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The Female Athlete Triad: A Literature Review

A culturally influenced physiological manifestation of the combination of amenorrhea, osteoporosis, and disordered eating among female athletes.

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Paper Advisor: Dr. Stephen Glass
Introduction:

A Culture of Sport and Competition in America

“Winning isn’t everything. It’s the only thing.” –Vince Lombardi

The words of a man who is revered for his success as a professional football coach ring true today in a society where competition drives most everything- especially sports. To be the best is to be unstoppable, to have insurmountable strength, and to carve your name into history. This attitude towards sports has created phenomenal athletes that achieve things not thought possible. Today men are breaking 10.00 seconds in the 100m dash and approaching the 2 hour mark in the ultimate test of endurance strength- the marathon. Women are not far behind in these athletic achievements. In fact, since the passage of Title IX in 1972, which provided increased access and opportunity for women’s participation in sport, women have established their own athletic prowess (Troy, Hoch, & Stravrakos, 2006). The desired increase in participation and opportunities for women at all levels of sport, which propelled the proponents of Title IX, was hoped to empower young women through improved physical fitness, character development opportunities, and increased self-esteem (Doherty, 1999). Today it is clear that the intent of Title IX and its’ proponents have been realized. Women’s athletics has grown significantly since the 1970s at all levels: high school, collegiate, and professional. With the increased numbers participating, the level of competition is much higher. A higher level of competition requires a greater performance and fitness. The current athletic emphasis is on improving physical fitness- at any cost; driven by parents, coaches, fans, cultural pressures and internal motivation to see success.

Prior to the 1970’s women in sport and athletics was limited, with the majority of high school, college, and professional sport participation being men. The male dominate sport culture
both prevented access to sport for women, and also limited attention given to the female athlete. With the passage of Title IX of the Education Amendments of 1972 which states that: “No person in the United States shall, on the basis of sex, be excluded from participation in, be denied the benefits of, or be subjected to discrimination under any education program or activity receiving Federal financial assistance,” increased access and attention was paid to women’s sports (United States House Subcommittee on Higher Education of the Education and Labor Committee, 1972). Title IX mandates women not be withheld the same opportunities afforded to men in the classroom, in admissions, in scholarship, and in extra-curricular activities (i.e. sports teams). Title IX has increased the number of female participants in sport and athletics due to increased access and facilities for women in athletics. The improvement in women’s athletics has led to great performances, incredible athletes, and increased participation of young girls in sport. Girls’ participation in high school sports has increased from 7% in 1972 to 42% today (Thein-Nissenbaum, 2013). Title IX opened the door for women’s athletics; it allowed women to be seen as capable of competing at as high a level as men and gave women opportunities to achieve their own level of success within their own sport(s).

At the time of the release of Title IX it was determined that “athletic women receive the same significant cardiovascular, musculoskeletal, and psychological benefits as men” (Nazem & Ackerman, 2012). The range of participation across sports for women has increased dramatically, with women playing in once male-only sports such as ice hockey, soccer, and rugby. The first women Olympic marathon winner in 1984, Joan Bennoite-Samuelson, held a world record of 2 hours, 22 minutes, while the current record is 2 hours, 15 minutes. Clearly the progression in participation and performance of women has seen dramatic changes in the past 40 years. However, the culture of female athletics is always judged by how far behind women fall
from the men in their respective sport. This gap between male and female performance, skill, speed, and ability level has bred a culture of perfection within female athletics. This performance gap exists because estrogen does not allow for the same musculoskeletal growth and strength development that testosterone allows in men (ACSM, 2007). Also, women menstruate every month, which lowers hemoglobin levels, and thus, lowers the ability to transport oxygen. The difference in male and female performance comes directly from maximal oxygen consumption decreases in women due to lower hemoglobin levels (Torstveit & Sundgot-Borgen, 2005). Unjustifiably based on this performance gap, a culture of female incompetence has developed in the last 30 plus years (Leburn, 2007). The current sport culture has forced women to believe that the only way to achieve success and prestige within athletics, especially where petite physiques are valued, is to adjust their eating and training habits to try to close the performance gap with men.

Regrettably, women in today’s culture (both sport culture and media culture) do not value their natural bodies nor appreciate that they can be competitive without going to extremes to physically modify their bodies (Sherman & Thompson, 2006). Women and men have different bodies due to hormones, structure, and purpose. Women are pressured by society to have the perfect physique in order to be compared to their male counterparts; the athletic build (broad shoulders, narrow hips, minimal body fat, defined musculature) is determined by the male athlete and is applied to female athletes. This standard is nearly impossible in a woman; she has hips, curves, and body fat. But those natural attributes don’t make her weak and unable to perform in athletics. Women are athletically capable in their own right; their success should be praised and not compared to that of men.
An increased prevalence of menstrual dysfunction, diminished bone health, and inadequate energy availability due to disordered eating behaviors was observed among female athletes after the passage of Title IX (Powell, 2011). These three conditions exist predominantly among female athletes due to the social ideals and the exacerbation of the exercise induced physiological adaptations because of over training and dieting (Lo, Herbert & McClean, 2003). Female athletes have succumbed to these pressures and have taken extreme and dangerous methods in order to see greater performances and success within their respective sport. The female athlete triad was defined less than 20 years age as the combination of menstrual dysfunction, diminished bone health, and inadequate energy availability (NATA, 2008). These components perpetuate one another and take on a cyclical nature. The female athlete triad is a relatively unknown disorder; it is under detected, under diagnosed and under treated. The female athlete triad is not only physiological in root; social attitudes have led to its development. This review of literature on the female athlete triad will present the current clinical definition, the pathophysiology & physiological components, the risks & warning signs, the prevalence, the diagnosis & treatment methods, permanent consequences, and sociological parameters of the female athlete triad in order to better understand the importance of addressing this condition from both a health and a social-cultural perspective.

**History and Definition of the Female Athlete Triad:**

Sport in American culture is one that praises athletes who perform at the highest levels, and rewards additionally the cultural appeal of the athlete. Male athletes are portrayed in advertising as masculine and confident, while female athletes are highlighted for their beauty as well as athleticism (Sherman & Thompson, 2006). In fact, some sport activities for women
praise the female physique as it enhances the beauty of movement, like figure skating, gymnastics, and some track and field events (sprinting and distance running). Physical activity provides innumerable benefits: improved cardiovascular fitness, increased anaerobic strength and capacity, low resting heart rate, low blood pressure, low body fat, low cholesterol levels, increased energy and improved sleep and concentration. However, sport and athletics require a high level of activity that can poses risks to athletes, both male and female. Women are more susceptible to succumbing to the potential dangers of exercise. Exercise inherently lowers body fat through the increased metabolic processing of food energy, and women require body fat and adequate nutrition to maintain normal menstrual function. Menses is essential for bone remodeling and prevention of diminished bone health. The physiological absence of menses (amenorrhea) is only healthy and normal in pregnant women. Amenorrhea unrelated to pregnancy was first noted among female athletes in the late 1970s.

The prevalence of amenorrhea increased as aerobic and endurance sports for women increased in popularity in the 1980s with the first Olympic marathon in 1984 and 10K in 1988. Experts noted that the increased incidence of amenorrhea among athletes was likely due to their increased exercise intensity and duration. Studies in the early 1980s showed a correlation between amenorrhea and a decreased bone health; in the late 1980s a correlation was established between both amenorrhea and diminished bone health and low energy availability (Powell, 2011). Sports medicine professionals began to notice a pattern among female athletes that verified the relationships determined by the literature at the time.

With the increase of female participation and the emergent culture surrounding female athletics that emphasizes leanness in success and in beauty, it was found that women’s susceptibility towards amenorrhea, disordered eating, and osteoporosis was exacerbated with
increased activity level. Increasingly, medical professionals observed the prevalence of these three conditions in conjunction with one another. In 1992 exercise, sport, and medical professional organizations began to confirm the coincidence of these three conditions. This newly confirmed condition was undefined, but identified as being specific to female athletes of all levels, ages, and of multiple sports. After further research the trio of amenorrhea, disordered eating, and osteoporosis was observed to have the highest prevalence among sports that emphasized a lean body type, i.e. gymnastics, ballet, cheerleading, dancing, diving, and long distance running. In 1997 the American College of Sports Medicine (ACSM) defined the Female Athlete Triad as the combination of amenorrhea, disordered eating, and osteoporosis. The female athlete triad (or the triad) was correlated to infertility, irreversible bone loss, and early onset osteoporosis with poor long-term bone health. The female athlete triad is a condition developed from the cascading effects of the physiological consequences of a previous condition or disease. The ACSM updated their position stand in 2007 to encompass more female athletes that did not “qualify” for the diagnosis of female athlete triad, but those with less severe states of the three components were found to have detrimental effects on both health and performance. The current position stand consists of the same components, but defined on a broader spectrum of energy availability, menstrual function, and bone mineral density (ACSM, 2007). This new definition allows for a larger pool of athletes considered to be suffering from the triad, and thus in theory, more women could be diagnosed and treated at the onset of the condition prior to developing the serious pathophysiology of amenorrhea, disordered eating, and osteoporosis. The same permanent consequences are still highly likely among female athletes who do not suffer from the full severity of the three conditions (disordered eating, amenorrhea, osteoporosis) the female athlete triad was previously defined with (Thein-Nissenbaum, 2013). The female athlete
The female athlete triad may be an outcome of both overtraining and cultural expectations of the female athlete, and need not occur. It is essential that awareness increase and prevalence of the triad decrease so that benefits of exercise continue to outweigh the risks.

