The female athlete is faced with unique challenges, as she lives in a society that values an “ideal” body shape and competes in a sporting arena where an ideal body weight or lean appearance equates with success. The “female athlete triad” (Otis et al. 1997) is the combination of disordered eating, menstrual irregularity and osteoporosis/osteopenia, which are interrelated in etiology, pathogenesis and consequences. Disordered eating is central to the triad, and refers to a wide spectrum of eating attitudes and behaviours used in an attempt to lose weight in order to achieve a low body weight and/or lean appearance.

Cognitive dietary restraint is one aspect of the continuum of disordered eating attitudes and behaviours. It is seen in women who consciously try to limit their food intake in order to maintain or achieve an “ideal” or desired body weight (Herman and Mack 1975; Stunkard and Messick 1985). Previous studies have generally found similar physical characteristics and energy intakes among women with differing restraint scores (Barr, Prior and Vigna 1994; Barr, Janelle and Prior 1994; Lebenstedt, Platte and Pirke 1999; McLean, Barr and Prior 2001b). The major differences between women with high and low restraint scores that have been reported are those in menstrual cycle characteristics (Barr, Prior and Vigna 1994; Barr, Janelle and Prior 1994; Lebenstedt, Platte and Pirke 1999; McLean and Barr 2003) and cortisol excretion levels (Anderson et

al. 2002; McLean, Barr and Prior 2001a). The constant monitoring of food intake and concerns about weight, characteristic of restrained eaters, may represent a stressor, which in turn increases cortisol which can affect the menstrual cycle and bone health. Preliminary evidence suggests there may be a link between high restraint and lower bone mineral density (BMD) in women (Van Loan and Keim 2000; McLean, Barr and Prior 2001b).

Amenorrhea/oligomenorrhea have been clearly associated with low BMD, in athletes and non-athletes alike (Drinkwater et al. 1984; Myburgh, Bachrach, and Lewis 1993; Otis et al. 1997). Some research suggests that more subtle menstrual cycle irregularities such as luteal phase deficiencies (LPD) and anovulation, may also be associated with bone loss (Prior et al. 1990). These findings are potentially significant, as such irregularities of the menstrual cycle are asymptomatic and not easily recognized by women.

Low BMD has been established as one risk factor for stress fractures (Myburgh et al. 1990; Bennell et al. 1996a; Lauder et al. 2000). If women athletes with high restraint scores experience more subtle menstrual irregularities, increased cortisol, and lower BMD, they may be at increased risk for stress fractures. To date, no studies have examined a possible relationship between dietary restraint and stress fractures in women athletes reporting “regular” menstrual cycles.

Therefore, the purpose of this study was to determine if a sample of regularly-menstruating female runners with lower extremity stress fractures would have higher scores on the dietary restraint subscale of the Three-Factor Eating Questionnaire (TFEQ),

compared to a sample of injury-free regularly-menstruating female runners who had similar activity levels, physical, and lifestyle characteristics.

2.2 Subjects and Methods

In this cross-sectional, descriptive study, differences between groups were analyzed. This study was a non-experimental analysis of differences, where the independent grouping variable was the presence of a recent stress fracture. The dependent variable is the score on the restraint scale of the TFEQ described below.

Participants and Protocol

The sampling method used in the study for both the study group and the controls was a *sample of convenience. Participants in the study group consisted of female runners with a current (less than three months previously) or past (more than three months but less than one-and-a-half years previously) stress fracture, while the control group consisted of female runners without a running-related injury. Runners with a diagnosed stress fracture were recruited through the Allan McGavin Sports Medicine Centre located at the University. Those with a current stress fracture were recruited through posters (Appendix 1) at the centre or at the suggestion of the treating physician during a patient visit. Interested participants contacted the primary investigator by telephone. Women with past stress fractures were identified through medical chart searches, initially contacted by letter (Appendix 2), and responded to the primary investigator if interested. Female runners for the control group were recruited through announcements at running club meetings, and by posters (Appendix 3) distributed to

running apparel stores, on campus and at local fitness clubs. The University of British Columbia Behavioural Research Ethics Board approved the study prior to participant recruitment (Appendix 4), and all participants provided written informed consent.

Inclusion criteria for all runners included: female, 20-40 years of age, nulliparity, menstrual cycles of normal length (21-35 days), stable body weight with a Body Mass Index (BMI) range of $18.5\text{--}25\text{ kg}\cdot\text{m}^{-2}$, recreational or competitive runner (running distance/frequency: $\geq 20\text{ km/week}$ for nine or more months per year for one or more years). Exclusion criteria included runners who were: cigarette smokers, using medications which may affect bone (e.g. steroids), ever diagnosed or treated for an eating disorder, presently dieting (on a specific weight loss diet), diagnosed with clinical hirsutism (excessive facial hair), or consuming more than seven drinks per week.

The sample size was determined by power analysis calculations using a power of 0.8, a medium size effect of 0.5 and a difference of 2.54 in restraint score means (Portney and Watkins 1993). The sample size calculated was to include 40 participants in each of the two groups; stress fracture and injury-free.

Eligible participants met with the primary investigator and received the questionnaire and three-day food record forms and instructions on how to complete them. They completed the questionnaire and diet record at home, and were provided with a postage-paid envelope to mail their forms back to the university within 10 days. All information was kept confidential.

Questionnaire

The questionnaire (Appendix 5) was designed to take 20-30 minutes to complete. It included previously validated, standardized scales designed to assess eating behaviours,

physical activity, and perceived stress. Additional questions on physical and lifestyle characteristics included age, weight, height, dieting history, menstrual cycle information, special diets (e.g. vegetarian), caffeine and alcohol use, use of medications, and use of supplements (vitamins, minerals, herbal formulas etc.).

Eating behaviours

The 51-item TFEQ (Stunkard and Messick 1985) was used to assess three dimensions of human eating behaviour: 1) cognitive restraint of eating, 2) disinhibition, and 3) hunger. The cognitive restraint scale (21 items) measures the intent to control food intake in order to achieve or maintain a desired body weight. The disinhibition scale (16 items) assesses overeating and binge eating in response to a variety of situations associated with loss of control of food intake. The hunger scale (14 items) measures perceived hunger. The score on the restraint subscale of TFEQ was the main outcome measure in this study. The first question on the TFEQ was changed from "When I smell a sizzling steak...." to "When I smell my favourite food...." in order to make it suitable for those individuals who do not consume meat. Responses to items on the TFEQ were scored according to instructions provided by the authors (Stunkard and Messick 1985) and summed to obtain scores for restraint, disinhibition and hunger.

Physical activity

The Baecke Questionnaire of Habitual Physical Activity (BQHPA) consists of three sections: work activity, sports activity, and non-sports leisure activity (Baecke, Burema, and Frijters 1982; Jacobs et al. 1993; Philippaerts, Westerterp and Lefevre 1999). The questionnaire includes 16 items scored on a Likert scale, ranging from never

to always or very often. For the two most frequently reported sport activities, additional questions query the number of months per year and hours per week of participation.

The work, sports, leisure and total activity indices were calculated according to the authors' instructions, with higher values indicating higher levels of activity by section and/or by total activity score. Specific questions regarding weekly running mileage and number of years participants had been running were also included as additional questions, in the "other information" section of the questionnaire. Runners with a current stress fracture reported their usual running activity prior to their fracture, while all others reported their current usual running activity.

Perceived stress

The Perceived Stress Scale (PSS) is a measure of the degree to which situations in one's life are appraised as stressful (Cohen, Kamarck, and Mermelstein 1983). PSS items were designed to assess the degree to which respondents found their lives unpredictable, uncontrollable, and overloaded. These three factors have been repeatedly found to be central components of the experience of stress, and the PSS can be used to determine whether "appraised" stress is an etiological (or risk) factor in behavioural disorders or disease (Cohen, Kamarck, and Mermelstein 1983). Items were scored and totalled according to instructions provided by the authors, with higher scores reflecting higher perceived stress. The PSS consists of 14 items that are scored and then added together to give a total score, which can range from 0-56 (Cohen, Kamarck, and Mermelstein 1983). In the present study one item was inadvertently excluded from the questionnaire, so a transformed score was calculated to account for the omission and to allow for comparison to other studies.

Physical and lifestyle characteristics

Participants reported their present age, height, and weight, as well as the weight at which “they feel their best”. BMI was calculated from these values. Participants were also asked whether they were currently trying to lose weight, had ever tried to lose weight, or had ever been diagnosed with or treated for an eating disorder, to ascertain that exclusion criteria were met. Weight fluctuation was determined by the number of times that more than five pounds was lost in the past two years.

Menstrual cycle information included age of menarche, whether they were currently having menstrual cycles, and if so, were they regular (21-35 days) or irregular. They were also asked the average length of their menstrual cycle, and if they were currently, or had in the past six months used oral contraceptives.

