Nutritional Considerations of Sports Medicine

Presented by: Ron Grabowski, D.C., R.D.
Nutritional Considerations of Sports Medicine
Nutritional Ergogenic Aids are Classified into 4 categories:

1) Substances that promote anabolism and improved body composition (e.g., dietary amino acids)

2) Substances that provide quickly utilizable energy (e.g., dietary carbohydrates)

3) Substances that facilitate recovery from physical exhaustion (e.g., dietary antioxidants)

4) Substances that fill other critical roles in exercise physiology (e.g., vitamins, sodium bicarbonate).
Supplement Usage

Surveys

• 76% of college athletes
• 100% of body builders

Protein Supplements

- Resistance-training athletes consuming 0.8 g/kg/day were in negative nitrogen balance.
- 1.4 g/kg/day – zero balance (equilibrium)
- 1.6 to 1.7 g/kg per day is optimal
CREATINE

Muscle – creatine phosphate

- Phosphate for adenosine diphosphate to restore adenosine triphosphate
- CP – buffer during high intensity exercise where lactic acid is produced.
Dosage

• 20 grams creatine (5g, qid)

• Marked increases of muscle creatine levels in 4 to 5 days.

• Can increase muscle phosphocreatine stores by 6 to 8 percent
Studies

• 20 to 30g of creatine per day for 1 week

• Enhanced performance of short term strenuous exercise.
Creatine Monohydrate

- Increased power in short-term, high intensity exercise
- Increased weight gain
CARNITINE

Mechanisms

- Increase fatty acid oxidation
- Spares glycogen
- Convert acetyl-coenzyme A (CoA) into acetyl-L-carnitine and CoA.
CoA Availability

- Essential for optimal functioning of the Kreb’s cycle.

- Decrease in ratio of acetyl-CoA:CoA may stimulate pyruvate dehydrogenase to increase glucose oxidation thereby preventing lactic acid production.
Eccentric Exercise

- Increased O₂ demand/O₂ supply
- Hypoxia
- Increased ATP consumption/ATP production
- Altered energy metabolism
- ATP depletion
- Altered ionic homeostasis
- Intracellular calcium overload
- Ultrastructural alterations
- Sarcolemma damage
- Enzyme release
- Symptoms (pain, soreness)

Vasodilation

- L-Carnitine

Accumulation of inhibitory metabolites

Mechanical stress
L-Carnitine vs D-Carnitine

**L-Carnitine:**
- Active
- Safe

**D-Carnitine**
- Depletes L-carnitine
- Muscle dysfunction
L-Carnitine

Dosage

- 2g/day for 4 weeks – increases muscle carnitine levels.
- Supplementation of 14 days or less had no effect on muscle carnitine levels.
Choline

- Muscle cramps/spasms

- Homocysteine

- Mixed research, inconclusive in regards to ergogenic effects.
Choline and Carnitine Connection

• These experiments indicate that free-living humans respond to supplementary choline by conserving carnitine through decreased urinary excretion of most fractions of carnitine and through a decreased renal clearance and fractional clearance of NEC (nonesterified carnitine).

• Carnitine reabsorption is likely to be a multistep process;
  ✓ choline has the opportunity to enhance carnitine transport through the tubular cells or the basolateral membrane into the circulation.

  ✓ It has also been suggested the possibility that choline is needed to position the carnitine carrier on the plasma membrane. Therefore, choline may aid the transport of carnitine into cells.
ASCORBATE & EXERCISE

• May function by preventing oxidation of tetrafolates to 10-formylfolic acid.

AJCN

• Ascorbate requirements are known to increase when the body is subjected to heat stress.

J. Nutrition

• Megaloblastic anemia occurs in some athletes, which is correctable by Ascorbate supplementation alone.

BMJ
Thiamine

- Thiamine pyrophosphate, plays an important role in the metabolism of both carbohydrate and the branched-chain amino acids.

- Coenzyme for pyruvate dehydrogenase (lipoamide), which catalyzes the conversion of pyruvate to acetyl CoA.

- Thiamine is also a coenzyme for oxoglutarate dehydrogenase (lipoamide), an enzyme responsible for the formation of succinyl CoA in the tricarboxylic acid cycle, and for branched-chain decarboxylase, an enzyme responsible for the catabolism of the branched-chain amino acids.

- Physical activity stresses these energy-producing metabolic pathways.

[Br J Nutr 1993]
Thiamin Deficiency

- Decreased wound strength (55% of normal)
- Decreased Lysyl oxidase activity in skin and wound (78% normal)
- Decreased Hydroxyproline content in collagen
- Abnormal maturation of collagen fibers
- Decreased Collagen accumulation in wounds
- Decreased Type III collagen in wounds (1st collagen type synthesized in wounds)
- Decreased differentiation of fibroblasts
- Derangement in macrophage ultra structure and phagocytosis
- Decreased wound healing
Riboflavin

• Necessary for the synthesis of 2 important coenzymes—flavin mononucleotide and flavin adenine dinucleotide (FAD).