**Amenorrhea and the Female Athlete:**

A menstrual cycle or menses, in a woman, is common between the ages of 11 and 51. Prior to menarche (a woman’s first menstrual cycle) and during menopause, estrogen and progesterone hormone levels are not high enough to sustain reproduction, thus, there exists no need for menstruation. Normal menstrual cycles last, on average, 28 days. Days 1-6 is when menstrual bleeding occurs; days 7-15 is the follicular phase; ovulation is different for each woman, but typically occurs between days 13-17; days 16-28 is the luteal phase. In the follicular phase high levels of estrogen along with luteinizing hormone and follicle stimulating hormone are released to support the growth of the ovarian follicle and to build up the endometrial lining of the uterus in preparation for a fertilized ovum (or egg). At mid-cycle ovulation occurs; a surge of luteinizing hormone and follicle stimulating hormone are released, which stimulates the release of the ovum from the ovarian follicle into the fallopian tube. During the luteal phase the remnants of the follicle (corpus luteum) continues to release estrogen and progesterone to ready the uterus for childbearing; if the ovum is fertilized, it becomes embedded within the endometrium and the luteal phase continues throughout pregnancy. If the ovum is not fertilized, the corpus luteum dies, the uterus sheds the endometrial lining, and a new menstrual cycle begins (Witkop & Warren, 2010). Menstruation is an essential part of normal physiological function within females due to the release of estrogen that stimulates other bodily functions through
endocrine communication (i.e. bone growth and deposition, musculoskeletal growth and strengthening, normal cardiac function, etc.).

Amenorrhea, or the absence of menses can be primary or secondary. Primary amenorrhea is defined in three ways: the loss of menses by age 16 with normal pubertal development; loss of the normal menstrual cycle (menses) two years after complete sexual maturation; loss of menses by age 14 without secondary sexual characteristics or delayed puberty. Secondary amenorrhea is also defined in three ways: the absence of menstrual bleeding for six months after establishing regular menses; the absence of menstrual bleeding for three cycle lengths in a female who has oligomenorrhea (infrequent or light menses that occur at intervals of 35 days or more after the establishment of normal menses); the absence of menstrual bleeding 18 months post menarche. Maternal genetic factors are the strongest indicators for menarche; average age of menarche is 12.9 years, but for female athletes could be as late as 15 years (Lo et al., 2003). However, leanness (low body mass and/or low body fat) is also an indicator of menstrual function and menarche; menses could cease if body fat drops below 22%, but eumenorrheic (normal menstrual cycles and function) menses has been found to exist in females with body fat as low as 4% (Witkop & Warren, 2010). Typically amenorrheic individuals have a Body Mass Index (BMI) lower than 18 kg/m\(^2\) and body fat below 17% (Powell, 2011). Prevalence of menstrual dysfunction in adolescents can be a challenge to determine due to the irregularity of menses for six months to two years after menarche (Thein-Nissenbaum, 2013). Normalization of menses should occur naturally 24 months post menarche (Witkop & Warren, 2010). The prevalence of menstrual dysfunction beyond the two-year window is estimated to be upwards of 54% among female athletes as a whole, 69% among dancers, 65% among long distance runners, and 5% among the general population (Thein-Nissenbaum, 2013). This increased incidence evidenced
among female athletes can be attributed to training intensity and volume. The ACSM found that as training intensity increased 10 fold, from 8 to 80 miles a week, among long distance runners, amenorrhea increased from 3% to 60%. Therefore, training intensity was found to have a negative correlation to menstruation. It was also found that luteal deficiency (expressed as anovulation - the absence of ovulation and menses) with significant decreases in estrogen, progesterone, luteinizing hormone, and follicle stimulating hormone was found among 78% of eumenorrheic recreational runners in at least one of three menstrual cycles (ACSM, 2007). The prevalence of amenorrhea among female athletes has been empirically established within the literature. The importance of normal menstruation function cannot be understated, especially among female athletes who need estrogen to maintain bone health, musculoskeletal growth and repair, and normal cardiac function to sustain training.

Pathophysiology of Amenorrhea:

Amenorrhea among female athletes results from low body fat elicited from excessive training and low caloric availability; body fat in women stimulates and maintains the ovarian release of estrogen. Low body weight, low body fat, and low body mass index are not the sole indicators for estrogen release and menstruation; nutrition, eating habits, stress and over training all have the possibility to lead to amenorrhea or oligomenorrhea (Lo et al., 2003). Nazem and Ackerman looked at physiological markers within female athletes that presented with one or more of the conditions of the female athlete triad. Through their research an overall negative energy balance (caloric intake less than expenditure) was observed and hypothesized to be the most likely culprit of amenorrhea among female athletes. This chronic energy deprivation state derived from excessive exercise and inadequate nutrition leading to a lower metabolic rate and
thermal response to food (Nazem & Ackerman, 2012). The literature also states other physical risk factors unrelated to exercise and energy availability for developing amenorrhea or other menstrual dysfunction. Delayed menarche (after 16 years of age), a history of missed periods or episodes of amenorrhea, and lack of spontaneous menstrual cycles after the cessation of oral contraceptives were all found to be physical markers linked to amenorrhea (Alleyne, 2004; Witkop & Warren, 2010; Nazem & Ackerman, 2012; Thein-Nissenbaum, 2013). These risks in conjunction with a high level of exercise duration and intensity should be red flags for menstrual dysfunction to all members of a sports medicine team (any professional working with and providing care for athletes; i.e. coaches, physicians, athletic trainers, psychologists, nutritionists, physical therapists, etc.)

The pathophysiology of amenorrhea is complex and has two potential origins, which may work independently or simultaneously to cause amenorrhea. Both over training and diminished energy availability induce a stress response in the body which affects on endocrine function negatively. An energy deprivation state has an effect of the hypothalamic-pituitary-gonadal axis through the depletion of the amplitude and frequency of the pulsatile release of gonadotropic releasing hormone (produced in the arcuate nucleus of the hypothalamus) (Lo et al., 2003). This decreased release of gonadotropic releasing hormone subsequently decreases the release of luteinizing hormone and follicle stimulating hormone from the pituitary gland (Nazem & Ackerman, 2012). When luteinizing hormone is not released mid-cycle, ovulation ceases causing estrogen and progesterone production decreases due to a lack of ovarian follicle stimulation; the missing surge of luteinizing hormone could lead to irregular menses or a complete lack of menses (Witkop & Warren, 2010). Neural pathways have been traced from receptors in the brain for several metabolic hormones and substrates to the gonadotropic releasing hormone pulse
generator. The low energy availability alters levels of metabolic hormones, such as insulin, cortisol, growth hormone, insulin-like growth factor-I, triiodothyronine, and leptin, and substrates, such as glucose, fatty acids, and ketones, which can eliminate the proper function of neural pathways essential to the endocrine initiation of menstruation (ACSM, 2007). Weight loss can lower leptin levels, which may constitute a change in the metabolic signal to gonadotropic releasing hormone-secreting neurons (Nazem & Akerman, 2012). The reduction in insulin, cortisol, insulin-like growth factor-I, triiodothyronine, and leptin results in suppressed bone formation; this may also be due to suppressed estradiol increasing bone resorption and decreasing bone deposition resulting from restricted energy availability (ACSM, 2007). Many sports that both require a light body weight and expend a large number of calories may create the conditions that push the female athlete towards amenorrhea. Runners, gymnasts, and dancers may maintain relatively strict caloric intakes to avoid weight gain, yet have very high caloric outputs from training. Limiting calories and excessive training leads to an energy deprived state that inhibits the normal endocrine initiation of menstrual function. Therefore, amenorrhea may be one of the first signs of inadequate energy intake or excessive training. It should not be viewed as a normal female response to training.