Lifestyle information included questions regarding alcohol and caffeinated beverage use, cigarette usage, as well as medication and supplement (vitamins, minerals, herbal formulas etc.) use. Participants were also asked if they followed a lacto-ovo vegetarian, vegan or other special diet.

Dietary Intake

Participants recorded their food and fluid intake for three consecutive days, including one weekend day (Appendix 6). Food intake records were analyzed using the computer program Nutritionist Five Version 1.6 (First Databank, Inc., 1998). Total energy, carbohydrate, protein, fat, fiber, calcium, vitamin D, and iron intakes were calculated and averaged over the three days.

Statistical analysis

Statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS) for Windows, Version 10.1 (SPSS Inc. 2000). Group comparisons for all variables were made between stress fracture and non-stress fracture groups. Mean characteristics of the two groups were made using independent t-tests; p value was set at 0.05. Comparisons involving group proportions were made using chi-square.

2.3 Results

Eighty-six women completed the study. Seven participants (four with a stress fracture and three controls) who admitted to being diagnosed with or treated for an eating disorder (by a “yes” response in the questionnaire) were excluded from analysis. Thirty-eight female runners with a diagnosed stress fracture and 41 non-injured runners were used in the analysis.

Descriptive and lifestyle characteristics and perceived stress

Descriptive physical, activity and lifestyle characteristics of the two groups are presented in Table 1 (Appendices 7-12 report mean comparisons for all variables). There were no significant differences in age, physical and menstrual cycle characteristics or BMI values between the two groups. Best weight and weight fluctuation (number of five pound weight losses in the last two years) were also similar between the groups, as were perceived stress scores.

Physical Activity

Physical activity and running characteristics are also presented in Table 1. Total activity scores for both groups were similar; the sub-scales for job, sport and leisure activity were also similar when compared by section. The length of time participants had been running and their weekly running distance were also similar between runners with and without history of a stress fracture.

Eating Behaviour and Dietary Intakes

Participants' scores on the TFEQ subscales are presented in Table 1. There were no differences on the TFEQ hunger and disinhibition subscales between the two groups. Restraint scores were significantly higher in the stress fracture group compared to the non-stress fracture group. Restraint scores of the stress fracture group did not differ between those with current ($n = 12$) or past ($n = 26$) stress fractures (9.8 ± 5.5 and 11.5 ± 5.4 , respectively, $t = -1.1$, $p = .29$).

Participants' daily nutrient intakes are presented in Table 2. Nutrient intake values for vitamins and minerals include intakes from both diet and supplements. Intakes of energy, protein, carbohydrate, fat, fibre and iron were similar between the two groups. However, women in the stress fracture group had significantly higher total daily calcium and vitamin D intakes compared to the women in the non-stress fracture group. Consumption of caffeinated beverages (daily) and alcoholic beverages (weekly) were similar between the two groups (Table 2). Approximately one-third of the participants were using a vitamin-mineral or other supplement (Table 2), with no differences seen between the two groups. A vegetarian diet was followed by approximately one-quarter of the participants (Table 2), with no differences seen between the two groups.

Table 1. Physical and menstrual cycle characteristics, running activity, and BQHPA^a, TFEQ^b, PSS^c scores in women runners grouped according to presence of stress fracture.^d

Characteristics	Non-Stress Fracture (n = 41)	Stress Fracture (n = 38)
Physical and menstrual cycle characteristics		
Age (yr)	29.1 ± 5.0	29.2 ± 5.5
Height (cm)	165.1 ± 5.3	167.0 ± 6.9
Weight (kg)	60.2 ± 7.8	59.1 ± 6.6
Body mass index (kg·m ⁻²)	22.0 ± 2.5	21.2 ± 1.8
Best weight (kg) ^e	57.9 ± 6.7	56.8 ± 5.2
Number of 5lb weight losses in last 2 years	1.2 ± 1.2	1.3 ± 1.8
Age of menarche (yr)	13.0 ± 1.5	13.4 ± 1.9
Menstrual cycle length (d)	28.6 ± 2.7	28.0 ± 2.9
Use of oral contraceptive in past 6 mo (%)	34	50
Running activity		
Running distance (km·wk ⁻¹)	33.4 ± 13.4	35.7 ± 13.5
Length of time running (yr)	6.7 ± 4.5	8.2 ± 4.9
BQHPA scores		
Total activity index	7.7 ± 0.7	7.8 ± 0.7
Job activity index	2.5 ± 0.4	2.6 ± 0.4
Sport activity index	2.2 ± 0.4	2.3 ± 0.4
Leisure activity index	2.9 ± 0.4	2.9 ± 0.5
TFEQ scores		
Restraint	8.4 ± 4.3	11.0 ± 5.4*
Disinhibition	6.7 ± 3.9	5.1 ± 3.6
Hunger	6.5 ± 2.7	6.1 ± 2.6
PSS score	23.9 ± 5.0	25.1 ± 4.7

^a Baecke Questionnaire of Habitual Physical Activity (Baecke, Burema and Frijters 1982).

^b Three-Factor Eating Questionnaire (Stunkard and Messick 1985).

^c Perceived Stress Scale (Cohen, Kamarck and Mermelstein 1983).

^d Mean ± SD.

^e Best weight: weight at which participants indicated they felt their best.

*p < 0.05, t-test.

Table 2. Daily energy and nutrient intakes^a (including intake from supplements), vegetarianism and supplement use in women runners grouped according to presence of stress fracture.

Characteristics	Non-Stress Fracture (n = 41)	Stress Fracture (n = 38)
Energy (kcal)	1948 ± 317	1920 ± 375
Protein (g)	79.5 ± 17.5	83.7 ± 23.3
Carbohydrates (g)	278.5 ± 73.7	269.7 ± 73.2
Fat (g)	57.2 ± 15.2	53.9 ± 14.5
Fibre (g)	25.8 ± 10.5	22.9 ± 8.2
Total calcium (mg)	1023.5 ± 361.4	1289.6 ± 524.0*
Total vitamin D (IU)	104.9 ± 74.1	213.7 ± 212.3*
Total iron (mg)	18.0 ± 6.9	20.8 ± 15.2
Caffeinated beverage consumption (cup/d)	1.6 ± 1.3	1.5 ± 0.9
Alcoholic beverage consumption (drinks/wk)	2.2 ± 2.4	2.6 ± 2.6
Vitamin-mineral/other supplement use (%)	36.6	34.2
Vegetarian (%) ^b	24.4	23.7

^a Mean ± SD.

^b Vegetarian refers to those who exclude meat and poultry. Two vegetarians consumed fish occasionally.

* p < 0.05, t-test.

2.4 Discussion

This is the first study to investigate, and find an association between, cognitive dietary restraint and women runners with a current or past stress fracture. We hypothesized that a sample of regularly-menstruating female runners with lower extremity stress fractures (SF) would have higher scores on the dietary restraint subscale of the TFEQ (TFEQ-R)[Stunkard and Messick 1985], compared to a sample of injury-free (NSF) regularly-menstruating female runners who had similar activity level, physical, and lifestyle characteristics. In our study, SF and NSF runners had similar BMI, physical activity, perceived stress, and energy and macronutrient intakes. Our main finding was significantly higher cognitive dietary restraint, as assessed by the TFEQ-R, in SF compared to NSF runners (Table 1).

Stress fractures are common overuse injuries in female runners, and it is well documented that stress fractures are associated with disordered eating and amenorrhea/oligomenorrhea (Tomten et al. 1998; Barrow and Saha 1988; Otis et al. 1997), as well as osteopenia or low BMD (Myburgh et al. 1990; Bennell et al. 1996a; Lauder et al. 2000). These interrelated conditions have been referred to the female athlete triad (Otis et al. 1997), usually when the symptoms of each component reach a clinically recognizable pathological state. Therefore, less obvious subclinical levels in the triad components may be overlooked due to their non-symptomatic nature. However, less severe forms of disordered eating and menstrual irregularities may in fact predispose female athletes to an increased risk for stress fracture and/or sub-optimal long-term bone health, including premature osteoporosis.

