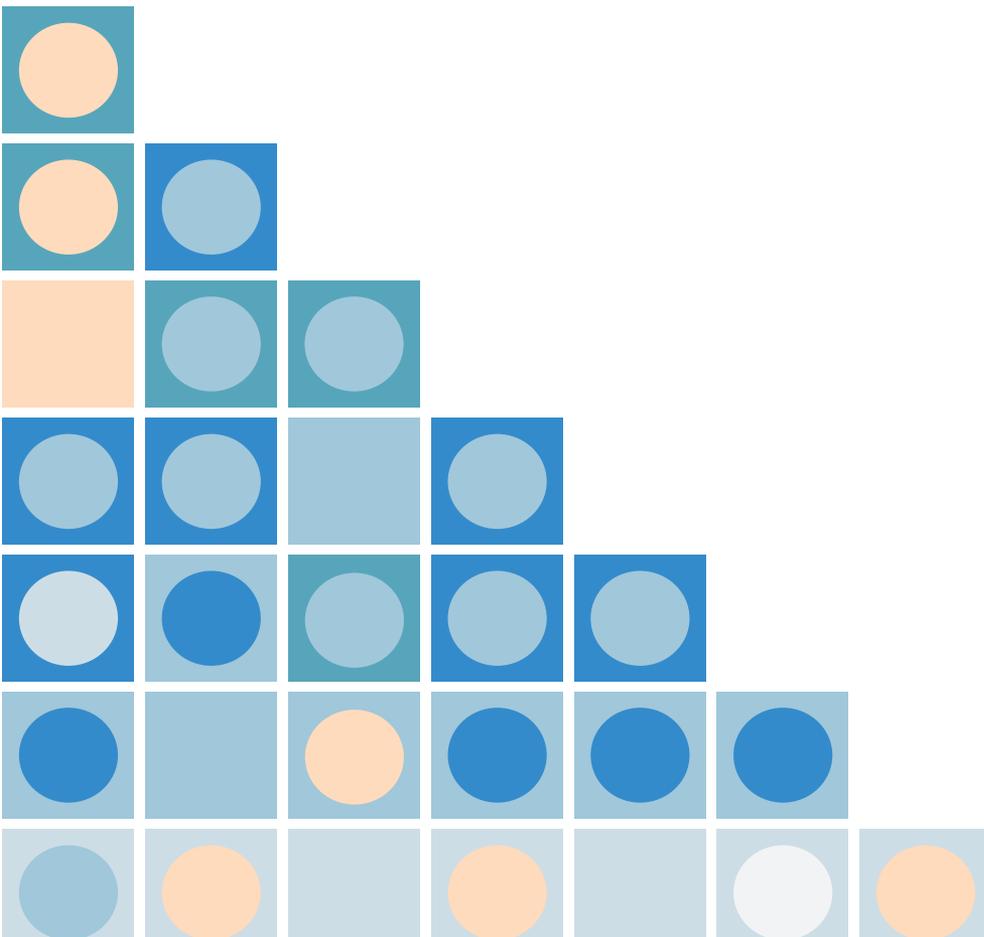


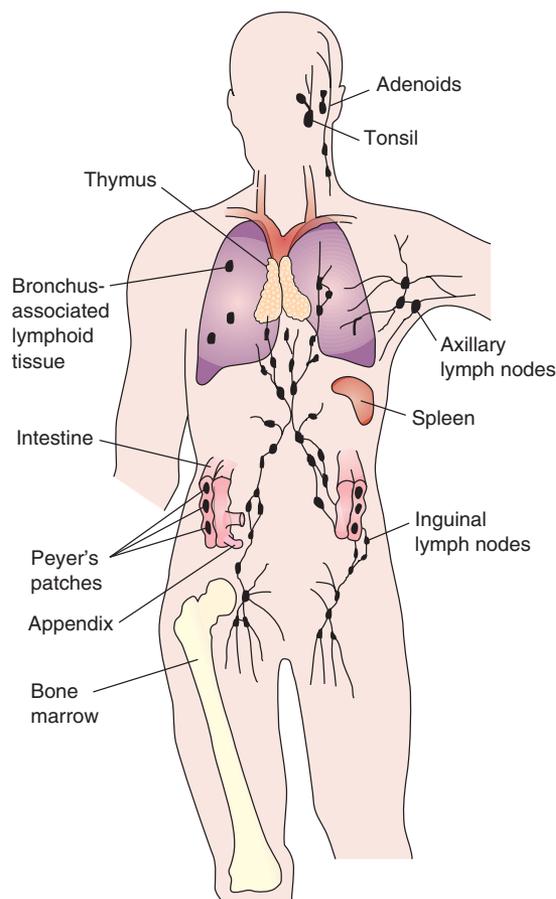
# Musculo-Skeletal and Collagen Disorders

## CHIEF ASSESSMENT FACTORS

- Actual Height, Measured Annually for Height Loss
- Arthritis—Warning Signs and Symptoms >2 Weeks: Early Morning Stiffness; Swelling in One or More Joints; Redness and Warmth in a Joint; Unexplained Weight Loss, Fever, or Weakness Combined with Joint Pain
- Bone Density Assessment
- Bone-Wasting Medications
- Contractures
- Easy Fatigue
- Edema
- Extremity Weakness
- Inflammation of Joints
- Movement Problems, Stiffness
- Pain in Muscles, Joints, Bones, Spine
- Psoriasis
- Unsteady Gait and Propensity to Fall
- Weight Loss, Anorexia, Depression, Insomnia
- Vitamin D<sub>3</sub> status (serum 25-OHD)



## OVERVIEW—RHEUMATIC DISORDERS



Adapted from: Porth CM. *Pathophysiology: Concepts of altered health states*, 5th ed. Philadelphia: Lippincott Williams & Wilkins, 1998.

Some rheumatic diseases involve connective tissues and others may be caused by autoimmune disorders, where the body attacks its own healthy cells and tissues. See Table 11-1. Rheumatic disorders include osteoarthritis (OA), rheumatoid arthritis (RA), juvenile RA, bursitis, tendonitis, infectious arthritis, spondyloarthropathies, polymyositis, psoriatic arthritis, systemic lupus erythematosus, scleroderma, polymyalgia rheumatica, polyarthritis nodosa, giant cell arteritis, gout, and fibromyalgia. Typically, treatment of these disorders includes a rheumatologist who specializes in the treatment of disorders that affect joints, soft tissue, bones and connective tissues.

Arthritis represents a group of more than 100 different rheumatic diseases that cause stiffness, pain, swelling in the joints, muscles, ligaments, tendons or bones. Over 15% (40 million) of Americans have some form of arthritis. *Spondylosis* is OA of the spine. *Infectious arthritis* is caused by bacterial invasion spread from nearby joints following chickenpox, rubella, or mumps. Autoimmune disorders, Crohn's disease, and psoriasis may cause *seronegative arthritis*. *Mixed connective tissue disease* shows features of RA, cutaneous systemic sclerosis, inflammatory myopathies and Raynaud's syndrome.

Mast cells and basophils are involved in several inflammatory and immune events and are known to produce a broad spectrum of cytokines (Rasheed et al, 2009). The activation of nuclear transcription factor- $\kappa$ B is linked with arthritis, osteoporosis, and psoriasis. The cytokine tumor necrosis factor

TABLE 11-1 Autoimmune Rheumatic Disorders<sup>a</sup>

<b>Blood and blood vessels</b>	Lupus Polyarteritis nodosa Temporal arteritis and Polymyalgia rheumatica
<b>Digestive tract and mouth</b>	Scleroderma Sjögren's syndrome
<b>Eyes</b>	Sjögren's syndrome Uveitis
<b>Heart</b>	Ankylosing spondylitis Lupus Rheumatic fever Scleroderma
<b>Joints</b>	Ankylosing spondylitis Lupus Osteoarthritis Rheumatoid arthritis
<b>Kidneys</b>	Gout Lupus
<b>Lungs</b>	Lupus Rheumatoid arthritis Scleroderma
<b>Muscles</b>	Polymyositis
<b>Nerves and brain</b>	Lupus
<b>Skin</b>	Lupus Scleroderma

<sup>a</sup>When the immune system does not work right, the immune cells can mistake the body's own cells as invaders and attack them; these are called autoimmune diseases. In this table a sample list of body systems affected by autoimmune rheumatic disorders. Adapted from: National Institutes of Health (NIH). NIH Publication No. 02-4858. Available at <http://www.niams.nih.gov/hi/topics/autoimmune/autoimmunity.htm>.

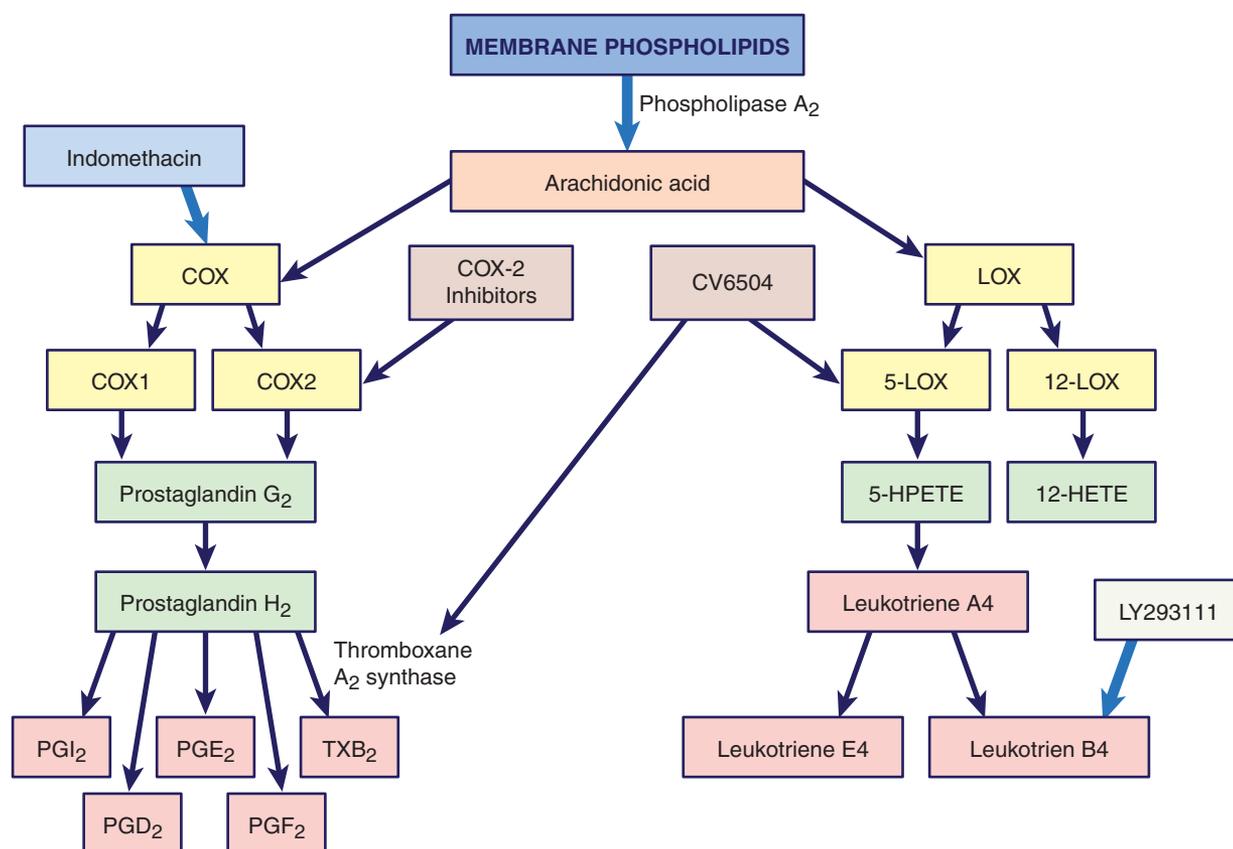
alpha (TNF $\alpha$ ) plays a key role in chronic inflammatory and rheumatic diseases.

Early recognition and treatment of these disorders are important. RA, juvenile idiopathic arthritis, the seronegative spondyloarthropathies, and lupus may have skeletal pathology (Walsh et al, 2005) and an inflammatory atherosclerosis. A multidisciplinary, multipronged approach is best. Physical and occupational therapies are beneficial for maintaining as much independence as possible in these conditions.

Most rheumatic conditions are managed by use of nonsteroidal anti-inflammatory drugs (NSAIDs) and TNF $\alpha$  antagonists. Etanercept, infliximab, and adalimumab significantly reduce symptoms and improve both functionality and quality of life (Braun et al, 2006; Nash and Florin, 2005). Fortunately, research is on-going for the autoimmune diseases. Gene profiling is helpful, especially in pediatrics (Jarvis, 2005). Osteoimmunology is a new branch of medical science, and anti-inflammatory therapies promise new treatments.

### Role of Inflammation and Fatty Acids

Excessive and inappropriate inflammation contributes to acute and chronic human diseases. It is characterized by the production of inflammatory cytokines, arachidonic



acid-derived eicosanoids (prostaglandins, thromboxanes, leukotrienes, and other oxidized derivatives), other inflammatory agents (e.g., reactive oxygen species), and adhesion molecules (Calder, 2006). Three major types of omega-3 fatty acids are ingested in foods: alpha linolenic acid (ALA), eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA). The body converts ALA to EPA and DHA, which are readily used by the body. Omega-3 fatty acids help reduce inflammation, while omega-6 fatty acids tend to promote inflammation. The precursor ALA does not appear to exert anti-inflammatory effects at achievable intakes (Calder, 2006).

A balance between omega-3 and omega-6 fatty acids in the diet is needed. The proper balance helps maintain and even improve health; one to four times more omega-6 fatty acids than omega-3 fatty acids is desirable, yet people who follow a Western diet consume a higher percentage of omega-6 fatty acids than they should.

Long-chain omega-3 polyunsaturated fatty acids (PUFAs) act by replacing arachidonic acid as an eicosanoid substrate, inhibiting arachidonic acid metabolism; by altering the expression of inflammatory genes through effects on transcription factor activation; and by leading to anti-inflammatory mediators known as resolvins (Calder, 2006).

### Role of Phytochemicals and Total Diet

Phytochemicals known for their ability to protect tissue also appear to block the activity of an enzyme that triggers inflammation in joints. See Table 11-2.

### Complementary and Alternative Medicine (CAM) Therapies

Controlled scientific studies of many patients can prove that a particular treatment is beneficial or that an apparent improvement is incidental. The important consideration is that treatment should do no harm.

Some studies have been done in alternative therapies, particularly diet in the treatment of arthritis, but none have shown any real long-term benefit. Patients often do benefit from complementary therapies, either because the treatment truly works or because of psychological (placebo) effects. While there is evidence of benefit for vitamin C, vitamin D, and nutraceuticals such as glucosamine, chondroitin, *S*-adenosylmethionine, ginger, and avocado/soybean unsaponifiables (McAlindon, 2006), specific diets and herbal or botanical products should only be used with medical consultation.

While the best nutrition-based strategy for promoting optimal health and reducing the risk of chronic disease is to wisely choose a wide variety of foods, additional nutrients from supplements can help some people meet their nutrition needs (American Dietetic Association, 2009). Physicians reported familiarity with acupuncture (80%), yoga (74%), and Tai-Chi (72%) yet almost all of their patients use CAM therapies (Mak et al, 2009). It is logical, then, that dietetics practitioners must keep up to date on the efficacy, safety, and the regulatory issues in order to provide the best advice.

**TABLE 11-2 Phytochemicals and Dietary Factors Affecting Rheumatic Disorders**

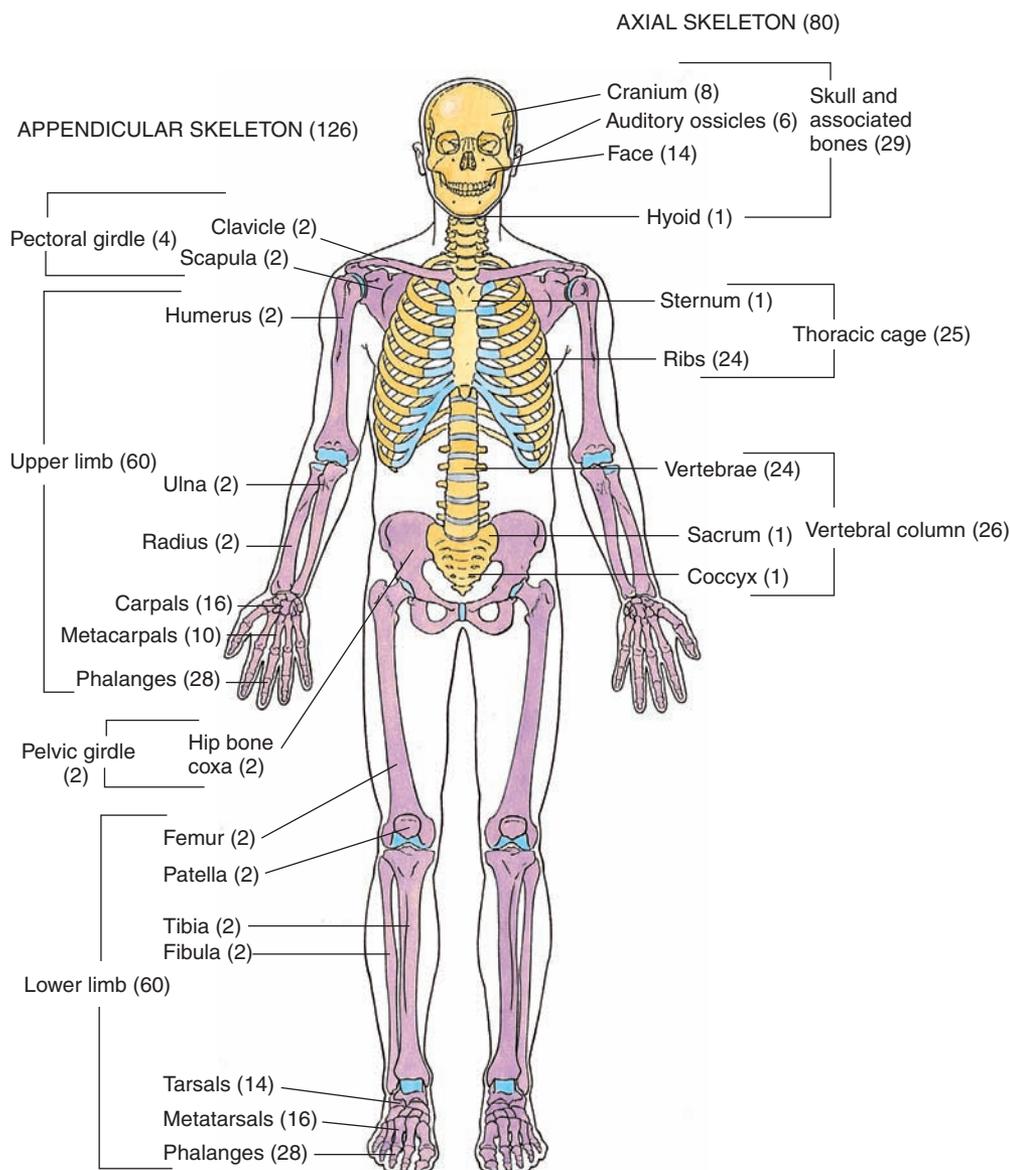
Component	Foods or Ingredients	Role
Cruciferous vegetables: broccoli, cauliflower, cabbage, bok choy	Sulforaphane	Boost phase 2 enzymes
Dairy products, low fat	To be identified; vitamin D?	Protective factors against gout (Choi, 2005).
Fruits: pomegranate, cranberry	Anthocyanins, tannins; ellagic acid; resveratrol; quercetin; vitamins A, C; selenium	Potent anti-inflammatory activity (Rasheed et al, 2009).
Long-chain polyunsaturated fatty acids	EPA and DHA	Replace arachidonic acid as an eicosanoid substrate, inhibiting arachidonic acid metabolism. Alter expression of inflammatory genes through effects on transcription factor activation, leading to anti-inflammatory mediators termed resolvins (Calder, 2006).
Mediterranean diet	Resveratrol, olive oil, lower intake of red meat	Protects against severity of rheumatoid arthritis (Choi, 2005).
Spices	Turmeric (curcumin) Red pepper (capsaicin) Cloves (eugenol) Ginger (gingerol) Cumin, anise, and fennel (anethol) Basil, rosemary (ursolic acid) Garlic (diallyl sulfide, ajoene, S-allylmercaptocysteine)	Interrupts pathway for transcription factor- $\kappa$ B (Aggarwal and Shishodia, 2004).
Vitamin D	Hormone affects over 2000 genes	Needed for healthy immune system, gene expression, strong bones.
Total protein and purine-rich vegetables	Neutral	Do not tend to promote gout (Choi, 2005).
Vitamin E, beta-carotene, and retinol.	Neutral	Have NOT been shown to halt the progression of rheumatic disorders
Red meats, seafood, beer, and liquor	Undesirable	Tend to promote symptoms of gout, inflammatory polyarthritis, or rheumatoid arthritis (Choi, 2005).

Sources: Aggarwal BB, Shishodia S. Suppression of the nuclear factor-kappa B activation pathway by spice-derived phytochemicals: reasoning for seasoning. *Ann N Y Acad Sci.* 1030:434, 2004.

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- Walsh NC, et al. Rheumatic diseases: the effects of inflammation on bone. *Immunol Rev.* 208:228, 2005.

## OVERVIEW—BONE DISORDERS



Adapted from: Moore KL, Agur AMR. *Essential Clinical Anatomy*, 2nd ed. Baltimore. Lippincott Williams & Wilkins

Bones are living, growing, and changing parts of the body. The human skeletal system consists of bones, cartilage, ligaments, and tendons and accounts for about 20% of the body weight. Osteoblasts are bone-forming cells, osteoclasts resorb or break down bone, and osteocytes are mature bone cells. The osteoblast is an endocrine cell type.

There is a reciprocal regulation of bone and energy metabolism by leptin and osteocalcin. Leptin inhibits insulin secretion by beta cells while osteocalcin favors it (Hinoi et al, 2009). Leptin deficiency leads to increased osteoblast activity and increased bone mass. Expression of the *Esp* gene, exclusive to osteoblasts, regulates glucose homeostasis and adiposity through controlling osteoblastic secretion of osteocalcin (Wolf, 2008). Osteocalcin deficiency leads to decreased insulin and adiponectin secretion, insulin resistance, higher serum glucose levels, and increased adiposity (Wolf, 2008). This recently understood concept has implications for diabetes and the metabolic syndrome.

There are 206 bones in the adult skeleton. The two types of bone tissue (compact and spongy) differ in density. Bone strength is derived from quantity (density and size) and quality (structure, consistency, and turnover). Bone mass is dependent upon individual genetic background. Adequate nutrient intake is needed from birth to achieve maximal bone mass and to prevent osteoporosis later in life.

The trace elements, calcium and phosphorus, are involved in skeletal growth. Parathyroid hormone (PTH) regulates calcium and bone homeostasis; it is expressed in the placenta, regulates the placental expression of genes involved in calcium and other solute transfer, and may directly stimulate placental calcium transfer (Simmonds et al, 2010).

Magnesium and fluoride are matrix constituents while zinc, copper and manganese are components of enzymatic systems involved in matrix turnover. A sufficient protein intake, along with adequate calcium, supports stronger

**TABLE 11-3 Recommendations for Prevention of Osteoporosis**

- Get the recommended amounts of calcium and vitamin D<sub>3</sub> for age and sex; use supplements when diets are inadequate.
- Maintain a healthy weight and be physically active 30+ minutes a day for adults and 60+ minutes a day for children, including weight-bearing activities to improve strength and balance.
- Minimize the risk of falls by removing items that might cause tripping, improving lighting, and encouraging regular exercise and vision tests to improve balance and coordination.
- Risks for patients of all ages should be evaluated by health care professionals. Obtain bone density tests for women over the age of 65 and for any man or woman who suffers even a minor fracture after the age of 50. "Red flags" for someone is at risk include a history of multiple fractures, those who take certain medications, and those who have a disease that can lead to bone loss.
- A BMD test is used to detect osteoporosis before fractures occur, predict chances of future fractures, or determine rate of bone loss and monitor the effects of treatment. The DEXA scan is most common.
- Normal BMD: within 1 standard deviation (SD) of a "young normal" adult.
  - Low bone mass (osteopenia): BMD is between 1 and 2.5 SD below that of a "young normal" adult.
  - Osteoporosis: BMD is 2.5 SD or more below that of a "young normal" adult.

bone density; this fact contradicts past suggestions that high-protein diets deplete bone strength.