Exercise puts a stress on the body that can contribute to amenorrhea via the hypothalamic-pituitary-adrenal axis; stress results in an increased release of corticotropin-releasing hormone (Witkop & Warren, 2010). The stress origin of amenorrhea also leads to a decrease in luteinizing hormone levels because corticotropin-releasing hormone release decreases the pulsatile release of gonadotropic releasing hormone (Nazem & Ackerman, 2012). The reduced estrogen levels seen as a result of caloric restriction and overtraining leads to amenorrhea. Additionally this hypoestrogenic state leads to losses of boney tissue, reduced
musculoskeletal strength, and impairment of reproductive health (Allyene, 2004). Energy deficiency before puberty suppresses growth and delays sexual development. Moderately or recurrently reduced energy availability can elicit subclinical menstrual disorders through the suppression of estrogen and metabolic hormones, which in turn can result in a reduced bone mineral density over time (ACSM, 2007). Many research studies conducted on the female athlete triad examined the changes in the physiological response when amenorrhea existed in the female athlete. Through blood testing, magnetic resonance imaging and physical examination these two origins of the female athlete triad have been established and verified numerous times (Lo et al., 2003; Allyne, 2004; Sherman & Thompson, 2006; ACSM, 2007; Witkop & Warren, 2010; Nazem & Ackerman, 2012; Thein-Nissembaum, 2013).

**Repercussions of Amenorrhea:**

Unfortunately, the majority of female athletes affected by the triad do not fully understand the physiological stresses that the body has experienced. A hypoestrogenic state is dangerous for women of childbearing age because it hinders endocrine, neurological, musculoskeletal, cardiopulmonary, and reproductive functions. The most substantial and permanent damage that results from amenorrhea is infertility. Amenorrheic women are infertile due to the absence of ovarian follicular development, ovulation, and luteal function (ACSM, 2007). The damage does not just affect reproductive health; because reproductive health is directly tied to both skeletal and nutritional health, the consequences could potentially affect body systems ranging from gastrointestinal, to muscular, to neurological, to psychological health. Therefore, to fully understand why menstruation is so important and why amenorrhea is
so significantly damaging, it is crucial to expose the most significant damage amenorrhea can cause.

Prolonged amenorrhea leaves women at a significantly increased risk for stress fractures, scoliosis, low bone mineral density, and early-onset osteoporosis (Lo et al., 2003). A study conducted by Nazem & Ackerman that examined physiological measures among female athletes presenting with symptoms of the female athlete triad and its’ different conditions, found that female athletes with amenorrhea had a 29% decrease in spinal bone mineral density (Nazem & Ackerman, 2012). Keen and Drinkwater’s research on female athletes presenting with diminished bone health found an inverse dose-response relationship between the severity of amenorrhea and bone mineral density; as amenorrhea severity increases, bone mineral density decreases (Keen & Drinkwater, 1997). For example, the National Association of Athletic Trainers cite in their position stand on the female athlete triad that the prevalence of decreased bone mineral density among dancers is highest among female athletes at 24%, and, of those with decreased bone mineral density, 83% have presented with amenorrhea. Of the 76% of dancers without decreased bone mineral density, 54% were amenorrheic (NATA, 2008). Secondary amenorrhea among dancers and other female athletes at a high risk for the female athlete triad is correlated with low bone mineral density and high risk for stress fractures. Kazis and Iglesias found through their research on the triad that a female athlete suffering from amenorrhea (secondary or primary) has a 4.5 times greater risk for stress fracture than an eumenorrheic athlete. If the hypoestrogenic state persists for two or more years permanent bone loss and osteoporosis is highly likely (Kazis & Iglesias, 2003). Amenorrhea develops due to hypoestrogenism, which can result in a myriad of non-reproductive related consequences. The lack of estrogen in amenorrheic females can lead to impaired endothelium-dependent arterial
vasodilation, causing diminished perfusion of the working muscle, impaired skeletal muscle oxidative metabolism, elevated low-density lipoprotein cholesterol levels and vaginal dryness (ACSM, 2007). Therefore, if the musculoskeletal system cannot receive the adequate oxygen needed to function, fatigue sets in (resulting in lactic acid build up), and recovery can be nearly impossible. Athletes need their muscles to work properly and efficiently; if the compromises that female athletes engage in to “improve performance” result in amenorrhea, that amenorrhea can actually hinder performance and eliminate their ability to compete. The permanent damage specific to amenorrhea and the female athlete triad is significant and should not be understated. This damage can change a woman’s future activity levels, her ability to have a family, and her life expectancy.

**Disordered Eating:**

A lack of energy availability results from disordered eating habits (i.e. inadequate caloric intake and insufficiency among macronutrient, mineral, and vitamin needs). Energy availability is energy intake minus energy expenditure, and post-exercise energy availability is crucial for recovery, repair, and normal physiologic functions (i.e. menstruation, bone remodeling and growth, cardiovascular function, thermoregulation, cellular maintenance) (ACSM, 2007). The compensation of shutting off physiological functions is a survival mechanism and restores energy balance, but results in severely impaired health (Black, Larkin, Coster, Leverenz, & Abood, 2003). Intentional energy reduction can occur through excessive exercise and/or decrease in caloric intake. Manipulation of caloric intake, such as fasting, skipping meals, restrictive eating, diet pills, laxatives, diuretics, enemas, binging and purging, is defined as disordered eating (Kerr, Berman, & DeSouza, 2006). The desire to manipulate one’s own nutrition is the
same desire to manipulate one’s individual athletic performance; thus, explaining why the female athlete triad involves disordered eating behaviors. The desire to control energy availability and the action to do so may occur at any time, but the behaviors to do so are formed in adolescents; adolescence is also the crucial time period for the development of the female athlete triad.

Athletes at the greatest risk for low energy availability and disordered eating exhibit restricted dietary intake, prolonged exercise sessions, are vegetarian, perpetually engaged in dieting, and limit the types of food they consume (ACSM, 2007). Black and colleagues’ research on eating disorders and disordered eating habits among female athletes found that female athletes to have a 2 to 3 times greater risk for developing eating disorders than male athletes and non-athletes (Black et al., 2003). Other risk factors for disordered eating include low energy level with complaints of fatigue, inability to achieve performance goals and benchmarks, frequent gastrointestinal complaints like nausea or bloating, frequently cold, intolerance to temperature changes, low BMI (BMI < 20 kg/m$^2$), and weight loss for more than 5% of body weight in six months (Kerr, et al., 2006). Among all female athletes, it is estimated that upwards of 62% have a disordered eating problem while the general population prevalence lies in the range of 0.5% – 1.0% for anorexia and 2% – 5% for bulimia (Black et al., 2003). Risks must be known and frequently screened for among female athletes by a sports medicine team to eliminate the potential for eating disorders to develop and to correct disordered eating behaviors.

Disordered eating takes on many forms, not just “eating disorders”. Two of the most commonly known of said “eating disorders” are very severe and are defined in the Diagnostic and Statistical Manual of Mental Disorders: anorexia nervosa (refusal to maintain 85% of minimal body weight) and bulimia nervosa (binging and purging at least 2 times per week) (American Psychiatric Association, 2013). The literature on the causes and treatments of eating
disorders among female athletes, conducted by Kerr and colleagues, indicated that recovery from anorexia only occurred in 46% of patients, while 20% remained chronically ill (Kerr et al., 2006). Eating disorder not otherwise specified is a third category that is a catchall for other disordered eating behavior that has a pattern and pathology unlike anorexia nervosa or bulimia nervosa (American Psychiatric Association, 2013). Eating disorder not otherwise specified is the most common eating disorder that is diagnosed, but because eating behaviors are self-reported, is challenging to diagnose.

Disordered eating causes electrolyte imbalances, slower mental processing, mental illness, and (eventually) a decreased ability to compete (NATA, 2008). Disordered eating directly results in a chronic energy deprivation state and suffering from such a condition leads to thermoregulatory problems, cardiac abnormalities and dysfunctions, nutritional deficiencies, compromised immune system function, depression, and hypoestrogenism. Disordered eating also leads to menstrual dysfunction and compromises in skeletal structure and function (Thein-Nissenbaum, 2013). One of the most severe consequences of disordered eating is mortality; the rate of mortality ranges from 6-12%, with suicide being the number one cause followed by cardiac arrhythmias, infection and starvation (Black et al., 2003). Cardiovascular complications, pulse rate and quality, blood pressure, orthostatic measurements, and body temperature should be monitored to prevent against the potential for systemic bodily functions to shut down (NATA, 2008). A majority of female athletes suffering from eating disorders also have co-existing medical conditions, i.e. amenorrhea and osteoporosis (NATA, 2008). It becomes vital to the health of the athlete when a suspected eating disorder or low energy availability exists to monitor for bone health and menstrual function simultaneously. Primarily, it protects the athlete against
systemic failure and long-term health related consequences. It also allows for the sports medicine team to screen the athlete for the triad.