Cognitive dietary restraint is one type of subclinical disordered eating behaviour that is occurring in women who are chronically preoccupied with trying to limit their food intake in order to maintain or achieve an “ideal” or desired body weight (Herman and Mack 1975). Bone health may be affected in women with high scores for restraint, despite normal weight and outwardly normal menstrual cycle patterns (Barr, Prior and Vigna 1994; McLean, Barr and Prior 2001b; Van Loan and Kiem 2000). It is well established that oligo/amenorrhea in female athletes is associated with spinal trabecular bone loss (Marcus et al. 1985; Cook et al. 1987; Myburgh, Bachrach and Lewis et al. 1993; Mickelsfield et al. 1995; Tomten et al. 1998, Cobb et al. 2003) as well as bone loss in whole body and appendicular sites (Myburgh et al. 1993; Mickelsfield et al. 1995; Rencken, Chestnut and Drinkwater 1996; Tomten et al. 1998; Pettersson et al. 1999). However, women without the overt form of menstrual irregularity (oligo/amenorrhea) may suffer from less obvious disturbances such as anovulatory cycles and shortened luteal phases, and cognitive dietary restraint has been associated with these subclinical menstrual disturbances (Schweiger et al. 1992; Barr, Prior and Vigna 1994; Barr, Janelle and Prior 1994; Lebenstedt, Platte and Pirke 1999; McLean and Barr 2003). Anovulation and shortened luteal phases, reported in highly restrained eaters, have been associated with bone loss. A prospective study conducted by Prior et al. (1990), involving regularly-menstruating women, mostly runners, found that recurrent, short luteal phase cycles and anovulation were associated with spinal trabecular bone loss of approximately 2-4% per year. Therefore, restrained eaters with subclinical menstrual irregularities may also be at risk for bone loss.

Preliminary evidence has suggested that high levels of cognitive dietary restraint are associated with lower BMD and/or bone mineral content (BMC) [Van Loan and Kiem 2000; McLean, Barr and Prior 2001b], and low BMD has been implicated in stress fracture risk. Many experts propose that low BMD may contribute to the development of stress fractures by reducing bone strength and allowing the microdamage from repetitive loading to accumulate; several studies have reported an association between low BMD and stress fracture risk (Myburgh et al. 1990; Bennell et al. 1996a; Lauder et al. 2000).

In a recent study Cobb et al. (2003) reported that disordered eating as assessed by the Eating Disorder Inventory (EDI)[Garner and Olmsted 1984], which measures attitudes about food and body size, was associated with low BMD in women reporting regular menstrual cycles. The authors noted that ovulatory and luteal phase characteristics were not measured, but may have been present, and this may have contributed to bone loss. The key finding in this study, the association of disordered eating to lowered BMD in the absence of overt menstrual dysfunction, supports the hypothesis that disordered eating practices may result in *subclinical* menstrual irregularities, and the potential for corresponding bone loss, and/or increased risk for stress fractures.

To date, only one study that examined the prevalence of stress fractures in a group of female athletes included dietary behaviour as a possible risk factor. Bennell et al. (1995) investigated the prevalence and nature of stress fractures retrospectively in a group of 53 female competitive track and field athletes, where dietary related factors were one of several risk factors assessed. A total of 45 fractures were reported in 22 women, and the results showed a significantly higher score on the Eating Attitudes Test (EAT-40) in the stress fracture group compared to the non-stress fracture group. The

EAT-40 is a 40-question test relating to three factors - dieting, bulimia and food preoccupation, and oral control – and is useful for identifying women with abnormal weight and eating concerns (Garner and Garfinkel 1979). The higher EAT-40 score (higher scores reflect higher levels of weight and eating concerns) in the stress fracture group was also supported by positive responses to other questions related to dieting and “carefulness about weight”. In a multivariate analysis, the questions regarding “carefulness about weight” were an independent predictor of a history of stress fracture (Bennell et al. 1995). Although Bennell et al.’s study did not directly assess dietary restraint, the results support the plausible association of disordered eating behaviours to stress fractures in female athletes, as reported herein.

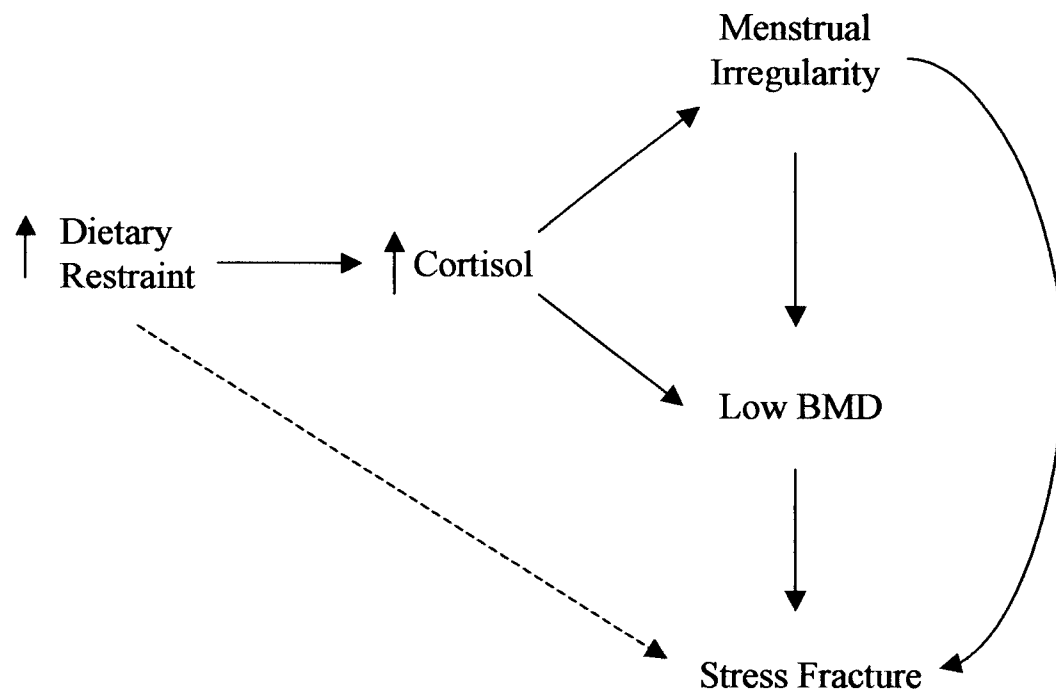
Cognitive dietary restraint has also been associated with increased cortisol levels (McLean, Barr and Prior 2001a; Anderson et al. 2002). The constant monitoring of food intake and chronic concerns about weight, characteristic of restrained eaters, may represent a stressor, which in turn increases cortisol levels which may affect the menstrual cycle and bone health. Elevated cortisol levels have been associated with menstrual and reproductive disturbances, as they represent a peripheral marker for hypothalamic-pituitary-adrenal (HPA) axis activation (Berga 1996). Stress, whether physical or psychological, stimulates the release of corticotropin releasing-hormone (CRH) from the hypothalamus, which in turn releases cortisol from the adrenal gland (Rivier, Rivier and Vale 1986). High concentrations of cortisol are associated with reproductive disturbances due to the inhibitory effect of CRH on hypothalamic and hence pituitary secretions required for normal menstrual function (Barbarino et al. 1989). However, the physiological consequences associated with higher circulating levels of

cortisol are not restricted to menstrual disturbances. Cortisol is the primary glucocorticoid produced in the human adrenal gland, and it is well documented that excessive glucocorticoid levels, both from endogenous (Cushing 1932; Greendale et al. 1999) as well as therapeutic exogenous sources (Ziegler and Kasperk 1998), have been implicated in bone loss. Glucocorticoid excesses can result in hypogonadism as well as directly affecting bone formation processes and calcium homeostasis (Canalis 1996). Therefore, higher cortisol levels reported in restrained eaters (McLean, Barr and Prior 2001a; Anderson et al. 2001) are of concern, as they are associated with disturbances of the menstrual cycle that may indirectly affect bone, in addition to the direct effects they exert on bone metabolism. Further, these two scenarios may have additive effects.

In short, stress fractures have been associated with low BMD, clinical disturbances of the menstrual cycle and disordered eating. Cognitive dietary restraint is one form of disordered eating that has been associated with subclinical MCI and elevated cortisol levels, which are both implicated in bone loss and thereby, stress fracture risk (Figure 4). Our finding that restraint scores are higher in women runners with a history of stress fracture supports this concept.

Although runners with stress fractures may be more likely to have eating attitudes that may increase stress, nutritionally, both groups of runners generally reported adequate intakes of macro- and micronutrients. In our study, we found that intakes of energy, protein, carbohydrate, fat, fibre and iron were similar between the stress fracture and non-stress fracture groups. However, women in the stress fracture group had significantly higher total daily calcium and vitamin D intakes compared to the women in the non-stress fracture group. We suggest that the reason for the differences in vitamin D and calcium

Fig 4. Proposed pathways among cognitive dietary restraint, the menstrual cycle, BMD and stress fracture risk. *Solid lines* represent associations suggested by previous studies; *dashed line* represents association suggested by current study.



intake between the groups was due to the treating physician's recommendations to increase these bone-specific nutrients at the time of stress fracture diagnosis. Most of the runners reported their dietary intake weeks or months after their diagnosis, when dietary changes (i.e. increased vitamin D and calcium) would have presumably been implemented. Runners diagnosed with a stress fracture would have had greater awareness of the importance of these nutrients to bone health, having had a bone injury, compared to the non-stress fracture group.