Changes in bone turnover markers may become accurate predictors of fracture risk. Assessing risk factors for low bone mass is important in monitoring the etiology of fracture in older individuals (Kelsey et al, 2006). In general, women's bone health has been studied more extensively than that of men. Studies on the predictors of fractures in men are needed, such as bone architecture, morphology, biochemical markers of bone turnover, and hormonal levels (Szulc et al, 2005).

Vitamins are important. Vitamin D<sub>3</sub> plays a role in calcium metabolism. Vitamins C and K are cofactors of key enzymes for skeletal metabolism. Another indicator of bone health is heart health. There are similar pathophysiological mechanisms underlying cardiovascular disease (such as dyslipidemia, oxidative stress, inflammation, hyperhomocysteinemia, hypertension, and diabetes) and low bone mineral density (BMD). Sufficient folic acid, vitamins B<sub>6</sub> and B<sub>12</sub> can help improve bone health by lowering elevated homocysteine levels. Antioxidant nutrients, including vitamins A and C and selenium, play a role in bone health.

While calcium is widely recognized for bone health, other minerals are equally important. Iron promotes production of collagen in bone structure; 18 mg is most protective for women but balance is also critical as too much iron may throw off calcium balance. Finally, silicon in the form of choline-stabilized orthosilicic acid is the bioavailable form that enhances calcium and vitamin D<sub>3</sub> in bone health.

Omega-3 fatty acids such as EPA help increase levels of calcium in the body, deposit calcium in the bones, and improve bone strength. People who are deficient in EFAs EPA and gamma linolenic acid (GLA) are more prone to bone loss.

Former U.S. Surgeon General Richard H. Carmona (2005) warned in a landmark report that, by 2020, half of all American citizens older than 50 would be at risk for fractures from osteoporosis and low bone mass if immediate action is delayed by individuals at risk, doctors, health systems, or policymakers. At least 10 million Americans over the age of 50 have osteoporosis, another 34 million are at risk for developing osteoporosis, and roughly 1.5 million people have suffered a bone fracture related to osteoporosis. About 20% of senior citizens who suffer a hip fracture die within a year of fracture; another 20% of individuals with a hip fracture end up in a nursing home. Hip fractures account for 300,000 hospitalizations each year. See Table 11-3 for recommendations to prevent osteoporosis.

### For More Information

- American Academy of Orthopaedic Surgeons  
<http://www.aaos.org/>
- American Academy of Physical Medicine and Rehabilitation  
<http://www.aapmr.org>
- American Autoimmune-Related Diseases Association (AARDA)  
<http://www.aarda.org/>
- American College of Rheumatology  
<http://www.rheumatology.org/>
- American Osteopathic Association  
<http://www.do-online.osteotech.org/>
- American Pain Foundation  
<http://www.painfoundation.org/>
- American Society for Bone and Mineral Research  
<http://www.asbmr.org/>
- Arthritis Foundation  
<http://www.arthritis.org/>
- Autoimmunity Resources  
<http://www.aarda.org/links.php>
- CAM Therapy Resources  
<http://nccam.nih.gov/health/bydisease.htm>
- CDC—Calcium for Bone Health  
<http://www.cdc.gov/nutrition/everyone/basics/vitamins/calcium.html>
- Clinical Trials Research Trials  
<http://www.aarda.org/links.php>
- Drug List  
<http://www.rxlist.com/alternative.htm>
- Journal of Immunology  
<http://www.jimmunol.org/>
- National Institute of Arthritis and Musculoskeletal and Skin Disorders  
<http://www.niams.nih.gov/hi/index.htm>
- Quack Watch for Unproven Remedies  
<http://www.quackwatch.com/>
- Rheumatic Diseases Internet Journal  
<http://www.rheuma21st.com/>

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### SAMPLE NUTRITION CARE PROCESS STEPS

#### Unintentional Weight Loss

**Assessment:** Loss of 15 lb this past 6 months, much pain and inflammation with ankylosing spondylitis, taking numerous medicines that cause GI distress and anorexia.

**Nutrition Diagnosis (PES):** Unintentional weight loss (NC-1.4) related to pain, inflammatory processes, and GI distress and evidenced by 15-lb unplanned weight loss in past 6 months.

#### Interventions:

Food and Nutrient Delivery: ND 1.2 Alter diet as tolerated. ND 32.3 and 32.4 Initiate vitamin and mineral supplementation.

Education: E-1.2 Discuss ways to increase energy and nutrient density in food choices.

Counseling: C 2.2 Agree to goal of consuming only nutrient-dense foods for the coming month until next visit. C-2.3 Keep a food diary for one month.

**Monitoring and Evaluation:** Review food diary after 1 month. Monitor weight for resolution of weight loss; goal is gain of 1–2 lb weekly.

- Improve ability to participate in physical activities of choice to maintain lean body mass.



## FOOD AND NUTRITION

- A normal diet is useful. Support gradual weight loss, if needed, to normalize weight. Some patients claim relief while using a vegetarian diet with less red meat.
- Preferred foods should be offered to stimulate appetite.
- Increase intake of foods rich in antioxidants such as vitamins E and C, selenium, and fish oils for rich sources of omega-3 fatty acids. Sufficient calcium and vitamin D are also important.
- Include phytochemicals derived from spices such as turmeric (curcumin); red pepper (capsaicin); cloves (eugenol); ginger (gingerol); cumin, anise, and fennel (anethol); basil and rosemary (ursolic acid); garlic (diallyl sulfide, S-allylmercaptocysteine, ajoene); and pomegranate (ellagic acid) (Aggarwal and Shishodia, 2004).

### Common Drugs Used and Potential Side Effects

- Sulfasalazine, methotrexate, azathioprine, cyclosporine, leflunomide, and tumor necrosis factor-alpha blocking agents can be considered as first-line therapy but there are possible harmful effects on intestinal integrity, permeability, and even on gut inflammation (Rodriguez-Reyna et al, 2009).

- Etanercept (Enbrel), an anti-TNF therapy, may improve mobility and quality of life (Braun et al, 2006; Davis et al, 2005; Temel et al, 2005). Infliximab (Remicade), another monoclonal antibody, also targets TNF $\alpha$  and provides clinical improvement. Upper respiratory infections, psoriatic rashes, and allergic reactions can occur.

### Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician. Ginger, corn, pineapple, and pigweed have been recommended; no clinical trials prove efficacy.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Exercise is crucial, especially swimming, to relieve back pain.
- Patient should practice deep breathing exercises for pain relief. Stretching and strengthening exercises also are important.
- Patient will likely find that sleeping on a hard bed, supine, is most helpful.
- Discuss role of energy intake for weight control.

### Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

#### For More Information

- Ankylosing Spondylitis International Federation <http://www.asif.rheumanet.org/>
- National Ankylosing Spondylitis Society (NASS)—United Kingdom <http://www.nass.co.uk/>
- Spondylitis Association of America <http://www.spondylitis.org>

### ANKYLOSING SPONDYLITIS—CITED REFERENCES

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# GOUT

## NUTRITIONAL ACUITY RANKING: LEVEL 2



Adapted from: Rubin E MD and Farber JL MD. *Pathology*, 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 1999.



### DEFINITIONS AND BACKGROUND

Uric acid is the end product of purine metabolism. Because humans have lost hepatic uricase activity, this leads to uniquely high serum uric acid concentrations when compared with other mammals. About 70% of daily urate disposal occurs via the kidneys; in 5–25% of the human population, impaired renal excretion leads to hyperuricemia.

Gout is a disorder of sudden and recurring attacks of painful arthritis with inflamed joints (usually the big toe, ankle, knees, and feet). Hyperuricemia promotes deposition of monosodium urate crystals in the joints and tendons. Gout affects more than 1% of adults in the United States and is the most common form of inflammatory arthritis among men (Saag and Choi, 2006). The disease tends to affect men between the ages of 30 and 50 years and is often hereditary.

Risks include genetic factors; high intake of seafood and red meats (Choi et al, 2005; Johnson et al, 2005) as well as beer and fructose (Doherty, 2009). Higher intakes of coffee, low-fat dairy products, and vitamin C are associated with lower risk (Doherty, 2009). See Table 11-4 for other etiologies of hyperuricemia. Gout prevalence increases in direct association with age, metabolic syndrome, hypertension, and use of thiazide diuretics (Saag and Choi, 2006). There is increased incidence in postmenopausal women, with polyarticular onset, hand involvement, and development of tophi (Ene-Stroescu and Gorbien, 2005). Tophi are hard lumps of urate crystals that are deposited under the skin around the joints and may be permanent.

Acute attacks may be triggered by surgery, sudden and severe illness, fasting, chemotherapy, or joint injury. Acute gout most commonly affects the first metatarsal joint of the foot, but other joints may also be involved. The joint swells, and skin turns warm, red, purplish, and shiny. Severe pain usually occurs, more so at night.

Gout progresses from asymptomatic hyperuricemia to acute gouty arthritis, intercritical gout (intervals between acute attacks), and finally to chronic tophaceous gout. Tophi

**TABLE 11-4 Acquired Causes of Hyperuricemia**

Cause	Description
<b>Increased urate production</b>	
Nutritional	Excess ethanol or fructose intake
Hematological	Myeloproliferative and lymphoproliferative disorders, polycythemia
Drugs	Ethanol, cytotoxic drugs, vitamin B <sub>12</sub> (treatment of pernicious anemia)
Miscellaneous	Obesity, psoriasis, hypertriglyceridemia
<b>Decreased renal excretion of urate</b>	
Drugs	Ethanol, cyclosporine (Sandimmune), thiazides, furosemide (Lasix) and other loop diuretics, ethambutol (Myambutol), pyrazinamide, aspirin (low-dose), levodopa (Larodopa), nicotinic acid (Nicolar)
Renal	Hypertension, polycystic kidney disease, chronic renal failure (any etiology)
Metabolic/endocrine	Dehydration, lactic acidosis, ketosis, hypothyroidism, hyperparathyroidism
Miscellaneous	Obesity, sarcoidosis, toxemia of pregnancy

Adapted from: Harris M, et al. Gout and hyperuricemia. *Am Fam Physician*. 59:925, 1999.

may develop if the condition goes untreated. Although attacks of gout can subside in a few days, repeated attacks can cause permanent joint damage, and the disease often results in substantial disability and frequent medical care.

Treatment includes the pain-relieving NSAIDs and, for more serious outbreaks, corticosteroids. Most patients with gout eventually require long-term treatment with medications that lower blood uric acid levels. Patients with asymptomatic hyperuricemia should lower their urate levels by changes in diet or lifestyle.



### ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** About 10% of people with hyperuricemia develop gout. Genetic variants within the transporter gene, SLC2A9 (GLUT9), affect both fructose and uric acid transport. Other renal urate transporters have been identified, including URAT1.

Clinical/History	Obesity	Urate crystals in urine
Height	Swollen, painful big toe (podagra)	Use of thiazide diuretics?
Weight	Arthritis	
BMI		

Tophus, suspected or proven	Birefringent crystals in the synovial fluid	Ca <sup>++</sup> , Mg <sup>++</sup> Na <sup>+</sup> , K <sup>+</sup>
Asymmetrical swelling within a joint on X-ray	BUN (increased) Cholesterol (Chol) Triglycerides (increased?)	Albumin (Alb) Creat Glucose (Gluc) AST, ALT Vitamin D <sub>3</sub> status (serum 25-OHD)
<b>Lab Work</b>		
CRP		
Uric acid (increased)		

## INTERVENTION



### OBJECTIVES

- Lower bodily stores of uric acid crystal deposits to prevent the inflammatory processes and structural alterations. Increase excretion of urates and force fluid intake to prevent uric acid kidney stones.
- Data from NHANES III show a remarkably high prevalence of the metabolic syndrome among individuals with gout, along with an increased risk of myocardial infarction and cardiovascular mortality (Hak and Choi, 2008). Encourage lifestyle changes including reduction in energy intake, weight, alcohol intake, red meat intake.
- Promote gradual weight loss. In the obese, controlled weight management has the potential to lower serum urate (Schlesinger, 2005).
- Correct any existing dyslipidemia and prevent complications such as renal disease, hypertension, and stroke.



### FOOD AND NUTRITION

- A low-fat, high-carbohydrate (CHO) diet increases excretion of urates. Vegetables such as peas, mushrooms,

cauliflower, and spinach yield a protective effect (Choi, 2005).

- Develop a weight loss plan if needed.
- Avoid excessive intake of seafood such as anchovies, sardines, caviar, and herring. Reduce intake of beef, pork, duck, bacon, turkey, and ham.
- Ensure a high-fluid intake, especially water and skim milk. Nonfat milk, low-fat yogurt, dairy products, fruits such as cherries, and high intakes of vegetable protein may reduce serum urate (Schlesinger, 2005).
- Use of 4+ cups of coffee per day should be recommended (Choi and Curhan, 2007).
- Exclude alcoholic beverages (Schlesinger, 2005) and fructose or sugar-sweetened soft drinks (Choi et al, 2008).
- Use antioxidant-rich foods such as pomegranate, raspberries, and strawberries.

## Common Drugs Used and Potential Side Effects

- Uricosuric drugs: Probenecid (Benemid) and sulfinpyrazone (Anturane) block renal absorption of urates. Serum uric acid levels should be kept below 360  $\mu\text{mol/L}$  (6 mg/dL). Use adequate fluid.

Anorexia, nausea, vomiting, and sore gums may result.

- The medication febuxostat (Uloric) shows promise.
- Xanthine oxidase inhibitors: Allopurinol (Aloprim) blocks uric acid formation. Adequate intake of fluid is needed. Mild gastrointestinal (GI) upset, taste changes, or diarrhea can occur; take after meals. Febuxostat is even more effective than allopurinol; side effects are transient (Schumacher, 2005).
- During more serious outbreaks, NSAIDs, colchicine (Colcrys), and corticosteroids (prednion) may be prescribed for short-term use.
- Medications that can increase uric acid levels include hydrochlorothiazide (a diuretic) and some transplantation medications (cyclosporine and tacrolimus). Monitor for signs of gout.

## Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Celery, avocado, turmeric, cat's claw, chiso, and devil's claw have been recommended; there are no clinical trials that prove efficacy.
- Vitamin C shows some effectiveness; 1000 mg may be beneficial in preventing gouty attacks (Gao et al, 2008).



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- The inflammatory response may be suppressed by omega-3 fatty acids from fish oils and from walnuts, flaxseed, and cherries. Use these foods several times a week.
- Alcohol, beef, sardines, anchovies, and pork may precipitate a gouty attack (Choi et al, 2005). Otherwise, there is little need for a traditional "low purine" diet (Hayman and Marcason, 2009).

## SAMPLE NUTRITION DIAGNOSES

### Excessive Alcohol Intake

**Assessment Data:** Diet history and food records, medication history, alcohol and fluid intake.

**Nutrition Diagnosis (PES):** Excessive alcohol intake related to consuming large amounts of alcohol (36 oz whiskey daily) as evidenced by recent painful flare of gout with hyperuricemia.

**Intervention:** Food-nutrient delivery—Decrease alcohol intake (ND 3.3). Education: Discuss role of proteins, alcohol, diet, fluid intake, and medications in managing gout. Counseling: Motivational interviewing and goal setting with patient (C 2.1, 2.2) to implement recommended lifestyle modifications into daily plan.

**Monitoring and Evaluation:** Evaluation of alcohol intake records; improvement in symptoms of gout. Monitor need for additional education/counseling. Evaluate for decrease in uric acid levels and lower frequency of gouty attacks.

- Weight loss may be helpful, but avoid fasting. Instruct patient to lose weight gradually.
- Discuss the importance of adequate fluid ingestion. Recommend coffee intake (Choi and Curhan, 2007). Avoid sugar-sweetened soft drinks and fructose, but diet soft drinks are acceptable (Choi et al, 2008; Hak and Choi, 2008).
- Aim to drink at least a half gallon of water and skim milk daily.

### Patient Education—Food Safety

If enteral or parenteral nutrition is needed, sanitation and handwashing are essential.

### For More Information

- American College of Rheumatology  
<http://www.rheumatology.org/>
- Arthritis—Gout  
<http://www.arthritis.org/conditions/diseasecenter/gout.asp>
- Diet for Gout  
[http://www.gout.com/diet\\_gout/gout\\_friendly\\_foods.aspx](http://www.gout.com/diet_gout/gout_friendly_foods.aspx)
- Mayo Clinic—Gout  
<http://www.mayoclinic.com/health/gout/DS00090/rss=1>

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## IMMOBILIZATION

### NUTRITIONAL ACUITY RANKING: LEVEL 2



#### DEFINITIONS AND BACKGROUND

Extended periods of immobilization, for various reasons, may be nutritionally depleting. Patients with orthopedic injuries may lose 15–20 lb from stress, immobilization, trauma, and bed rest. Prolonged immobilization and nonuse of lower and upper limb muscles may cause atrophy. Nitrogen depletion can be extensive. A large nitrogen loss and high protein oxidation can be related to extensive injury and elevated energy expenditure.

Unloading of weight-bearing bones induced by immobilization has significant impacts on calcium and bone metabolism. Immobilization hypercalcemia involves nausea, vomiting, abdominal cramps, constipation, headache, and lethargy.

Persons with physical disabilities frequently are nonambulatory and have bone loss due to immobility. Prevention of osteoporosis and related fractures in this population includes calcium and vitamin D supplementation and risk-based screening. With careful attention to functional capacity enhancements, bone mass can be restored (Rittweger et al, 2005).

In older individuals, sarcopenia is the result of excessive loss of muscle mass and strength, loss of mobility, neuromuscular impairment, and balance failure. Falls and fractures can lead to immobilization, which induces more loss of muscle mass.

One final group at risk for the consequences of immobilization are those individuals who are in intensive care units (ICU) for a prolonged period. There is a need for physical therapy, as possible, to avoid a long recovery.



#### ASSESSMENT, MONITORING, AND EVALUATION



#### CLINICAL INDICATORS

**Genetic Markers:** Immobilization is usually from injury or other nongenetic causes, but may be a side effect of certain diseases with a genetic origin, such as spina bifida.

Clinical/History	Dual-energy x-ray absorptiometry (DEXA)	Lab Work
Height or arm length/knee length	Decreased range of motion?	H & H
Weight	Contractures; stiff joints?	Alb
BMI	Blood clots	Transthyretin, retinol-binding protein (RBP)
Weight changes	Pressure ulcers	CRP
Triceps skinfold (TSF)	Constipation	Nitrogen (N) balance
Midarm muscle circumference (MAMC)	Indigestion, anorexia?	Ca <sup>++</sup>
Midarm circumference (MAC)	Depression?	(increased?)
	Change in quality of life?	Parathormone (PTH)

Urinary Ca <sup>++</sup> (high?)	Alk phos Mg <sup>++</sup>	BUN, Creat Na <sup>+</sup> , K <sup>+</sup>
Vitamin D <sub>3</sub> status (serum 25-OHD)	Red blood cell (RBC) count	

## INTERVENTION



### OBJECTIVES

- Correct negative nitrogen balance from increased losses (perhaps up to 2–3 g of nitrogen per day) to prevent pressure ulcers and infections. Moderate exercise is beneficial in altering the inflammatory milieu associated with immobility, and in improving muscle strength and physical function (Truong et al, 2009).
- Correct anorexia, indigestion, constipation.
- Prevent deossification and osteoporosis of bones. Prevent hypercalcemia from low serum levels of albumin, which normally binds calcium.
- Prevent kidney and bladder stones, urinary tract infections.
- Provide adequate fluid intake to aid excretion of nutrients.
- Prevent constipation, impactions, and obstruction.
- Prevent anemias that result from inadequate nitrogen balance.
- Prevent venous thrombosis (McManus et al, 2009).
- Improve or sustain a positive quality of life.



### FOOD AND NUTRITION

- Diet should provide adequate intake of high-biological value proteins to correct nitrogen balance. An intake of 1.2 g protein/kg body weight is often recommended. Provide adequate energy to spare protein; use sufficient carbohydrates and fats, including 1–2% total kilocalories as essential fatty acids (EFAs).
- Encourage adequate intake of calcium since a high-protein diet raises the body's calcium requirements. Increased

### SAMPLE NUTRITION DIAGNOSES

#### Physical Inactivity

**Assessment Data:** Diet history, food records, medication history, fluid intake. New paraplegia following motorcycle accident.

**Nutrition Diagnosis (PES):** Physical inactivity (NB 2.1) related to paraplegia as evidenced by inability to walk voluntarily after motorcycle accident.

**Intervention:** Food-nutrient delivery—Offer food preferences to maintain desired intake; monitor calcium and protein intake in particular. Education: Discuss importance of physical therapy and nutrition in maintaining as much lean body mass as possible.

**Monitoring and Evaluation:** Evaluate ability to tolerate sufficient physical therapy to maintain adequate skin integrity, muscle mass, and urinary tract function; ability to achieve desirable nitrogen and calcium balance.

intake of phosphorus during the first few weeks may be useful.

- Diet should provide a high-fluid intake.
- Intake of vitamin C and zinc should be adequate to protect against skin breakdown.
- Diet should provide adequate amounts of fiber to prevent constipation. Avoid overuse of fiber in cases where there is impaction.

### Common Drugs Used and Potential Side Effects

- Medications may be used to treat underlying conditions; they may have side effects that contribute to nutrient losses.
- Take pain medications as directed to maintain relief of pain, rather than only taking them when you feel very badly.
- Immobilization-induced hypercalcemia affects bone metabolism in Parkinson's disease; this inhibits secretion of PTH, which in turn suppresses 1,25-dihydroxyvitamin D production (Sato et al, 2005). These abnormalities may be corrected by the suppression of bone resorption with bisphosphonate; supplementations of calcium and vitamin D should be avoided in these patients (Sato et al, 2005).

### Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain that calcium and nutrient intakes will have to be monitored for patients who will be tube fed or on a liquid diet for extended periods of time.
- Explain the need for adequate fiber and fluid (2–3 L) to prevent constipation, urinary tract infections, and so on. Early ambulation is the best treatment possible.
- Because prolonged bed rest in the ICU affects the development of ICU-acquired weakness, early mobility requires a reduction in heavy sedation and bed rest (Truong et al, 2009). Identify strengths and limitations, and alternate rest periods with activity. Do range of motion exercises every day.
- Monitor and report to a physician any symptoms such as pain and fatigue upon movement, new numbness in legs or arms, loss of motor strength, increased weakness, loss of bowel or bladder control, increased pain on movement.

#### Patient Education—Food Safety

If enteral or parenteral nutrition is needed, sanitation and handwashing are essential.

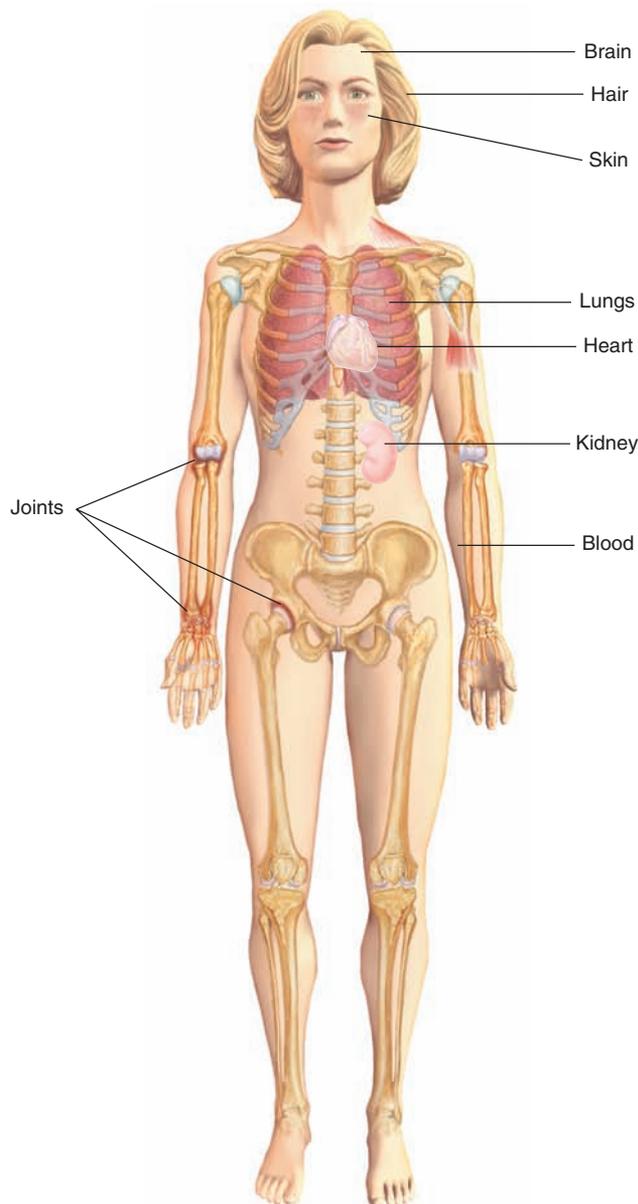
#### For More Information

- Family Care Research Program—Immobility and Movement <http://www.cancercare.msu.edu/patients-caregivers/symptoms/immobility.htm>
- Rehab Classworks <http://www.rehabclassworks.com/mobility.htm>

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**LUPUS****NUTRITIONAL ACUITY RANKING: LEVEL 2**

Asset provided by Anatomical Chart Co.