The etiology for disordered eating is multidimensional including factors such as psychological, social, personality, family, self-identity, and self-esteem (Lo et al., 2003). Young, highly compulsive, overachieving, controlling, perfectionist girls who have an attitude of “the ends justifying the means” and will do “whatever it takes” when it comes to “success” are at a high risk for disordered eating. These high-risk female athletes lack the necessary coping skills, support system, and self-esteem to overcome the temptation to confine body weight. The emphasis in athletics, especially for women, on leanness, speed, agility, flexibility, and fitness is ever increasing; with the escalating pressure on female athletes to compete at an (exponentially increasing) high level it is easy to understand why some succumb to the temptation that is disordered eating. There exists a social standard that associates thinness with success and beauty; in sports culture for female athletes, that standard is reflected in the pressures coaches and the athletes put on themselves to maintain a slim, muscular physique (NATA, 2008). This emphasis has a high prevalence among aesthetic sports (i.e. body building, dance, etc.), and sports where physique directly manifests into performance results (i.e. long distance running). These social and sport related pressures manifest into disordered eating habits. Eating habits are developed in adolescence, as it is a very formative time period in life on many levels (i.e. social, body image, exercise, diet, growth, etc.). The pressures on young, adolescent athletes today are great, especially among aesthetic-physique related sports.

If disordered eating develops during these formative years, it becomes increasingly more challenging to treat and recover from in the future. It also puts the athletes at a much greater risk for developing menstrual dysfunction and poor bone health (NATA, 2008). As many as 62% of
athletes are estimated to engage in pathogenic weight-control behaviors; the majority of these athletes have low body weights and body fat percentages because their sports emphasize trim, lean figures (Black et al., 2003). Gymnasts, ballet dancers, long distance runners, divers, cheerleaders, and figure skaters are particularly vulnerable to disordered eating due to the nature of their sport in which success comes with a lean, muscular physique. A study done by Constantini and Warren on the female athlete triad in 1994 found that 74% of gymnasts, 35% of track athletes, and 33% of dancers suffered from an eating disorder (anorexia, bulimia, eating disorder not otherwise specified) (Constantini & Warren, 1994). Among those gymnasts who were suffering from disordered eating, 75% of them were instructed by a coach specifically to lose weight (in whatever way possible) to improve performance (Constantini & Warren). Kerr, Berman & De Souza’s research on the triad found that 54% of coaches determined whether or not a gymnast “needed” to lose weight by visual appearance alone, and while no coaches admitted to doing this themselves. An impressive 82% of those “innocent” coaches admitted to knowing other coaches in the sport who implemented regular weighing of their athletes and 75% of them admitted to knowing other “guilty” coaches who regular conducted body fat assessments (Kerr et al., 2006). Even more startling, a study conducted by Drummer and associates in 1987 studied the eating habits of 427 elite female swimmers between the ages of 9 to 18; this study reported that 80% dieted to lose weight, 12.7% vomited to lose weight, 2.5% used laxatives, 1.5% use diuretics, and 62% skipped meals (Drummer, 1987). The three studies discussed previously have all established the prevalence of disordered eating behaviors among female athletes. Athletes not among aesthetic, lean body-type sports are also at risk for developing the female athlete triad due to the sociological pressure on competitive females in today’s world of sports. The body image ideals of today are significant and ubiquitously, and young girls are
impressionable. The combination of the two can elicit a desire to manipulate body physique through disordered eating behaviors. The female athlete triad exists as a result of the energy availability exploitation due to the pressures within sport to be thin and to win.

**Skeletal Problems: Low Bone Mineral Density, Osteopenia, & Early Onset Osteoporosis:**

Osteoporosis as defined by the ACSM in the Female Athlete Triad Position Stand is a skeletal disorder characterized by compromised bone strength predisposing a person to an increased risk of fracture (ACSM, 2007). Osteoporosis (as defined by the World Health Organization) is a decrease in bone mass and tensile strength resulting in microarchitectural deterioration and increased risk of fragility and fracture. The World Health Organization also defines osteoporosis as a bone mineral density at least 2.5 standard deviations below mean for women (by age) measured at hip, spine, wrist; exists in patients with history of atraumatic (stress) fracture(s). Osteopenia is defined as a bone mineral density between 1.0 and 2.5 standard deviations below the norm. Bone mineral density for a menstruating population (non-menopausal) is measured differently (expressed as Z-scores, not the normal T-scores) to compare individuals to age and sex-matched controls; bone mineral density at least 2.0 standard deviations below the norm is “low bone density for chronological age” and poses a high risk for future osteopenia and osteoporosis among a premenopausal population. Research has shown prevalence of low bone mineral density among high school aged female athletes to be 22%. It is crucial when evaluating the bone mineral density of young female athletes in weight bearing sports to consider that their bone mineral density should be 12-15% higher than the norm (based on sedentary controls) (Thein-Nissenbaum, 2013).
Risk factors for osteoporosis and poor bone health include: female sex, Asian or white race, age greater the 65 years, sedentary lifestyle, ectomorphic body habitus, tobacco use, decreased bone mineral density, prolonged corticosteroid use, decreased calcium and vitamin D intake, chronic malnutrition, dietary absorption problems (i.e. lactose intolerance, gluten intolerance, etc.), dietary pathologies (i.e. Crohn’s, Ulcerative Colitis, Celiac, etc.), involvement in non-weight-bearing sports (i.e. swimming, cycling, etc.), low BMI (< 20 kg/m²), current or past eating disorders, previous fracture history, and estrogenic deficiency. Menstrual history has been identified by research as the primary indicator for bone mineral density; girls with delayed menarche and low body mass index (BMI) during adolescence have displayed the lowest bone mineral density compared to peers. Exercise (exceptions- swimming, waterpolo, etc.) is weight bearing activity and, therefore, should stimulate bone growth and repair to increase mass. However, chronic calcium deprivation and hypoestrogenism offsets the positive benefits of weight bearing exercise. Research has shown the reversibility of bone loss with resumption of menses; however, after two years, the rate of bone deposition becomes negligible and these women have overall lower bone mineral density in the long run than normally menstruating athletes (Lo et al., 2003). The majority of bone deposition occurs between 11 and 14 years with 95% of total bone mineral density attained by 18 years (Thein-Nissenbaum, 2013). Peak bone mass is site specific, but it is reached between 25 and 30 years; therefore, because bone mineral density deposition occurs relatively early on in the life cycle it is essential that high risk persons be identified as early as possible to help monitor bone mineral density, treat, repair and reverse poor bone health (Lo et al., 2003). Osteoporosis is not always caused by accelerated bone mineral loss in adulthood; it can be caused by the lack of adequate bone mineral density accrual during childhood and adolescence (ACSM, 2007). Bone mass acquisition involves the
interaction of numerous hormones and environmental factors including diet and exercise; the amount of bone mass acquired in adolescence is the most important factor in determining osteoporosis and fracture risk later in life (Thein-Nissenbaum, 2013).

The skeleton is a metabolically active organ that undergoes continuous remodeling throughout life. Bone remodeling involves the removal of mineralized bone by osteoclasts followed by the formation of bone matrix through the osteoblasts that become mineralized. The remodeling cycle consists of three phases: resorption, reversal, and formation. During resorption osteoclasts (cells that break down bony tissue) digest old bone. During reversal, new osteocytes appear on the bone surface. During formation osteoblasts create new osteocytes. Bone remodeling serves to recreate bone architecture to meet the evolving needs of the individual; it also repairs damage to the matrix of the bone to prevent the accumulation of osteoclasts (Hadjidakis & Androulakis, 2006). The process of bone remodeling is compromised in a hypoestrogenic state because estrogen stimulates the initiation of this process. The development of bone mineral density to reach a peak by age 25 is dependent upon the bone growth velocity, or the rate at which bone mineral density is accrued. If there is a decrease in bone growth velocity due to compromises in bone remodeling and bone health, it has a permanent reduction on peak bone mineral density overall and that bone mineral density can never be regained (Keen & Drinkwater, 1997). In the female athlete triad, the process of bone remodeling is slowed or even stopped due to the endocrine changes that occur from menstrual dysfunction and the lack of energy availability to sustain the chemical and molecular processes (Hadjidakis & Androulakis, 2006). Estrogen via appropriate menstrual function and adequate energy availability are essential in the establishment of appropriate bone growth velocity to establish necessary peak bone mineral density to sustain a woman’s skeletal system throughout her lifetime.
Hypoestrogenic women have accelerated bone loss due to increased ratio of bone resorption to bone formation. A prolonged state of hypoestrogenism during the crucial window of bone deposition (prior to 25-30 years of age) causes permanent damage because one can never reach expected peak bone mass past 30 years of age. Low peak bone mass can be caused by genetics, medication, inadequate nutrition, and sedentary lifestyle, but hypoestrogenism is the most significant risk factor for poor bone health long-term beginning prematurely (prior to menopause). Women recovering from amenorrhea may never be able to reach normal bone mass due to the cessation of bone deposition during amenorrhea leaving these women at a high risk for early onset osteoporosis (Lo et al., 2003). Amenorrhea occurring prior to 18 years of age results in lower peak bone mineral densities than if amenorrhea occurs after 18 years of age.

