Neuroendocrine system alterations in female athletes: an update

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Overview
Historically difficult to define, stress is, in one sense, the factor that stressors have in common in their impact on the body. Menstrual function is disrupted by stressors that activate the hypothalamic-pituitary-adrenal (HPA) axis; this activation is part of a catabolic response of the whole body that mobilizes metabolic fuels to meet energy demand. Functional menstrual disorders are associated with an increase in cortisol and with a broad spectrum of other symptoms of energy deficiency (Loucks AB and Redman LM 2004).

Two hormones with relevance to the reproductive system will be analysed in more detail in the presentation under the aspects of exercise stress and hypocalaric nutrition: luteinizing hormone (LH) and leptin.

To investigate the dependence of LH pulsatility on energy availability (dietary energy intake minus exercise energy expenditure), Loucks and Thuma (2003) measured LH pulsatility after manipulating the energy availability of 29 regularly menstruating, habitually sedentary, young women of normal body composition for 5 d in the early follicular phase. Subjects expended 15 kcal/kg of lean body mass (LBM) per day in supervised exercise at 70% of aerobic capacity while consuming a clinical dietary product to set energy availability at 45 and either 10, 20, or 30 kcal/kg LBM.d in two randomized trials separated by at least 2 months. Blood was sampled daily during treatments and at 10-min intervals for the next 24 h. Samples were assayed for LH, FSH, estradiol (E2), glucose, beta-hydroxybutyrate, insulin, cortisol, GH, IGF-I, IGF-I binding protein (IGFBP)-1, IGFBP-3, leptin, and T3. LH pulsatility was unaffected by an energy availability of 30 kcal/kg LBM.d, but below this threshold LH pulse frequency decreased, whereas LH pulse amplitude increased. This disruption was more extreme in women with short luteal phases. According to the authors, these incremental effects most closely resembled the effects of energy availability on plasma glucose, beta-hydroxybutyrate, GH, and cortisol and contrasted with the dependencies displayed by the other metabolic hormones. These results demonstrate that LH pulsatility is disrupted only below a threshold of energy availability deep into energy negative balance and suggest priorities for future investigations into the mechanism that mediates the nonlinear dependence of LH pulsatility on energy availability (Loucks AB, Thuma JR 2003). These results are resembled by our own studies on the effects of exercise and diets on LH pulsatility (Platen et al. 1998).

Because the effect of exercise on leptin was not established, Hilton and Loucks (2003) controlled energy intake (I) and exercise energy expenditure (E) to distinguish the independent effects of energy availability (A = I - E) and exercise stress (everything associated with exercise except its energy cost) on the diurnal leptin rhythm in healthy young women. In random order, the authors set A = 45 and 10 kcal. kg lean body mass(-1) (LBM) x day(-1) for 4 days during the early follicular phase of separate menstrual cycles in sedentary (S, n = 7) and exercising (X, n = 9: E = 30 kcal x kg LBM(-1) x day(-1)) women. Low energy availability suppressed the 24-h mean and amplitude, whereas exercise stress did not. Suppressions of the 24-h mean (-72 +/- 3 vs. -53 +/- 3%) and amplitude (-85 +/- 3 vs. -58 +/- 6%) were more extreme in S vs. X than previously reported effects on luteinizing hormone pulsatility and carbohydrate availability. Thus the diurnal rhythm of leptin depends on energy, or carbohydrate availability, not intake, and exercise has no suppressive effect on the diurnal rhythm of leptin beyond the impact of its energy cost on energy availability (Hilton LK, Loucks AB 2000).

Many athletes, especially female athletes and participants in endurance and aesthetic sports and sports with weight classes, are chronically energy deficient. This energy deficiency impairs performance and health. Reproductive disorders in female athletes are caused by low energy availability (defined as dietary energy intake minus exercise energy expenditure), perhaps specifically by low carbohydrate availability, and not by the stress of exercise. These reproductive disorders can be prevented or reversed by dietary supplementation in compensation for exercise energy expenditure without any modification of the exercise regimen. Energy balance is not the objective of athletic training. To maximize performance, athletes strive to achieve an optimum sport-specific body size, body composition and mix of energy stores. To pursue these objectives, athletes need to manage fat, protein and carbohydrate balances separately, but it is impractical for athletes to monitor these balances directly, and appetite is not a reliable indicator of their energy and macronutrient needs. To guide their progress, athletes need to eat by discipline and to monitor specific, reliable and practical biomarkers of their objectives. Skinfolds and urinary ketones may be the best biomarkers of fat stores and carbohydrate deficiency, respectively. Research is needed to identify and validate these and other markers (Loucks AB 2004).

References
Loucks, A. B. and Thuma, J. R. Luteinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating women. J Clin Endocrinol Metab. 2003; 88(1):297-311; ISSN: 0021-972X.