Nutrition and Wound Healing - Cheat Sheet

- **Well-balanced, whole foods diet**
  - consume nutrient-dense foods to support the anabolic process of wound healing
  - Foods to avoid limit
    - trans-fat, saturated fat, cholesterol
  - Reduce sodium intake (DASH - <2300 mg/day)
- **Protein**
  - 1.25 - 1.5 g/kg/day
- **Adequate Hydration**
  - 1 mL of fluid / 1 kcal ingested (30-35 mL/kg/day)
- **Caloric intake**
  - 30-35 kcal / kg of body weight/day
- **Oral nutritional supplementation (as needed)**
  - Multivitamin (micronutrients and minerals)
  - Arginine - a precursor to collagen formation and NO production
  - Glutamine - increases collagen production at the wound
  - HMB (hydroxymethyl butyrate) - helps with protein synthesis

- ***specific nutrition recommendations should be individualized based on each patient's preexisting conditions, lab results, age***

The Role of Nutrition in Chronic Wound Care Management

What patients eat affects how they heal.

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The multi-faceted approach to the management of chronic wounds can be a daunting challenge. Treatment strategies for chronic wounds of the lower extremity often entail a multidisciplinary approach focusing on therapies, including off-loading modalities, serial wound debridements, assessment of arterial vascular inflow, and managing bacterial bioburden. While these do represent the major pillars of chronic wound management, evaluation and optimization of nutritional status should be included as part of the comprehensive treatment of the chronic wound patient.

Hippocrates in 5th century B.C. recognized the value of nutrition and the potential of certain foods for good health and said "Let food be thy medicine and medicine be thy food." Wound healing requires the body to be in an anabolic state that necessitates specific macro- and micronutrients as well as proper hydration to fuel the physiologic process. Since dietary depletion may contribute to wound chronicity,[1] proper assessment of nutritional status is paramount to predict healing potential.

The complex wound healing cascade progresses from hemostasis, inflammation, proliferation, and ultimately tissue remodeling. Among other factors, this process relies on adequate levels of macro-and micro-nutrients to ensure a timely progression through these stages. Patients who are malnourished may lack the necessary fundamental building blocks to promote tissue regeneration, leading to wound chronicity.[1] Patients may be malnourished for a number of reasons, including limited financial resources, lack of family support, dietary constraints, anxiety, depression, and diminished appetite. Malnourishment ensues if a patient's nutritional intake does not meet the body's metabolic demands.

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Malnutrition

Malnutrition is defined as loss of body tissue and diminished body functions caused by inadequate nutritional intake.[2] Firstly, during a period of malnourishment the patient will deplete the liver's glycogen stores to ensure glucose is continually supplied to vital body cellular functions. However, glycogen stores are often depleted within the first 24 hours following starvation. The continued stress of starvation will increase the metabolic rate and result in the release of cortisol from the adrenal glands.[3] This will improve the body's ability to mobilize amino acids from protein sources to support hepatic gluconeogenesis and continually supplying the body with glucose. During this stress response with increased cortisol production, elevated levels of inflammatory cytokines, including interleukin 1 (IL-1), interleukin 6 (IL-6), and tumor necrosis factor alpha will be elevated, contributing to a delay in wound healing.[4]

This systemic inflammation will also cause a decrease in negative acute phase reactants including albumin and pre-albumin.[4] With continual lack of nutritional intake, the body will begin metabolizing fat for energy, sparing the sustained breakdown of muscle tissue for protein.[3] This transition from primarily protein to fat metabolism will minimize muscle and overall weight loss. A consequence of adipose tissue depletion, however, may contribute to increased pressure on osseous prominences leading to pressure ulcer formation.

Following the depletion of fat stores, the body will again rely primarily on skeletal muscle for energy demands, resulting in rapid weight loss and overall cachexia. Clinically, a malnourished status will contrib-

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