**DEFINITIONS AND BACKGROUND**

Lupus is an autoimmune disorder that involves areas of inflammation of the joints, tendons, other connective tissues, and skin. A pathologic CD4+T cell subset with impaired extracellular signal-regulated kinase (ERK) pathway signaling, DNA hypomethylation, and consequent aberrant gene expression contributes to disease pathogenesis (Gorelik and Richardson, 2010).

There are four types of lupus: neonatal, discoid, systemic, and drug induced. The systemic form (SLE) is the most common. One to two million people have lupus, especially Latino, African American, and Native American women, with onset in the late teens to thirties. For most people, lupus is a mild disease affecting only a few organs; for some, it may cause serious and even life-threatening problems. Because lupus has symptoms that mimic other disorders, careful diagnosis is important. Lupus may show symptoms similar to those of celiac disease.

Infections can bring on a lupus flare, increasing the risk of even more infections. Other environmental factors that may trigger the disease include antibiotics (especially sulfa and penicillin), other drugs, and exposure to phthalate in toys, plastics, and beauty products.

Active lupus contributes to coronary heart disease (CHD) risk (Haque et al, 2010). Premature cardiovascular disease in SLE patients is a consequence of inflammation. Type I interferons stimulate the cascade of atherosclerotic development, starting with endothelial damage and abnormal vascular repair (Von Feldt, 2008).

SLE is characterized by autoantibodies to nuclear antigens and immune complex deposition in organs such as the kidney (Gorelik and Richardson, 2010). Lupus nephritis is the term for this form of kidney disease that occurs. About a third of patients with lupus will develop it, requiring medical evaluation and nutritional management.

A cure for lupus is not yet possible, but treatments allow a more normal life. The use of methotrexate can reduce the dependency on steroids, which is desirable (Fortin et al, 2008). Antioxidant interventions have been studied extensively and show promise. Finally, supplementation with fish oil may reduce symptomatic disease activity.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Persons with close family members who have lupus have a 10 times greater frequency than the general population. Alleles in the TYK2 gene have been associated with SLE as well as multiple sclerosis.

<b>Clinical/History</b>	Achy joints (arthralgia)	Antibodies to double-stranded DNA
Height	Swollen and painful joints (nonerosive arthritis)	Serum copper (increased)
Weight	Protein or cellular casts in urine	Total protein (decreased)
BMI	Swollen ankles	WBC (decreased)
BP	Dry eyes	Gluc (increased)
I & O	Easy bruising	Low platelet count
Fever over 100°F		H & H, serum ferritin (decreased)
Seizures and cognitive dysfunction		Transferrin
Butterfly rash across cheeks and nose		Chol (increased)
Skin rashes, red raised patches	<b>Lab Work</b>	BUN, Creat
Photosensitivity	LE prep	Specific gravity, urine (decreased)
Painless mouth or nose ulcers	ESR or CRP (elevated?)	Alb, transthyretin
Pale or purple fingers from cold or stress (Raynaud's syndrome)	Complement protein test (C3, C4, CH50, CH100)	Transferrin
Unusual hair loss	INR, abnormal blood clotting	Ca <sup>++</sup> , Mg <sup>++</sup> , Na <sup>+</sup> , K <sup>+</sup>
Pleuritis or pericarditis	Positive anti-nuclear antibody test (ANA)	Vitamin D <sub>3</sub> status (serum 25-OHD)
Fatigue, prolonged		

## INTERVENTION



### OBJECTIVES

- Counteract steroid therapy; replenish potassium and nutrient reserves.
- Reduce fever and replace nutrient losses and weight loss.
- Control disease manifestations.
- Manage cardiac effects. Accelerated atherosclerosis and premature CHD are recognized complications (Haque et al, 2010). Pericarditis is also common, with shortness of breath and chest pain.
- Rule out gluten intolerance.

## SAMPLE NUTRITION CARE PROCESS STEPS

### Drug–Nutrient Interaction

**Assessment Data:** Weight and medical histories; medications; altered lab values for calcium, potassium. Complaints of swollen ankles and fluid retention.

**Nutrition Diagnosis (PES):** Drug–nutrient interaction related to prolonged use of corticosteroids for lupus as evidenced by osteopenia, low serum calcium and potassium, negative nitrogen balance and sodium–fluid retention.

**Intervention:** Food–nutrient delivery—Alter dietary intake to increase protein-rich foods, sources of potassium and calcium; decrease sodium intake. Education about the importance of managing specific nutrients while taking steroid medications (i.e., protein, calcium, potassium) and decreasing sodium-rich foods. Counseling about how to apply the DASH diet principles, which may be helpful.

**Monitoring and Evaluation:** Fewer complaints of swollen ankles and fluid retention; improved lab values related to calcium, potassium, and nitrogen balance studies.

- Prevent or manage infections, such as urinary tract infections, shingles, respiratory infections such as colds, yeast infections, salmonella and herpes.



## FOOD AND NUTRITION

- Diet should be adequate in protein and energy during fever.
- When renal disease is present, diet should be adjusted. Check lab values regularly.
- Alter diet, if needed, to lower blood pressure (BP) levels or excess weight. Mildly restrict sodium intake and monitor for potassium and phosphorus changes.
- Dietary nutrients may modify clinical course of disease. Vitamin C intake may prevent the occurrence of active disease; use a multivitamin–mineral supplement.
- Anemia is often present. Vitamin B<sub>12</sub>, dietary fiber, iron, calcium, and folate may be low in the diets of lupus patients. However, avoid excessive doses of supplements; use DRI levels.
- Use a nutrient-rich diet that includes nuts, fish and fish oils, olive oil, fruits, vegetables, and whole grains that are rich in phytochemicals, omega-3 fatty acids, and antioxidants. Include phytochemicals derived from spices (see Table 11-2).
- If gluten intolerance is present, provide a gluten-free nutrition plan.

### Common Drugs Used and Potential Side Effects

- Benlysta (belimumab), is a new drug developed specifically for people with systemic lupus. Many other drugs are in clinical trials.

- Steroid therapy may cause sodium retention, hyperglycemia, potassium and calcium depletion, and negative nitrogen balance. Side effects include weight gain, a round face, acne, easy bruising, fractures or osteoporosis, hypertension, cataracts, hyperglycemia or onset of diabetes, increased risk of infection, and stomach ulcers. Fish oil supplements may allow gradual reduction in use of steroids.
- Methotrexate (Rheumatrex) confers an advantage in participants with moderately active lupus by lowering daily prednisone dose and slightly decreasing lupus disease activity (Fortin et al 2008).
- Corticosteroid and cytotoxic drugs affect the immune system over time, making the individual prone to more infections. Immunosuppressive agents such as azathioprine (Imuran) and cyclophosphamide (Cytoxan) or methotrexate are used to control the overactive immune system but they have GI side effects.
- NSAIDs and acetaminophen may be useful.
- Sunscreens are needed to protect against the sun's harmful rays; there are no systemic side effects.
- Antimalarials, such as chloroquine (Aralen) or hydroxychloroquine (Plaquenil), may be used for skin and joint symptoms of lupus. Side effects are rare and consist of occasional diarrhea or rashes. Chloroquine can affect the eyes. Hydroxychloroquine may cause anorexia, nausea, abdominal cramps, and diarrhea.

## Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Coumestrol, a natural phytoestrogen, may relieve some symptoms.
- Use of indoles, conjugated linolenic acid (CLA), and vitamins C, E, and D may be beneficial.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Ensure patient has an adequate intake of fluids during febrile periods.
- Explain which foods are sources of sodium and potassium in the diet.

- Adequate rest is needed during flare-ups.
- Cortisone creams may be needed for persistent skin rashes. Sunblock should be used outdoors.
- Discuss how to manage diet for elevated blood glucose; insulin may be needed. Carbohydrate counting may be useful.
- Regular doctor visits and lab tests are important, especially blood and urine testing.
- Dietary strategies for the prevention of obesity, osteoporosis, and dyslipidemia deserve attention. Weight loss plans may be needed.

### Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

### For More Information

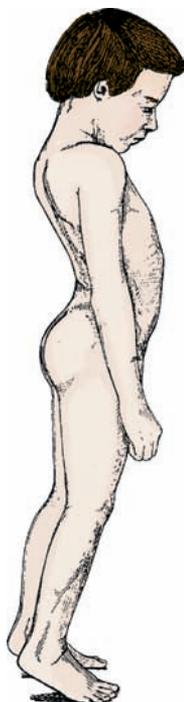
- Lupus Alliance of America  
<http://www.lupusalliance.org/>
- Lupus Canada  
<http://www.lupuscanada.org/>
- Lupus Foundation of America  
<http://www.lupus.org/>
- Lupus Library  
<http://www.lupusny.org/library.php>
- Lupus Organizations  
<http://www.lupusny.org/links.php#lupusorg>
- SLE Foundation, Inc.  
<http://www.lupusny.org/>

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# MUSCULAR DYSTROPHY

## NUTRITIONAL ACUITY RANKING: LEVEL 2



### DEFINITIONS AND BACKGROUND

Actually a group of nine disorders, muscular dystrophy (MD) involves a hereditary condition with progressive degenerative changes in the muscle fibers, leading to weakness and atrophy. Most of the disorders are described in Table 11-5.

Muscular biopsy is required for the definitive diagnosis of the specific congenital type. BMI should be used with caution for the evaluation of the nutritional status of patients with Duchenne MD (DMD); assessment of the compartmental distribution of muscle and fat are more sensitive. Extremely elevated serum creatine kinase (CK) levels may indicate muscle disease. In the late stages, fat and connective tissue may replace muscle fibers.

Patients with MD may be prone to nutrient deficiency due to mobility limitations or oropharyngeal weakness (Motlagh et al, 2005). Micronutrient requirements are yet to be determined but, as a result of corticosteroid treatment, vitamin D and calcium should be supplemented (Davidson and Truby, 2009).

Many patients demonstrate inadequate nutrient intake of protein, energy, vitamins (especially E), and minerals (calcium, selenium, and magnesium), and significant correlations exist between measures of strength and copper and water-soluble vitamins (Motlagh et al, 2005).

Delayed growth, short stature, muscle wasting, and increased fat mass are characteristics that impact on nutritional status and energy requirements (Davidson and Truby, 2009). There may be loss of muscle mass, wasting, which may be hard to see because some types of MD cause a build-up of fat and connective tissue that makes the muscle appear larger (pseudohypertrophy).

Gene therapy, gene silencing, and cell therapy are potential therapies for MD patients. Some evidence exists supporting supplementation with creatine monohydrate to improve muscle strength (Davidson and Truby, 2009). Creatinine as a marker of renal function has limited value in DMD because of reduced muscle mass. There is potential value of cystatin C as a biomarker for monitoring renal function (Violett et al, 2009).

The prognosis of MD varies according to type and progression. Some cases may be mild and very slowly progressive, with a normal lifespan. Other cases may have more marked progression of muscle weakness, functional disability, and loss of ambulation. Life expectancy often depends on the degree of progression and late respiratory deficit. In DMD, death often occurs in the late teens or early twenties. Rehabilitation, orthopedic, respiratory, cardiovascular, gastroenterology, nutrition, pain issues, as well as general surgical and emergency-room considerations are essential to address (Bushby et al, 2010).



### ASSESSMENT, MONITORING, AND EVALUATION



#### CLINICAL INDICATORS

**Genetic Markers:** Dystrophinopathies are due to a genetic defect of the protein dystrophin. Genetic counseling is advised when there is a family history of MD. Note that DMD can be detected by genetic studies performed during pregnancy.

<b>Clinical/History</b>	Eyelid drooping (ptosis)	(LDH), increased
Height	Dysphagia?	Cystatin C
Weight	Drooling	Myoglobin-urine/serum
BMI (use only with other parameters)	Chewing difficulty?	Creat (often decreased)
MAC, MAMC, TSF	Hand-to-mouth coordination	Aldolase, AST (altered)
Muscle weakness	Electromyography (EMG)	BUN
Apparent lack of coordination	Electrocardiography (ECG)	N balance
Progressive crippling	DEXA	Alb, transthyretin
Scoliosis?		CRP
Contractures of the muscles around the joints	<b>Lab Work</b>	H & H, serum ferritin
Clubfoot or clawhand?	Muscle biopsy	Serum P
Hypotonia	Creatine phosphokinase (CPK), increased?	Gluc
Loss of mobility	Lactate dehydrogenase	AST, ALT
		Ca <sup>++</sup> , Mg <sup>++</sup>
		Na <sup>+</sup> , K <sup>+</sup>
		Vitamin D <sub>3</sub> status (serum 25-OHD)

**TABLE 11-5 Types of Muscular Dystrophy and Nutritional Implications**

Type of MD	Comments	Nutritional Implications
Becker muscular dystrophy (BMD)	Very similar to DMD (see below), but onset is later (adolescence or adulthood). BMD patients live longer.	Weakness makes it difficult for self-feeding.
Congenital muscular dystrophy (CMD)	Caused by genetic mutations affecting some of the proteins necessary for muscles and some proteins related to the eyes and/or brain. Onset is at or near time of birth. Indicators include generalized muscle weakness with possible joint stiffness or looseness. Depending on the type, CMD may involve spinal curvature, respiratory insufficiency, mental retardation or learning disabilities, eye defects, and seizures.	Weakness makes it difficult for self-feeding.
Distal muscular dystrophy (DD)	DD is caused by a mutation in any of at least seven genes that affect proteins necessary to the function of muscles; it is usually passed on as an autosomal dominant or autosomal recessive trait. DD (Miyoshi form) first shows signs between ages 40 and 60 years, with weakness and muscle wasting of the hands, forearms, and lower legs; it progresses slowly.	Weakness and muscle wasting of the hands and forearms make self-feeding difficult.
Duchenne muscular dystrophy (DMD)	DMD primarily affects young boys, who inherit the disease through their mothers (X-linked recessive). Also called pseudohypertrophic. Caused by absence of dystrophin, a protein that helps keep muscle cells intact. DMD is the most severe form of dystrophinopathy. Aggressive forms appear in age 2–3, with frequent falls, difficulty in getting up from sitting or lying position. Generalized weakness and muscle wasting affect hip, thigh, shoulder, and trunk muscles first; large calf muscles. Weakness in lower leg muscles, resulting in difficulty running and jumping; waddling gait; mild mental retardation or scoliosis, in some cases. Survival is rare after the late twenties.	Facial muscles are involved; patient cannot suck, close lips, bite, chew, or swallow. DMD eventually affects all voluntary muscles, the heart, and lung muscles.
Emery-Dreyfus muscular dystrophy (EDMD)	EDMD is caused by gene mutations that produce emerin, lamin A, or lamin C, which are proteins in the membrane that surrounds the nucleus of each muscle cell. Onset in childhood, usually by age 10. Weakness and wasting of shoulder, upper arm, and shin muscles and joint deformities are common. Disease usually progresses slowly. Frequent cardiac complications are common, and a pacemaker may be needed.	Self-feeding becomes difficult.
Fascioscapulohumeral muscular dystrophy (FSHMD)	FSHMD (also called Landouzy-Dejerine) begins in childhood to early adulthood, with facial muscle weakness and weakness and wasting of the shoulders and upper arms. Caused by a missing piece of DNA on chromosome 4, it progresses slowly with some periods of rapid deterioration. Usually evident by age 20, it may span many decades. Inheritance is autosomal dominant, which means it can be passed on by either parent. It is considered to be the most common form.	Self-feeding becomes difficult; loss of skeletal muscle occurs. Abdominal muscles are affected.
Limb-girdle muscular dystrophy (LGMD)	LGMD is caused by a mutation in any of at least 15 different genes that affect proteins necessary for muscle function. LGMD has an onset in late childhood to middle age. Weakness and wasting affects shoulder and pelvic girdles first. Progression is slow, with cardiopulmonary complications often occurring in later stages of the disease. It is inherited as an autosomal recessive, autosomal dominant trait.	Self-feeding becomes difficult.
Myotonic dystrophy (MyD)	MyD (Steinert's disease) has onset anywhere from birth to middle age. Congenital myotonic form is more severe. Generalized weakness and muscle wasting affect the face, feet, hands, and neck first. Delayed relaxation of muscles after contraction. Progression is slow, sometimes spanning 50–60 years. Inheritance is autosomal dominant; there is a repeated section of DNA on either chromosome 19 or chromosome 3. Individuals with MyD have long faces and drooping eyelids; men have frontal baldness.	Progression is slow. Often complicated by diabetes. Prone to nutritional deficiencies from associated dysmotility of the entire GI. Handgrip is significantly lower; knee extension is higher compared to other dystrophies (Motlagh et al, 2005).
Oculopharyngeal muscular dystrophy (OPMD)	OPMD has onset in early adulthood to middle age. It affects muscles of eyelids (causing droopy eyelids) and throat. It progresses slowly, with swallowing problems common. Inheritance is autosomal dominant, and onset is usually in the fourth or fifth decade. The gene that is defective in OPMD is called the <i>poly(A) binding protein2</i> gene; extra amino acids in the protein made from the defective <i>PABP2</i> gene cause the protein to clump together in the muscle cell nuclei, interfering with cell function. OPMD can be diagnosed with a DNA test.	Swallowing difficulty is common. Tube feeding should be considered before wasting occurs.

Type of CMD	Cause	Inheritance Pattern
Merosin-deficient CMD	Lack of merosin (laminin 2) or other defect leading to merosin deficiency	Chromosome 6 gene Other genes
Ullrich CMD	Abnormalities in collagen 6	Chromosome 2 or 21 genes, recessive or dominant
Bethlem myopathy	Abnormalities in collagen 6	Chromosome 2 or 21 genes, dominant
Integrin-deficient CMD	Lack of integrin alpha 7	Chromosome 12 gene, recessive
Fukuyama CMD (FCMD)	Lack of fukutin	Chromosome 9 gene, recessive
Muscle-eye-brain disease (MEB)	Lack of POMGnT1, fukutin or fukutin-related protein	Chromosome 1, 9, or 19 genes, recessive
Walker–Warburg syndrome (WWS)	Lack of POMT1, POMT2, fukutin or fukutin related protein	Chromosome 9, 14, or 19 genes, recessive
CMD with rigid spine syndrome	Lack of selenoprotein N1	Chromosome 1 gene, recessive

## INTERVENTION



### OBJECTIVES

- Encourage patient to lead a relatively active life; exercise programs can help prevent contractures.
- Prevent obesity, from inactivity; obesity complicates physical therapy.
- Encourage activities other than eating to prevent dependency on food as a source of pleasure.
- Malnutrition is a serious threat, especially with respiratory muscle weakness. Monitor nutritional intake and deficits

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Abnormal GI Function—Constipation

**Assessment Data:** Weight and physical activity histories. Medical history, medications and lab values. Complaints of chronic constipation and GI discomfort. Diet hx showing low fiber and intake of <4 cups of fluid per day to avoid the need to urinate.

**Nutrition Diagnosis (PES):** Abnormal GI function related to constipation and physical inactivity as evidenced by infrequent evacuation, hard stools, and GI discomfort.

**Intervention:** Food and Nutrient Delivery—Increase fluid and fiber sources from tolerated foods such as whole grain cereals, fresh fruits, and vegetables. Educate about the desirable foods and fluid intake. Counsel about the importance of daily range of motion and physical exercises. Coordinate care with other disciplines according to needs, including PT, OT, nursing, medical team.

**Monitoring and Evaluation:** Alleviation of constipation. No further complaints of GI discomfort related to infrequent stooling pattern.

on a regular basis. Prevent aspiration pneumonia or nasal regurgitation. Use a multidisciplinary approach, especially for feeding difficulties such as texture modification and supplemental feeding (Davidson and Truby, 2009).

- Avoid constipation because fecal impaction is frequent.
- Prevent osteoporosis and fractures, which can occur in this population.
- Manage long-term consequences, such as cardiomyopathy or respiratory failure.



### FOOD AND NUTRITION

- Work with the MyPyramid food guidance system as a basic guide. Check patient's BMI and adjust intake accordingly. Use a low-energy diet if necessary to control or lessen obesity. Some patients' requirements may be 30% lower than normal (Munn et al, 2005).
- Use foods that are easy to chew and swallow for DMD, such as pureed or blenderized foods. Tube feed only if necessary.
- Provide adequate fiber (prune juice, bran and other whole grains, fruits, and vegetables) if constipation becomes a problem.
- Ensure adequate intake of fluid to prevent fecal impaction, dehydration, and related effects.
- Adequate sodium chloride is important (Yoshida et al, 2006). Manage carefully if there are cardiac side effects or problems with BP.

### Common Drugs Used and Potential Side Effects

- The myotonia (delayed relaxation of a muscle after a strong contraction) may be treated with medications

such as phenytoin or quinine. Side effects can include folic acid depletion.

- Early introduction of steroids can exacerbate weight gain in a population already susceptible to obesity (Davidson and Truby, 2009).
- It may be useful to try beta<sub>2</sub>-adrenergic agonists, which can increase muscle mass. Albuterol may be needed for some individuals prior to exercise and strength training.

## Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.

Approximately 50% of children are on herbal preparations, 30% of adolescents take herbal medications, and 70% of adults use some aspect of complementary medicine (Buehler, 2007).

- Green tea extract may improve muscle health by reducing or delaying necrosis by an antioxidant mechanism (Dorchies et al, 2006).
- Traditional Chinese medicine has been advocated for treatment of types of MD, but studies are needed to identify active ingredients.
- Supplementation with creatine monohydrate may improve muscle strength (Davidson and Truby, 2009).



## NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Provide low-calorie snack tips for patients who are obese.
- Help patient modify food textures to meet needs.
- Discuss problems related to inactivity or weight gain.
- Discuss the importance of adequate fluid intake.
- Discuss methods to prevent aspiration and pneumonia.
- Comprehensive management strategies can improve function, quality of life, and longevity (Bushby et al, 2010). Work with the occupational therapist and other therapists to maintain optimal levels of function.

## Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

### For More Information

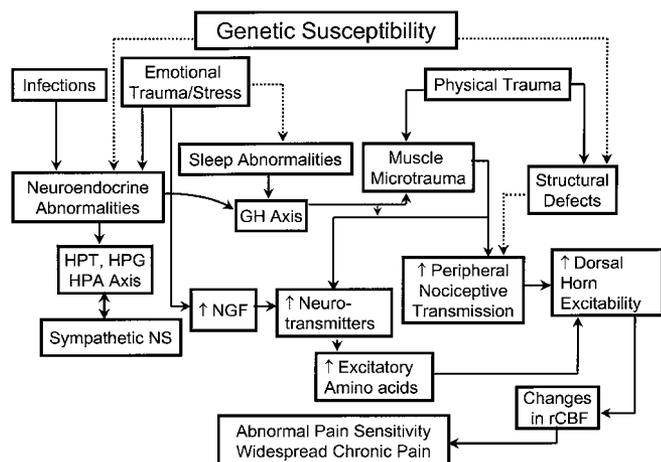
- Facioscapulohumeral Dystrophy (FSHD) Society  
<http://www.fshsociety.org>
- Muscular Dystrophy Association (MDA)  
<http://www.mdaua.org/>
- Muscular Dystrophy Association of Canada  
<http://www.mdac.ca/>
- Muscular Dystrophy Family Foundation  
<http://www.mdff.org/>
- National Institute—MD  
[http://www.ninds.nih.gov/health\\_and\\_medical/disorders/md.htm](http://www.ninds.nih.gov/health_and_medical/disorders/md.htm)
- Neuromuscular Disorders in the MDA Foundation  
<http://www.mdaua.org/disease/>
- Parent Project for Muscular Dystrophy Research  
<http://www.parentprojectmd.org>
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# MYOFASCIAL PAIN SYNDROMES: FIBROMYALGIA AND POLYMYALGIA RHEUMATICA

## NUTRITIONAL ACUITY RANKING: LEVEL 1–2



Adapted from: William J. Koopman, Larry W. Moreland, *Arthritis and Allied Conditions A Textbook of Rheumatology*, 15th ed. Philadelphia: Lippincott Williams & Wilkins, 2005.