Limitations of our study include the absence of measured data on BMD, cortisol levels and subclinical menstrual disturbances in our participants. Therefore, the possibility could be raised that dietary restraint scores were increased as a consequence of experiencing stress fractures rather than being associated with their occurrence. We do not believe this to be the case, primarily due to the fact that associations among dietary restraint and subclinical menstrual disturbances, cortisol and BMD have been demonstrated in a number of other studies, as previously described. In addition, the fact that our participants included those recovering from stress fractures as well as recovered individuals who had resumed running provided an opportunity to examine this issue. One might speculate that restraint scores in those with a current stress fracture would increase to compensate for decreased activity during recovery (although in most cases these women were remaining active by participating in non-weight bearing activities). However, a comparison of restraint scores between runners whose stress fractures had occurred within the past three months to those who had fully recovered revealed that restraint scores were not different. Further, if anything, there was a tendency for restraint scores to be slightly higher in those who had recovered and resumed running.

In conclusion, we found higher levels of cognitive dietary restraint in women runners with a recent stress fracture, compared to those without. This observation is a concern for women runners, as stress fractures are presently a common injury in this population. High levels of cognitive dietary restraint (and the corresponding likelihood of subclinical menstrual disturbances and increased cortisol) may be additional risk factors for stress fractures that are overlooked by both the athlete herself as well as health care providers, due to their non-symptomatic nature.

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Chapter 3:

Conclusion, Study Limitations and Future Directions

3.1 Conclusion

Societal emphasis on body image and the 'ideal' body weight drives many women to make conscious *efforts* to limit their food intake in order to achieve or maintain a desired body weight. Female athletes are faced with these 'body image' challenges, as well as the pressures to achieve the desired body weight for optimal performance in their sport. This attitude and preoccupation with food-related issues, referred to as *dietary restraint* or *cognitive dietary restraint* (Herman and Mack, 1975), has been associated with menstrual disturbances (Schweiger et al. 1992; Barr, Prior and Vigna 1994; Barr, Janelle and Prior 1994; Lebenstedt, Platte and Pirke 1999; McLean and Barr 2003), elevated cortisol levels (McLean, Barr and Prior 2001a; Anderson et al. 2002), and lower values for BMD/BMC (Van Loan and Kiem 2000; McLean, Barr and Prior 2001b) in previous studies. We have established that some female athletes, in this case runners, are experiencing restrained eating patterns, perhaps to achieve the combined effect of having the 'ideal body' as well as enhanced performance. This eating behaviour may be affecting their bone health and could potentially increase their risk for a stress fracture, as we have found an association between dietary restraint score and the occurrence of stress fractures.

Stress fractures are a common injury in female runners. The cause of stress fractures is multifactorial and the prevention of these fractures has proven to be difficult. An association between high levels of dietary restraint and the occurrence of stress

fractures was detected, and this finding may elucidate another risk factor presently overlooked due to the non-symptomatic nature of restrained eating patterns. Health professionals may benefit from research in this area through developing a better understanding of restrained eating, its potential association to bone health, and being able to recognize and evaluate these eating behaviours in the female athletic population.

3.2 Limitations

There are some limitations to the present study that must be considered when interpreting the data. Firstly, our data, which indicates that there is an association between high cognitive restraint and stress fractures, is limited to the sample population of women runners with characteristics reported elsewhere. Secondly, all information is self-reported. And thirdly, we did not measure menstrual cycle characteristics, cortisol excretion levels and BMD/BMC. Contingent to our hypothesis, that restraint may be a risk factor for stress fractures, was the presence of anovulation and/or short luteal phases and elevated cortisol levels resulting in lowered BMD and/or BMC as the mediating factors contributing to the cause of subsequent stress fracture. Menstrual cycle characteristics, BMD and hormonal levels were not measured in this study, therefore, we cannot conclude that these factors contributed to the stress fractures. Lastly, the cross-sectional design of this study precludes the ability to detect a cause and effect relationship (i.e. restraint causing stress fractures), and does not allow us to determine the occurrence, if any, of future stress fractures in the control group.

One additional item that warrants discussion was the finding that highest adult weight differed between the SF and NSF groups. There does not appear to be any logical

rationale for this observation, other than the fact that we completed numerous comparisons of means between the stress fracture and non-stress fracture groups, and an error level set at .05 would allow for one in 20 significant differences to happen by chance alone. Therefore, we dismiss any alternative explanation, and attribute the significantly higher value for highest adult weight in the SF compared to NSF group to chance alone.

Finally, there are some concerns regarding the tools that were chosen to measure various characteristics in the participants. Firstly, the TFEQ is presently the best available tool to assess dietary restraint, however this questionnaire was found may be somewhat flawed in a few ways. The questionnaire appears to be outdated with regard to the wording of numerous questions. For example, an active lifestyle, along with the eating habits of many individuals today, would commonly include eating more than three meals per day. However, one question asks whether true or false: "I am usually so hungry that I eat more than three times a day". This assumes that one is "hungry" if responded as "true", however, it may be very common to eat four or five smaller meals in a day. This "grazing" may be their chosen eating style, and not necessarily indicative of a positive association to the hunger construct.

Another drawback, is the fact that many questions make an assumption that an individual diets, at least some of the time. For example, some true or false statements begin with "While on a diet..." or "Dieting is so hard for me because..." Many individuals may never diet, and therefore an appropriate response to such questions is difficult to reach. These concerns are valid, as some participants did in fact state that they were confused by these questions, as such "dieting assumptions" did not apply to them.

In addition, the PSS appeared to be appropriate for the purpose of the study, and was easily understood by the participants. However, there were significant differences found between the groups in two of the questions (see appendix 5, questions 9 and 13 in PSS section of questionnaire) which may indicate that certain aspects of “daily stress” are more positively associated to those with high compared to low restraint.

3.3 Future Directions

Regardless of the limitations described above, this study reveals important relationships between restraint and stress fractures that are consistent with the literature surrounding restraint and bone. However, future studies could improve on and strengthen our findings. Ideally, future cross-sectional studies would compare three groups of women runners: 1) those with a current or past stress fracture; 2) those with other non-bony overuse injuries; and 3) those with no running related injuries (controls). Based on comparisons of these three groups, if restraint was significantly higher in the stress fracture compared to the other two groups, this would strengthen the hypothesis of restraint’s influence on bone. This relates to the possibility that disordered eating may be associated with personality traits, such as perfectionism (as assessed by the Eat-40 described previously), that may perhaps mediate a runner’s increased risk for all types of running related over-use injuries due to overtraining and/or “pushing themselves” to run through injuries. Body dissatisfaction has also been reported in restrained eaters (Lautenbacher et al. 1992; Davis et al. 1993), which again may be conducive to overtraining and increased risk of many types of injuries, not just bone. In addition, future studies would benefit from assessing luteal phase characteristics, through basal

body temperature readings or progesterone measurements taken from daily collections of saliva/urine (less invasive than blood samples) to confirm or dismiss the presence of menstrual cycle irregularities in restrained eaters. Measurements of urinary cortisol excretion levels, if elevated in restrained eaters, would also provide evidence of the mechanism through which the HPA axis activation may be impacting the menstrual cycle and BMD/BMC, and therefore stress fracture risk. Comparisons made from the aforementioned three-group cross-sectional study design, and collection of additional data (luteal phase characteristics and cortisol values) would provide a substantial amount of evidence to establish a clearer association or lack thereof, between restraint and stress fracture risk.

In addition, future research using a prospective design, which measures restraint (as well as related characteristics such as ovulation, luteal phase characteristics, and cortisol) and other risk factors, would be beneficial to determine independent risk factors for stress fractures and to establish a cause and effect relationship.

In summary, it is important that female athletes, as well as coaches and health professionals, are aware of the negative impacts of disordered eating behaviours as well as attitudes. Although it is well established that disordered eating *behaviours* and *practices* often result in menstrual cycle disturbances and bone loss, *attitudes* may also be detrimental to bone health over the long term.

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Questionnaire:

Eating Attitudes & Behaviour, and Athletic Injuries in Female Runners

The relationship between women and their eating habits and how this may be associated with athletic injuries is currently being researched.

Your participation in this study involves completing this questionnaire and a 3-day food diary. The questionnaire, which will take 15-20 min. to fill out, is a measure of a variety of attitudes, feelings and behaviours. The questions are related to food, physical activity and other information about yourself. There are no right or wrong answers so try to be completely honest in your answers. Results are completely confidential.

Please mail this questionnaire along with your 3-day food diary to U.B.C. with the pre-addressed, postage-paid envelope provided, within 10 days of receiving these documents. These documents are both coded with a number that was issued to your name when you received them from the researcher. This will allow you to mail them back to U.B.C. *without* including your name. Upon receiving the completed food diary and questionnaire from you, the researcher will match the code number to your name. This will allow us to mail you feedback on your diet.