Adolescents suffering from anorexia nervosa have significantly lower bone mineral density than their peers; anorexic adolescents also have a decreased peak bone mineral density, even after resumption of menses and recovery from anorexia. Women who currently or previously suffered from bulimia nervosa or eating disorder not otherwise specified also show a significant decrease in bone mineral density compared to controls; eating disorder not otherwise specified is correlated to having the lowest bone mineral density when compared to bulimia or anorexia, especially among long distance runners (Thein-Nissenbaum, 2013). Low bone mineral density was shown in eumenorrheic athletes when coupled with disordered eating. Studies have estimated among female athletes the prevalence osteopenia to be 50% and osteoporosis to be 13%, compared to 12% and 2.3% in an age-matched population; these are alarming figures, as osteopenia and osteoporosis are not commonly seen among healthy premenopausal women (ACSM, 2007). An athlete’s bone mineral density reflects her cumulative history of energy availability, menstrual status, genetic endowment, and environmental factors. Therefore,
encouraging healthy nutritional behaviors and encouraging women to adopt health body image ideals could reduce the risk of hypoestrogenism and low bone mineral density due to disordered eating and menstrual dysfunction.

**Prevalence:**

All female athletes are at risk for developing the triad due to the inherent nature of exercise to reduce body fat, put a substantial stress on boney structures and the need for adequate caloric intake. If the female’s exercise regimen increases in intensity and in volume it can become too stressful on the skeletal system; if this is in conjunction with an inadequate increase in caloric intake, the risk of developing diminished bone mineral density, deficient energy availability and menstrual dysfunction increases exponentially. Most existing research utilizes self-report questionnaires to establish risk for further evaluation; the premise of a questionnaire requires honesty and accuracy by the respondents, which is not always guaranteed (Kleposki, 2002). Many female athletes of all ages and levels are unaware of the triad’s existence, and therefore, do not realize the loss of menstrual dysfunction, frequent musculoskeletal injuries, and weight loss are large red flags signaling a serious health problem (Hoch et al., 2009). There is also an anxiety on the part of female athletes in reporting their symptoms because the athletes fear if their disordered eating, amenorrhea, or poor bone health is discovered- athletic competition and participation may be halted (Kleposki, 2002). Prevalence is largely unknown due to the lack of awareness by athletes, coaches, physicians, physical therapists, nurses, athletic trainers, etc.

The lack of awareness of the female athlete triad can be attributed to the lack of awareness among health professionals and coaches. In a study conducted by Troy, Hoch &
Stavrakos it was determined that of the 240 adult professionals who work with female athletes (largely health professionals and coaches), only 48% of physicians, 43% of physical therapists, 38% of athletic trainers, and 8% of coaches could identify all the components of the female athlete triad. Of the physicians who were aware of the triad, only 36% of pediatricians and 17% of gynecologists could identify the triad’s components. The most startling result of the study, which looked determine awareness and comfort in treating the female athlete triad, was that only 9% of physicians felt comfortable treating the triad (Troy, Hoch & Stavrakos, 2006). This lack of awareness and treatment knowledge is alarming, especially in a health profession population.

Of the prevalence research conducted the results are largely estimates that lack internal validity and could potentially underestimate how many female athletes actually suffer from the female athlete triad. Hoch and colleagues conducted a study and found that 78% of high school athletes had at least one component of the triad (Hoch et al., 2009). Nichols and colleagues conducted a study to determine the prevalence of the triad among high school athletes; it was found that 32% of the 170 participants identified with any one of the components of the triad, 7% met criteria for both and 2% met the criteria for all three components. Despite the prevalence of the triad being low in this study, 89% of the girls were identified as being “at risk” for one or more of the components of the female athlete triad (Nichols et al., 2006). Both research studies aimed to assess prevalence in high school athletes because of the sensitivity to developing the female athlete triad between 13 and 18.

Torstveit and Sundgot-Borgen conducted a study to determine the prevalence of the female athlete triad among elite athletes and to determine if they were at an increased risk due to the high intensity of training required to be an elite athlete. It was found that a high percentage of non-elite athletes (69.2%) were at risk for the triad compared to elite athletes (60.4%). Elite
athletes reported more menstrual dysfunction and stress fracture(s) than non-elite athletes. It was found that 66.4% of elite athletes in aesthetic sports were at risk for the triad, while only 52.6% of elite athletes in ball game sports were found to be at risk (Torstveit & Sundgot-Borgen, 2005). There was no significant risk involved in elite athletics and the development of the female athlete triad, as most of the behaviors leading to the triad begin in adolescence, which is prior to professional athletics beginning and typically at the onset of competitive sport participation.

The literature depicts the increased prevalence and risk of the triad among athletes of all levels within aesthetic sports. The increase of female athlete triad can also be attributed to the increase in women of all ages in physical activity and competitive sports. The exact prevalence of the female athlete triad among all female athletes of all ages and levels is unknown due to the lack of comprehensive and longitudinal research available (ACSM, 2007). This lack of knowledge is due to the differences of screening, assessment tools (i.e. self-report questionnaires vs. in-depth interviews), parameters for diagnosing disordered eating and menstrual dysfunction, failure to control oral contraceptive and hormone replacement therapy use, and lack of accurate determination of bone mineral density (Leburn, 2007). There is no gold standard for assessment of the female athlete triad that currently exists; there are a variety of types of assessments, but most rely on the premise that the athlete is open and honest with all members of a sports medicine team (Kleposki, 2002). It has become critical that sports medicine professional develop a standardized method of diagnosis and treatment for the female athlete triad due to the ever-increasing prevalence.

Diagnosis of the Female Athlete Triad:
Permanent bone loss can be prevented if the female athlete triad is recognized early and diagnosed to allow for the earliest possible intervention; diagnosis is possible with increased awareness in coaches, parents, athletes, and physicians. An appropriate screening of female athletes at a pre-participation physical is necessary; a questionnaire completed by athletes before a physical exam can help physicians target high-risk individuals for each component of the female athlete triad (ACSM, 2007). A questionnaire regarding menstruation history and reproductive health, (stress) fracture history, osteoporosis risks, dietary habits, and body image would be appropriate and essential; a potential questionnaire could help physicians initiate discussions that the adolescent may not feel comfortable bringing up, especially if a risk is exposed. Further diagnostic testing including, but not limited to, a complete blood cell count, sedimentation rate, complete metabolic profile, cardiac examination and urinalysis is essential to unequivocally diagnose each of the components of the female athlete triad. These tests screen for evidence of systemic processes such as anemia, electrolyte abnormalities, systemic inflammation, multi-organ involvement (with potential renal insufficiency), bone marrow suppression, and metabolic acidosis or alkalosis. Cardiac examinations could include electrocardiogram and/or echocardiography to determine if dysrrhythmias, cardiomyopathies, or rare complications to chronic nutritional deprivation exist (Lo et al., 2003). Cardiac complications are linked to severe disordered eating.

Eating disorder diagnosis requires examination of vital signs, BMI, body habitus, thyroid evaluation, signs of virilism (hirsuitism, acne), and Tanner staging (ACSM, 2007). Pituitary gland and thyroid abnormalities can be evaluated through visual field and cranial nerve examination (Lo et al., 2003). A pelvic examination is essential if amenorrhea exists to rule out physical abnormalities preventing menstruation. Serious physiological damage requiring
intervention can be detected through a physical examination that results in findings of any of the following: hypotension, hypertension, hypothermia, bradycardia, tachycardia, lanugo, thin scalp hair, carotenemia, pale or dry skin, and poor muscle development (ACSM, 2007). Hypertension in female athletes suggests excess androgens; hypotension, cachexia, brittle nails, bradycardia, and hypothermia in female athletes indicate severe malnutrition (i.e. anorexia). Symptoms of bulimia include parotid enlargement, eroded tooth enamel, rotting teeth, cheilosis and finger and/or nail calluses on first and second digits of dominant hand (Russell’s sign) (Lo et al., 2003). Diagnosis based on questionnaires or interviews can be skewed due to the subjective nature of the method; the answers rely on the honesty and accuracy in part by the patient.

