



Female Athlete Triad

The Flip Side of Active Living

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In 1997, the American College of Sport Medicine coined the term “female athlete triad” to represent an interrelated syndrome of three factors: disordered eating, amenorrhea, and osteoporosis.^{1,2}

At the time, it was believed this syndrome affected elite athletes and produced osteoporosis as a result of poor nutritional habits. Today, we understand we are dealing with a continuum of physiologic states that affects the recreational and elite active woman.

Female athlete triad has now been broadened to include:

1. Disordered eating as an energy deficit
2. Amenorrhea as an array of menstrual abnormalities
3. Osteoporosis to cover the continuum of compromised bone health leading to stress fracture

There is growing evidence this clinical sequelae of hypoestrogenism includes detrimental affects on the cardiovascular system.³⁻⁵

Terry's case

Terry, 16, is a competitive figure skater. She trains approximately six hours per week.



One day, during training, she experiences knee pain that becomes persistent and interferes with her ability to continue skating. Despite physical therapy, medication, and modification of her skating routine, there is no improvement and she is advised to stop skating for six weeks.

Her physician recommends water training and cycling as a way to cross train. Terry begins to modify her diet, as she feels her physical training has been reduced and she doesn't want to gain weight and lose her competitive edge.

After four weeks, she eliminates grains, cereals, and dairy and is consuming high-protein meals with a variety of vegetables and salads. She weighs herself three months later and notices she's lost 7 lbs. In addition, she's missed two menstrual periods and is now experiencing fatigue, decreased endurance, and a left shin pain that wakes her at night.

For more on Terry's case, go to page 65.

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Table 1

Prevention and treatment strategies for female athlete triad

Triad components	Prevention	Treatment
Disordered eating	Psychoeducation Dealing with food myths and facts Early detection of energy deficit	Menu planning Small, frequent meals Dietary and psychoeducational counselling
Menstrual dysfunction	Inclusion of healthy carbohydrates and fats Yearly physical exams, including menstrual history	Reversal through resumption of energy intake to output Refrain from early intervention of the OCP
Compromised bone health	Inclusion of weight-bearing, high-impact activities Inclusion of high dietary calcium and vitamin D Full investigation of any stress fractures	Gaining natural menstrual cycles through food energy balance to produce normal levels of cyclical estrogen Short-term calcitonin nasal spray (Miacalcin®) may be indicated Increased calcium and vitamin D

OCP: Oral contraceptive pill

1 *Disordered eating*

The definition of disordered eating is often misunderstood to mean a Diagnostic and Statistical Manual IV psychiatric diagnosis of eating disorder accompanied by intent to lose weight and high body dissatisfaction. In fact, disordered eating refers to the inappropriate choices of food, the elimination of food groups, and the inadequate caloric intake as compared with the energy output achieved on a regular basis. Many young women do not realize they are in an energy deficit. The restoration of this balance is the key to prevention and treatment of female athlete triad.

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2 *Menstrual dysfunction*

When a women is in energy deficit, her body begins to conserve metabolic energy by slowing down the basal metabolic rate (BMR) and altering the normal hormonal levels regulating the body. This includes, but is not limited to, the estrogen axis. Hypoestrogenism leads to:

- abnormal menstrual cycles,
- cessation of menstrual cycles,
- delayed onset of menarche after age 16, and
- luteal phase defects in an ovulatory cycle.

Other indicators of metabolic deficiency include low levels of triiodothyronine (T3), leptin, and growth hormone, as well as elevated cortisol and creatinine kinase.

The etiology of disordered eating may not be clinically significant, yet when combined with high-energy output leading to weight loss, there is a central suppression of reproductive function and metabolic rate.

3

*Compromised
bone health*

Bone density is maximized during adolescence, as approximately 90% of peak bone mass is achieved before the age of 20. This critical time of bone building can collide with episodes of poor calcium and vitamin D intake, poor nutritional balance, and lack of available estrogen. When this occurs, we begin to see signs of poor bone health, such as stress fracture, osteopenia, and in some cases, osteoporosis.

Women who regain their natural menstrual cycles supported by natural estrogen, as opposed to synthetic oral contraceptives, produce higher bone densities and faster recovery. The stimulation of bone density building requires impact and force stress in the form of exercises like jumping, hopping, skipping, and running. Walking is not enough to stimulate bone density development.

The role of calcium has been well understood in the past, but we are now realizing vitamin D is critical to healthy bones. Abnormal bone mass is not easily detected in adolescents by traditional methods; new forms of imaging are being studied, including quantitative computed tomography scans that measure cortex strength and magnetic resonance imaging analysis of bone density.

Table 1 provides a short summary of prevention and treatment strategies for the three factors involved with female athlete triad. **Dx**

A followup on Terry

During times of transition in training and routine, an athlete can often lose her "food routine", sacrificing healthy nutrition under the misconception that less is better to avoid gaining weight.

In this case, Terry may have unintentionally slowed down her metabolic rate, causing a state of hypoestrogenism. The rate of conversion to amenorrhea can be quick and relate to early changes in bone density, causing signs of potential stress fracture.

Although Terry reduces her skating, she continues to train in off-ice routines, maintain her daily activities, and increase her calisthenics exercises through her rehabilitation. She is likely already in a state of deficit prior to her injury and further tips the scales during her injury transition.

Terry's treatment includes:

- education on the connections between energy balance and metabolic health;
- increase in energy intake, while implementing more recovery time into her training schedule; and
- further investigations into her physical state, including:
 - a bone scan to rule out stress fracture,
 - initial blood work (complete blood count, iron, ferritin, cortisol, T3, creatine kinase, electrolytes, and phosphate), and
 - menstrual cycle monitoring.

References

1. Drinkwater BL, Bruemner B, Chesnut CH: Menstrual history as a determinant of current bone density in young athletes. *J Am Med Assoc* 1990; 263(4):545-8.
2. Otis CL, Drinkwater BL, Johnson M, et al: ACSM Position stand on the female athlete triad. *Med Sci Sports Exerc* 1997; 29(5):i-ix.
3. Friday KE, Drinkwater BL, Bruemner B, et al: Elevated plasma low-density lipoprotein and high-density lipoprotein cholesterol levels in amenorrheic athletes: Effects of endogenous hormone status and nutrient intake. *J Clin Endocrinol Metab* 1993; 77(6):1605-9.
4. Lamon-Fava S, Fisher EC, Nelson ME, et al: Effect of exercise and menstrual cycle status on plasma lipids, low density lipoprotein protein particle size and apolipoproteins. *J Clin Endocrinol Metab* 1989; 68(1):17-21.
5. Williams NI, Caston-Balderrama A, Helmreich DL, et al: Longitudinal changes in reproductive hormones and menstrual cyclicity in cynomolgus monkeys during strenuous exercise training: Abrupt transition to exercise induced amenorrhea. *Endocrinol* 2001; 142(6):2381-9.

Further references available—contact
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