You may decide at any point to discontinue your involvement in this study, with no consequences to your present or future treatment at the Allan McGavin Sports Medicine Centre or any other sports medicine clinic where you may have received treatment. It is assumed that if you complete this questionnaire, your consent has been given to participate in the study. For additional information or questions please contact Nanci Guest at 604-818-8348 or email: nanci@powerplayweb.com

Please be sure to answer ALL questions. Your best guess at least.
Thank you!

Part I. Activity

The following questions are related to your physical activity level at work, in sports (running is included as a sport) and in your leisure time.

Please fill in the blanks with your response or CIRCLE your response when given a choice.

Don't use blanks

1. What is your main occupation?

2. At work I sit:

1) never 2) seldom 3) sometimes 4) often 5) always

3. At work I stand:

1) never 2) seldom 3) sometimes 4) often 5) always

4. At work I walk:

1) never 2) seldom 3) sometimes 4) often 5) always

5. At work I lift heavy loads:

1) never 2) seldom 3) sometimes 4) often 5) always

6. After work I am tired:

1) never 2) seldom 3) sometimes 4) often 5) always

7. At work I sweat:

1) never 2) seldom 3) sometimes 4) often 5) always

8. In comparison with others my own age I think my work is physically:

1) much heavier 2) heavier 3) as heavy 4) lighter 5) much lighter

9a. Do you play sport? (Running is included as a sport)

_____ no (If no, go to question 10 on the next page and continue)

_____ yes: If yes, continue... Which sport do you play most frequently?

9b. How many hours a week do you play/perform this sport ?

_____ less than 1 hr/wk

_____ 1 – 2 hr/wk

_____ 3 – 4 hr/wk

_____ more than 4 hr/wk

9c. How many months a year do you play/perform this sport?

_____ less than 1 month

_____ 1-3 months

_____ 4-6 months

_____ 7-9 months

_____ more than 9 months

9d. Do you play/perform a second sport?

- ☐ no (If no, go to question 10 below and continue)
☐ yes: If yes, continue.... What is this second sport?

9e. How many hours a week do you play/perform this sport?

- ☐ less than 1 hr/wk
☐ 1 - 2 hr/wk
☐ 2 - 3 hr/wk
☐ 3 - 4 hr/wk
☐ more than 4 hr/wk

9f. How many months a year?

- ☐ less than 1 month
☐ 1-3 months
☐ 4-6 months
☐ 7-9 months
☐ more than 9 months

➔ 10. In comparison with others my own age I think my physical activity during **leisure** time is:

- 1) much more 2) more 3) the same 4) less 5) much less

11. During leisure time I sweat:

- 1) very often 2) often 3) sometimes 4) seldom 5) never

12. During leisure time I play sport:

- 1) very often 2) often 3) sometimes 4) seldom 5) never

13. During leisure time I watch television:

- 1) very often 2) often 3) sometimes 4) seldom 5) never

14. During leisure time I walk:

- 1) very often 2) often 3) sometimes 4) seldom 5) never

15. During leisure time I cycle:

- 1) very often 2) often 3) sometimes 4) seldom 5) never

16. How many minutes do you walk and/or cycle per day to and from work, school, and shopping?

- ☐ less than 5 min/d
☐ 5 - 15 min/d
☐ 15 - 30 min/d
☐ 30 - 45 min/d
☐ more than 45 min/d

Part II. Eating Behaviour.

Please CIRCLE whether the statements below are true (T) or false (F) for you.

Don't use blanks

- | | | | |
|--------------------------------------------------------------------------------------------------------------------------------------------------------------------|---|---|-------|
| 1. When I smell the aroma of my favourite food, I find it very difficult to keep from eating, even if I have just finished a meal | T | F | _____ |
| 2. I usually eat too much at social occasions, like parties and picnics | T | F | _____ |
| 3. I am usually so hungry that I eat more than three times a day | T | F | _____ |
| 4. When I have eaten my quota of calories, I am usually good about not eating any more | T | F | _____ |
| 5. Dieting is so hard for me because I just get too hungry | T | F | _____ |
| 6. I deliberately take small helpings as a means of controlling my weight | T | F | _____ |
| 7. Sometimes things just taste so good that I keep on eating even when I am no longer hungry | T | F | _____ |
| 8. Since I am often hungry, I sometimes wish that while I am eating, an expert would tell me that I have had enough or that I can have something more to eat | T | F | _____ |
| 9. When I feel anxious, I find myself eating | T | F | _____ |
| 10. Life is too short to worry about dieting | T | F | _____ |
| 11. Since my weight goes up and down, I have gone on reducing diets more than once | T | F | _____ |
| 12. I often feel so hungry that I just have to eat something | T | F | _____ |
| 13. When I am with someone who is overeating, I usually overeat too | T | F | _____ |
| 14. I have a pretty good idea of the number of calories in common food | T | F | _____ |
| 15. Sometimes when I start eating, I just can't stop | T | F | _____ |
| 16. It is not difficult for me to leave something on my plate | T | F | _____ |
| 17. At certain times of the day, I get hungry because I have gotten used to eating then | T | F | _____ |
| 18. While on a diet, if I eat food that is not allowed, I consciously eat less for a period of time to make up for it | T | F | _____ |
| 19. Being with someone who is eating often makes me hungry enough to eat also | T | F | _____ |
| 20. When I feel blue, I often overeat | T | F | _____ |
| 21. I enjoy eating too much to spoil it by counting calories or watching my weight | T | F | _____ |
| 22. When I see a real delicacy, I often get so hungry that I have to eat right away | T | F | _____ |

- | | | | |
|----------------------------------------------------------------------------------------------------------------------|---|---|-------|
| 23. I often stop eating when I am not really full as a conscious means of limiting the amount I eat..... | T | F | _____ |
| 24. I get so hungry that my stomach often seems like a bottomless pit | T | F | _____ |
| 25. My weight has hardly changed at all in the last two years | T | F | _____ |
| 26. I am always hungry so it is hard for me to stop eating before I finish the food on my plate..... | T | F | _____ |
| 27. When I feel lonely, I console myself by eating..... | T | F | _____ |
| 28. I consciously hold back at meals in order not to gain weight..... | T | F | _____ |
| 29. I sometimes get very hungry late in the evening or night..... | T | F | _____ |
| 30. I eat anything I want, any time I want | T | F | _____ |
| 31. Without even thinking about it, I take a long time to eat..... | T | F | _____ |
| 32. I count calories as a conscious means of controlling my weight | T | F | _____ |
| 33. I do not eat some foods because they make me fat | T | F | _____ |
| 34. I am always hungry enough to eat any time..... | T | F | _____ |
| 35. I pay a great deal of attention to changes in my figure..... | T | F | _____ |
| 36. While on a diet, if I eat a food that is not allowed, I often then splurge and eat other high calorie foods..... | T | F | _____ |

Please answer the following questions by CIRCLING the response that is appropriate to you.

- | | | | | | |
|------------------------------------------------------------------------------------------------------------------|-----------------------|---------------|-------------------|--|-------|
| 37. How often are you dieting in a conscious effort to control your weight? | | | | | _____ |
| 1) never | 2) sometimes | 3) usually | 4) always | | |
| 38. Would a weight fluctuation of 5 lbs affect the way you live your life? | | | | | _____ |
| 1) not at all | 2) slightly | 3) moderately | 4) very much | | |
| 39. How often do you feel hungry? | | | | | _____ |
| 1) never | 2) sometimes | 3) usually | 4) always | | |
| 40. Do your feelings of guilt about overeating help you to control your food intake? | | | | | _____ |
| 1) never | 2) rarely | 3) often | 4) always | | |
| 41. How difficult would it be for you to stop eating halfway through dinner and not eat for the next four hours? | | | | | _____ |
| 1) easy | 2) slightly difficult | 3) moderately | 4) very difficult | | |

42. How conscious are you of what you are eating?
1) not at all 2) slightly 3) moderately 4) extremely _____
43. How frequently do you avoid 'stocking up' on tempting foods?
1) almost never 2) seldom 3) usually 4) almost always _____
44. How likely are you to shop for low calorie foods?
1) unlikely 2) slightly likely 3) moderately likely 4) very likely _____
45. Do you eat sensibly in front of others and splurge alone?
1) never 2) sometimes 3) often 4) always _____
46. How likely are you to consciously eat slowly in order to cut down on how much you eat?
1) unlikely 2) slightly likely 3) moderately likely 4) very likely _____
47. How frequently do you skip dessert because you are no longer hungry?
1) almost never 2) seldom 3) at least once/week 4) almost daily _____
48. How likely are you to consciously eat less than you want?
1) unlikely 2) slightly likely 3) moderately likely 4) very likely _____
49. Do you go on eating binges though you are not hungry?
1) never 2) rarely 3) sometimes 4) at least weekly _____
50. On a scale of 0 to 5, where 0 means no restraint in eating (eating whatever you want, whenever you want it) and 5 means total restraint (constantly limiting food intake and never 'giving in'), what number would you give yourself? (please circle the number)
0) eat whatever you want, whenever you want it _____
1) usually eat whatever you want, whenever you want it
2) often eat whatever you want, whenever you want it
3) often limit food intake, but often 'give in'
4) usually limit food intake, rarely 'giving in'
5) constantly limit food intake, never 'giving in'
51. To what extent does this statement describe your eating behaviour? "I start dieting in the morning, but because of any number of things that happen during the day, by evening I have given up and eat what I want, promising myself to start dieting again tomorrow."
1) not like me 2) a little like me 3) pretty good description of me 4) describes me perfectly _____

Part III: Feelings and Thoughts

The questions in this scale ask you about your feelings and thoughts during the last month. In each case you will be asked how *often* you felt or thought in a certain way.

