Overtraining Markers

I. Symptom markers
   A. Inflammation
      1. repeated trauma results in chronic inflammation (Smith 2004)
      2. traumatized tissue synthesizes cytokines
      3. cytokines promote recovery
   B. Reduced oxidative capacity of mitochondria (Newcomer 2005)
      1. decreased ability to generate ATP from oxidative phosphorylation
      2. increased reliance on anaerobic ATP production
      3. increased rates of fatigue
      4. muscles less economical
      5. muscle required more AYP to produce same amount of work
      6. not associated with whole body oxygen uptake
   C. Reaction time increased (Nederhof 2006)
      1. psychomotor slowness present in both non functional and functional overtraining syndrome
      2. Reaction time very sensitive marker of cognitive brain functioning (Rietjens 2005)
   D. Rating of Perceived exertion
      1. greater effort sense
      2. combines with decreased feelings of well being on POMS
   E. Altered autonomic cardiovascular activity (Baumert 2006)
      1. reduced resting heart rate variability
      2. reduced blood pressure variability
      3. sympathetic activation
      4. parasympathetic inhibition
      5. sympathetic activation
   F. Altered or dysfunctional hypothalamic-pituitary axis (Meeusen 2004)
      1. No increase, or decrease in hormones following two max test protocol
      2. Slight increase on second test in prolactin
      3. Marked decrease in adrenocorticotrophic hormone (ACTH)
   G. Increased levels of oxidative stress biomarkers (Margonis 2007)
      1. increase in urinary isoprostanes (7x)
      2. increased protein carbonyls
      3. increased TBARS
   H. Increased blood viscosity (iron depleted athletes) (Khaled 1998)(Alissa Benhaddad 1999)
      1. ferritin negatively related to blood viscosity
      2. RBC aggregability increases with iron deficiency
      3. Lower zinc levels
   I. Insulin like growth factor binding protein (IGFBP-3)
      1. fall in IGFBP-3 following an intense bout of exercise represents a index of tiredness
      2. fall of 25% in fatigued subjects
      3. rise of 40% in fit subjects
   J. “Heavy Legs” (Variet-Marie 2003)
      1. associated with mild plasma hyperviscosity
      2. associated with mild erythrocyte hyperaggregability
   K. Mild iron deficiency without anemia (Brownie 2004)
      1. impairs adaptation of endurance capacity in untrained women
      2. indicator was serum transferrin receptor
   L. Mucosal immunoglobulins Gleeson (2000)
      1. salivary IgA and IgM decline following a bout of intense exercise
      2. normal recovery within 24 hours
      3. continuous intense training results in chronic suppression
      4. associated with increased risk for URTI
II. Causes
A. Chronic intense training (Mackinnon 2000)
   1. impaired neutrophil function
   2. impaired natural killer cell activity
   3. increased incidence of URTI
   4. decreased endocrine response to training (Halson 2004)
   5. reduces circulating choline concentrations (precursor for neurotransmitter Acetylcholine) (Jager 2007)
   6. immune system dysfunction most pronounced when exercise continuous and prolonged (>1.5h) (Gleeson 2007)
   7. depression of immune function following exercise persists for 24 hours (Gleeson 2006)
B. Altered sleep cycles (Reilly 2007)
   1. results in immuno-suppression
   2. increased cortisol levels
   3. sleep deprivation has greater negative effect on females (Oginska 2006)
   4. large individual variations in sleep requirements
C. Stress (Christian 2006)
   1. academic exams
   2. even brief stress impedes healing
   3. caregiving (chronic stress)
D. Malnutrition
   1. iron deficiency is most common nutrient deficiency in the western world (Portal 2003)
   2. central suppression of reproductive function
   3. hypoestrogenism (De Souza 2005)
      a. loss of bone mineral density
      b. stress fractures
      c. osteoporosis
III. Solutions
A. Iron supplementation (Hinton 2007)
   1. prevented decline in ventilatory threshold
   2. significantly increased serum ferritin (sFer)
   3. increase was greater for those with low sFer prior to supplementation
   4. group who supplemented had significant improvements in muscular effiency
   5. iron supplementation necessary in iron deficiency as stores difficult to refill iron stores on diet alone (Portal 2003)
   6. Some disagreement on need for supplementation (Rodenberg 2007)
   7. Athletes should be screened and monitored if receiving iron therapy (Zoller 2004)
B. Dietary intervention
   1. carbohydrate supplementation (Halson 2007)
      a. reduces symptoms of overtraining
      b. does not prevent overtraining
      c. oxidative stress reduced by CHO supplementation (McAnulty 2007)
   2. carbohydrate and protein supplementation (Kreider 2007)
      a. increases insulin levels
      b. optimizes glycogen resynthesis
      c. enhances protein synthesis
      d. lessens immuno-suppressive effects of intense exercise
      e. attenuates rise in stress hormones (Gleeson 2006)
   3. Phospholipids and Phosphatidyl-phospholipids (Jager 2007)
      a. Phosphatidylcholine(PC) and Phosphatidylserine (PS) necessary for cellular function
      b. Supplementation with PS has been reproted to: attenuate circulating cortisol levels, improve perceived well-being, and reduce perceived muscle soreness after exercise
   4. Antioxidants cannot now be recommended for endurance athletes (Williams 2006)
C. Sleep

1. 2 hour mid-afternoon nap
   a. improves alertness
   b. improves psychomotor performance
   c. reverses one night’s sleep loss effects on cortisol and IL-6

2. Sleep is age dependant (Oginska 2006)
   a. 9 hr 23 minutes school children
   b. 8 hr 22 min college students
   c. 7 hr 37 minutes for young employees