Black and colleagues conducted a study to develop and evaluate a physiologic screening tests specifically designed for female collegiate athletes engage in competition in order to detect eating disorders or disordered eating. Black and colleagues tested two different eating disorder screening methods on 148 female collegiate athletes of varying levels (NCAA, club, intramural). Through the research, it was determined that the 18-item Physiologic Screening Test developed yielded the highest sensitivity (87%) and specificity (78%) and was superior to the Eating Disorders Inventory-2 and Bulimia Test-Revised. Through the use of the physiologic screening test, 62% of these athletes were determined to be suffering from disordered eating. The premise of the physiologic screening test developed was to: 1) involve cogent domains that make physiologic sense with respect to the disorder in question, 2) contain variables that have been identified several times in the literature as warning signs for eating disorders or disordered eating, and 3) include recommendations for diagnosis and treatment by experts. This developed physiologic screening test was created by thorough examination of the literature on disordered eating and utilization of experts. The final physiologic screening test contained six questionnaire
items: dizziness when standing up quickly, gaseousness or bloating in abdominal area unrelated to menstrual period, cramps and dull pains in abdominal area unrelated to menses, frequency of bowel movements, consistency of stools and number of menstrual periods annually. It contained eight self-report items: hours exercised outside of practice, whether menstrual cycles have every been irregular, lowest weight at current height, age at this weight, highest weight, difference between lowest and highest weights, perception of every being overweight, and whether currently trying to change or maintain weight. The final four items were four physiologic measurements: percentage of body fat, waist:hip ratio, standing diastolic blood pressure, and enlarged parotid glands (Black et al., 2003). The ideology of the physiologic screening test would be wisely utilized to develop a similar style and type of diagnostic tool for the female athlete triad. It could potentially identify where on the spectrum of the three components athletes lie in their pathology of the triad. Also, it could direct a sports medicine team in the proper direction for treatment based on what appears to be the primary component of the triad that led to the development of the triad in that athlete.

Bone loss increases with the duration of amenorrhea; therefore, the ACSM suggest medical evaluation of patients with amenorrhea exceeding three months (ACSM, 2007). In the evaluation of amenorrhea, it is important to remember that pregnancy is the most common cause of secondary amenorrhea, so a pregnancy test is essential to determine the cause of amenorrhea. A full screening of thyroid-stimulating hormone, prolactin levels, luteinizing hormone, and follicle stimulating hormone levels are essential to diagnose and to understand the severity of each component. Thyroid-stimulating hormone levels can be indicative of hypothyroidism or hyperthyroidism, and thus, the etiology of amenorrhea. Elevated prolactin levels suggest hypothyroidism due to elevated thyrotropin-releasing hormone causing prolactinoma. If prolactin
levels exceed 100 ng/mL and patient exhibits visual disturbances, galactorrhea, and/or severe headaches, it is essential to rule out pituitary microadenoma with computer imaging. Inadequate follicle stimulating hormone, luteinizing hormone levels, hypogonadotrophic hypogonadism or hypergonatrophic hypogonadism (ovarian failure) are all indicative of amenorrhea. Low follicle stimulating hormone and luteinizing hormone levels are most commonly caused by chronic systemic illness, disordered eating (anorexia), drug use, over training, psychological stress (depression, anxiety). Elevated follicle stimulating hormone and luteinizing levels are linked to ovarian failure. When female athlete associated amenorrhea is suspected, a “progesterone challenge” can be initiated. An oral treatment 5-10 mg of medroxyprogesterone is completed for five to ten days and if menses occurs within a week of completing the oral progesterone regimen it is indicative that the endometrium is primed by estrogen from functional ovaries. This indicates that anovulatory amenorrhea results from excess estrogen; two other anovulatory amenorrhea etiologies result from polycystic ovarian syndrome and adrenal hyperplasia. If menstruation does not resume post progesterone challenge it can be concluded that both estrogen and progesterone are deficient (Lo et al., 2003). Both progesterone and estrogen can be inadequate from a number of etiologies including exercise associated amenorrhea, anorexia nervosa, chronic disease, drug use, stress, hypothalamic dysfunction, pituitary dysfunction, or ovarian failure; however, hormone replacement of both estrogen and progesterone can be used to treat the resulting amenorrhea (Lo et al., 2003).

Currently there is no recommendation with prolonged amenorrhea with dual energy x-ray absorptiometry (DEXA) to evaluate the degree of bone loss due to amenorrhea. Osteoporosis is the primary cause for concern in amenorrheic athletes; therefore, it is essential to assess the degree of bone loss so that an appropriate treatment can be instated for the athlete. If amenorrhea
exists for longer than one year, the patient has a BMI of less than 18 kg/m², and a documented history of stress fracture, the evaluation of bone loss via DEXA could be immensely helpful to determine the severity and chance of recovery from the subsequent bone loss. A DEXA scan can serve to diagnose bone mineral density, assessment of bone mineral density during therapy, and encouragement to comply with treatment by informing the patient of the improvements through therapy to bone mineral density (Lo et al., 2003). In diagnosis of the triad, it will be essential to understand the risks for stress fracture, which are indicative of low bone mineral density. These risks include menstrual disturbances, late menarche, dietary insufficiency, genetic predisposition, biomechanical abnormalities training errors, narrow tibia width, and shortened tibia length (ACSM, 2007). If an athlete has presented with symptoms of disordered eating or menstrual dysfunction, it is essential for long-term health that they be screened for osteoporosis.

**Treatment for the Female Athlete Triad:**

Educating coaches, athletic trainers, physicians, parents, and athletes can help to highlight the psychosocial issues that led to the development and persistence of the female athlete triad; it may also facilitate a forum where female athletes can develop healthier attitudes towards exercise, body image and eating habits. Dr. Kathleen Pantano, a physical therapist at Cleveland State University, conducted a study to determine what methods of treatment and prevention were effective in treating the female athlete triad. It was determined that education, athletic screening, physician consultation, dietician consultation, resistance training programs, metabolic testing, contractual treatment parameters, body fat assessments, and observation of risks and signs of the triad are all effective in the prevention and treatment of the triad. It was concluded that physical therapists must be responsible for the recognition, diagnosis, treatment,
and prevention of the female athlete triad as well as coaches, physicians, athletic trainers, and parents (Pantano, 2008). To decrease the prevalence of the components of the triad, it is essential that the triad be watched out for with respect of the risks and warning signs by multiple parties (i.e. physicians, coaches, athletic trainers, parents, physical therapists). Early screening with a pre-participation questionnaire could identify high-risk female athletes and help physicians initiate early intervention to prevent irreversible damage (ACSM, 2007). Psychological difficulties (depression, anxiety, suicidal ideations, etc.) in athletes should not be taken lightly, as unhealthy mental status could be indicative of disordered eating and poor body image (both of which are risk factors for the triad) (Lo et al., 2003). Seeking psychiatric help for these athletes is essential to protect their well-being and to prevent the development of psychological disorders and/or the components of the female athlete triad (ACSM, 2007). If the athlete is resistant to treatment, is symptomatic, or is at risk a dietician should be consulted. Referral to a dietician is designed to be diagnostic through the observation of current diet and compliance to a new diet that meets caloric and nutritional needs for health and performance (Sherman & Thompson, 2006).

Treatment for the female athlete triad requires a change in eating habits, moderation of exercise regimen, realistic expectations of performance in their respective sport, and balanced energy input and output. Adequate nutrition will lead to resumption of menses, which is the ultimate goal for treating the female athlete triad (Alleyne, 2004). Normalization of menses is the only physiological indicator that unequivocally displays improvement in all three areas of the female athlete triad and prevents the permanent damage from hypoestrogenism. The Committee on Sports Medicine of the American Academy of Pediatrics recommends that amenorrheic females with a gynecological age of 3 years of less decrease physical activity and increase
calcium and protein. Hormone replacement therapy in a female athlete that has not reached menarche is only advised if she is older than 16 years of age (Kazis & Iglesias, 2003). It is recommended that a three-day food intake log be recorded to evaluate caloric, calcium and vitamin D intake. A pelvic exam should be performed to rule out pathological abnormalities. DEXA should be utilized to assess bone mineral density if a history of multiple stress fractures, menstrual irregularity, and/or a history of disordered eating exist; bone mineral density should be reevaluated through DEXA every 6-12 months post-diagnosis to establish if treatment is effective (Thein-Nissenbaum, 2013).