Although some of the questions are similar, there are differences between them and you should treat each one as a separate question. The best approach is to answer each question fairly quickly.

Please answer each question by **CIRCLING** the number above the response which best applies to you.

1. In the last month, how often have you been upset because of something that happened unexpectedly? Don't use blanks
- | | | | | | |
|-------|-----------------|-----------|-----------------|---------------|-------|
| 0 | 1 | 2 | 3 | 4 | |
| never | almost
never | sometimes | fairly
often | very
often | _____ |
2. In the last month, how often have you felt that you could not control the important things in your life?
- | | | | | | |
|-------|-----------------|-----------|-----------------|---------------|-------|
| 0 | 1 | 2 | 3 | 4 | |
| never | almost
never | sometimes | fairly
often | very
often | _____ |
3. In the last month, how often have you dealt successfully with irritating life hassles?
- | | | | | | |
|-------|-----------------|-----------|-----------------|---------------|-------|
| 0 | 1 | 2 | 3 | 4 | |
| never | almost
never | sometimes | fairly
often | very
often | _____ |
4. In the last month, how often have you felt you were effectively coping with important changes that were occurring in your life?
- | | | | | | |
|-------|-----------------|-----------|-----------------|---------------|-------|
| 0 | 1 | 2 | 3 | 4 | |
| never | almost
never | sometimes | fairly
often | very
often | _____ |
5. In the last month, how often have you felt confident about your ability to handle your personal problems?
- | | | | | | |
|-------|-----------------|-----------|-----------------|---------------|-------|
| 0 | 1 | 2 | 3 | 4 | |
| never | almost
never | sometimes | fairly
often | very
often | _____ |
6. In the last month, how often have you felt that things were going your way?
- | | | | | | |
|-------|-----------------|-----------|-----------------|---------------|-------|
| 0 | 1 | 2 | 3 | 4 | |
| never | almost
never | sometimes | fairly
often | very
often | _____ |
7. In the last month, how often have you found you could not cope with all of the things you had to?
- | | | | | | |
|-------|-----------------|-----------|-----------------|---------------|-------|
| 0 | 1 | 2 | 3 | 4 | |
| never | almost
never | sometimes | fairly
often | very
often | _____ |
8. In the last month, how often have you been able to control irritations in your life?
- | | | | | | |
|-------|-----------------|-----------|-----------------|---------------|-------|
| 0 | 1 | 2 | 3 | 4 | |
| never | almost
never | sometimes | fairly
often | very
often | _____ |

9. In the last month, how often have you felt that you were on top of things?

0	1	2	3	4	_____
never	almost never	sometimes	fairly often	very often	

10. In the last month, how often have you been angered because of things that happened that were outside of your control?

0	1	2	3	4	_____
never	almost never	sometimes	fairly often	very often	

11. In the last month, how often have you found yourself thinking about things you have to accomplish?

0	1	2	3	4	_____
never	almost never	sometimes	fairly often	very often	

12. In the last month, how often have you been able to control the way you spend your time?

0	1	2	3	4	_____
never	almost never	sometimes	fairly often	very often	

13. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?

0	1	2	3	4	_____
never	almost never	sometimes	fairly often	very often	

Part IV. Information About You

The following information will help us interpret the results of the questionnaire. It's very important that all questions be completed. If you don't know the exact value for any of the questions, give your best estimate.

Demographic Data

1. What is your present age?

_____ years _____

2. How tall are you (without shoes)?

_____ cm, or _____ feet, inches _____

3. What is your present weight (without clothes)?

_____ kg, or _____ lbs _____

Weight History

4. What has been your highest adult (18 yrs. or older) weight?

_____ kg, or _____ lbs _____

5. At what weight do you feel your best?

_____ kg, or _____ lbs _____

6. Have you ever tried to lose weight?

_____ yes

_____ no

7. Are you presently trying to lose weight?

_____ yes

_____ no

8. How many times, in the past 2 years, have you lost more than 5 pounds?

Menstrual Cycle Information

9. Are you currently having menstrual cycles?

_____ no (if no, skip to question 15 below and continue)

_____ yes, irregularly (continue to question 10)

_____ yes, regularly (every 21-35 days) (continue to question 10)

10. Are you currently taking birth control pills?

_____ yes

_____ no

11. Have you taken birth control pills in the past 6 months?

_____ yes

_____ no

12. On what day did your last menstrual cycle begin?

13. What is today's date?

14. What is the average length of your cycle? (* this is the number of days from the beginning of one menstrual cycle to the start of the next i.e. March 2 - March 28 = 26 days)

_____ days

15. Have you ever been pregnant?

_____ yes

_____ no

16. What was your age of menarche? (when you began your first menstruation)

_____ years

Other Information

17. What is the approximate distance that you run per week?

_____ km or _____ miles

18. How many years have you been running?

_____ years

19. Have you ever been diagnosed with or treated for an eating disorder?

_____ yes
_____ no

20. Are you currently on any of the following diets?

_____ Lacto-ovo vegetarian (no meat, poultry or fish but including dairy products such
as milk, cheese, yogurt and eggs)
_____ Vegan (no animal products of any kind)
_____ Other (please describe):

21. How many cups of caffeinated beverage (coffee/tea/colas) do you drink in a day?

_____ cups/d

22. Do you smoke cigarettes?

_____ yes
_____ no

23. What is the average number of drinks of alcohol you consume in a week? (eg: 1 drink = 1
beer or cider, 3oz (100ml) wine, 1oz (30ml) hard liquor)

_____ drinks per week

24. Please list any medications that you are currently taking: (if none, write "NONE")

25. Are you currently taking any vitamin, mineral or herbal supplements?

_____ no
_____ yes

If yes, please list which vitamin, mineral or herbal supplement (or for a multivitamin or
herbal formula the brand name), in what dosage and how frequently you take it (if known).

<u>Vitamin/Mineral/Herb</u>	<u>Dosage</u>	<u>Frequency</u>
(Example: Calcium	500 mg	1 per day)
_____	_____	_____
_____	_____	_____
_____	_____	_____

If more space is needed, please use the back of this page.

PLEASE CHECK THAT YOU HAVE ANSWERED ALL QUESTIONS!

THANK-YOU!

.....

If you would like a summary of our results, please check: ____ Yes ____ No

Appendix 6

Three-day Dietary Intake Guidelines

An accurately completed dietary intake record can provide valuable information about the nutritional content of an individual's usual diet. Please try and maintain your normal eating patterns in terms of content and quantity of foods consumed during this 3-day period. Please keep record of everything you eat or drink on the attached forms for three days in a row (including 1 weekend day). Please be as specific and as detailed as possible.

- To ensure accuracy please try to record immediately after eating.
- The more accurately you record, the more meaningful is the analysis!

Be sure to include:

1. ALL FOODS AND DRINKS consumed including snacks, soft drinks, alcohol, cream and sugar in coffee/tea, butter/sauces on vegetables, jams, relishes, candies, butter/margarine/mayonnaise on sandwiches, salad dressing. Break combination foods down into their constituents (e.g. ham and cheese omelette = 3 eggs + 1 oz. Cheddar cheese + 1 slice Oscar Meyer Packaged ham slices + 1 tsp butter in pan)

2. THE AMOUNT OF FOOD that was consumed. It is extremely important for assessment purposes that accurate measurements be recorded. It may be helpful to measure the volume of your regular glasses, bowls and cups before you begin.

- Use VOLUME measures such as cups, tablespoons (tbs.), teaspoons (tsp.) or millilitres (mls) for soup, pasta, cereals, rice, other grains, small or cut vegetables, cut fruit, tinned foods, drinks, sauces, salad dressings, butter, mayonnaise, margarine, jams, peanut butter etc. Please be as accurate as possible. For example, record if a tablespoon is 'heaping' as opposed to 'level'.
- Use WEIGHTS (ounces or grams) or meat, fish, poultry, cheese. Use the labels on packages to help you. If you are dining out, record the size of the piece of the meat eg sirloin steak 3" by 4" by ½", or hamburger patty 3" diameter by ½".
- Use SIZES for whole fruits, whole vegetables, cookies, cakes, eggs, cheese pieces etc. Either specify small, medium or large or give dimensions. In some cases it may be more appropriate to give size in relation to a whole. E.g. ½ medium pepperoni pizza, piece of cheddar cheese 2" by 3" by 1", 1 small apple, 1 large bran muffin.