## DEFINITIONS AND BACKGROUND

**Myofascial pain syndromes** are a group of disorders characterized by achy pain and stiffness in soft tissues, including muscles, tendons, and ligaments. In the United States, **fibromyalgia (FM)** is estimated to occur in 2% of adults (Mease, 2005). Diagnosis is difficult, and the etiology is not clear. Corticotropin-releasing hormone (CRH) and substance P (SP) are found in increased levels in the cerebral spinal fluid (CSF) of FM patients, and increased interleukin (IL)-6 and IL-8 are found in the serum where they release proinflammatory and neurosensitizing molecules (Lucas et al, 2006).

FM, or “fibrositis,” is a central sensitivity syndrome with abnormalities in the peripheral, central, sympathetic nervous systems, and the hypothalamo–pituitary–adrenal axis stress response system (Mease, 2005). Etiology theories abound, including inadequate sleep, physical or psychological trauma, or exposure to viruses such as hepatitis B or C, or HIV infection. Serotonin and dopamine levels may be lower than normal. Insulin-like growth factor-1 (IGF-1) levels may also be low; they are a surrogate marker for low growth hormone secretion during stage 3 and 4 of sleep, when tissue repair occurs (Rosenzweig and Thomas, 2009).

FM causes widespread pain and stiffness either throughout the body or localized along the spine. Persistent symptoms may be disruptive but are not life threatening. Symptoms include sleep disturbance, depression, fatigue, headaches, irritable bowel syndrome, numbness in the hands and feet, and mood disorders. Acupuncture may offer relief (Martin et al, 2006).

**Polymyalgia rheumatica (PMR)** affects people over age 70 years, usually women. It causes aching, severe muscle stiffness, and pain. Symptoms start suddenly and may affect several areas in the neck, shoulders, hips, and/or thighs. It

usually goes away with treatment but may reoccur. Symptoms include mild joint stiffness and swelling, face pain, anemia, extreme fatigue, unintentional weight loss, and anorexia. The cause of PMR is not known but may be related to aging. Diagnosis is difficult. Many people with PMR also have giant-cell arteritis with double vision, severe headaches, or vision loss. Low-dose corticosteroids may be needed for up to a year (Hernandez-Rodriguez et al, 2009).

Treatment of myofascial pain disorders may include exercise, medications such as glucocorticoids and NSAIDs, a healthy diet rich in antioxidants, and adequate rest. Massage and cognitive behavioral therapy (CBT) are helpful (Mease, 2005). After a warm-water swimming program, a significant decrease in IL-8, IFN $\gamma$ , and CRP has been noted (Ortega et al, 2009). Use of a phytochemical-rich diet results in a decrease in joint stiffness and pain as well as an improvement in self-reported quality of life. Plant foods are rich natural sources of antioxidants (quercetin, myristin, and kaempferol) in addition to fiber and other nutrients. A vegan diet often shows highly increased serum levels of beta- and alpha-carotenes, lycopene, lutein, and vitamins C and E.

Rapid-paced discovery is taking place in genetics, patient assessment, new therapeutic targets, and novel methods of treatment delivery (Williams and Clauw, 2009). The best multidisciplinary team includes a rheumatologist, physical therapist, exercise therapist, dietitian, and massage therapist (Lemstra and Olszynski, 2005).



## ASSESSMENT, MONITORING, AND EVALUATION



## CLINICAL INDICATORS

**Genetic Markers:** A multigenic or genome-wide approach may be needed to alter individualized pain therapy according to the patient’s genotype. For example, DRD2 polymorphisms decrease the functioning of the dopaminergic reward system; this could cause an individual to require more pain medicine. Research is ongoing to determine whether individuals with FM have the genetic tendency toward lower pain thresholds.

Clinical/History	Morning	FM-pain in
Height	stiffness	shoulders,
Weight	Fatigue, sleep	pelvis, and
BMI	disturbances	hips (pain in
Tender areas,	Fibromyalgia	11/18 trigger
back pain	Impact	points)
Headache	Questionnaire	Carpal tunnel
Depression,	(FIQ)	syndrome
mood		(in PMR)
disorders		

<b>Lab Work</b>	Trig, Chol Alb, transthyretin	Alk phos Vitamin D <sub>3</sub> status (serum 25-OHD)
CRP or ESR (may be high)	BUN, Creat	
Plasma adreno- medullin (high in PMR)	Ca <sup>++</sup> , Mg <sup>++</sup> Na <sup>+</sup> , K <sup>+</sup> Gluc	

## INTERVENTION



### OBJECTIVES

- Relieve pain. Acupuncture, massage, cognitive-behavior therapy (CBT) and varied exercises may be recommended (Assefi et al, 2005; Rosenzweig and Thomas, 2009).
- Lose weight, if obese.
- Correct underlying problems such as hypertension.
- Support lifestyle changes, including stress reduction, relaxation techniques, and exercise.
- Prevent blindness in PMR when there is giant-cell arteritis.



### FOOD AND NUTRITION

- Use a balanced diet. The MyPyramid food guidance system is another useful tool for planning a healthy diet. Include phytochemicals; dietary quercetin should be encouraged (Lucas et al, 2006). Table 11-2 is also a useful reference.
- A vegan diet may be beneficial with berries, fruits, vegetables, roots, nuts, germinated seeds, and sprouts.
- A weight loss plan may be needed.

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Inadequate Intake of Bioactive Substances

**Assessment Data:** Weight and physical activity histories. Medical history, medications and lab values. Much pain; diagnosed FM. Diet hx and 3-day food record shows intake of <2 fruits and vegetables daily. No food allergies.

**Nutrition Diagnosis (PES):** Inadequate intake of bioactive substances related to low intake of fruits and vegetables as evidenced by diet history and intake records.

**Intervention:** Food and Nutrient Delivery—Provision of more spices, fruits, vegetables and juices. Education about the role of antioxidants, spices, and phytochemicals in reducing inflammation and the possibility of lessening pain symptoms. Counseling about menus, recipes and cooking tips for including more bioactive ingredients. Encourage intake of fish oils, walnuts, fatty fish such as salmon for omega-3 fatty acids.

**Monitoring and Evaluation:** Diet Hx showing improved intake of spices (such as turmeric, cumin, cinnamon), cocoa and coffee, fruits including berries and apples and pomegranates, vegetables such as broccoli and cabbage on a daily and weekly basis. Fewer complaints of overt pain.

- Increased intake of omega-3 fatty acids may help to reduce inflammation and relieve pain in some individuals. Increase intake of fatty fish, walnuts, and flaxseed.

## Common Drugs Used and Potential Side Effects

- Medications that decrease pain and improve sleep may be prescribed. Low doses of tricyclic antidepressants (amitriptyline, Elavil; cyclobenzaprine, Flexeril) and the serotonin-3 receptor antagonist tropisetron may be helpful (Lucas et al, 2006; Rosenzweig and Thomas, 2009). Opioids, NSAIDs, sedatives, muscle relaxants, and antiepileptics have been used to treat FMS (Mease, 2005).
- Pregabalin (Lyrica) and duloxetine (Cymbalta) are used in FMS. Milnacipran (Savella) is a dual norepinephrine and serotonin reuptake inhibitor that has been shown to be safe (Arnold et al, 2009; Mease et al, 2009).
- Opioids are not recommended for FM (Rosenzweig and Thomas, 2009).
- For PMR, a trial of low-dose corticosteroids is given, usually in the form of 10–15 mg of prednisone (Deltasone, Orasone) per day. Side effects may include sleeplessness, weight gain, loss of nitrogen and calcium, cataracts, thinning of the skin, easy bruising. NSAIDs, such as ibuprofen (Advil, Motrin) and naproxen (Naprosyn, Aleve), are ineffective in the treatment of PMR.

## Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician. CAM is popular for musculoskeletal conditions. Some CAM modalities show significant promise, such as acupuncture (Martin et al, 2006). Excellent resources are available on the Internet from the National Center for Complementary and Alternative Medicine (<http://nccam.nih.gov>).
- Magnesium; sulfur compounds such as SAME, dimethylsulfoxide (DMSO), taurine, glucosamine, and chondroitin sulfate; and reduced GSH may have clinical applications in the treatment of FMS; controlled trials are needed.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Aerobic exercise, patient education and CBT are quite effective. Daily exercise will be important for strengthening weak muscles. Exercise adherence can help reduce the need for pain medications (Lemstra and Olszynski, 2005).
- Discuss weight management, as needed.
- Discuss the role of omega-3 fatty acids in reduction of inflammation.

#### Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

**For More Information**

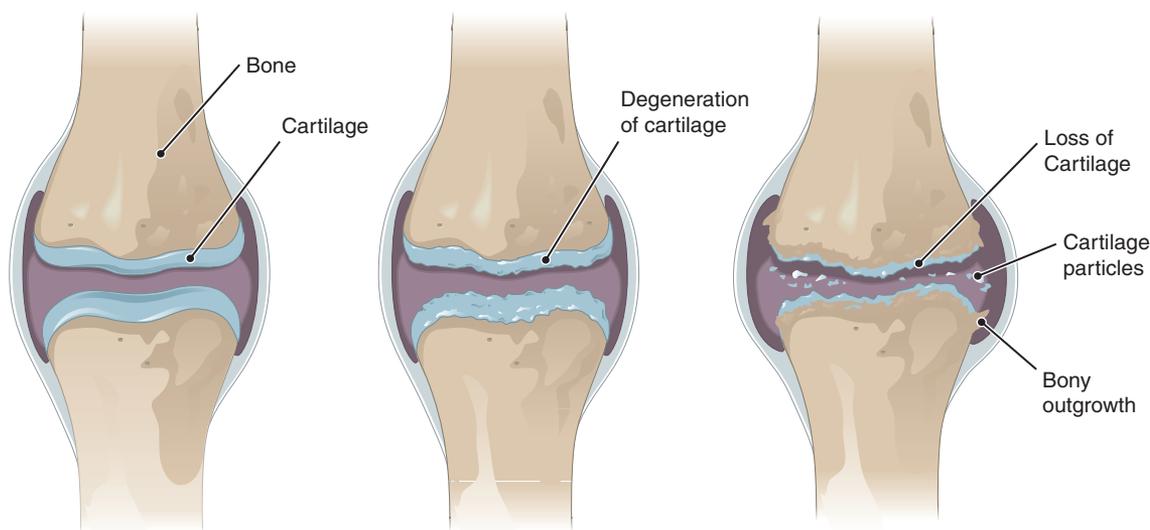
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<http://www.afsafund.org/>
- Fibromyalgia Network  
<http://www.fmnetnews.com/>
- Mayo Clinic  
<http://www.mayoclinic.com/health/myofascial-pain-syndrome/DS01042>
- Myositis Association  
<http://www.myositis.org/>
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- National Institute of Arthritis and Musculoskeletal and Skin Diseases  
<http://www.niams.nih.gov/hi/topics/fibromyalgia/fibrofs.htm>
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[http://www.rheumatology.org/public/factsheets/diseases\\_and\\_conditions/polymyalgiarheumatica.asp](http://www.rheumatology.org/public/factsheets/diseases_and_conditions/polymyalgiarheumatica.asp)
- Polymyalgia Rheumatica and Giant Cell Arteritis  
[http://www.niams.nih.gov/Health\\_Info/Polymyalgia/default.asp](http://www.niams.nih.gov/Health_Info/Polymyalgia/default.asp)

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# OSTEOARTHRITIS AND DEGENERATIVE JOINT DISEASE

**NUTRITIONAL ACUITY RANKING: LEVEL 1–2**

Adapted from: Cohen BJ. *Medical Terminology*, 4th ed. Philadelphia. Lippincott Williams & Wilkins 2003.

**DEFINITIONS AND BACKGROUND**

OA may be primary (in older individuals) or may follow an injury or disease involving the articular surfaces of synovial joints. The joint may lose its normal shape and bone spurs may grow on the edges of the joint. OA is a common health problem in populations over age 40 years, and it is a leading cause of pain and disability. OA mostly affects cartilage where the surface layer breaks down and wears away. The hands, knees, hips, and spine are most commonly affected.

Over 40 million Americans report that they have arthritis and many indicate that it limits their daily activities. High serum concentrations of tumor necrosis factor are associated with lower physical function and more pain, stiffness, and physical disability. Over a third of adults with arthritis experience limitations in their ability to work.

Treatments for OA combine nonpharmacological modalities, pharmacological agents, and surgical procedures. Exercise, weight control, rest, and relief from stress on joints, nondrug pain relief, and various types of complementary

medical techniques may be useful. Continuous passive motion (CPM), massage, and heat treatments may be used. Surgery is reserved for those persons for whom other treatments have been unsuccessful.

Weight loss is a primary treatment for OA for individuals who are obese. Obesity is a significant risk factor for and contributor to increased morbidity and mortality from chronic diseases, including OA (Pi-Sunyer, 2009). Overweight causes strain on joints and should be managed early by health professionals (Gasbarrini and Piscaglia, 2005). An average weight loss of 5% in overweight and obese older patients brings an 18% gain in overall function (Messier et al, 2005), and a 10% weight loss improves function by 28% (Christensen et al, 2005).

While vitamins A, C, and E have major roles in modulating oxidative stress, immune responses, and cell differentiation, controlled trials found that these vitamins do not halt progression of OA (Choi, 2005). Vitamins D and K play a protective role (Bergink et al, 2009; Oka et al, 2009). Diets rich in omega-3 fatty acids may reduce joint stiffness and pain, increase grip strength, and enhance walking pace. Pomegranate fruit extracts can block interleukin-1 $\beta$  (IL-1 $\beta$ ) enzymes that contribute to cartilage destruction and OA. Finally, cadgerin-11 is a protein that contributes to joint destruction and a related fabric has been developed for cartilage replacement.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** OA is the breakdown and inflammation of joint cartilage, usually brought on by aging and repetitive joint usage. OA and cardiovascular disease share age and obesity as risk factors, but may also be linked by pathogenic mechanisms involving metabolic abnormalities and systemic inflammation (Puenpatom and Victor, 2009).

<b>Clinical/History</b>	X-rays; DEXA OA Index	Gluc Alk phos Uric acid
Height		
Weight		
BMI		
Obesity?	Antistreptolysin titer (ASO)	Ca <sup>++</sup> , Mg <sup>++</sup> Na <sup>+</sup> , K <sup>+</sup>
Pain, swelling of joints (arthralgia)	Antirheumatoid factor	Serum folate and B <sub>12</sub> Vitamin D <sub>3</sub> status (serum 25-OHD)
Synovial joint stiffness	BUN, Creat Sedimentation rate	
Crunching sound of bone against bone	CRP	

## SAMPLE NUTRITION CARE PROCESS STEPS

### Overweight

**Assessment Data:** Weight and physical activity histories; symptoms diary; medical history, medications, and lab values.

**Nutrition Diagnosis (PES):** Overweight with joint pain related to eating double portions at lunch and dinner meals as evidenced by diet history and BMI of 29.

**Intervention:** Food and Nutrient Delivery—Offer choices of lower fat and low energy foods and snacks. Educate about the role of excess weight and joint pain. Counsel and offer weight management tips, tips for dining out or snacking. Coordinate care with referral to physical therapy for exercises to reduce pain and stiffness in affected joints.

**Monitoring and Evaluation:** Weight changes, food intake records, symptoms diary.

## INTERVENTION



### OBJECTIVES

- Control pain and improve joint function. If joint replacement is necessary, prepare for surgery accordingly.
- Maintain a normal body weight. If needed, weight loss may be beneficial to lessen pressure on weight-bearing joints.
- Maintain an active lifestyle as much as possible.
- Encourage patient (especially if older) to consume adequate amounts of vitamins D and K, protein and calcium from a healthy, nutrient-dense, antioxidant-rich diet.
- Maintain integrity of cartilage in affected joints. Omega-3 fatty acids may reduce the activity of enzymes that destroy cartilage. Include fish oils and certain plant seed oils that impact immune and inflammatory responses as precursors of eicosanoids.



### FOOD AND NUTRITION

- Use a calorie-controlled diet if obesity is present. Use of a meal replacement may help to promote weight loss.
- The inflammatory response may be suppressed by an increase in omega-3 fatty acids, as found in fatty fish (mackerel, herring, and salmon) and from walnuts and flaxseed. Use these foods several times a week.
- Calcium is found in dark green, leafy vegetables, such as kale and broccoli; canned sardines and salmon with bones; fortified orange juice; milk and dairy products, such as cheese and yogurt; fortified bread, tofu or soy milk.
- Vitamin C is needed for healthy collagen and cartilage. Good sources include citrus fruits, bell peppers, tomatoes, watermelon, strawberries, and kiwifruit.
- Low dietary vitamin D intake increases the risk of progression of knee OA, particularly in subjects with low baseline BMD (Bergink et al, 2009). Include vitamin D from sardines, herring, fish-liver oil; butter and cream; egg yolks; liver; fortified cow's milk and dairy

products, such as cheese and yogurt; and fortified cereals.

- Vitamin K may be found in leafy greens such as kale, Swiss chard, broccoli, spinach, raw parsley. It is also found in small amounts in olive, soybean or canola oils and in mayonnaise.
- Boron may help OA. Sources include apples, legumes, leafy vegetables, carrots, pears, grapes, and some drinking water.
- Include plenty of phytochemicals. Pomegranates and cranberries are especially protective because of the ellagic acid. See Table 11-2.

## Common Drugs Used and Potential Side Effects

- Because of GI risks (including ulcer complications) and cardiovascular risks, including hypertension and thrombotic events associated with NSAIDs, acetaminophen is the first choice anti-inflammatory agent (Berenbaum, 2008). Table 11-6 gives more details on medicines used for OA. Evaluate risks as well as benefits for all drug therapies.

**TABLE 11-6 Medications Commonly Used for Osteoarthritis**

Medication	Comments	Side Effects
Anti-inflammatory drugs, nonsteroidal (NSAIDs)	Indomethacin (Indocin), aspirin piroxicam (Feldene), naproxen (Naprosyn), nabumetone (Relafen) and Ibuprofen (Advil, Motrin) may be recommended.	Nausea, GI distress, anorexia, flatulence, or vomiting can occur. Take with food. Prolonged use may cause GI bleeding or ulcers. Indomethacin may also cause renal failure, or diarrhea; naproxen may cause heartburn or increased risk of cardiovascular disease.
COX-2 Inhibitors	Cyclooxygenases are needed for the synthesis of prostaglandins. The COX-2 enzyme mediates inflammation and pain. Celecoxib (Celebrex) is the only FDA-approved drug at this time.	These agents may promote increased risk of heart attack and stroke. Rofecoxib (Vioxx) and valdecoxib (Bectra) were removed from the market in 2004 and 2005.
Misoprostol (Cytotec)	Misoprostol reduces stomach acid if NSAIDs are used.	Abdominal cramps may occur.
Frankincense (Boswellia frereana)	Interestingly, this herb may lessen arthritic pain. Epi-lupeol is the principal constituent of B. frereana. B. frereana prevents collagen degradation, and inhibits the production of pro-inflammatory mediators (Blain et al, 2010).	Fewer side effects than glucosamine and chondroitin.
Glucosamine sulfate and chondroitin	Glucosamine reduces cartilage damage and decreases pain associated with osteoarthritis. Taken with chondroitin, it may help relieve symptoms of osteoarthritis. Some pills do not contain sufficient levels to be effective; check brand with <a href="http://www.consumerlab.com">www.consumerlab.com</a> to select the best choice.	Glucosamine can increase blood glucose levels and aggravate shellfish allergy because it is made from these shells. Chondroitin may alter blood clotting activity in a manner similar to that of aspirin.
Hyaluronic acid substitutes (viscosupplements)	These injections are designed to replace a normal component of the knee joint involved in joint lubrication and nutrition.	A series of injections are required. When used with methotrexate, the benefits may be greater (Homma et al, 2009).
Omega-3 fatty acids	Supplementation causes a decrease in both degradative and inflammatory aspects of chondrocyte metabolism.	May increase effects of blood-thinning drugs and herbs.
Steroids	Corticosteroids may cause sodium retention; calcium, nitrogen, and potassium depletion; truncal obesity; and hyperglycemia.	Corticosteroids may have a short-term effect in osteoarthritis (Bellamy et al, 2005). Injections are required.
Topical pain relievers (Zostrix, Icy Hot, Therapeutic Mineral Ice, Aspercreme, and Ben Gay)	Creams and rubs stimulate nerve endings to relieve pain; deplete the amount of neurotransmitter (substance P) that sends pain messages to the brain; and block prostaglandins that cause pain and inflammation.	No internal side effects.
Tramadol (Ultram)	Pain reliever that is prescribed when over-the-counter medications do not provide relief.	Potentially addictive.

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- National Institute of Arthritis and Musculoskeletal and Skin Disorders. Web site accessed December 13, 2009, at [http://www.niams.nih.gov/Health\\_Info/Osteoarthritis/default.asp#4](http://www.niams.nih.gov/Health_Info/Osteoarthritis/default.asp#4)

**TABLE 11-7 Side Effects of Herbs Commonly Used for Arthritis**

<b>Bromelain</b>	May increase effects of blood-thinning drugs and tetracycline antibiotics.
<b>Echinacea</b>	Might counteract immunosuppressant drugs, such as glucocorticoids, taken for lupus and rheumatoid arthritis; might increase side effects of methotrexate.
<b>Evening primrose oil</b>	Can counteract the effects of anticonvulsant drugs.
<b>Folic acid</b>	Interferes with methotrexate.
<b>Gamma linoleic acid (GLA)</b>	May increase effects of blood-thinning drugs and herbs.
<b>Garlic</b>	Can increase effects of blood-thinning drugs and herbs
<b>Ginger</b>	Can increase nonsteroidal anti-inflammatory drug (NSAID) side effects and effects of blood-thinning drugs and herbs.
<b>Ginkgo</b>	May increase effects of blood-thinning drugs and herbs.
<b>Ginseng</b>	May increase effects of blood-thinning drugs, estrogens, and glucocorticoids; should not be used by those with diabetes; may interact with monoamine oxidase (MAO) inhibitors.
<b>Kava</b>	Can increase effects of alcohol, sedatives, and tranquilizers.
<b>Magnesium</b>	May interact with blood pressure medications.
<b>S-adenosylmethionine (SAMe)</b>	SAMe may rebuild eroded joint cartilage. Enteric coating is needed because of gastrointestinal (GI) side effects.
<b>Soy and avocado extracts</b>	Antioxidant effects in reducing the symptoms of osteoarthritis; avoid excessive use in patients with hormonal cancers.
<b>St. John's wort</b>	May enhance effects of narcotics, alcohol, and antidepressants; increases risk of sunburn; interferes with iron absorption.
<b>Valerian</b>	May enhance effects of sedatives and tranquilizers.
<b>Vitamin E</b>	Gamma-tocopherol may worsen osteoarthritis; alpha-tocopherol is better.
<b>Zinc</b>	Can interfere with glucocorticoids and other immunosuppressive drugs.

Note: Herbs and botanical supplements should not be used without discussing with physician. Excerpted from The Arthritis Foundation's guide to alternative therapies, Web site accessed December 6, 2009, at <http://www.arthritis.org>.