• These coenzymes are especially important in the metabolism of glucose, fatty acids, glycerol, and amino acids for energy.

• Riboflavin is also involved in the conversion of vitamin B6 to its functional coenzyme.

CRC Press, 1998
Riboflavin Deficiency

- Increased ratio of alpha/beta chains in collagen
- Decreased content of total collagen (by 28%)
- Decreased aldehyde content of collagen (by 26%)
- Decreased elastin and elastin cross-links (by 50-60%)
- Decreased wound healing
- Granulation tissue much less dense, more vascular, more cellular
- Decreased pyridoxal phosphate levels (by 22%)
Pyridoxine & Exercise

- Average Intake of Pyridoxine in the USA
  Males = 1.87 mg/day (RDA = 2.0)
  Females = 1.16 mg/day (RDA = 1.6)

- Athletes Risks for Pyridoxine Deficiency
  Plays a pivotal role in the formation of heme.
  Deficiency impairs cobalamin absorption.
  Endurance exercise increases excretion.

AJCN (1984)
Pyridoxine

- A major function of vitamin B-6 is the metabolism of proteins and amino acids.
- The most biologically active form of vitamin B-6 is pyridoxal 5'-phosphate (PLP).
- PLP is a cofactor for transaminases, decarboxylases, and other enzymes used in the metabolic transformations of amino acids and nitrogen-containing compounds.
- During exercise, the gluconeogenic process involves the breakdown of amino acids for energy in the muscle and the conversion of lactic acid to glucose in the liver.
- Vitamin B-6 is directly related to energy production during exercise is the breakdown of muscle glycogen.
Zinc

Low zinc intakes and reduced serum zinc concentrations have been associated with:

- Impaired muscle function
- Reduced strength
- Increased propensity to fatigue
- Decreased power output during peak work capacity testing.

Zinc

• Inhibits histamine and leukotriene release.

• Involved in collagen formation.

• Antioxidant
  – Superoxide dismutase
Copper

- Important for collagen formation
  - Cross-linking

- Important for elastin formation
  - Cross-linking

- Superoxide dismutase
Iron

• Iron deficiency is the most common single nutrient deficiency disease in the world and is a major concern for 15% of the world's population.

• Heme iron - 5% to 35% of heme iron is absorbed from a single meal.

• Nonheme iron - absorption from a single meal can range from 2% to 20%.

• Required for collagen synthesis.
L-GLUTAMINE & EXERCISE

Protein Synthesis
The amount of protein synthesized in the muscle depends largely on the amount of L-Glutamine found in that muscle.

Considered a conditionally essential amino acid.
Skeletal Muscle produces most of the L-Glutamine in the body.
Glutamine

- Central to muscle function.
- The concentration of free glutamine in muscle is about 20 mmol/L, making it by far the amino acid present in the highest concentrations in the free amino acid pool of skeletal muscle (60% of the total free amino acid pool).
- A negative arterio-venous difference in the plasma glutamine concentration occurs across muscle and becomes particularly pronounced after prolonged exercise.
- In slow-twitch muscle, the intracellular concentration of glutamine is 3-fold higher than in fast-twitch muscle, suggesting a greater demand for glutamine in the muscle fibers most associated with endurance training.
- In prolonged and high-intensity exercise, plasma glutamine rises during exercise and then falls during the postexercise recovery period.
- This decline in glutamine following exercise has been implicated in the onset of acidosis, and in diminished immune response, particularly in the case of overtraining.
Human Studies - Stress

- Surgery
- Starvation
- Severe burns
- Trauma
- Severe infections
- Exercise
  - Significant reduction in L-Glutamine concentrations in the muscle.
Pro & Cons of Stress on L-Glutamine

• Synthesis is increased

• Release/Efflux is also increased and to a greater extent.

• Higher concentration than any other free amino acid in human skeletal muscle.
BCAA & L-Glutamine

• Even when BCAA were decreased by 75%, muscle protein synthesis did not change except when L-Glutamine levels were altered.
L-Glutamine & Muscle Mass

• Amount of L-Glutamine found in muscle may significantly influence the amount of protein synthesized in the muscle which in turn determines the rate at which you will increase muscle mass.
Anabolic Steroids & L-Glutamine

Study conducted by Dr. Max at Univ. Maryland Medical school, discovered that anabolic steroids block the effects of stress.

Exercise

Generates free radicals by various means;

1) increases in epinephrine and other catecholamines that can produce oxygen radicals when they are metabolically inactivated.

2) production of lactic acid that can convert a weakly damaging free radical (superoxide) into a strongly damaging one (hydroxyl).

3) inflammatory responses to secondary muscle damage incurred with overexertion.

Free Radic Biol Med 1995
J Appl Physiol 1995
Eur J Appl Physiol 1998
Delayed Onset Muscle Soreness (DOMS)
Definition of Delayed Onset Muscle Soreness

• Classified as a type I muscle strain injury.