3. THE BRAND NAMES OR TYPE OF FOOD.

Examples:

- Sunrise soft tofu - 1/2 cup
- Benny's whole wheat bagel
- 1% milk - 1.5 cups
- 4 Oreo cookies

4. THE TIME OF DAY the foods and beverages were consumed.

Page 1 of Day 1 Three-Day Food Record Forms

Date	Time	Complete Description of Food or Beverage	Portion Size

Page 2 of Day 1 Three-Day Food Record Forms

Date	Time	Complete Description of Food or Beverage	Portion Size

Was this a typical day for you? Yes _____ No _____

If not please give reason(s):

Page 1 of Day 2 Three-Day Food Record Forms

Date	Time	Complete Description of Food or Beverage	Portion Size

Page 2 of Day 2 Three-Day Food Record Forms

Date	Time	Complete Description of Food or Beverage	Portion Size

Was this a typical day for you? Yes _____ No _____

If not please give reason(s):

Page 1 of Day 3 Three-Day Food Record Forms

Date	Time	Complete Description of Food or Beverage	Portion Size

Page 2 of Day 3 Three-Day Food Record Forms

Date	Time	Complete Description of Food or Beverage	Portion Size

Was this a typical day for you? Yes _____ No _____

If not please give reason(s):

Appendix 7

BQHPA^a question comparisons grouped according to stress fracture^{b,c}.

	Non-Stress Fracture (n=41)	Stress Fracture (n=38)	t	P-value
Sitting at work	3.5 ± .93	3.5 ± 1.0	-.18	.87
Standing at work	3.0 ± .79	3.1 ± .90	-.82	.42
Walking at work	3.3 ± .78	3.2 ± .71	.33	.74
Lifting heavy loads at work	1.9 ± .93	2.0 ± .96	-.34	.73
Tired after work	3.0 ± .76	3.3 ± .70	-1.8	.08
Sweating at work	1.9 ± .82	1.7 ± .76	1.07	.29
Physical demands at work in comparison to others my age	3.3 ± 1.1	3.4 ± .97	-.22	.82
Total job activity index	2.5 ± .40	2.5 ± .40	-.41	.68
Amount of time per week main sport played or performed (hr)	4.2 ± .86	4.3 ± .92	-.10	.92
Amount of time per year main sport played or performed (mo)	4.8 ± .59	4.8 ± .44	-.11	.91
Total sport activity index	2.2 ± .41	2.3 ± .43	-1.1	.26
Physical activity during leisure time in comparison to others my own age	1.7 ± .61	1.9 ± .76	-1.4	.18

	Non-Stress Fracture (n=41)	Stress Fracture (n=38)	t	P-value
Sweating during leisure time	2.0 ± .90	2.3 ± .80	-1.6	.11
Sport playing during leisure time	2.0 ± .72	2.0 ± .72	-.31	.76
Watching television during leisure time	3.2 ± .74	3.5 ± .86	-1.8	.07
Walking during leisure time	2.5 ± .84	2.5 ± .90	-.20	.84
Cycling during leisure time	3.1 ± 1.1	3.3 ± 1.1	-.88	.38
Daily time spent walking and/or cycling to and from work, school, and shopping	3.3 ± 1.1	3.3 ± 1.1	.11	.92
Total leisure activity index	2.9 ± .39	2.9 ± .47	.25	.80
Total activity index	7.7 ± .65	7.8 ± .68	-.81	.42
Running distance (km·wk ⁻¹)	33.4 ± 13.4	35.7 ± 13.5	-.75	.46
Length of time running (yr)	6.7 ± 4.5	8.2 ± 4.9	-1.45	.15

^a Baecke Questionnaire of Habitual Physical Activity (Baecke, Burema and Fritjers 1982).

^b Mean ± SD.

^c A higher value indicates a greater positive association to the question.

Appendix 8

BHQPA^a secondary sport questions grouped according to presence of stress fracture^b

	Non-stress Fracture (n=39)	Stress Fracture (n=38)	t	P-value
Do you play a second sport? (%)	78.0	81.6	1.91	.39
Amount of time per week second sport played/performed (hr)	2.7 ± 1.7	2.6 ± 1.7	.34	.74
Amount of time per year second sport played/performed (mo)	3.4 ± 1.9	3.4 ± 1.9	.17	.87

^a Baecke Questionnaire of Habitual Physical Activity (Baecke, Burema and Fritjers 1982).

^b Means ± SD.

Appendix 9

TFEQ^a question comparisons grouped according to presence of stress fracture^{b,c}

	Non-Stress Fracture (n=41)	Stress Fracture (n=38)	t	P-value
When I smell the aroma of my favourite food, I find it very difficult to keep from eating, even if I have just finished a meal	1.5 ± .51	1.5 ± .51	.10	.93
I usually eat too much at social occasions, like parties and picnics	1.3 ± .47	1.6 ± .50	-2.4	.02
I am usually so hungry that I eat more than three times a day	1.3 ± .45	1.3 ± .47	-.46	.65
When I have eaten my quota of calories, I am usually good about not eating any more	1.8 ± .44	1.6 ± .50	1.9	.06
Dieting is so hard for me because I just get too hungry	1.5 ± .51	1.6 ± .50	-.80	.42
I deliberately take small helpings as a means of controlling my weight	1.7 ± .47	1.6 ± .49	.48	.64
Sometimes things just taste so good that I keep on eating even when I am no longer hungry	1.2 ± .42	1.3 ± .48	-1.2	.23
Since I am often hungry, I sometimes wish that while I am eating, an expert would tell me that I have had enough or that I can have something more to eat	1.7 ± .48	1.8 ± .41	-1.3	.20
When I feel anxious, I find myself eating	1.6 ± .50	1.7 ± .48	-.66	.51
Life is too short to worry about dieting	1.5 ± .51	1.5 ± .51	-.09	.93
Since my weight goes up and down, I have gone on reducing diets more than once	1.7 ± .45	1.8 ± .41	-.59	.55
I often feel so hungry that I just have to eat something	1.4 ± .49	1.5 ± .51	-.96	.34
When I am with someone who is overeating, I usually overeat too	1.5 ± .51	1.8 ± .39	-3.0	< .01
I have a pretty good idea of the number of calories in common food	1.4 ± .50	1.3 ± .48	.66	.51
Sometimes when I start eating, I just can't stop	1.6 ± .50	1.7 ± .45	-1.4	.16

	Non-Stress Fracture (n=41)	Stress Fracture (n=38)	t	P-value
It is not difficult for me to leave something on my plate	1.5 ± .51	1.4 ± .50	1.3	.21
At certain times of the day, I get hungry because I have gotten used to eating then	1.4 ± .49	1.3 ± .46	.72	.48
While on a diet, if I eat food that is not allowed, I consciously eat less for a period of time to make up for it	1.7 ± .45	1.7 ± .48	.71	.48
Being with someone who is eating often makes me hungry enough to eat also	1.5 ± .51	1.7 ± .48	-1.5	.13
When I feel blue, I often overeat	1.6 ± .49	1.7 ± .45	-.97	.33
I enjoy eating too much to spoil it by counting calories or watching my weight	1.6 ± .49	1.7 ± .48	-.22	.83
When I see a real delicacy, I often get so hungry that I have to eat right away	1.8 ± .44	1.8 ± .39	-.64	.53
I often stop eating when I am not really full as a conscious means of limiting the amount I eat	1.9 ± .33	1.7 ± .47	2.1	.04
I get so hungry that my stomach often seems like a bottomless pit	1.7 ± .45	1.7 ± .45	-.05	.96
My weight has hardly changed at all in the last two years	1.3 ± .46	1.3 ± .46	.03	.98
I am always hungry so it is hard for me to stop eating before I finish the food on my plate	1.8 ± .44	1.8 ± .43	-.07	.94
When I feel lonely, I console myself by eating I consciously hold back at meals in order not to gain weight	1.7 ± .47	1.8 ± .41	-1.07	.29
I consciously hold back at meals in order not to gain weight	1.8 ± .40	1.5 ± .50	3.5	< .01
I sometimes get very hungry late in the evening or night	1.5 ± .51	1.4 ± .50	.59	.56
I eat anything I want, any time I want	1.6 ± .50	1.8 ± .39	-2.3	.03