## Herbs, Botanicals, and Supplements

- *Boswellia frereana* (frankincense) suppresses cytokine-induced matrix metalloproteinase expression and production of pro-inflammatory molecules; studies are on-going (Blain et al, 2010).
- Glucosamine sulfate combined with omega-3 fatty acids may reduce OA symptoms, including morning stiffness and pain in hips and knees (Gruenwald et al, 2009). Undenatured type II collagen (UC-II) may be more effective in the treatment of OA than glucosamine and chondroitin (Crowley et al, 2009).
- Green tea's anti-inflammatory properties and ginger may aid in pain relief.
- Table 11-7 provides a description of some side effects of products often used for OA.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Physical and occupational therapies, diet, and exercise play an extremely important role. Long-term exercise and dietary weight loss are most effective. Acupuncture may be used to relieve pain.
- Because OA allows muscles around the joints to become weak, exercise and stretching should be suggested to maintain flexibility. Repetitive, high-impact movements are not recommended whereas Tai Chi helps balance and protects bones. Exercises include a series for strengthening, aerobic, agility, and range of motion. Long-term

weight training, walking programs, swimming, and flexibility exercises are helpful.

- Encourage patient to avoid fad diets for "arthritis cure." Ensure that the patient's diet is balanced and includes all nutrients. A weight-loss plan may be needed.
- To alleviate stress on the joints, pharmacological and behavioral techniques with self-monitoring, should be included (Berkel et al, 2005).
- Pain initiates and exacerbates sleep disturbance; therefore, improving the sleep of OA patients helps to reduce the pain (Vitello et al, 2009). Transcutaneous electrical nerve stimulation (TENS) directs mild electric pulses to nerve endings that lie beneath the skin in the painful area; it block messages to the brain and by modifies pain perception. CBT is also useful.
- Focus on abilities and strengths rather than on disabilities and weaknesses.

### Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

### For More Information

- Arthritis Foundation  
<http://arthritis.about.com/>
- Arthritis Resource Center at Healingwell  
<http://www.healingwell.com/arthritis>
- NIAMS—Osteoarthritis  
<http://www.niams.nih.gov/hi/topics/arthritis/oahandout.htm>
- Johns Hopkins Arthritis Center  
<http://www.hopkins-arthritis.som.jhmi.edu/>

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## OSTEOMYELITIS

### NUTRITIONAL ACUITY RANKING: LEVEL 1–2



#### DEFINITIONS AND BACKGROUND

Acute osteomyelitis may be caused by localized infection of the long bones or injury to bone and surrounding soft tissue. *Staphylococcus aureus* is implicated in most patients with acute osteomyelitis; *S. epidermidis*, *S. aureus*, *Pseudomonas aeruginosa*, *Serratia marcescens*, and *Escherichia coli* may be found in the chronic form. When a bone is infected, the bone marrow swells and compresses against the rigid outer wall of bone, and blood vessels may be compressed or die; abscesses may form. Osteomyelitis and inflammatory arthritis affect many children (Pruthi and Thapa, 2009).

Some diseases predispose patients to osteomyelitis, including diabetes mellitus, sickle cell disease, acquired immunodeficiency virus (AIDS), intravenous drug abuse, alcoholism, chronic steroid use, immunosuppression, and chronic joint disease. Use of prosthetic orthopedic devices and recent orthopedic surgery or open fracture may also place a patient at risk for osteomyelitis. Patients with diabetes mellitus with poor glucose control may experience infections of the lower extremities, from superficial cellulitis to deep soft tissue infections and osteomyelitis. Because osteomyelitis is prevalent after diabetic foot ulcers, careful treatment is crucial to avoid amputation (Schinabeck and Johnson, 2005).

Prompt treatment is important. If not treated properly, the condition may become chronic with a poor prognosis. Treatment generally involves evaluation, staging, determination of etiology, antimicrobial therapy, and debridement or stabilization of bone. In children, serious musculoskeletal infections include osteomyelitis, septic arthritis, pyomyositis, and necrotizing fasciitis (Frank et al, 2005).



#### ASSESSMENT, MONITORING, AND EVALUATION



#### CLINICAL INDICATORS

**Genetic Markers:** Deficiency of the interleukin-1-receptor antagonist (DIRA) promotes neonatal osteomyelitis, an autosomal recessive autoinflammatory disease caused by mutations in IL1RN (Aksentijevich et al, 2009).

<b>Clinical/History</b>	Local swelling, redness	White blood cell (WBC) count (increased)
Height	Contractures in affected extremities	Gluc
Weight	Magnetic resonance imaging (MRI)	Alb,
BMI	Bone densitometry or x-rays	transthyretin
Intake and output (I & O)	Pressure ulcers or lesions with a sinus tract?	BUN, Creat
BP		Alk phos
Bone pain		AST, ALT
Sudden, acute pain in joints near the infection		Ca <sup>++</sup> , Mg <sup>++</sup>
Fever, chills		Na <sup>+</sup> , K <sup>+</sup>
Tachycardia, diaphoresis		Vitamin D <sub>3</sub> status (serum 25-OHD)

#### Lab Work

CRP and ESR (increased)

**SAMPLE NUTRITION CARE PROCESS STEPS****Underweight**

**Assessment Data:** Weight and physical activity histories. Medical history, medications, and lab values.

**Nutrition Diagnosis (PES):** Underweight related to poor nutrition quality of life and inadequate oral intake as evidenced by insufficient pain medicine before meals and BMI of 18.

**Intervention:** Food and nutrient delivery—offer nutrient and energy-dense favorite foods. Educate about the benefits of gaining weight to tolerate medical therapies more effectively, to gain energy, and to improve nutritional quality of life. Coordinate timing of medicines with meals to assure that pain medicine is given 20–30 minutes before meals.

**Monitoring and Evaluation:** Weight gain in 3–6 months; improved timing of meals with pain medicine to decrease anorexia at mealtime.

**INTERVENTION****OBJECTIVES**

- Characterize and treat the infection. Prevent further infection, dehydration, and other complications.
- Promote recovery and healing of any skin lesions or pressure ulcers.
- Correct defective blood flow to allow nutrients and oxygen to reach all tissues.
- Control serum glucose and alleviate hyperglycemia with insulin if needed.
- Correct anorexia, poor intake, weight loss, nausea and vomiting where present.

**FOOD AND NUTRITION**

- Encourage adequate fluid intake.
- Maintain a normal to high intake of calories, protein, zinc, vitamin A, and vitamin C in particular. A multivitamin–mineral supplement may be needed.
- With diabetes, control carbohydrate to promote more effective healing.

**Common Drugs Used and Potential Side Effects**

- For optimal results, antibiotic therapy must be started early, with antimicrobial agents administered parenterally

for at least 4–6 weeks if needed. Vancomycin or amphotericin B may be used; monitor for side effects related to timing and meals.

- Analgesics may be used for pain. GI distress is a common side effect.

**Herbs, Botanicals, and Supplements**

- Herbs and botanical supplements should not be used without discussing with physician.
- It is reasonable to include phytochemical-rich foods each day. See Table 11-2.

**NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT**

- Discuss role of nutrition in wound healing, immunity, and other conditions related to this disorder.
- Discuss signs that may indicate reversal of status or recovery, such as increased fever, elevated glucose levels, additional infections, more redness in affected areas.
- Promote use of nutrient-dense foods that are rich in antioxidants, phytochemicals, protein, zinc and vitamins.

**Patient Education—Food Safety**

- If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used. Infection control is extremely important to avoid additional microbial contamination.

**For More Information**

- Cleveland Clinic—Osteomyelitis  
[http://my.clevelandclinic.org/disorders/osteomyelitis/hic\\_osteomyelitis.aspx](http://my.clevelandclinic.org/disorders/osteomyelitis/hic_osteomyelitis.aspx)
- Mayo Clinic—Osteomyelitis  
<http://www.mayoclinic.com/health/osteomyelitis/DS00759>
- National Institutes of Health—Osteomyelitis  
<http://www.nlm.nih.gov/medlineplus/ency/article/000437.htm>

**OSTEOMYELITIS—CITED REFERENCES**

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- Frank G, et al. Musculoskeletal infections in children. *Pediatr Clin North Am.* 52:1083, 2005.
- Pruthi S, Thapa MM. Infectious and inflammatory disorders. *Radiol Clin North Am.* 47:911, 2009.
- Schinabeck MK, Johnson JL. Osteomyelitis in diabetic foot ulcers. Prompt diagnosis can avert amputation. *Postgrad Med.* 118:11, 2005.

# OSTEOMALACIA

## NUTRITIONAL ACUITY RANKING: LEVEL 2



Adapted from: Yochum TR and Rowe LJ. *Yochum and Rowe's Essentials of Skeletal Radiology*, 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2004.



### DEFINITIONS AND BACKGROUND

**Osteomalacia**, adult rickets, causes softening and demineralization of the bone from insufficient vitamin D. Osteomalacia may occur in conjunction with bone loss and hip fractures. It more commonly results from intestinal malabsorption as from Crohn's disease, colon resection, cystic fibrosis, celiac disease, or chronic use of anticonvulsants. It is also seen in kidney failure, liver disease, and some types of cancer. Severe vitamin D deficiency leads to secondary hyperparathyroidism, increased bone turnover and losses.

Derangements in serum phosphate level result in osteomalacia (Saito and Fukumoto, 2009). Matrix extracellular phosphoglycoprotein (MEPE) inhibits mineralization; altered expression is associated with oncogenic osteomalacia and hypophosphatemic rickets (Boskey et al, 2010). In addition, deficient actions of fibroblast growth factor, FGF23, result in hypophosphatemic osteomalacia; FGF23 works as a hormone (Saito and Fukumoto, 2009).

Osteomalacia often occurs in older people, in dark-skinned individuals who live in northern latitudes, and in those who have limited sunlight exposure. Vitamin D is produced in response to sun exposure, so the process works faster in pale individuals. Sun exposure of about 15 minutes without sunscreen a few times a week is needed. Darker skinned individuals may need to take supplements.



### ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** The vitamin D receptor (VDR) is responsible for the expression of over 900 genes, or about 3% of the human genome.

Clinical/History	Lab Work	
Height	Serum P	PTH
Weight	(decreased)	Mg <sup>++</sup>
BMI	Serum Ca <sup>++</sup>	Na <sup>+</sup> , K <sup>+</sup>
Bone pain, aches	(decreased)	Serum phos
Softened, deformed bones	Urinary Ca <sup>++</sup>	Alk phos (increased)
Muscular weakness, listlessness	Vitamin D <sub>3</sub> (serum 25-OHD): normal >30 ng/mL;	Alb, transthyretin
Numbness of arms, feet	low between 15 and 30 ng/mL;	CRP
Easy bone fractures	very low at less than 15 ng/mL	BUN, Creat
Bone densitometry, DEXA		
Bone biopsy		

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Inadequate Vitamin D Intake

**Assessment Data:** Weight, physical activity histories, medical history, medication use, lab values. Limited sunlight exposure; works indoors in a sedentary job. Complains of bone pain. Low serum vitamin D<sub>3</sub> at 17. Diet Hx shows limited to no intake from milk and milk products, dislike of fish, no use of multivitamin supplements.

**Nutrition Diagnosis (PES):** Inadequate vitamin D intake related to limited sunlight exposure, indoor job, and diet low in vitamin-D rich foods as evidenced by low serum vitamin D levels.

**Intervention:** Food-nutrient delivery—Encourage intake of more fortified dairy products, fish; use daily supplement containing the active form of vitamin D (cholecalciferol) in dose as prescribed by physician. Educate about the need to get sunlight exposure (20 minutes without sunscreen) several times weekly. Counsel about ways to use vitamin-D rich foods in menu planning and recipes that are acceptable to patient and/or family members.

**Monitoring and Evaluation:** Serum levels of 25-hydroxyvitamin D (25-OHD) at more desirable level after 2–3 months. Patient statement of acceptance of foods rich in vitamin D, such as cream soups and casseroles made with milk. No further signs of osteomalacia.

**INTERVENTION****OBJECTIVES**

- Provide correct amount of calcium, phosphorus, and vitamin D<sub>3</sub>. Include other nutrients that support bone health; meet DRI levels. See Table 11-8.
- Prevent or reverse, if possible, bone density loss resulting from calcium loss in the bone matrix.
- Prevent heart disease and stroke, which may be consequences of severe vitamin D deficiency.

**FOOD AND NUTRITION**

- Diets should be high in calcium; adults will need 1200–1500 mg. If patient is lactose intolerant, try Lactaid

or other forms of lactose-free milk, broccoli, greens, and other sources of calcium.

- Vitamin D is administered at high levels. Dietary sources include fish (particularly salmon, tuna, and mackerel) and fish liver oils. Small amounts are found in egg yolk and beef liver.
- Potassium, magnesium, vitamins C and K and other potentially important nutrients should be highlighted.

**Common Drugs Used and Potential Side Effects**

- Monitor treatment with calcium salts to prevent hypercalcemia; use with plenty of liquids. Avoid taking with iron supplements or bulk-forming laxatives. High-calcium diets may reduce zinc absorption and balance and may, therefore, increase zinc intake.

**TABLE 11-8 Nutrients and Bone Health**

Nutrient	Comments
Alcohol	Moderate drinking (1–2 glasses of wine daily) is associated with increased trochanteric bone mineral density (BMD), but higher intakes may be associated with lower BMD. Heavy alcohol consumption may be linked to tobacco use, poor dietary habits, and poor bone health.
B-complex vitamins	Folic acid and vitamins B <sub>6</sub> and B <sub>12</sub> help to lower homocysteine when elevated.
Boron	Some role but not clearly defined.
Caffeine	Over 300 mg/d of caffeine can negatively impact the vitamin D receptor gene ( <i>VDR</i> ), and the Site Testing Osteoporosis Prevention and Intervention Trial (STOP-IT) found that greater amounts of caffeine affect BMD negatively (Rapuri et al, 2007). Limit intake to three cups of coffee daily and five servings of caffeinated soft drinks or tea; be sure to include adequate amounts of calcium.
Calcium and vitamin D	Dietary supplementation with calcium (1200 mg or more) and vitamin D (800–1000 IU) supports strong bone matrix, moderately reduces bone loss, and reduces the incidence of fractures. Vitamin D may actually be more important than calcium.
Copper	Copper is integral to the process of cross-linking of collagen and elastin molecules, and may have other roles in bone cells as well. Copper is found in meat, poultry, shellfish, organ meats; chocolate; nuts; cereal grains; dried legumes and dried fruits.
Dietary Fiber	A high intake of dietary fiber may interfere with calcium absorption; this may impact vegans, who consume 50 or more grams of fiber per day.
Iron	Iron is important for collagen maturation, and has other roles in osteoblasts and osteoclasts. Iron is found in organ meats, such as liver, kidney, heart; seafood; lean meat, poultry; dried beans; egg yolks; dried fruits; dark molasses; whole-grain and enriched breads or cereals.
Magnesium	Low intakes of magnesium contribute to bone loss. More than 50% of the total magnesium in the body is found in the bone, mostly in bone fluids. Magnesium is found in seeds, nuts; legumes; milled cereal grains; dark-green leafy vegetables such as spinach, broccoli, turnip greens, dark lettuces; milk.
Manganese	Manganese is necessary for the formation of bone matrix and is found in whole grains, nuts, legumes, tea, instant coffee, fruits, and vegetables.
Phosphorus	Meat, poultry, fish, eggs; cereals, grains; legumes; milk and dairy products, nuts.
Protein	70–100 g/d provides more bone building. Avoid larger doses, which can lead to excessive urinary calcium losses.
Silicon	There may be a role for silicon in stimulation of collagen synthesis and osteoblast differentiation. Intake of biologically active silicon, orthosilicic acid, enhances bone density and may help to preserve bone mass (Devine et al, 2005).
Sodium	Excesses can increase calcium excretion. Avoid using salt at the table, and limit total intake to 2400 mg/d.
Soy	Soy seems to be protective against fractures. Isoflavones increase bone density; use dietary sources.
Vitamin A	Too much retinal (not derived from the carotenoids found in plant sources) may contribute to hip fractures, especially in postmenopausal Caucasian women. Preformed vitamin A is found in liver, milk fat, fortified skim milk, eggs.
Vitamin C	Part of collagen, which supports healthy bone structure. Tissues saturate at 200 mg, therefore large doses are wasted.
Vitamin K	Supports osteocalcin for bone strength, reduces urinary calcium excretion, and modifies bone matrix proteins. A low intake of this fat-soluble vitamin increases the risk for bone fracture. Supplement with 120 µg if needed. Vitamin K is found in dark-green leafy vegetables, dairy products, meat, and eggs.
Zinc	The enzymes in osteoblasts require zinc in order to synthesize collagen. Zinc is found in animal products such as meat, fish, poultry; fortified and whole-grain cereals; milk and milk products; shellfish; liver. Zinc is also found in dry beans; nuts.

**REFERENCES**

Devine A, et al. Protein consumption is an important predictor of lower limb bone mass in elderly women. *Am J Clin Nutr.* 81:1423, 2005.

- Anticonvulsant therapy, tranquilizers, sedatives, muscle relaxants, and oral diabetic agents may deplete vitamin D. Phosphate binders with aluminum may precipitate osteomalacia; calcium carbonate may be useful, but do not take it with whole grains, bran, high-oxalate foods, or iron tablets.

### Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Many doctors believe that between 1000–2000 IU of vitamin D per day may be needed to maintain adequate serum levels of this hormone. The upper limit for vitamin D is 10,000 IU per day.

### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain which foods are good sources of vitamin D. Encourage use of cholecalciferol (vitamin D<sub>3</sub>) and not vitamin D<sub>2</sub>.
- Encourage patient to spend time in the sun for skin synthesis of vitamin D; 15–20 minutes may be needed, after which use a sunscreen to avoid sunburn.

- A spoonful of cod liver oil contains about 1300 IU of vitamin D; an 8-oz glass of fortified milk contains about 100 IU.
- Vegetarians who avoid dairy products may be at risk for calcium and vitamin D depletion; discuss alternative sources from diet or from necessary supplementation.

### Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

### For More Information

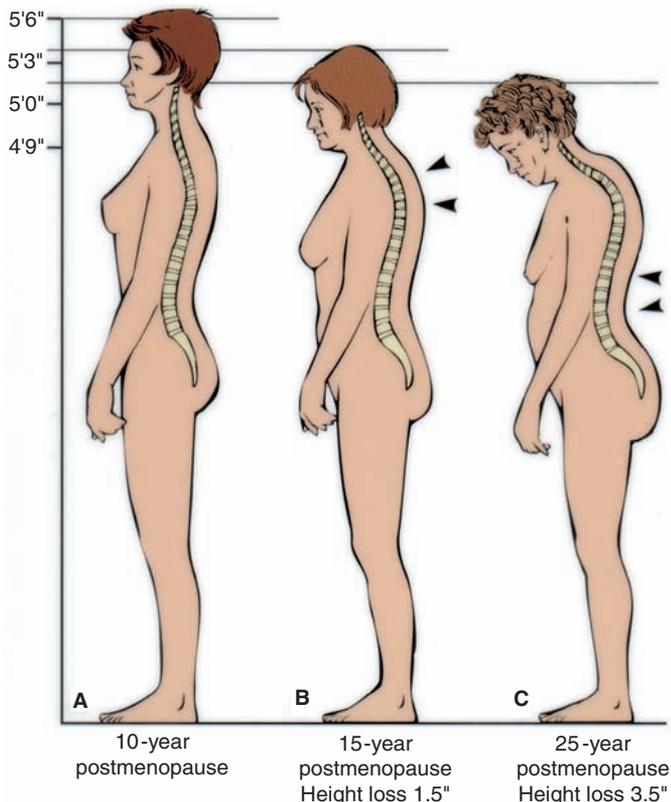
- Mayo Clinic—Osteomalacia  
<http://www.mayoclinic.com/health/osteomalacia/DS00935>
- Medline—Osteomalacia  
<http://www.nlm.nih.gov/medlineplus/ency/article/000376.htm#Definition>

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- Saito T, Fukumoto S. Fibroblast Growth Factor 23 (FGF23) and Disorders of Phosphate Metabolism. [published online ahead of print October 7, 2009] *Int J Pediatr Endocrinol*. 2009:496514.

## OSTEOPENIA AND OSTEOPOROSIS

### NUTRITIONAL ACUITY RANKING: LEVEL 2



Adapted from: Smeltzer SC, Bare BG. *Textbook of Medical-Surgical Nursing*, 9th ed. Philadelphia: Lippincott Williams & Wilkins, 2000.



### DEFINITIONS AND BACKGROUND

**Osteopenia** is a decrease in the amount of calcium and phosphorus in the bones. It is identified by a decrease in bone density, which is evident through a DEXA scan. It can occur in premature infants or in adults as a result of long-term inflammatory bowel disease, especially Crohn's disease, or from low BMI. Plasma 25-hydroxyvitamin D (25-OHD) is the most sensitive indicator of BMD and clinical vitamin D<sub>3</sub> status.

**Osteoporosis** is the most common bone disease in humans; it is characterized by low bone mass, structural deterioration, and decreased bone strength in an estimated 10 million Americans (NOF, 2009).

The aging population is highly affected. Seven percent of non-Hispanic white and Asian men aged 50 and older are estimated to have osteoporosis, and 35% are estimated to have low bone mass. Men are especially vulnerable when they have renal failure, smoke, or take medications on a regular basis, such as anticonvulsants, corticosteroids, or barbiturates.

The World Health Organization (2009) defines osteoporosis as a BMD value that is 2.5 standard deviations or more below the mean of a young adult of the same sex. The lower the BMD, the greater the fracture risk. Osteoporosis can be a silent disease until a fragility fracture occurs at the hip and proximal humerus, when significant physical disability can result.

**TABLE 11-9 Risk Factors for Osteoporosis***Factors That Cannot Be Changed*

Advanced age	History of fracture in a first-degree relative
Caucasians (e.g., Northern European and Asian)	Low body mass index (BMI) and low muscle mass
Female gender	Personal history of fracture after age 50 years
Family history of osteoporosis	

*Factors That Might Be Altered*

Anorexia nervosa	Hypogonadism, as from low estrogen levels or anorexia nervosa
Current smoking	Lifetime diet low in calcium (poor diet, excess fiber)
Depression, past or current	Low testosterone levels in men
Diabetes	Low vitamin D intake or sunlight exposure
Estrogen deficiency (premature menopause, amenorrhea)	Sedentary lifestyle or extended bed rest (immobilization)
Excessive use of alcohol	Use of chemotherapy, tamoxifen, glucocorticoids, lithium, and some anticonvulsants
Homocysteine, elevated plasma levels	Total parenteral nutrition, long-term use
Hypertension	

*Conditions or Diseases That May Lead to Osteoporosis*

AIDS/HIV	Gastrectomy	Liver disease, severe	Primary biliary cirrhosis
Amyloidosis	Gaucher's disease	Lymphoma or leukemia	Rheumatoid arthritis
Ankylosing spondylitis	Hemochromatosis	Malabsorption syndromes	Spinal cord transection
Celiac disease	Hemophilia	Mastocytosis	Stroke
Chronic obstructive pulmonary disease	Hyperparathyroidism	Multiple myeloma	Thalassemia
Congenital porphyria	Hypophosphatasia	Multiple sclerosis	Thyrotoxicosis
Cushing's syndrome	Idiopathic scoliosis	Osteomalacia	Tropical sprue
<b>Diabetes, type 1</b>	<b>Inflammatory bowel disease</b>	Pernicious anemia	

Women can lose up to 20% of their bone mass in the 5–7 years following menopause; 50% of will experience an osteoporotic fracture at some point in time. About 20% of postmenopausal white women in the United States have osteoporosis and 1.5 million fractures occur annually, especially of the hip and spine. Falls are associated with a high risk of frailty fractures (Schwartz et al, 2005).

Spinal or vertebral fractures may lead to loss of height, severe back pain, and spinal deformities such as kyphosis or stooped posture. Hip fractures require hospitalization and major surgery; they impair the ability to walk and may cause disability or death. By 2050, the annual number of hip fractures is expected to triple (World Health Organization, 2009).

Awareness and management of risk factors is important for preventing osteoporosis and the related disability. Both genetic and lifestyle factors play a role. A family history of hip fracture carries a twofold increased risk of fracture among descendants; genetic factors play a major role in BMD and in osteoporosis risk (Ferrari, 2008). Yet BMD is just one of many contributors to bone strength and fracture risk reduction. Dairy, fruit, and vegetable intakes have

emerged as an important modifiable protective factor for bone health (Tucker, 2009). Women may lose bone during lactation if their diets are low in calcium and other nutrients. Magnesium, potassium, vitamin C, vitamin K, several B vitamins, and carotenoids are important (Tucker, 2009); see Table 11-8.