Nutritional education integrating the necessity for balanced energy intake to sustain the demands of participation in sport is a great platform to begin treatment with; athletes need to be educated on their treatment so that they understand the necessity of reestablishing normal physiological functions and overall health. To combat disordered eating, initially establishing small frequent meals and snacks to increase caloric intake and repay caloric deficits without bloating and digestion dysfunction is necessary and proven effective in reinstitution of balanced energy intake (Alleyne, 2004). Decreasing exercise by a minimum of 10% and increasing body weight by 2%-3% along with an intake of 1200-1500mg of calcium and 400-800 IU of vitamin D per day is the standard treatment for female athletes suffering from the female athlete triad (Lo et al., 2003). Reducing energy output by decreasing the intensity of exercise and adding in 2-3 recovery days per week until an energy balance is achieved (Alleyne, 2004). A diet consisting of adequate calories from fats, carbohydrates, and proteins to sustain a high-intensity training program is the most non-invasive way to restore menstruation and prevent bone loss (Lo et al., 2003). Focusing on intake of calcium, protein, and complex carbohydrates (grains, starch, etc.) will help establish goals for a new diet that meets the needs in the deprived athlete (Alleyne,
Cessation of normal physiologic functions and skeletal compromises are evident within 5 days of caloric intake below \( < 30 \text{ kcal} \cdot \text{kg} \cdot \text{FFM}^{-1} \cdot \text{day}^{-1} \); therefore, it is recommended that daily caloric intake should be \( 45 \text{ kcal} \cdot \text{kg} \cdot \text{FFM}^{-1} \cdot \text{day}^{-1} \) for at least 90 days and/or until normal physiological functions (especially menses) resume (Manore, Kam & Loucks, 2007). It is also essential that female athletes receive 5-6 g carbohydrates per kg of body weight to maintain glycogen stores during training; if training exceeds moderate duration with low intensity towards high intensity and prolonged duration, carbohydrate intake should increase to 7-12 \( \text{CHO} \cdot \text{kg BW}^{-1} \). Protein requirements for female athletes should range between 1.0 and 1.7 \( \text{g PRO} \cdot \text{kg BW}^{-1} \). There is no current research on amenorrheic or eumenorrheic athletes to recommend daily fat consumption, which for the general population is between 20-35%; however, it is essential that female athletes monitor the intake of essential fatty acids because they are important to regulate vasoconstriction, inflammation, and blood clotting. Many female athletes do not receive the adequate macronutrients necessary for normal energy, blood measures, and bone building materials. Therefore, it would increase recovery through the resumption of menses, normalized eating habits, and increased bone health if B12, B-complex vitamins, iron, folic acid, calcium, magnesium, and vitamin D were monitored within the diet and supplemented if the dietary recommendations were not met (Manore et al., 2007). Using a practical approach to nutritional behavioral changes and establishment of energy balance for at least six months is recommended prior to considering oral contraceptive usage. While utilizing a dietary intervention to combat the components of the triad, it would be helpful to monitor menstrual cycle patterns as they relate to exercise imbalances as well as energy intake changes.

If such dietary changes do not reestablish menstruation, further testing (as addressed earlier) will reveal the etiology of the amenorrhea and the appropriate treatment. It is important
to remember that amenorrhea can take longer than 6 months to reverse itself. Estrogen replacement therapy is recommended until amenorrhea returns to eumenorrhea due to the hypoestrogenic state. A postmenopausal dose of estrogen (0.625 mg) can prevent further bone loss, but will not restore bone mineral density to normal; a lower dose of estrogen (less than 50 mcg) and a progesterone regimen can regulate menstruation and can improve bone mineral density (Lo et al., 2003). Oral contraceptive and/or hormone replacement therapy is not empirically shown to have an effect on the reduction in stress fracture risk in women with the triad. Research on 150 competitive female long distance runners between 18 and 26 years received oral contraceptives or no treatment for two years. There was no significant difference in stress fracture incidence; it also provided a false means of resumption of menses due to the lack of nutritional deficiency not being corrected (Witkop & Warren, 2010). Reinstatement of natural menses supported by natural estrogen as opposed to synthetic treatments (oral contraceptives, hormone replacement therapy) has been shown to produce higher bone mineral density and faster recovery (Alleyne, 2004). Oral contraceptives and/or hormone replacement therapy may mask the components of the triad and not clinically treat them so that the patients fully recover. It is essential that oral contraceptives and/or hormone replacement therapy be utilized in conjunction with other treatment techniques to sufficiently address the components and provide clinical recovery.

An increased bone mineral density can prevent the incidence of future stress fracture(s) in athletes recovering from or suffering from the female athlete triad. However, any compromise in energy availability and menstrual function at any point in the life span, but especially during adolescence or prior to peak bone mass accrual, can result in permanent, irreversible low bone mineral density reduction that has devastating long term consequences in both premenopausal
and post-menopausal (Thein-Nissenbaum, 2013). Current treatment methods to combat poor bone health are essential in the treatment of the female athlete triad. These treatments include the essential increases in vitamin D and calcium (both dietary and supplement forms), caloric energy balance, integration of weight-bearing exercises (i.e. running, jumping, skipping, etc.; not walking due to lack of bone stimulation incurred), and potential short-term use of calcitonin nasal spray (Alleyne, 2004). Treatments for the female athlete triad, especially for diminished bone health, are essential to prevent the long-term consequences. The triad could result in permanently compromised bone health due to menstrual dysfunction and disordered eating behaviors. Therefore, if treatment is to be successful and preventative for further complications, females should resume menses, have an increase in bone mineral density close to the norms for their age, and maintain a weight that is no less than 5% of their projected healthy weight (NATA< 2008). The main goals for treatment of the female athlete triad are to revert current complications back to previous healthy norms, to establish healthy behaviors related to menstrual function, bone health, and energy availability, and to prevent future complications.

**Short and Long Term Consequences:**

The female athlete triad leads to many problematic long-term consequences. Bone mineral density is crucial for function throughout the life span. To compromise the acquisition of that bone mineral density compromises and limits the function of individuals later in life. Women are at a greater risk for osteoporosis; therefore, reaching and developing the highest peak bone mass possible is the only way to combat the onset of osteoporosis. The presence of either amenorrhea or disordered eating result in low bone mineral density and low peak bone mass. The majority of bone mass is accumulated in adolescence and disordered eating results in low energy
availability which interferes with normal bone acquisition. The risk of osteopenia and osteoporosis among athletes with disordered eating at any point in the life span (not just post-menopause) is exponentially higher than in healthy controls; these individuals are also at a seven times greater risk for fracture in adulthood (Thein-Nissenbaum, 2013). Cobb and colleagues conducted a study on female distance runners between 18 and 26 to examine the relationship between disordered eating, menstrual dysfunction and bone mineral density. They found that “catching up” on bone mass to norms by age may not be possible; it was also concluded that runners exhibited a suppressed bone accrual pattern and are at risk for inadequate bone mass gains and low peak bone mineral density. The research concluded that disordered eating was associated with low bone mineral density, even among eumenorrheic athletes (Cobb, Bachrach, & Greendale, 2003). The presence of menstrual dysfunction is correlated with low bone mineral density in the pelvis and lumbar spine due hypoestrogenic state (Thein-Nissenbaum, 2013). Keen and Drinkwater conducted a study on amenorrheic athletes and found that after 8 years of normal menses and/or the use of oral contraceptives, the formerly amenorrheic athletes continued to have significantly lower bone mineral density at the lumbar spine when compared to eumenorrheic athletes. It was also found that vertebral bone mineral density at all levels of the spine in formerly amenorrheic athletes was 85% of that of eumenorrheic athletes at the onset of the study and 8 years later (Keen & Drinkwater, 1997). Therefore, if bone mineral density is compromised within the formative bone mass growth period (prior to 18 years) it is likely that they will never acquire adequate mass when compared to healthy, eumenorrheic age-matched controls.