	Non-Stress Fracture (n=41)	Stress Fracture (n=38)	t	P-value
Without even thinking about it, I take a long time to eat	1.8 ± .44	1.7 ± .48	.95	.34
I count calories as a conscious means of controlling my weight	1.9 ± .36	1.8 ± .43	1.0	.32
I do not eat some foods because they make me fat	1.4 ± .49	1.3 ± .45	.97	.33
I am always hungry enough to eat any time	1.9 ± .36	1.8 ± .37	.14	.89
I pay a great deal of attention to changes in my figure	1.3 ± .46	1.3 ± .45	.23	.77
While on a diet, if I eat a food that is not allowed, I often then splurge and eat other high calorie foods	1.7 ± .45	1.8 ± .39	-.88	.38
How often are you dieting in a conscious effort to control your weight?	1.7 ± .79	1.9 ± .88	-1.3	.21
Would a weight fluctuation of 5 lbs affect the way you live your life?	2.1 ± .86	2.2 ± .93	-.43	.67
How often do you feel hungry?	2.3 ± .58	2.3 ± .48	-.01	1.0
Do your feelings of guilt about overeating help you to control your food intake?	2.2 ± .69	2.4 ± .83	-1.6	.11
How difficult would it be for you to stop eating halfway through dinner and not eat for the next four hours?	2.9 ± .94	2.6 ± .85	1.1	.28
How conscious are you of what you are eating?	3.3 ± .51	3.3 ± .66	-.17	.86
How frequently do you avoid 'stocking up' on tempting foods?	2.7 ± .81	3.2 ± .81	-2.6	.01
How likely are you to shop for low calorie foods?	2.6 ± 1.0	3.1 ± 1.0	-2.4	.02
Do you eat sensibly in front of others and splurge alone?	1.7 ± .69	1.6 ± .64	.70	.50
How likely are you to consciously eat slowly in order to cut down on how much you eat?	1.7 ± .64	1.6 ± .82	.46	.65

	Non-Stress Fracture (n=41)	Stress Fracture (n=38)	t	P-value
How frequently do you skip dessert because you are no longer hungry?	3.2 ± 1.0	3.3 ± .89	-.43	.67
How likely are you to consciously eat less than you want?	1.9 ± .69	2.0 ± .87	-.70	.49
Do you go on eating binges though you are not hungry?	2.2 ± .95	2.1 ± .80	.45	.65
On a scale of 0 to 5, where 0 means no restraint in eating (eating whatever you want, whenever you want it) and 5 means total restraint (constantly limiting food intake and never 'giving in'), what number would you give yourself?	2.1 ± 1.0	2.6 ± 1.1	-2.2	.03
Response to: "I start dieting in the morning, but because of any number of things that happen during the day, by evening I have given up and eat what I want, promising myself to start dieting again tomorrow."	1.6 ± .74	1.5 ± .69	.21	.83

^a Three-Factor Eating Questionnaire (Stunkard and Messick 1985).

^b Means ± SD.

^c These questions were answered true or false. A higher value indicates a greater positive association to the question.

Appendix 10

PSS^a question comparisons grouped according to presence of stress fracture^b.

	Non-Stress Fracture (n=41)	Stress Fracture (n=38)	t	P-value
In the last month, how often have you been upset because of something that happened unexpectedly?	1.6 ± .77	2.0 ± .77	-1.8	.07
In the last month, how often have you felt that you could not control the important things in your life?	1.6 ± 1.0	1.7 ± .78	-.23	.82
In the last month, how often have you dealt successfully with irritating life hassles?	2.7 ± .66	2.7 ± .78	-.16	.87
In the last month, how often have you felt you were effectively coping with important changes that were occurring in your life?	2.8 ± .77	2.8 ± .58	.26	.80
In the last month, how often have you felt confident about your ability to handle your personal problems?	3.0 ± .79	2.9 ± .85	.30	.77
In the last month, how often have you felt that things were going your way?	2.7 ± .74	2.7 ± .85	.41	.68
In the last month, how often have you found you could not cope with all of the things you had to?	1.4 ± .97	1.6 ± .94	-1.2	.22
In the last month, how often have you been able to control irritations in your life?	2.7 ± .69	2.8 ± .66	-.70	.49
In the last month, how often have you felt that you were on top of things?	2.7 ± .75	2.3 ± .87	2.3	.02
In the last month, how often have you been angered because of things that happened that were outside of your control?	1.8 ± .89	1.9 ± .82	-.86	.39
In the last month, how often have you found yourself thinking about things you have to accomplish?	3.2 ± .77	3.2 ± .75	.50	.62
In the last month, how often have you been able to control the way you spend your time?	2.8 ± .73	2.7 ± .81	.41	.68

	Non-Stress Fracture (n=41)	Stress Fracture (n=38)	t	P-value
In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?	1.1 ± .93	1.6 ± .92	-2.3	.02
Perceived Stress Score	23.9 ± 5.0	25.1 ± 4.7	- 1.1	.29

^a Perceived Stress Scale (Cohen, Kamarck and Mermelstein 1983).

^b Means ± SD

Appendix 11

Physical and menstrual cycle characteristics, weight history and running activity grouped according to presence of stress fracture^a.

Characteristic	Non-stress Fractures (n=41)	Stress Fracture (n=38)	t	P-value
Age (yr)	29.1 ± 5.0	29.2 ± 5.5	-.05	.96
Height (cm)	165.1 ± 5.3	166.8 ± 6.9	-1.2	.24
Weight (kg)	60.2 ± 7.8	59.1 ± 6.6	.64	.53
Body mass index (kg·m ⁻²)	22.0 ± 2.5	21.2 ± 1.8	1.6	.12
Highest adult weight (kg)	66.3 ± 10.4	62.0 ± 7.2	2.1	.04
Best weight (kg) ^b	58.0 ± 6.7	56.8 ± 5.2	.16	.40
Attempted to lose weight in the past (%)	92.7	78.9	3.1	.08
Presently attempting to lose weight (%)	48.8	44.7	.13	.72
Number of 5 lb weight losses in the past 2 years	1.2 ± 1.2	1.3 ± 1.8	.60	.84
Currently using oral contraceptive (%)	34.1	50.0	2.0	.15
Use of oral contraceptive in past 6 mo (%)	43.9	57.9	1.5	.21
Menstrual cycle length (d)	28.6 ± 2.7	28.0 ± 2.9	.86	.39
Age of menarche (yr)	13.0 ± 1.5	13.4 ± 1.9	-1.1	.26
Previous pregnancy (%)	9.8	5.3	.57	.45
Running distance (km·wk ⁻¹)	33.4 ± 13.4	35.7 ± 13.5	-.75	.46
Length of time running (yr)	6.7 ± 4.5	8.2 ± 4.9	-1.45	.15

^a Means ± SD.

^b Best weight: weight at which participants indicated they felt their best.

^c Pregnancy occurred, but not carried to term (all women were nulliparous).

Appendix 12

Daily nutrient intakes^a, vegetarianism, and alcohol^a, caffeine^a cigarette and supplement use grouped according to presence of stress fracture.

Characteristics	Non-Stress Fracture (n = 41)	Stress Fracture (n = 38)	t	P-value
Calories (kcal)	1948.1 ± 316.5	1919.7 ± 375.4	.37	.72
Protein (g)	79.5 ± 17.5	83.7 ± 23.3	-.91	.37
Carbohydrates (g)	278.5 ± 73.7	269.7 ± 73.2	.54	.59
Fat (g)	57.2 ± 15.2	53.9 ± 14.5	.98	.33
Fibre (g)	25.8 ± 10.5	22.9 ± 8.2	1.4	.18
Dietary calcium (mg)	916.4	1049.4	-1.6	.10
Supplemental calcium (mg)	107.1	240.2	-1.8	.07
Total calcium (mg)	1023.5 ± 361.4	1289.6 ± 524.0	-2.6	.01
Dietary vitamin D (IU)	100.0	164.2	-2.8	.01
Supplemental vitamin D (IU)	4.9	49.6	-1.6	.13
Total vitamin D (IU)	104.9 ± 74.1	213.7 ± 212.3	-3.0	<.01
Dietary iron (mg)	16.7	15.9	.67	.51
Supplemental iron (mg)	1.2	4.9	-1.7	.10
Total iron (mg)	18.0 ± 6.9	20.8 ± 15.2	-1.1	.30
Caffeinated beverages (cup/d)	1.6 ± 1.3	1.5 ± 0.9	.44	.66
Alcoholic beverages (drinks/wk)	2.2 ± 2.4	2.6 ± 2.6	-.67	.50
Vitamin-mineral/supplement use (%)	36.6	34.2	1.1	.57
Vegetarian (%) ^b	24.4	23.7	.01	.94
Cigarette Smokers (%)	2.4	2.6	2.0	.37

^a Mean ± SD.

^b Vegetarian refers to those who exclude meat and poultry. Two vegetarians consumed fish occasionally.