In the skeleton, interleukin-1 protein causes an increase in the number and activity of osteoclastic cells—the cells that break down bone tissue. Depression and elevated plasma homocysteine levels are also associated with osteoporosis. See Table 11-9 for the full list of risk factors for osteoporosis.

Physical activity has different effects depending on its intensity, frequency, and duration, and the age at which it is started, with greater effects in adolescence and as a result of weight-bearing exercise. In addition, diet contributes significantly. Building strong bones during childhood and adolescence can be the best defense against developing osteoporosis later. By about age of 20, most women have acquired 98% of total bone mass. Acquisition of a high peak bone mass (reaching genetic potential) by 30 years of age helps reduce bone losses later in life.

Markers of bone turnover can be used to predict the rate of bone loss in post-menopausal women and can also be used to assess the risk of fractures (Eastell and Hannon, 2008). Markers of bone formation include serum bone alkaline phosphatase, total osteocalcin and the procollagen type I N-terminal propeptide assay (Eastell and Hannon, 2008).

Serotonin can serve as a marker for low bone mass. Circulating levels of the neurotransmitter serotonin are inversely associated with bone mass in women; this may have implications when using SSRIs. Bone formation is inhibited by serotonin in the gut. Inactivation of the leptin receptor in serotonergic neurons identifies a molecular basis for the common regulation of bone and energy metabolisms (Yadav et al, 2009). A drug that stops the gut from synthesizing serotonin may be able to reverse severe bone loss and prevent osteoporosis.

Measurements of the urinary excretion of N- and C-terminal cross-linked telopeptides and of serum C-terminal cross-linked telopeptides are sensitive and specific for bone resorption (Eastell and Hannon, 2008). These measures are as important as measurement of BMD.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Genes coding for the LDL-receptor related protein 5 (LRP5), estrogen receptor alpha (ESR1), and osteoprotegerin, OPG (TNFRSF11b) are known to pose a risk for osteoporosis (Ferrari, 2008). In a large-scale study, single nucleotide polymorphisms (SNPs) from nine gene loci (ESR1, LRP4, ITGA1, LRP5, SOST, SPP1, TNFRSF11A, TNFRSF11B, and TNFRSF11) were associated with BMD, but not always in a predictable manner (Richards et al, 2009).

<b>Clinical/History</b>	Mg <sup>++</sup>	TSH
Height	Na <sup>+</sup> , K <sup>+</sup>	Total
Weight	Vitamin D <sub>3</sub>	testosterone
BMI	status (serum	in men
Back pain	25-OHD)	Serum homocys-
BP	Alb	teine
Bone densitome-	CRP	Serum folate
try, DEXA	PTH (useful in	and vitamin
	some patients)	B <sub>12</sub>
<b>Lab Work</b>	Serum P	
Ca <sup>++</sup>	(decreased	
Urinary Ca <sup>++</sup>	with	
(24 hours)	hyperparathy-	
	roidism)	

## INTERVENTION



### OBJECTIVES

- Preserve height, support independence, and improve functional status. Prevent fractures (Tussing and Chapman-Novakofski, 2005).

## SAMPLE NUTRITION CARE PROCESS STEPS

### Inadequate Vitamin Intake

**Assessment Data:** Food records, DEXA scan showing two deviations below normal. Weight and physical activity histories. Medical history, medications, and lab values.

**Nutrition Diagnosis (PES):** Inadequate intake of vitamin D (NI5.9.1.3) related to poor diet, no food sources, and no use of supplements as evidenced by very low BMD on DEXA.

**Intervention:** Food and Nutrient Delivery: ND 1.3—add calcium containing beverages and foods, vitamin-D rich foods, adequate protein. ND 3.2.3.3—Supplement use for Vitamin D.

Education: E 2.2—Educate patient concerning calcium and vitamin D food and beverage sources to meet needs along with supplement, sunlight exposure 10–30 min/d, protein needs, increased physical activity.

Counseling: Good sources of vitamin D from diet and supplements, meal planning and shopping tips, dining out guide, referral to Meals-on-Wheels or other social agencies as appropriate, appropriate role for sunshine exposure.

**Monitoring and Evaluation:** Improvements in dietary and supplemental intake of vitamin D as shown in food records, lab values. DEXA scan report showing no further decline, and a slight improvement in risk level. No fractures.

- Optimize bone health: choose a balanced diet rich in calcium and vitamin D; use weight-bearing and resistance-training exercises. Follow a healthy lifestyle with no smoking.
- Decrease precipitating factors, such as use of anticonvulsants or corticosteroids, lactase deficiency, low intake of fruits and vegetables and dairy, calcium malabsorption, sedentary lifestyle, and low BMI. Provide adequate time for evaluating improvement (6–9 months at least).
- Assure adequate intake of protein. Rather than having a negative effect on bone, protein intake appears to benefit bone status, particularly in older adults (Tucker, 2009).
- Intake of magnesium, potassium, fruit, and vegetables is positively associated with bone health and total bone mass.



## FOOD AND NUTRITION

- Advise all patients to consume adequate amounts of calcium ( $\geq 1200$  mg/d, including supplements if necessary) and vitamin D. Women after menopause or over age 65 years will need 1500 mg calcium daily. To fulfill the requirement, 1 quart of milk daily can be consumed. If fluid milk is not consumed, dry skim milk powder can be added to many foods. Aged cheeses and yogurt are sources as well.
- Calcium supplements can be used if dairy products are not tolerated; calcium absorption averages approximately 30–40% from most sources See Table 11-10. Space the supplements throughout the day; take no more than 500–600 mg two or more times daily with meals. Use with vitamin D and magnesium.
- For vitamin D, choose fortified milk, cod liver oil, egg yolks, and fatty fish. Supplements may be needed. Do not exceed 10,000 IU/d.

**TABLE 11-10 Tips on Calcium Supplements<sup>a</sup>**

Product	Source of Calcium (mg)	No. of Tablets/Day to Provide About 900–1000 mg Calcium Per Tablet
Caltrate 600	Carbonate (600 mg)	1.5
Os-Cal 500	Carbonate from oyster shell (500 mg)	2
Os-Cal 500 + Vitamin D	Carbonate from oyster shell (500 mg)	2
Posture (600 mg)	Phosphate (600 mg)	1.5
Posture-Vitamin D	Phosphate (600 mg)	1.5
Citracal	Citrate (200 mg)	5
Citracal + Vitamin D	Citrate (315 mg)	3
Citracal Liquitab	Citrate (500 mg)	2
Tums 500 mg	Carbonate from limestone (500 mg)	2
Tums E-X	Carbonate from limestone (300 mg)	3.5
Tums Ultra	Carbonate from shell (400 mg)	2.5
Calcet + Vitamin D	Carbonate, lactate, gluconate (300 mg)	3.5
Fosfree	Carbonate, gluconate, lactate (175 mg)	6

<sup>a</sup>Excesses of calcium supplements can cause hypercalcemia; monitor intakes carefully and take no more than 500–600 mg two or more times daily with meals. Avoid taking with iron supplements. Use extra water with supplements. Excess vitamin D can cause vitamin D calcinosis. Rates of calcium absorption vary, and dietary sources are the best absorbed; calcium maleate is also well absorbed. Elemental calcium varies in different supplements, as follows:

1. Calcium carbonate (Tums, Roxane, Os-Cal, Calciday, Oyst-Cal, Oysterical, Caltrate) contains 40%. Calcium carbonate temporarily decreases gastric acidity, which is needed for calcium absorption. Tricalcium phosphate provides 39%.
2. Calcium chloride contains 36%.
3. Bone meal or Dolomite contains 33% but should be avoided as it also contains lead.
4. Calcium acetate (Phos-Ex, PhosLo) contains 25%.
5. Calcium citrate (Citracal) contains 21%.
6. Calcium lactate contains 13%.
7. Calcium gluconate contains 9%.

Updated from: Shils M, et al, eds. *Modern nutrition in health and disease*. Baltimore: Lippincott Williams & Wilkins, 1999.

- Extra protein may be needed (Devine et al, 2005; Tucker, 2009).
- For sufficient intake of vitamin B<sub>12</sub>, include dairy products, meat, poultry, fish, and fortified cereals.
- Isoflavones may also be beneficial; use two to three servings of soy foods daily.
- If patient is obese, use a nutrient-rich, calorie-controlled diet that provides adequate protein, vitamins, calcium, and other minerals. Adequate manganese, vitamins C and K, potassium, and magnesium should be consumed to meet at least the DRI levels. Include fruits and vegetables that contribute to bone health.
- Assure that folic acid and vitamins B<sub>6</sub> and B<sub>12</sub> are adequate, especially if serum homocysteine levels are elevated.
- Sodium must be controlled. Keep sodium within desired limits while increasing potassium and magnesium.
- Beware of excesses of wheat bran because phytates may increase calcium excretion.
- Caffeine from coffee does not seem to be a problem if calcium (as from milk) is consumed in adequate amounts. However, cola drinks should be limited (Tucker, 2009).
- Moderate alcohol intake shows positive effects on bone, particularly in older women (Tucker, 2009).

### Common Drugs Used and Potential Side Effects

- Adequate calcium is crucial; supplementation in bioavailable forms is necessary in individuals who do not

achieve recommended intake from dietary sources (see Table 11-11). Side effects may include abdominal pain, anorexia, constipation, vomiting, nausea or dry mouth.

- Oral doses of vitamin D<sub>3</sub> in the range of 1800 to 4000 IU per day may be needed to take serum levels up to 75 to 110 nmol/L (Bischoff-Ferrari et al, 2010).
- Oral alendronic acid is the reference drug for menopausal women with osteopenia. It may be used with parathormone as well, but this has a 2-year limit (Black et al, 2005). See Table 11-12 for more guidance.
- The once monthly injections of risedronate (150 µg) are beneficial for those for which daily or weekly dosing is a challenge (Rackoff, 2009). Bone markers can be used to monitor the efficacy of antiresorptive therapy such as hormone-replacement therapy, raloxifene and bisphosphonates (Eastell and Hannon, 2008).
- SSRIs have been associated with lower BMD and increased rates of bone loss, as well as increased rates of fracture (Haney et al, 2010). Their use should be closely monitored.

### Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Cabbage, pigweed, dandelion, avocado, and parsley have been recommended, but have not shown efficacy.

**TABLE 11-11 Medications Commonly Used for Management of Osteoporosis<sup>a</sup>**

Medication Comments	Effects	Comments
Bisphosphonates: risedronate (Actonel); alendronate (Fosamax)	Effective agents for reducing vertebral and nonvertebral fracture risk. Alendronate is approved for the treatment of osteoporosis in men. Alendronate and risedronate are approved for use by men and women with glucocorticoid-induced osteoporosis. Zoledronic acid is under study. Bisphosphonates inhibit atherogenesis.	Risedronate may cause dysphagia, esophageal ulcer, and stomach ulcer. Take on an empty stomach 30 minutes before meals; sit upright. Take additional vitamin D and calcium. Headache, gastrointestinal (GI) distress, diarrhea, nausea, constipation, and rash may occur, although rarely. Alendronate may cause metallic taste, nausea, diarrhea, and decreased potassium and magnesium. Avoid in severe renal disease, pregnancy, or breastfeeding. Nausea, heartburn, irritation or pain of the esophagus, anorexia, vomiting, dysphagia, sensation of fullness, and constipation or diarrhea may occur.
Calcitonin-salmon (Miacalcin)	Bone loss is reduced, and bone mass increases, although not in the hip. A modest increase in bone mass occurs.	200 IU/d, the recommended regimen, reduces vertebral fracture risk by 33% in women with low bone mass. Calcitonin makes calcium more available to bones. It is given as an injection or nasal spray; it may cause allergic reactions and flushing of the face and hands, urinary frequency, anorexia, nausea, constipation, or skin rash.
Calcitriol (1,25-dihydroxyvitamin D)	Active form of vitamin D hormone that increases GI absorption of calcium from the gut, kidney reabsorption of calcium, stimulates bone resorption, decreases PTH production, and stimulates skeletal osteoblasts/osteoclasts. Larger doses than the DRI for vitamin D may be needed; 700–800 IU may be beneficial along with 500–1200 mg calcium (Cranney et al, 2007).	Anorexia, abdominal cramping, headache, lethargy, nausea, weight loss, and weakness may result from larger doses.
Ibandronate (Boniva)	Ibandronate is used to treat or prevent osteoporosis in women after menopause; it may increase bone mass by slowing loss of bone.	Should not be taken if hypocalcemia is a problem.
PTH (teriparatide; Forteo)	PTH is the only anabolic osteoporosis agent available for clinical use to lower vertebral fracture incidence by triggering formation of new bone.	Use only in ambulatory patients.
Raloxifene (Evista)	Significantly reduces vertebral fracture risk but not nonvertebral fracture risk.	Protects against thin, weak bones and fractures; also lowers serum cholesterol by 7% and low-density lipoprotein (LDL) by 11%. It may trigger menopausal symptoms, including hot flashes, but is less likely to have an estrogen-like increase in cancer risk.
Sodium fluoride	The slow-release form may increase bone formation and decrease the risk of fractures.	In patients with mild-to-moderate osteoporosis, long-term supplements with fluoride plus calcium result in lower rates of vertebral fracture than supplementation with calcium alone. Intake of fluoride in drinking water at 1 ppm does not appear to be associated with increased risk of hip fracture. Side effects may include abdominal pain, diarrhea, nausea, vomiting.
Statins	Statins, agents that reduce atherogenesis, stimulate bone formation.	Cardiovascular disease and low bone mineral density have some common etiologies.

<sup>a</sup>The FDA approves calcitonin, alendronate, raloxifene, and risedronate for the treatment of postmenopausal osteoporosis; alendronate, risedronate, and raloxifene are approved for the prevention of the disease. Current pharmacological options for osteoporosis prevention and/or treatment are bisphosphonates (alendronate and risedronate), calcitonin, estrogens and/or hormone therapy, parathyroid hormone (PTH 1–34), and raloxifene.

Source: National Osteoporosis Foundation Web site accessed December 14, 2009, at: <http://www.nof.org/patientinfo/medications.htm>.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Prevention is the best medicine. Encourage patient to stand upright, rather than sit or recline, as often as feasible. Measures to decrease fall frequency and to slow down the rapid life pace of healthy people with low bone mass should prevent some fractures (Kelsey et al, 2005).
- Change a sedentary lifestyle. Regular resistance and high-impact exercise, contributes to development of high peak bone mass and may reduce the risk of falls in older individuals (Moayeri, 2008). Aerobic and strengthening exercises are helpful as well.
- Walking or running is beneficial. However, excessive weight-bearing exercise can cause amenorrhea in premenopausal women when a low-calorie diet is consumed.

**TABLE 11-12 Features of Rheumatic Arthritis**

Tender, warm, swollen joints
Symmetrical pattern of affected joints
Joint inflammation <i>often</i> affecting the wrist and finger joints closest to the hand
Joint inflammation <i>sometimes</i> affecting other joints, including the neck, shoulders, elbows, hips, knees, ankles, and feet
Fatigue, occasional fevers, a general sense of not feeling well
Pain and stiffness lasting for more than 30 minutes in the morning or after a long rest
Symptoms that last for many years
Variability of symptoms among people with the disease

Source: National Institute of Arthritis and Musculoskeletal and Skin Diseases. Available at [http://www.niams.nih.gov/Health\\_Info/Rheumatic\\_Disease/default.asp#ra\\_14](http://www.niams.nih.gov/Health_Info/Rheumatic_Disease/default.asp#ra_14).

- An educational osteoporosis prevention program with hands-on activities can increase self-efficacy (Tussing and Chapman-Novakofski, 2005). Explain that calcium absorption declines with age and that adequate calcium and vitamin D are important throughout life. The overall benefit of healthful eating must be strongly emphasized. Consider calcium and vitamin D supplementation in the elderly.
- Describe importance of the use of milk, cheeses, yogurt, broccoli, kale and other greens, and soybeans. Provide recipes and shopping tips.
- Decrease the use of tobacco. Use only moderate amounts of alcohol.
- Caffeine poses a minimal risk unless it replaces calcium-containing beverages; BMD is not affected by caffeine if at least 1 glass of milk is consumed daily.
- Encourage adequate exposure to sunlight (10–30 min/d). Avoid sunburn and overexposure, with its risks of skin cancer.
- Remind all teenagers that osteoporosis is “kid stuff;” maintenance of weight-bearing activity is important during the growing years. Intake of carbonated beverages instead of milk is a big concern.
- Some mineral waters are excellent sources of calcium; bioavailability is good.
- Avoid long-term use of high doses of retinol from fortified foods or supplements.
- Persons with previous fractures are at risk and should be monitored carefully for osteoporosis. The National Osteoporosis Foundation supports an Awareness and Prevention Month in May of each year.

- When steroids are used, check on bone density changes; there is a high incidence of osteoporosis.
- Note that improvements in BMD may take up to 3 years to note improvement (Compston, 2009).

### Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

### For More Information

- Clinical Guidelines—Osteoporosis  
<http://www.nof.org/professionals/clinical.htm>
- National Osteoporosis Foundation  
<http://www.nof.org/>
- Osteopenia  
<http://www.nlm.nih.gov/medlineplus/ency/article/007231.htm>

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# PAGET'S DISEASE (OSTEITIS DEFORMANS)

## NUTRITIONAL ACUITY RANKING: LEVEL 1–2



### DEFINITIONS AND BACKGROUND

Paget's disease of the bone (PDB) is a disorder of skeletal remodeling, where areas on bone grow abnormally, enlarging and becoming soft. It is of unknown etiology, with excessive bone destruction and repairing. Of all persons older than 50 years of age, 3% have an isolated lesion; actual clinical disease is much less common. PDB is the second most common bone disease in the world. A systematic laboratory screening including serum alkaline phosphatase of an older subject complaining of bone pain, articular pain, or back pain is a strategy to improve the diagnosis of PDB (Varenna et al, 2009).

The disease tends to run in families. Genetic analysis indicates that 40% of patients with Paget's disease have an affected first-degree relative. Approximately 3 million Americans have the disease; it rarely occurs before age 40. Juvenile Paget's disease is very debilitating. Osteoclasts are larger than normal and increased in size (Defetos, 2005). Juvenile Paget's disease usually presents in infancy or childhood and results in progressive deformity, growth retardation, and deafness.

The disease is higher in frequency in people who are aged 65 or older. There is a slight male predominance. Prognosis is good in mild cases.

Sarcoma can also be found in this population (Mankin and Hornicek, 2005). There has been a decline in incidence of this complication but where it does occur, prognosis is still poor (Mangham et al, 2009).



### ASSESSMENT, MONITORING, AND EVALUATION



#### CLINICAL INDICATORS

**Genetic Markers:** There seem to be strong ties to European ancestry in Paget's disease, including Australia and New Zealand. A majority of cases harbor germline mutations in the SQSTM1, Sequestosome1 gene (Merchant et al, 2009).

#### Clinical/History

Headaches  
Tickening of long bones  
Bowing of limbs  
Reduced height  
Spontaneous fractures  
X-rays (denser, expanded bones)  
Bone scans

Height  
Weight  
BMI  
Deep "bone pain"  
Joint pain, neck pain  
Skull enlargement  
Hearing loss

#### Lab Work

Alk phos (increased)  
Urinary Ca<sup>++</sup> (altered)  
Vitamin D<sub>3</sub> status (serum 25-OHD)  
Uric acid (UA), elevated?

PTH (abnormal) Ca <sup>++</sup> , Mg <sup>++</sup> Na <sup>+</sup> , K <sup>+</sup>	Alb, transthyretin CRP Transferrin Serum P	H & H Serum B <sub>12</sub> Radiolabeled bisphosphonate
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### INTERVENTION



#### OBJECTIVES

- Prevent complications, especially related to the nervous system (e.g., fractures, spinal stenosis, paraplegia, cardiac failure, and deafness).
- Prevent side effects of drug therapy.
- Promote full recovery when possible.
- Differentiate from other conditions with bone lesions.
- Alleviate anemia and other complications.



#### FOOD AND NUTRITION

- Adequate protein is important, with adequate calories to spare protein.
- Adequate levels of calcium and vitamins C and D may be needed.
- To correct anemia, monitor serum levels of iron and vitamin B<sub>12</sub> to determine need for an altered diet.

#### Common Drugs Used and Potential Side Effects

- Drugs that inhibit bone resorption—bisphosphonates (etidronate, pamidronate, clodronate, or alendronate)—

#### SAMPLE NUTRITION CARE PROCESS STEPS

##### Abnormal Nutritional Labs

**Assessment Data:** Weight and physical activity histories. Medical history, medications, abnormal lab values for alk phos, uric acid, serum vitamin D<sub>3</sub>, and PTH.

**Nutrition Diagnosis (PES):** Abnormal nutritional labs related to metabolic changes from Paget's disease as evidenced by increased alk phos, altered urinary calcium, abnormal PTH, elevated uric acid levels, diagnosis of anemia, and bone pain.

**Intervention:** Treatment with bisphosphonates to alter serum and urinary labs; careful monitoring for side effects affecting intake and appetite. Food-nutrient delivery—Correct iron-deficiency anemia from poor intake and disease process.

**Monitoring and Evaluation:** Improvement in serum lab values; resolution of anemia; decreased bone pain.

may be used to slow the progression. Bisphosphonates are pyrophosphate analogs that bind to bone at active sites of remodeling. The bisphosphonate zoledronic acid (Zometa), given in a single injection, yields a rapid and long-lasting improvement in bone health (Reid et al, 2005). The nitrogen-containing BPs pamidronate (Aredia) and zoledronic acid (Zometa) are capable of causing bisphosphonate-associated osteonecrosis of the jaw (Grewal and Fayans, 2008).

- Risedronate (Actonel) can cause dysphagia, esophageal ulcer, and stomach ulcer. Take on an empty stomach 30 minutes before meals; consume additional vitamin D and calcium. Headache, diarrhea, nausea, constipation, and rash may occur, although they are rare.
- Osteoprotegerin may be used in managing the juvenile form of Paget's disease (Cundy et al, 2005).
- Thyrocalcitonin or synthetic calcitonin may be used to decrease passage of calcium from bones to bloodstream. Methods of administration include a nasal spray. Monitor for nausea or vomiting.
- Analgesics may be needed for pain.

### Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Unusual bone diseases may be associated with use of Chinese herbs.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss appropriate dietary alterations for patient's condition, individualized for the current condition and status. Include good food sources of calcium, B-complex

vitamins, iron, protein, and vitamin D. Monitor carefully, if supplements are used, in addition to dietary guidance.

- Discuss side effects for the specific drugs ordered.

### Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

### For More Information

- National Association for the Relief of Paget's Disease  
<http://www.paget.org.uk/>
- National Institute of Arthritis and Musculoskeletal and Skin Diseases  
<http://www.niams.nih.gov/bone/hi/paget/diagnosed.htm>
- National Institutes of Health Osteoporosis and Related Bones Diseases  
<http://www.niams.nih.gov/bone/>
- Paget's Disease  
<http://www.nlm.nih.gov/medlineplus/ency/article/000414.htm>
- Paget Foundation  
<http://www.paget.org/>

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## POLYARTERITIS NODOSA

### NUTRITIONAL ACUITY RANKING: LEVEL 1–2



### DEFINITIONS AND BACKGROUND

Polyarteritis nodosa (PAN) is characterized by necrotizing inflammation of medium- or small-sized arteries without glomerulonephritis or vasculitis in arterioles, capillaries, or venules (Colmegna and Maldonado-Cocco, 2005). Viral infections such as hepatitis B may trigger it.

In PAN, arteries become inflamed in several organs, causing damage in brain, heart, liver, GI tract, or renal tissues. Renal involvement develops and is accompanied by hyper-

tension in half of patients. PAN also commonly involves the gut (abdominal angina, hemorrhage, perforation), heart (myocarditis, myocardial infarction), or eye (scleritis); rupture of renal or mesenteric microaneurysms can also occur. PAN is two to three times more common in men and usually develops between ages 40 and 50 years. Rarely, it occurs after a Hepatitis-B vaccination.