In examining the relationship between high intensity training and bone mineral density, Nattiv and colleagues followed 211 collegiate track and field and cross country athletes over 5
years of participation. At the completion of the observational study, 34 athletes had sustained 61 stress fracture injuries; upon return to competition after recovery from injury, a significant increase in bone mineral density was observed. Bone mineral density was also observed to have a significant increase from 6 weeks of recovery to 12 weeks of recovery; recovery consisted of a reduced training intensity to allow healing of the stress fractures. The stress fractures were observed in the femoral neck, pubic bone, and sacrum; knowing the sites of trabecular bone damage it was determined that these athletes were at risk for the female athlete triad. A correlation between both menstrual dysfunction and diminished energy availability and compromised bone health leading to stress fracture(s) was established (Nattiv et al., 2013). This study solidified the prevalence of the female athlete triad among high intensity endurance running and proved the short-term repercussions if health is not maintained within the athlete.

Sustaining a musculoskeletal injuries at a young age and the increased prevalence of injuries in females lead to negative consequences that can be damaging long-term. Of the three components of the triad, menstrual dysfunction was correlated with having the strongest relationship to the development of injuries. Thein-Nissenbaum and colleagues conducted a study on 249 high school female athletes; results showed that menstrual irregularity correlated to a significant increase in the percentage of severe injuries when compared to non-athletes. All three triad components had a high correlation with musculoskeletal injuries. Severe injuries can require the cessation of exercise, which could lead to obesity, hypertension, and diabetes (Thein-Nissenbaum, 2013). Extensive research has also concluded that amenorrhea, commonly seen in long distance runners, is responsible for stress fracture(s), which is most commonly diagnosed in the tibia (63% of all stress fractures in distance runners) (Manore et al., 2007). Therefore,
resumption of menses and adequate nutrition while training are essential to establish sufficient bone mineral density for the long term.

Negative energy balance can cause a decrease in calcium and vitamin D, which are both essential to bone health. In a negative energy balance state many physiologic functions cease to persist normally, such as the reproductive system. The combination of a negative energy state and menstrual dysfunction causes hypoestrogenism; this leads to bone mineral density deterioration in the long run, which can shorten functionality during the life span and the condition will only worsen (Thein-Nissenbaum, 2013). If a female is being screened for the triad it becomes essential that the questionnaires assess adolescence to establish behaviors that may have developed into the components of the triad. If an athlete identifies as having one component of the triad, she should be assessed for the other two components to ensure that she is not at risk for or already suffering from the triad.

The International Olympic Committee’s Medical Commission’s (IOCMC) position stand on the female athlete triad emphasizes the message that the health of the individual is more important than her performance. The IOCMC aims to highlight the importance of long-term health by educating athletes, coaches, athletic trainers, physicians, psychiatrists, dieticians, physical therapists, and parents on the symptoms of and risks for the triad. The prominence of body image and composition in female sports should be avoided for the health argument. The IOCMC hopes that if athletes at the highest level of competition can adopt healthy behaviors and attitudes towards their current body and the need for that body to be healthy and functional long-term then it would influence all other levels of athletics to do the same (Sherman & Thompson, 2006). The IOCMC asked all National Olympic Committees to develop an educational program and a screening process for the female athletes and those who work with them, such as coaches,
physicians, athletic trainers, psychologists, physical therapists, and dieticians, to deter growing prevalence on the national and international sports stage (Leburn, 2007). Hoch and colleagues suggested that education on the triad begin in the formative elementary school years to prevent onset and improve health while averting long-term complications (Hoch et al., 2009). In order for society to encourage female athletes to develop a positive image of health as it relates to their future and their sport, it is imperative that the athletes themselves are educated on the consequences relating to the components of the female athlete triad.

Education would be most beneficial if it started with discussing the importance of nutrition as a means to fuel the athlete so that they can reach their performance goals. Discussing consequences of inadequate fuelling is an appropriate manner to begin the discussion on the female athlete triad. Emphasis on the importance of optimal nutritional behaviors to eliminate the possibility of medical and performance complications due to prolonged energy and nutrient deprivation is imperative in the prevention of the triad. Linking nutritional importance to reproductive health would aid in the discussion of amenorrhea and other menstrual irregularities; it becomes imperative that athletes seek medical attention at the first sign of abnormalities to avoid potential infertility, determine if a pathological condition explains the amenorrhea, and to prevent the early onset of osteoporosis and diminished bone mineral density. An educational program should aim to increase knowledge level, attitudes, and behaviors of athletes as well as for those who participate in their health maintenance and performance enhancement to better minimize, contain, manage, and prevent problems (NATA, 2008). It is through education that awareness will increase, which in turn may aide in better diagnostic methods and treatment protocols for dealing with the triad. Education is the only way to begin the process of decreasing the prevalence of the female athlete triad, which has been continuously growing since the 1970s.
Decreasing the prevalence of this new and relatively unknown condition will help in the prevention of the permanent consequences resulting from the current lack of awareness, diagnosis, and treatment.

**Conclusion:**

The female athlete triad began as a result of the increased pressures on women to compete at a high level and succeed, and also as a result of society’s value on thin, yet muscular women. The standards and expectations have been set very high for women. The pressure does not begin once the girl begins to participate in sports, or when she enters puberty/adolescence; this pressure has existed throughout her lifetime. Based on a study that examined the sociological factors contributing to the triad, it can be said that it is not just the outside society that affects female athletes— it results also from family and individual motivations. Westernized, contemporary society emphasizes the correlation between thinness and both beauty and happiness, an emphasis on self and body, and high demands that can be conflicting on the role of women in society. Western society also is capable of readily disseminating cultural values and styles through visual media, i.e. movies, television, magazines, etc. (Doherty, 1999). Changing the way an athlete thinks about her body, the way she trains, and how she eats has become challenging. Providing education may not be enough. Psychological therapy, especially cognitive behavioral therapy, has been shown to have high success with patients suffering from eating disorders (Waldrop, 2005). Psychological therapy could be successful in changing the way the athlete views her body from an emphasis on what society desires towards what is healthy and best for her long-term.
Unfortunately, because the female athlete triad is a “new” condition, there exists a lack of understanding as to what it is and how to treat it among health professionals, coaches, and athletic trainers. In the Troy, Hoch, and Stavrakos study, it was determined that very few physicians knew of the triad and the specific details, and a large majority was not comfortable in prescribing treatment for female athletes. Of the physicians who were aware of the triad, only 36% of pediatricians and 17% of gynecologists could identify the components (Troy et al., 2006). This is unsettling because those two types of physicians care for young female athletes during the essential formative years to establish or prevent the onset of the triad. The study established the need for further education among medical professionals and coaching staffs to increase awareness and encourage the proper treatment for recovery.

It is unfair to expect adolescent girls to recognize the signs of the triad in themselves or their teammates. However, it is not unreasonable to expect knowledge and understanding of this condition amongst coaches, athletic trainers, and medical professionals. How can a society attempt to eradicate a medical condition that only grows as participation by girls in sports grows if the professionals trusted to recognize and treat it are not informed? With increased awareness, individualized specific treatment plans to combat female athlete triad, and education to improve social attitudes towards female sports, the incidence of the female athlete triad can decrease. It remains primarily important to educate those responsible for the athletes and their diagnosis, treatment, and prevention in order to decrease prevalence. If the professionals who work with the female athletes can learn to spot the signs of the triad, in conjunction with the individual and her family, and provide a treatment plan, the end result of better athletic performance and life-long beneficial ramifications for bone health will arise. Establishing a team approach to treating the
The pressures of society place a “win at all costs” attitude in athletes’ minds, and for many female athletes that manifests into disordered eating which cascades into menstrual dysfunction and poor bone health. It is essential to change current attitudes to diminish the prevalence of the female athlete triad. It may never cease to exist because of the nature of exercise and the risks it poses to physiologic function if energy availability is not balanced. However, the inherent risks of exercise do not justify the lack of awareness that exists for the female athlete triad. In addition to creating standards for diagnosis and treatment, adding education to change social attitudes, increase knowledge and inform the public can aide in the reduction of the current prevalence of the female athlete triad. Many social movements of the
20\textsuperscript{th} century aimed to create equality; alas, these desires for equality have created new dichotomies within society. The female athlete triad is a result of the unequal standards that still exist between men and women within athletics. Forty years ago, a movement began to increase the participation of women in sport and athletics resulting in the Education Amendments of 1972 including Title IX. Unfortunately, this law opened doors for additional societal pressures to be placed on women within the sports culture. It is important to work towards the goals of equal opportunity in sport through decreasing the existence of a medical condition that has been exacerbated by societal influence. The female athlete triad is evidence of equality gone wrong. The triad is clearly defined; more research needs to be conducted to establish specific diagnosis and treatment standards, and to determine the damage left in the wake of the triad long after “recovery.” But there is no excuse for the lack of awareness that exists. Knowledge is power, and in this case that knowledge could eradicate the female athlete triad, both a social and physiological disease.
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