• Presents with tenderness or stiffness to palpation and/or movement.

• Tenderness is concentrated in the distal portion of the muscle and becomes progressively diffuse by 24 – 48 hours post exercise.

• DOMS is usually associated with unfamiliar high-force muscular work and is precipitated by eccentric actions.
Delayed-Onset Muscle Soreness (DOMS)

- Is an indicator of muscle damage induced by exercise.

- Kaminski and Boal had subjects ingest 3 g ascorbic acid/d or a placebo for 3 d before eccentric exercise of the calf muscles. The vitamin C supplement appeared to reduce the intensity of soreness.

  ✓ Approximately one-half of the subjects there was a > 33% reduction in soreness compared with the placebo.

Pain - 1992
Proposed Mechanisms Underlying DOMS

- Lactic acid
- Muscle spasm
- Connective tissue damage
- Muscle damage
- Inflammation
- Enzyme efflux
Lactic acid Theory

• Theory has largely been rejected.

• Lactic acid levels return to pre-exercised levels within 1 hour following exercises.

• Lactic acid may contribute to the acute pain associated with fatigue.
Muscle Spasm Theory

- Increased resting muscle activation indicated a tonic localized spasm of motor units.
  - Compression of local blood vessels
  - Ischaemia
  - Accumulation of pain substances
Connective Tissue Damage Theory

- Type I (slow twitch) fibres display a more robust structure than type II (fast twitch) fibres.

- Fast twitch fibres may demonstrate an increased susceptibility to stretched-induced injury and excessive strain of the connective tissue may lead to muscle soreness.

- Urine Samples
  - Hydroxyproline (HP) - elevations
  - Hydroxylysine (HL) – elevations

- HP and HL amino acids are a component of mature collagen and presence in the urine is the result of collagen degradation.
Muscle Damage Theory

- Disruption of the contractile component of the muscle tissue, particularly the level of the z-line, following eccentric exercise.

- Microscopic lesion
  - Broadening
  - Smearing
  - Total myofibrillar disruption of the z-line

- Particularly amongst the type II fibres, which have the narrowest and weakest z-lines.

- Creatine kinase elevations (up to 5 days).
Inflammation Theory

Few hours

- Significant elevation of circulating neutrophils.
- Substances released to attract monocytes between 6 – 12 hours that turn into macrophages.
- Monocytes/macrophages peak in number at 48 hours.
  - Macrophages produce PGE$_2$
    - Sensitizes type III & IV nerve endings to mechanical, chemical or thermal stimulation.
Enzyme Efflux Theory

Calcium
- Normally stored in the sarcoplasmic reticulum.
- Accumulates in injured muscles following sarcolemmal damage.
- Inhibition of cellular respiration at the mitochondrial level causing ATP regeneration.
  - Required for the active transport of Ca++ back into the sarcoplasmic reticulum, to slow.
- Ca++ accumulation
  - Activates proteases and phospholipases
    - Leukotrienes and prostaglandins
Mechanical

- Group III and IV afferent receptors are found mostly within the connective tissue of the muscle.
- Group IV receptors respond to mechanical, chemical and noxious stimuli.
- Noticeable histological variation by 24 hours in the proteoglycan component of the ECM following eccentric activity.
- Structural disruption of the proteoglycan component may result in attraction of water within the ECM as part of an osmotic force that leads to fluid accumulation.
- It has been suggested that the stiffness is due to connective tissue damage, including tissue oedema, which causes increased mechanical sensitivity of the muscle receptors giving rise to discomfort when tendons are activated by pressure or stretching.
Biochemical

- Biopsies of elbow flexors taken at 48 hours' after 70 maximal eccentric repetitions;
  - Separation of the ECM
  - Mast cell degranulation
  - Increase plasma constituents in the extracellular space.

- Mast cell degranulation
  - Release of histamine and the cause of pain.
  - Kinin may be responsible for the pain.
    - Kinins are released by the action of proteases on kininogens and it is possible that the same proteases involved in connective tissue damage could produce kinins.
Smith – Hypothesis

- Substances such as bradykinin, histamine, serotonin and acetylcholine may stimulate pain afferents.
- Most likely chemical stimulant may be prostaglandin E₂ (PGE₂).
- PE₂ causes increased sensitivity of the pain receptors.
- Macrophages have the capability of synthesizing PGE₂.
- Kent and Hart have reported that neutrophils provide essential products for the synthesis of prostaglandins.
- During tissue repair fibrinogen is converted to fibrin which also activates prostaglandins.
- Injured muscle there are cells and processes which may be responsible for the synthesis of PGE₂. This in turn may sensitize the afferent receptors of the muscle to mechanical stimuli (such as stretch or swelling) causing delayed muscle soreness.
Impact of DOMS on Athletic Performance

- Functional Impairment
- Joint Kinematics
- Strength and Power
- Altered Recruitment Patterns
- Injury Risk factors
Please join us next month, Thursday July 15th, for our webinar on, “Nutritional Considerations of ADHD and Autism.”

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