It is fatal if not treated. Treatment includes use of prednisone, plasmapheresis to remove immune complexes, and antiviral therapy (lamivudine) for the hepatitis B infection.



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Medium-sized artery vasculitides that occur in childhood manifest mostly as PAN, with high morbidity and mortality rates (Dillon et al, 2009). PAN is likely to have a genetic connection; MEFV is one gene under study.

<b>Clinical/History</b>	Myalgias, weakness	Hepatitis B antigen or antibody in serum
Height	Neuropathy	
Weight	Hematuria	
BMI	Fatigue	Ca <sup>++</sup> , Mg <sup>++</sup>
Weight loss >4 kg since onset of illness	Rash, nodules or Raynaud's disease	Na <sup>+</sup> , K <sup>+</sup>
Hematuria	Biopsy of medium vessels	Alb, transthyretin
Edema		BUN, Creat (elevated but not from dehydration)
Chest pain		
Tachycardia		
Shortness of breath	<b>Lab Work</b>	Transferrin
Fever?	ESR (elevated)	H & H
Abdominal pain	CRP	Vitamin D <sub>3</sub> status (serum 25-OHD)
BP (elevated)	Glucose	

## INTERVENTION



### OBJECTIVES

- Treat as soon as possible to decrease heart and renal damage.
- Improve appetite and intake.
- Prevent weight loss.
- Increase calorie intake when there is fever.

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Inadequate Oral Food and Beverage Intake

**Assessment Data:** Weight and physical activity histories. Medical history, medications, and lab values.

**Nutrition Diagnosis (PES):** Inadequate oral food and beverage intake related to anorexia, fever and abdominal pain as evidenced by weight loss of 3 kg in 8 weeks.

**Intervention:** Food-Nutrient delivery—Offer preferred foods, enhanced with energy kilocalories from milk powder, fats, etc. Educate about how to manage nausea; suggest small, frequent meals throughout the day and liquids separate from meals. Coordinate care with nursing, medical teams if medications are needed.

**Monitoring and Evaluation:** Resolution of weight loss; no further losses. Improvement in nausea and abdominal pain.

- Reduce edema, anorexia, hypertension, and other effects of the disorder.



### FOOD AND NUTRITION

- A high-energy intake may be beneficial in case of weight loss.
- A normal to high protein intake generally is required.
- Fluid or sodium intake may be limited with hypertension, kidney disease, or edema or with use of steroids.
- Include phytochemicals derived from spices such as turmeric (curcumin); red pepper (capsaicin); cloves (eugenol); ginger (gingerol); cumin, anise, and fennel (anethol); basil and rosemary (ursolic acid); garlic (diallyl sulfide, S-allylmercaptocysteine, and ajoene); and pomegranate (ellagic acid) (Aggarwal and Shishodia, 2004).

### Common Drugs Used and Potential Side Effects

- Steroids such as prednisone may be used for 2 weeks. Side effects of long-term use include negative nitrogen and potassium balances; decreased calcium and zinc levels; CHO intolerance; and excessive sodium retention. With weight gain, a calorie-controlled diet may be useful.
- Pain relievers may be needed; monitor individually for side effects such as GI distress.
- Immunosuppressive cyclophosphamide may be used; long-term effects can reduce the ability to fight infections. Corticosteroids plus cyclophosphamide is the standard of care, in particular for patients with more severe disease, in whom this combination prolongs survival (Colmegna and Maldonado-Cocco, 2005).
- Infliximab may be used as an alternative agent for the treatment of patients with PAN refractory to conventional therapy (Al-Bishri et al, 2005).

### Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss alternate dietary guidelines as appropriate for medications and side effects of the disease.
- Discuss sources of nutrients as appropriate for the ordered diet. Provide guidance on enhancing nutrient and energy density from meals and snacks.
- With abdominal pain and GI bleeding, PAN occasionally is mistaken for inflammatory bowel disease. Be certain to see a trained specialist as needed.

#### Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

**For More Information**

- Johns Hopkins Vasculitis Center  
<http://vasculitis.med.jhu.edu/typesof/polyarteritis.html>
- Polyarteritis Nodosa Foundation  
<http://www.angelfire.com/pa3/autoimmunedisease/aifeindex.html>
- Polyarteritis Nodosa  
<http://www.emedicine.com/ped/topic1844.htm>
- Vasculitis Foundation  
<http://www.vasculitisfoundation.org/polyarteritisnodosa>

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# RHABDOMYOLYSIS

**NUTRITIONAL ACUITY RANKING: LEVEL 3****DEFINITIONS AND BACKGROUND**

Rhabdomyolysis (RML) is a clinical and biochemical syndrome resulting from skeletal muscle injury with release of myoglobin into the plasma and breakdown of muscle fibers with release into the circulation. Some of these changes are toxic to the kidney, often resulting in kidney damage or acute renal failure. A disturbance in myocyte calcium homeostasis takes place.

RML may occur in infants, toddlers, and adolescents who have inherited enzyme deficiencies of carbohydrate or lipid metabolism, DMD, or malignant hyperthermia. RML may also occur from extensive muscle damage as from a crushing injury, major burn, electrical shock, toxins, bacterial infections, excessive exercise (Olpin, 2005), seizures, alcoholism, overdose of cocaine, or use of drugs such as cholesterol-reducing statins. The most common causes of RML in adults include crush injury, overexertion, alcohol abuse, use of certain medicines, and toxic substances. Postoperative RML in bariatric surgery occurs with prolonged muscle compression; potential consequences may lead to death (de Menezes Ettinger et al, 2005).

Muscle pain caused by RML may involve specific symptoms of groups of muscles or may be generalized throughout the body. Muscles in the calves and the lower back are commonly affected but each patient is different. Early complications of RML include severe hyperkalemia with cardiac arrhythmia and arrest. The most serious late complication is acute renal failure.

RML can be defined with CK values exceeding 10–25 times the upper limit of normal irrespective of renal function (Linares et al, 2009). Management of suspected drug-induced myopathy should include immediate discontinuation of the offending agent and supportive care (Mor et al, 2009).

inherited myopathies. Elevated CK levels have been found with a high-density SNP genotype in a p.Trp3X allele; this mutation is associated with a mild Becker phenotype of MD (Flanigan et al, 2009).

Clinical/History	Weakness of the affected muscles	Serum myoglobin test (positive)
Height	Muscle stiffness or aching (myalgia)	Urinary casts or hemoglobin
Weight	Seizures	Ca <sup>++</sup> , Mg <sup>++</sup>
BMI	Joint pain	Na <sup>+</sup>
Weight gain (unintentional)	Fatigue	K <sup>+</sup> (may be high from muscle breakdown)
I & O	Abnormally dark colored urine from excretion of myoglobin	Alb, transthyretin
Tea-colored urine		CRP
Temperature		BUN
BP (elevated)		Creat
Exposure to toxic substances or chronic alcohol use	<b>Lab Work</b>	Transferrin
Use of medications such as statins	Creatine phosphokinase (CPK) (very high)	H & H
Muscle tenderness		UA (elevated)
		Vitamin D <sub>3</sub> status (serum 25-OHD)

**ASSESSMENT, MONITORING, AND EVALUATION****CLINICAL INDICATORS**

**Genetic Markers:** RML may occur in infants, toddlers, and adolescents who have inherited enzyme deficiencies of carbohydrate or lipid metabolism or who have

**INTERVENTION****OBJECTIVES**

- Preserve renal function.
- Eliminate myoglobin out of the kidneys with early and aggressive hydration. Medicines may also be needed to make the urine more alkaline.
- Treat kidney failure or hyperkalemia if needed.

**FOOD AND NUTRITION**

- Hydration needs with muscle necrosis may approximate the massive fluid volume needs of a severely burned patient.

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Excessive Physical Activity

**Assessment Data:** Weight and physical activity histories. Medical history with diagnosis of RML.

**Nutrition Diagnosis (PES):** Excessive physical activity related to 2-hour workouts twice daily as evidenced by complaints of weakness, muscle stiffness and tenderness, and very high CPK levels.

**Intervention:** Education about a more desirable level of physical activity to lessen strain on muscles. Counseling about nutrition for athletics and maintenance of other healthy habits, including adequate rest and sleep. Physical activity logs.

**Monitoring and Evaluation:** Improvement in muscle stiffness and tenderness; CPK levels returning to a normal range. Physical activity logs showing workouts no longer than 30 minutes twice daily.

- Special dietary advice is required if there is renal disease or the need for dialysis.
- It is important to offer advice according to the medical condition that preceded RML. Avoidance of fasting, feeding with a high-carbohydrate and low-fat diet, and intravenous drip infusion soon after every onset of RML may be needed for children (Korematsu et al, 2009).

### Common Drugs Used and Potential Side Effects

- Statins block the enzyme in the liver that is responsible for making cholesterol, hydroxy-methylglutaryl-coenzyme A reductase (HMG-CoA reductase).
- Despite the withdrawal of cerivastatin because of fatal RML, the risk of this complication with other statins is extremely low (Waters, 2005). Options for managing statin myopathy include statin switching, particularly to fluvastatin or low-dose rosuvastatin; nondaily dosing regimens; nonstatin alternatives, such as ezetimibe and bile acid-binding resins; and coenzyme Q10 supplementation (Joy and Hegele, 2009).
- Diuretic therapy may be needed if there is hypertension.
- If there is hyperkalemia, calcium chloride or calcium gluconate may be used.

### Herbs, Botanicals, and Supplements

- Health care practitioners must take an active role in identifying patients who are using CAM and provide appropriate patient education (Gabardi et al, 2007). Herbs and botanical supplements should not be used without

discussing with physician. There are 17 dietary supplements that have been associated with direct renal injury, CAM-induced immune-mediated nephrotoxicity, nephrolithiasis, RML with acute renal injury, and hepatorenal syndrome (Gabardi et al, 2007).

- Even brief exposure to atorvastatin causes a marked decrease in blood coenzyme Q10 concentration, with commonly reported adverse effects of exercise intolerance, myalgia, and myoglobinuria.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss alternate dietary guidelines as appropriate for medications and side effects of the disease.
- Discuss how to use diet and exercise to manage high serum cholesterol if this information has not been given before. Reinforce what the patient has been doing well.
- After damage to any muscles, extra fluid is needed to dilute urine and to eliminate myoglobin. Among soldiers, RML occurs in 25% of those who are injured (Carter et al, 2005).

#### Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

#### For More Information

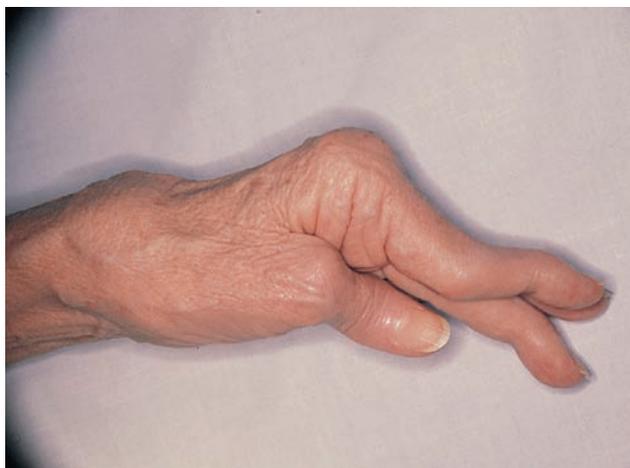
- E-medicine  
<http://www.emedicine.com/emerg/topic508.htm>
- Rhabdomyolysis  
<http://www.nlm.nih.gov/medlineplus/ency/article/000473.htm>

### RHABDOMYOLYSIS—CITED REFERENCES

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- de Menezes Ettinger JE, et al. Prevention of rhabdomyolysis in bariatric surgery. *Obes Surg.* 15:874, 2005.
- Flanigan KM, et al. DMD Trp3X nonsense mutation associated with a founder effect in North American families with mild Becker muscular dystrophy. *Neuromuscul Disord.* 19:743, 2009.
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- Joy TR, Hegele RA. Narrative review: statin-related myopathy. *Ann Intern Med.* 150:858, 2009.
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# RHEUMATOID ARTHRITIS

## NUTRITIONAL ACUITY RANKING: LEVEL 1



Adapted from: Strickland JW, Graham TJ. *Master Techniques in Orthopaedic Surgery: The Hand*, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2005.



### DEFINITIONS AND BACKGROUND

RA is a chronic polyarthritis mainly affecting the smaller peripheral joints and is accompanied by general ill health. Crippling deformities can occur. Of all cases, 75% are women. Most patients are between ages 20 and 40, and RA affects over 1.3 million Americans. To diagnose RA, symptoms must have been present for at least 6 weeks; see Table 11-12.

The cause of RA is increased inflammatory cytokine production, such as from mast cells, interleukin-6, tumor necrosis factor alpha (TNF $\alpha$ ), and acute-phase proteins. Inflammation of synovial tissues is the dominant manifestation. Antibodies against IgG and collagen are noted. Hand involvement and knees or ankles/feet are involved in most. Table 11-13 provides a list of the variant forms of RA.

Some studies show an improvement in RA symptoms over the short term with a diet high in omega-3s or fish oil supplements. Omega-3 fatty acids reduce tenderness in joints, decrease morning stiffness, and reduce the amount

**TABLE 11-13 Variant Forms of Rheumatic Arthritis (RA)**

Condition	Background	Nutritional Implications
Juvenile RA (JRA)	JRA causes joint inflammation and stiffness for more than 6 weeks in a child 16 years of age or less. It is classified into three types, depending on symptoms, number of joints involved, and presence or absence of antibodies in the blood. Pauciarticular JRA is most common and affects mainly the knees. The polyarticular form affects 30% of children with JRA. Still disease is the systemic form; it tests negative for the usual antibodies, may affect internal organs, may become chronic in adulthood and affects 20% of children with JRA. Both genetic factors and environmental factors, such as a virus, can trigger JRA. Because JRA often affects knees, limping can occur. Salicylates, gold salts, or glucocorticoids may be used.	Children suffering from JCA may have reduced serum levels of beta-carotene, retinol, and zinc.
Sjögren's syndrome	Dry eyes and dry mouth occur as a result of insufficient production of lacrimal and salivary secretions. Artificial tears and glucocorticoids may be needed. Sjögren's syndrome is relatively common and affects 4 million Americans, mostly women. It is most often related to RA, lupus, scleroderma, or polymyositis. Debilitating pain and fatigue can occur. Sensitivity to sunlight is common; sunscreen is helpful.	Plan meals and use artificial saliva for easier swallowing. Chewing sugar-free gum can stimulate saliva production if any is available. Gel-based saliva substitutes are useful. Sip water often, and avoid caffeinated drinks, which can be dehydrating. Drink water during meals to help with swallowing. Mouth infections are common; use good oral hygiene. With dry mouth or dysphagia, there is a risk for aspiration pneumonia. Weight loss and digestive problems are common.
Felty's syndrome	Felty's syndrome only affects about 1% of RA patients. This is a triad of RA, granulocytopenia, and splenomegaly. Painful, stiff, and swollen joints occur. Infections, leg ulcers, burning eyes, and anemia also can complicate the condition. Sometimes, splenectomy is indicated; drug therapy may be helpful to others.	Fever, weight loss, and brown pigmentation may occur. If immunosuppressive drugs are used, monitor for side effects.
Rheumatoid vasculitis	Rheumatoid vasculitis can be life threatening and usually occurs in patients with severe deforming arthritis and a high titer of rheumatoid factor. A majority have a strong human leukocyte antigen relationship. Vasculitic lesions include rheumatoid nodules, small nail fold infarcts, and purpura. Fatigue, weight loss, fever, organ ischemia, CNS infarctions, myocardial infarction, and peripheral neuropathy can occur.	Corticosteroids are the usual treatment. D-penicillamine and prednisone generally are used.

of medication needed; they also downregulate T-cell proliferation. People with RA who eat 4 oz of fish every day have less morning stiffness, swollen joints, and all-around pain. Fish oil and aspirin are blood thinners, and they should not be taken together for a long time.

Supplements of GLA, as from borage oil, may reduce generation of mediators of inflammation and attenuate symptoms but may cause potentially harmful increases in serum arachidonic acid unless EPA is also used. GLA increases prostaglandin E levels, which increase cyclic adenosine monophosphate (cAMP) levels which, in turn, suppress TNF $\alpha$  synthesis.

Epidemiological studies suggest that the antioxidant potential of dietary carotenoids may protect against the oxidative damage that can result in inflammation (Pattison et al, 2005). Proper antioxidant nutrients provide defense against increased oxidant stress. Supplementation of folate and vitamin B<sub>12</sub> is needed in patients treated with methotrexate to reduce side effects and to offset elevated plasma homocysteine.

Complications of RA may include osteoporosis and chronic anemia. Calcium and vitamin D reduce the bone loss in patients who take steroids. An iron supplement may prevent anemia, and serum ferritin levels may be low. Patients benefit from a basic dietary supplement.

Higher intakes of meat and total protein and lower intakes of fruit, vegetables, and vitamin C are associated with an increased risk of RA (Choi, 2005). However, dietary factors such as fruit, coffee, long-chain fatty acids, olive oil, vitamins A, E, C, and D, zinc, selenium, and iron need to be studied over a longer time period (Pedersen et al, 2005).

Rheumatoid cachexia, loss of muscle mass and strength and increase in fat mass, is very common in patients with RA and persists even after joint inflammation improves (Roubenoff, 2009). Cardiovascular disease is a concern. Body composition studies are as important as BMI and other traditional assessment measures (Elkan et al, 2009).

## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** B cell, cytokine and inflammation response, and antigen presentation pathways are associated with RA; this confirms the known biological mechanisms for auto-immunity (Ballard et al, 2009).

Clinical/History	Pain and	Lab Work
Height	stiffness >30	RBC
Weight	minutes in	CRP
BMI	the morning	LE prep
Temperature	or after a	Creat (may be
	long rest	decreased)
	Food allergies	

ESR (increases with inflammation)	Ceruloplasmin (may be increased)	Alb, transthyretin
ANA	H & H	Gluc
Rheumatoid factor (RF)	Serum ferritin	BUN
Antistreptococcal antibody titer	Serum B <sub>12</sub>	Ca <sup>++</sup> , Mg <sup>++</sup>
Immunoglobulins (may be elevated in Sjögren's)	Transferrin	Na <sup>+</sup> , K <sup>+</sup>
	Serum folate, RBC folate	Vitamin D <sub>3</sub> status (serum 25-OHD)
	Serum copper	

## INTERVENTION



### OBJECTIVES

- Preserve a high level of physical and social functioning to promote good quality of life; reduce the effects of pain and swelling.
- Maintain satisfactory nutritional status; malnutrition and loss of lean body mass are common in this condition. Monitor weight changes.
- Simplify meal preparation.
- Support the immune system. Consume foods rich in antioxidants, such as carotenoids (Pattison et al, 2005), vitamin E, selenium, and vitamin D. A vegetarian diet may have significant benefits.
- Promote adequate growth in children who have RA; stunting can occur from glucocorticoids.
- Promote return of fat-free body mass and improvement in muscle strength.
- Restrict sodium intake, if needed.

## SAMPLE NUTRITION CARE PROCESS STEPS

### Drug–Nutrient Interaction

**Assessment Data:** Weight and physical activity histories. Medical history, medications and lab values. DEXA scan results.

**Nutrition Diagnosis (PES):** Food-Medication interaction (NC-2.3) related to corticosteroid use secondary to diagnosis of RA as evidence by abnormal Ca<sup>++</sup> level <8.4, DEXA scan at 80% of desirable range for age, perimenopausal status, low calcium and vitamin D intake from diet history.

**Intervention:** Food-Nutrient Delivery—include extra calcium-rich foods. Education about use of steroid therapy and its impact on nutritional status. Counseling about good sources of calcium and vitamin D from diet and supplements, meal planning and shopping tips, dining out guide, referral to Meals-on-Wheels or other social agencies as appropriate. Coordinate care with nursing and physician to administer calcium and vitamin D supplements at different time than corticosteroids to help increase absorption.

**Monitoring and Evaluation:** Improvements in dietary and supplemental intake of vitamin D and calcium as shown in food records, lab values, and DEXA scan report.

- Modify patient's diet if hyperlipidemia is present or if there is elevated homocysteine.
- Avoid or correct constipation.



## FOOD AND NUTRITION

- Use a high-protein and high-calorie diet if patient is malnourished. Cachexia is common (Marcora et al, 2005).
- A diet that lessens inflammation is useful; olive oil should be used often because it contains oleocanthal, a natural anti-inflammatory agent.
- Eating fatty fish, such as salmon, sardines, mackerel, herring, and tuna, two times per week is suggested. In addition to fatty fish, other good sources of omega-3s include flaxseed, walnuts, soy, canola oils. Try to acquire 3–6 g of omega-3 fatty acids per day for 4 months.
- An uncooked vegan diet may be useful, with berries, fruits, vegetables, roots, nuts, and seeds; see Table 11-2. There is improvement in RA when eating a lactovegetarian, vegan, or Mediterranean diet (Skoldstam et al, 2005).
- Adequate fluid, fiber, vitamins, and minerals are important. Use foods high in beta-carotene, lutein lycopene, selenium, vitamins C and E; choose nutrient-dense foods. Antioxidants such as beta-cryptoxanthin (as from one glass of freshly squeezed orange juice daily) can reduce the risk of developing RA (Pattison et al, 2005).
- Increase vitamin D intakes to decrease the incidence and severity of RA. Provide adequate intake of calcium, magnesium, B-complex vitamins, potassium, and zinc.
- Increase folic acid if methotrexate is used; enhance diet or encourage folic acid supplements.
- Provide meals that are easy to tolerate when the drugs being used cause gastric irritation. Avoid acidic or highly spiced foods if needed.
- With dysphagia, tube feed or use soft/thick, pureed foods as needed.
- Identify and eliminate any food allergens. Individualize the diet accordingly.

## Common Drugs Used and Potential Side Effects

- With biologic therapies, such as TNF inhibitors, many patients with RA have seen significant improvement in symptoms, function, and quality of life (Barton et al, 2009). See Table 11-14.

**TABLE 11-14 Medications Used in Rheumatoid Arthritis**

Medications	Uses/Effects	Side Effects	Monitoring
Analgesics and nonsteroidal anti-inflammatory drugs (NSAIDs)	Analgesics relieve pain; NSAIDs relieve pain and reduce inflammation.	Upset stomach, peptic ulcer, bleeding, renal failure. Use of NSAIDs may increase rate of miscarriage for pregnant women.	For all traditional NSAIDs: avoid drinking alcohol or using blood thinners; avoid if there is sensitivity or allergy to aspirin or similar drugs, kidney or liver disease, heart disease, high blood pressure, asthma, or peptic ulcers.
Acetaminophen		Usually no side effects when taken as directed.	Not to be taken with alcohol or with other products containing acetaminophen. Not to be used for more than 10 days unless directed by a physician.
Aspirin: buffered, plain	Aspirin is used to reduce pain, swelling, and inflammation, allowing patients to move more easily and carry out normal activities. It is generally part of early and ongoing therapy.	Upset stomach; tendency to bruise easily; ulcers, pain, or discomfort; diarrhea; headache; heartburn or indigestion; nausea or vomiting.	Doctor monitoring is needed. Not used for children in whom Reye's syndrome is a risk, but otherwise useful in lessening inflammation.
Traditional NSAIDs: ibuprofen, ketoprofen, naproxen	NSAIDs help relieve pain within hours of administration in dosages available over the counter (available for all three medications). They relieve pain and inflammation in dosages available in prescription form (ibuprofen and ketoprofen). It may take several days to reduce inflammation.	For all traditional NSAIDs: abdominal or stomach cramps, pain, or discomfort; diarrhea; dizziness; drowsiness or light-headedness; headache; heartburn or indigestion; peptic ulcers; nausea or vomiting; possible kidney and liver damage (rare).	For all traditional NSAIDs: avoid drinking alcohol or using blood thinners; avoid if there is sensitivity or allergy to aspirin or similar drugs, kidney or liver disease, heart disease, high blood pressure, asthma, or peptic ulcers.
Cyclo-oxygenase (COX)-2 inhibitor NSAIDs: celecoxib, valdecoxib	COX-2 inhibitors, such as traditional NSAIDs, block COX-2, an enzyme in the body that stimulates an inflammatory response. Unlike traditional NSAIDs, however, they do not block the action of COX-1, an enzyme that protects the stomach lining. Vioxx was withdrawn by FDA.	Stomach irritation, ulceration, and bleeding may occur. Caution is advisable for patients with a history of bleeding or ulcers, decreased renal function, hepatic disease, hypertension, or asthma.	Doctor monitoring for possible allergic responses to valdecoxib and celecoxib is important.

(continued)

**TABLE 11-14 Medications Used in Rheumatoid Arthritis (continued)**

Medications	Uses/Effects	Side Effects	Monitoring
Corticosteroids	These are steroids given by mouth or injection. They are used to relieve inflammation and reduce swelling, redness, itching, and allergic reactions.	Increased appetite, indigestion, nervousness, or restlessness.	For all corticosteroids, advise the doctor if there is presence of the following: fungal infection, history of tuberculosis, underactive thyroid, herpes simplex of the eye, high blood pressure, osteoporosis, or stomach ulcer.
Methylprednisolone, prednisone	These steroids are available in a pill form or as an injection into a joint. Improvements are seen up to 24 hours after administration. There is potential for serious side effects, especially at high doses. They are used for severe flares or when the disease does not respond to NSAIDs and disease-modifying antirheumatic drugs.	Osteoporosis, mood changes, fragile skin, easy bruising, fluid retention, weight gain, muscle weakness, onset or worsening of diabetes, cataracts, increased risk of infection, and hypertension.	Doctor monitoring for continued effectiveness of medication and for side effects is needed.
Disease-modifying antirheumatic drugs (DMARDs)	These are common arthritis medications. They relieve painful, swollen joints and slow joint damage, and several DMARDs may be used over the disease course. They take a few weeks or months to have an effect and may produce significant improvements for many patients. Exactly how they work is still unknown.	Side effects vary with each medicine. DMARDs may increase risk of infection, hair loss, and kidney or liver damage.	Doctor monitoring allows the risk of toxicities to be weighed against the potential benefits of individual medications.
Azathioprine	This drug was first used in higher doses in cancer chemotherapy and organ transplantation. It is used in patients who have not responded to other drugs and in combination therapy.	Cough or hoarseness, fever or chills, loss of appetite, lower back or side pain, nausea or vomiting, painful or difficult urination, unusual tiredness or weakness.	Avoid with allopurinol or kidney or liver disease. May decrease immunity; contact doctor immediately with chills, fever, or a cough. Regular blood and liver function tests are needed.
Cyclosporine	This medication was first used in organ transplantation to prevent rejection. It is used in patients who have not responded to other drugs.	Bleeding, tender, or enlarged gums; high blood pressure; increase in hair growth; kidney problems; trembling and shaking of hands.	Avoid with sensitivity to castor oil (if receiving the drug by injection), liver or kidney disease, active infection, or high blood pressure. Using this drug may make you more susceptible to infection and certain cancers. Do not take live vaccines while on this drug. Avoid St. John's wort and echinacea.
Hydroxychloroquine	It may take several months to notice the benefits of this drug, which include reducing the signs and symptoms of rheumatoid arthritis.	Diarrhea, eye problems (rare), headache, loss of appetite, nausea or vomiting, and stomach cramps or pain.	Doctor monitoring is important, particularly with an allergy to any antimalarial drug or a retinal abnormality.
Gold sodium thiomalate (Ridaura)	This was one of the first DMARDs used to treat rheumatoid arthritis.	Redness or soreness of tongue; swelling or bleeding gums; skin rash or itching; ulcers or sores on lips, mouth, or throat; irritation on tongue. Monitor joint pain 1 or 2 days after injection.	Avoid with lupus, skin rash, kidney disease, or colitis. Periodic urine and blood tests are needed to check for side effects.
Leflunomide	This drug reduces signs and symptoms and slows structural damage to joints caused by arthritis.	Bloody or cloudy urine; congestion in chest; cough; diarrhea; difficult, burning, or painful urination or breathing; fever; hair loss; headache; heartburn; loss of appetite; nausea and/or vomiting; skin rash; stomach pain; sneezing; and sore throat.	Doctor must monitor for the following: active infection, liver disease, known immune deficiency, renal insufficiency, or underlying malignancy. Regular blood tests, including liver function tests, are needed. Leflunomide must not be taken during pregnancy; it may cause birth defects in humans.

(continued)

**TABLE 11-14 Medications Used in Rheumatoid Arthritis (continued)**

Medications	Uses/Effects	Side Effects	Monitoring
Methotrexate (Rheumatrex)	This drug can be taken by mouth or by injection and results in rapid improvement (it usually takes 3–6 weeks to begin working). It is very effective, especially in combination with infliximab or etanercept. It produces more favorable long-term responses compared with DMARDs such as sulfasalazine, gold sodium thiomalate, hydroxychloroquine and may be used in pediatrics.	Abdominal discomfort, chest pain, chills, nausea, mouth sores, painful urination, sore throat, and unusual tiredness or weakness.	Doctor monitoring is important, particularly with an abnormal blood count, liver or lung disease, alcoholism, immune system deficiency, or active infection. Methotrexate must not be taken during pregnancy because it may cause birth defects in humans. Avoid Echinacea. Extra folic acid is needed.
Sulfasalazine	This drug suppresses the immune system.	Abdominal pain, aching joints, diarrhea, headache, sensitivity to sunlight, loss of appetite, nausea or vomiting, and skin rash.	Doctor monitoring is important, particularly with allergy to sulfa drugs or aspirin or with a kidney, liver, or blood disease.
Biological response modifiers	These drugs selectively block cytokines, which play a role in inflammation. Long-term efficacy and safety are uncertain.	Increased risk of infection, especially tuberculosis. Increased risk of pneumonia, and listeriosis (a foodborne illness caused by the bacterium <i>Listeria monocytogenes</i> ).	Avoid eating undercooked foods (including unpasteurized cheeses, cold cuts, and hot dogs) to reduce listeriosis while taking biological response modifiers.
Tumor necrosis factor inhibitors: etanercept (Enbrel), golimumab (Simponi), infliximab (Remicade), and adalimumab (Humira)	Highly effective for treating patients with an inadequate response to DMARDs. Often prescribed in combination with methotrexate. Etanercept requires subcutaneous injections twice weekly. Infliximab is taken intravenously (IV) during a 2-hour procedure, along with methotrexate. Adalimumab requires injections every 2 weeks.	<i>Etanercept</i> : pain or burning in throat, redness, itching, pain, and/or swelling at injection site, runny or stuffy nose. <i>Infliximab</i> : abdominal pain, cough, dizziness, fainting, headache, muscle pain, runny nose, shortness of breath, sore throat, vomiting, wheezing. <i>Adalimumab</i> : redness, rash, swelling, itching, bruising, sinus infection, headache, nausea. <i>Golimumab</i> : respiratory infection, sore throat and nasal congestion.	Doctor monitoring is important, particularly with active infection, exposure to tuberculosis, or a central nervous system disorder. Evaluation for tuberculosis is necessary before treatment begins.
Interleukin-1 inhibitor: anakinra (Kineret)	This medication requires daily injections. Long-term efficacy and safety are uncertain.	Redness, swelling, bruising, or pain at the site of injection; headache; upset stomach; diarrhea; runny nose; and stomach pain.	Doctor monitoring is required.
Selective Costimulation Modulator: Abatacept	Abatacept is given intravenously in a 30-minute infusion. It may be given alone or with DMARDs.	Cough, dizziness, headache, infections, sore throat.	Doctor monitoring is needed.
CD20 Antibody: Rituximab	This medication is for people whose rheumatoid arthritis has not responded to other biologic agents. It is given by two IV infusions 2 weeks apart. It is given with methotrexate.	Abdominal pain, chills/shivering, fever, headache, infection, itching.	Doctor monitoring is needed.
Other medications	Pilocarpine hydrochloride (Salagen) and cevimeline (Evoxac).	Available to treat dry mouth associated with Sjögren's syndrome. They simulate the salivary glands.	

Adapted from: National Institutes of Health. Health topics. Accessed December 19, 2009 at [http://www.niams.nih.gov/Health\\_Info/Rheumatic\\_Disease/default.asp#ra\\_16](http://www.niams.nih.gov/Health_Info/Rheumatic_Disease/default.asp#ra_16).

## Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician. Some people have tried acupuncture and other alternatives to traditional medicine, but it is important not to neglect regular health care or treatment of serious symptoms. Female patients tend to use alternative treatments for RA more than males; psychosocial intervention may be beneficial.
- With borage oil, concomitant NSAID use may undermine the effects. Borage oil is contraindicated in pregnancy given the teratogenic and labor-inducing effects of prostaglandin E agonists.
- St. John's wort and echinacea should not be used with cyclosporine or methotrexate.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Adoption of a Mediterranean diet confers health benefits in this population because of greater consumption of fruits and vegetables, lower consumption of animal products, and use of olive oil, which modulates immune function (Wahle et al, 2005). Inclusion of omega-3 fatty acids is also important (Berbert et al, 2005); herring, salmon, sardines, tuna, and mackerel are good dietary sources.
- No evidence exists to prove that foods from the nightshade family (potatoes, tomatoes, eggplant, and sweet and hot peppers) should be excluded.
- Encourage nutrient-dense foods. If intake is poor, a vitamin–mineral supplement may be needed. Dietary quinones, phenolics, vitamins, amino acids, isoprenoids, and other compounds in functional foods have become very popular (Losso and Bawadi, 2005).
- Instruct patient about simplified planning and preparation tips. Sandwiches, prepared meals, precut fruits and vegetables are easy to use. Cook double portions and freeze leftovers for another day.
- Discourage quackery and substitute sound health practices.
- Carbohydrate intolerance occurs because of chronic inflammation and use of steroids; planning must reflect individual needs.
- A support group may be helpful for coping.
- Physical therapy and exercise are beneficial for most patients. Strengthening exercises may help improve patient's ability to walk and may decrease joint pain and fatigue. Dynamic exercise is beneficial in RA (Hurkmans et al, 2009).
- Check on bone density; there is a high incidence of osteoporosis when steroids are used.

## Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

### For More Information

- American Autoimmune Related Diseases Association  
<http://www.aarda.org>
- American College of Rheumatology  
<http://www.rheumatology.org>
- Arthritis Foundation  
<http://www.arthritis.org>
- Felty Syndrome  
<http://rarediseases.about.com/od/rarediseasesf/a/121104.htm>
- Information on Rheumatoid Arthritis  
<http://www.niams.nih.gov/hi/topics/arthritis/rahandout.htm>
- Juvenile Rheumatoid Arthritis  
[http://www.niams.nih.gov/hi/topics/juvenile\\_arthritis/juvarthr.htm](http://www.niams.nih.gov/hi/topics/juvenile_arthritis/juvarthr.htm)
- National Institute of Dental and Craniofacial Research—Sjögren's Syndrome  
[http://www.nidcr.nih.gov/GrantsAndFunding/See\\_Funding\\_Opportunities\\_Sorted\\_By/ConceptClearance/CurrentCC/SjogrenSynd.htm](http://www.nidcr.nih.gov/GrantsAndFunding/See_Funding_Opportunities_Sorted_By/ConceptClearance/CurrentCC/SjogrenSynd.htm)
- National Sjögren's Syndrome Association  
<http://www.sjogrenssyndrome.org/index.html>
- Rheumatoid Vasculitis  
<http://vasculitis.med.jhu.edu/typesof/rheumatoid.html>
- Sjögren's Syndrome Foundation—Food Tips  
<http://www.sjogrens.org/home/about-sjogrens-syndrome/living-with-sjogrens/diet-a-food-tips>

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# RUPTURED DISC

## NUTRITIONAL ACUITY RANKING: LEVEL 1



### DEFINITIONS AND BACKGROUND

Determining the cause of back pain is complicated as it is often multifactorial; anatomical abnormalities are common in the spine and may not necessarily translate into clinical symptoms (Sheehan et al, 2010).

A slipped or ruptured disc is called a cervical radiculopathy, herniated intervertebral disc, lumbar radiculopathy, or prolapsed intervertebral disc. In this condition, slipping or prolapse of a cervical or lumbar disc occurs, with neck, shoulder, or low back pain accordingly. Degenerating changes in the disks begin around 30 years of age. Overweight and obesity increase the risk of low back pain and the need for medical attention (Shiri et al, 2010).

With **lumbar radiculopathy**, ambulation may be painful, and limping can occur. Muscular weakness, severe back pain that radiates to buttocks or legs and feet, pain that worsens with coughing or laughing, tingling or numbness in legs or feet, and muscle contractions or spasms may also result. With **cervical radiculopathy**, neck pain in back and sides is deep; pain may radiate to shoulders, upper arms, or forearms and worsens with coughing or laughing. Spasm of neck muscles and pain that worsens at night may occur.

A laminectomy surgically removes the lamina of a vertebra. Percutaneous automated discectomy (PAD) surgery can be performed in some cases; this surgery breaks up the disc and removes fragments. There is no convincing medical evidence to support routine use of lumbar fusion, but it may be useful in patients with associated spinal deformity, instability, or associated chronic low-back pain (Resnick et al, 2005). Surgery for radiculopathy with herniated lumbar disc and symptomatic spinal stenosis is associated with short-term benefits compared to nonsurgical therapy, though benefits diminish with long-term follow-up in some trials (Chou et al, 2009).



### ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** This condition is acquired and not genetic.

<b>Clinical/History</b>	Myelography	Na <sup>+</sup> , K <sup>+</sup>
Height	Discography	Alb, transthyretin
Weight	Spinal or	BUN, Creat
BMI	neck x-rays	Alk phos
I & O	Nerve	Gluc
BP	conduction	Vitamin D <sub>3</sub>
Constipation	velocity test	status (serum
Edema		25-OHD)

#### Lab Work

MRI or computed tomography (CT) scan	H & H Ca <sup>++</sup> , Mg <sup>++</sup>
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### SAMPLE NUTRITION CARE PROCESS STEPS

#### Abnormal GI Function

**Assessment Data:** Weight and physical activity histories. Medical history and medications.

**Nutrition Diagnosis (PES):** Abnormal GI function related to constipation and infrequent stooling pattern as evidenced by GI distress and evacuation every 3–4 days.

**Intervention:** Food-nutrient delivery—Assure intake of adequate fluid and fiber at all meals. Education—Discuss tips for alleviating constipation through use of specific foods rich in fiber (fruits, vegetables, whole grains, beans and legumes). Coordinate care—Work with nursing and physicians to determine if any medications that cause constipation can be changed, or if some type of laxative can be added.

**Monitoring and Evaluation:** Improvement in bowel habits; alleviation of constipation.

### INTERVENTION



#### OBJECTIVES

- Maintain adequate rest and activity levels, as assigned by physician.
- Prevent weight gain from decreased activity.
- Encourage adequate hydration.
- Prevent constipation and straining.
- Assist with feeding, if patient is in traction.
- Relieve pain and promote healing.



#### FOOD AND NUTRITION

- A regular diet generally is sufficient. For some, a strict energy-controlled diet may be beneficial to promote weight loss.
- Increased fluid and fiber intake can be helpful to reduce constipation. Fresh fruits and vegetables, dried beans, legumes, whole grains, bran, and other foods may be needed.

#### Common Drugs Used and Potential Side Effects

- Anti-inflammatory drugs may be used. NSAIDs are used for long-term pain control, but narcotics may be given if the pain does not respond. Nausea, GI distress, and anorexia may result. Follow directions regarding when to take (e.g., before or after meals).
- Analgesics may be helpful to relieve pain. Chronic use of aspirin may cause GI bleeding.
- Muscle relaxants may be ordered. GI distress or nausea can occur.

## Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Instruct patient regarding effective methods of relieving constipation.
- Discuss role of nutrition and exercise in health maintenance. Weight loss may be needed.
- After surgery, the role of nutrition in wound healing should be discussed.

### Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

### For More Information

- Herniated Disk  
<http://www.nlm.nih.gov/medlineplus/ency/article/000442.htm>
- Lumbar Radiculopathy  
<https://health.google.com/health/ref/Herniated+nucleus+pulposus>

## RUPTURED INTERVERTEBRAL DISC—CITED REFERENCES

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# SCLERODERMA (SYSTEMIC SCLEROSIS)

## NUTRITIONAL ACUITY RANKING: LEVEL 1–2



Adapted from: Goodheart HP, MD. *Goodheart's Photoguide of Common Skin Disorders*, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2003.



### DEFINITIONS AND BACKGROUND

Scleroderma is a chronic disease characterized by fibrosis and autoantibodies. Approximately 2% of the population in Europe and North America suffers from disorders such as scleroderma (Chen and von Mikecz, 2005). Genetic, immunological, hormonal, and environmental factors are considered to be triggers (Molina and Shoenfeld, 2005).

The diffuse form affects a large area of the skin and several organs; it is also called **systemic sclerosis** (SSc). In SSc, pathological deposition of fibrous connective tissue in the skin and visceral organs occurs. Fibrosis involves an increase of hydroxylysine aldehyde collagen cross-linkages as well as an increase in inflammatory cytokines (Brinckmann et al,

2005). The GI tract is affected, and Raynaud's syndrome (ischemia of fingers) is common.

The limited form of scleroderma affects the skin and sometimes the lungs. The CREST syndrome (**limited cutaneous sclerosis**) is less severe than SSc and causes less internal organ damage. Calcium deposits, Raynaud's phenomenon, esophageal dysfunction, skin damage on fingers, and telangiectasia form the acronym for CREST.

As the disease progresses, large areas of the skin or just the fingers (sclerodactyly) may be affected. Skin on the face tightens and causes a mask-like appearance. Spider veins (telangiectasia) occur on the fingers, chest, face, lips, or tongue. Calcium deposits can occur on the fingers or other bony areas; sores or contractures may result from the scarring. Scarring of the esophagus may be especially detrimental, causing blockage or even cancer. Lungs can be affected, leading to shortness of breath with exercise.

Neurological involvement consists of epilepsy, central nervous system vasculitis, peripheral neuropathy, vascular malformations, headache, and neuroimaging abnormalities; ocular manifestations include uveitis, xerophthalmia, glaucoma, and papilledema (Zulian et al, 2005). SSc is characterized by vasculopathy, inflammation, vasospasm, microvascular involvement is common; an increased prevalence of distal peripheral artery disease in the digits has been found (Hetterna et al, 2008).

**Scleroderma renal crisis** (SRC) occurs in 5–10% of SSc patients, who may present with an abrupt onset of hypertension, acute renal failure, headaches, fevers, malaise, hypertensive retinopathy, encephalopathy, and pulmonary edema (Denton et al, 2009). Multiple organ system dysfunction may occur. Pulmonary hypertension, heart failure, and respiratory failure cause serious morbidity and mortality. There is no known cure, and SSc can be fatal.

There seems to be an increased prevalence of celiac disease in patients with scleroderma (Rosato et al, 2009). Both disorders require careful management. Current therapies for scleroderma target the immune system, with the goal of reducing inflammation, ischemic injury to the involved organs, and secondary tissue injury and fibrosis (Hennes and Wigley, 2007).



## ASSESSMENT, MONITORING, AND EVALUATION



### CLINICAL INDICATORS

**Genetic Markers:** Genetic factors contribute to disease susceptibility; transforming growth factor- $\alpha$  is a cytokine that contributes to fibroblast activation, collagen overproduction, and pathological tissue fibrosis (Varga, 2008). T-cell polarization is implicated in the lung disease of SSc (Boin et al, 2008).

<b>Clinical/History</b>	Nausea, vomiting Diarrhea, constipation Skinfold measurements	Gluc Prothrombin time (PT) Alb, transthyretin CRP GFR BUN, Creat Homocysteine Ca <sup>++</sup> , Mg <sup>++</sup> Na <sup>+</sup> , K <sup>+</sup> Alk phos Vitamin D <sub>3</sub> status (serum 25-OHD) Fecal fat test, hydrogen breath test for malabsorption
Height		
Weight		
BMI		
I & O		
Weight loss		
Fever?		
BP		
Thickening, swelling of the ends of the fingers		
Dysphagia		
Heartburn		
Fibrosis of salivary and lacrimal glands		
Abdominal pain, flatulence		
	<b>Lab Work</b>	
	ANA (high) RF (high) LDL Cholesterol (elevated) Trig (may be low) Anti-tTG antibody Serum folate H & H Serum B <sub>12</sub>	

### SAMPLE NUTRITION CARE PROCESS STEPS

#### Difficulty Swallowing

**Assessment Data:** Weight, medical history, medications. Low salivation and difficulty swallowing.

**Nutrition Diagnosis (PES):** Difficulty swallowing related to low saliva production as evidenced by fibrosis, inability to swallow solids.

**Intervention:** Food-Nutrient Delivery—Alteration in food choices to liquefy meals and make them easier to swallow. Educate about the use of saliva substitutes, more fluids, altered food choices as needed. Counseling about when to request changes, such as tube feeding.

**Monitoring and Evaluation:** Improvement in swallowing and tolerance for meals. No weight loss.

## INTERVENTION



### OBJECTIVES

- Prevent or correct protein-energy malnutrition and nutrient deficiencies.
- Correct xerostomia where present; decreased saliva, dysphagia, and difficulty in chewing will result.
- Monitor dysphagia with esophageal involvement; alter method of feeding as needed.
- Counteract vitamin B<sub>12</sub> and fat maldigestion and absorption, which may be common.
- Monitor hypomotility and gastroparesis; alter fiber intake as appropriate. For many patients, nutritional support and relief of symptoms remain the primary management goals.
- Improve quality of life and reduce fatigue; allow return to work or maintenance of energy levels.



### FOOD AND NUTRITION

- Diets high in energy (30–40 kcal/kg) and adequate to high in protein are often necessary. A soft diet with moistened foods and extra fluids is useful. Add fiber if constipation is a problem (such as adding crushed bran to hot cereal).
- Small, frequent feedings may be needed. Tube feed if patient is dysphagic or has obstruction.
- Use parenteral nutrition if GI tract is highly affected, with intractable diarrhea and severe malabsorption.
- If there is celiac sensitivity, omit gluten from the diet.
- Reduce lactose if intolerance occurs. Extra calcium may be needed if lactose is not tolerated orally.
- Give supplements of fat- and water-soluble vitamins.
- With hypertension and multiple organ system dysfunction, reduced sodium or fluid restriction may be needed.

### Common Drugs Used and Potential Side Effects

- Topical or systemic corticosteroids, vitamin D analogs (calcitriol and calcipotriol), photochemotherapy, laser therapy, antimalarials, phenytoin, D-penicillamine, and colchicine all have varying degrees of success. Topical tacrolimus cream is an immunosuppressive antibiotic.
- Interstitial lung disease can be treated with cyclophosphamide, vascular disease of the lungs and digits with endothelin receptor antagonists, and general symptoms with phosphodiesterase inhibitor sildenafil or prostacyclins (Hennes and Wigley, 2007).
- Early, aggressive treatment with angiotensin-converting enzyme inhibitors helps with a renal crisis (Denton et al, 2009).
- Anti-inflammatory agents, such as steroids, are often used in SSc. Monitor for nitrogen and calcium losses, altered electrolyte levels, and elevated glucose levels. Correct diet accordingly.
- Antihypertensives usually are needed; monitor BP results. Potassium supplements may or may not be required; determine need according to medication selected.
- Trental (pentoxifylline) is used for Raynaud's syndrome to improve circulation. Anorexia or GI distress may result.

## Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- For Raynaud's disease, evening primrose, ginkgo, mustard, garlic, borage, and red pepper have been suggested, but there are no clinical trials that prove effectiveness.
- CAM is frequently used to treat stress-related disorders such as scleroderma; some merit can be noted (Hui et al, 2009).



### NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Artificial saliva (Xero-Lube) or lemon glycerine may be useful.
- Chew sugarless gum.
- If eating orally, adequate chewing time will be required.
- Consume adequate fluids. Choose moist foods or foods with sauces/gravies.
- For heartburn, keep head elevated after meals; decrease or limit intake of chocolate, caffeine, fatty foods, alcohol, citrus, and tomatoes.
- Physical therapy and exercise may help maintain muscle strength but cannot totally prevent joints from locking into stiffened positions.

#### Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

#### For More Information

- Scleroderma Foundation  
<http://www.scleroderma.org/>
- Scleroderma Research Foundation  
<http://www.srfcure.org>

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