Medical Nutrition Therapy in Arthritis Diseases

Hadi Abdollahzad
Professor of Nutrition
Urmia University of Medical Sciences

- Rheumatoid arthritis
- Sjögren Syndrome
- Systemic Lupus Erythematosus

Rheumatoid arthritis

Medical Nutrition Therapy (MNT)

- A comprehensive **nutrition assessment** of individuals with RA is essential, with a review of systems to determine the systemic effects of the disease process.
- Current weight and history of weight change over time are the least expensive, least invasive, and most reliable assessment tools to use.
- **Weight change** is an important measure of RA severity.
- The characteristic progression of **malnutrition** in RA is attributed to excessive **protein catabolism** evoked by:
  - inflammatory **cytokines**
  - disuse **atrophy** resulting from functional impairment

MNT

- The **diet history** should review:
  - the usual diet
  - the effect of the handicap
  - types of food consumed
  - changes in food tolerance secondary to oral, esophageal, and intestinal disorders.
- The **effect of the disease** on food shopping and preparation, self-feeding ability, appetite, and intake also must be assessed.
- The **use of elimination or other diets** purported to treat or cure arthritis should be identified.

MNT

- Articular and extraarticular manifestations of RA affect the **nutrition status** of individuals in several ways:
- Articular involvement of the small and large **joints** may limit the ability to perform nutrition-related **ADLs,** including **shopping** for, **preparing,** and **eating** food.
- Involvement of the **temporomandibular** joint can affect the ability to chew and swallow and may necessitate changes in **diet consistency**.

MNT

- Extraarticular manifestations include:
  - increased metabolic rate secondary to:
    - the inflammatory process, SS, and changes in the **gastrointestinal mucosa**.

1. **Taste alterations** secondary to xerostomia and dryness of the nasal mucosa
2. **dysphagia** secondary to pharyngeal and esophageal dryness
3. **anorexia** secondary to medications, fatigue, and pain

- may reduce dietary intake.

MNT

- The association of **foods** with disease **flares** should be discussed.
- **Dietary manipulation** by either:
  - modifying food **composition** or
  - **reducing body weight** may give some clinical benefit in improving RA symptoms.
- A **vegan, gluten-free** diet causes improvement in some patients, possibly because of the reduction of immunoreactivity to food antigens.
- Identification of possible food allergies and use of an elimination diet may be useful.

MNT

- **Fasting** reduces:
  1. (its role in adaptive cellular responses) **oxidative damage and inflammation.**
  2. **hypertension, asthma,** and symptoms of RA.
- Thus fasting has the potential to:
  - **delay aging**
  - prevent and treat diseases
  - while minimizing the side effects caused by **medications.**
- **Intermittent fasting** during the acute phase of RA:
  - may provide some **pain relief.**

MNT

- The **antiinflammatory diet** similar Mediterranean-style eating plan includes foods that almost everyone should aim to consume on a daily basis, such as:
  - moderate amounts of lean meat
  - unsaturated fats instead of saturated fats
  - plenty of fruits and vegetables, and fish.
- These diets are also nutritionally adequate and cover all of the food groups.

Energy

- There are 3 unique aspects of energy metabolism in RA:

1. **elevated REE**. RA causes cachexia, a metabolic response characterized by loss of muscle mass and elevated resting energy expenditure.
2. elevated **whole-body protein catabolism**, a destructive form of muscle metabolism that translates to muscle wasting.
3. **low body cell mass**, which leads to increased fat mass.

Energy

- People with RA tend to be **less active** than people without it;
- the stiffness and swelling caused by inflammation naturally prompt them to pursue less physical, more sedentary lifestyles.
- Such habits lead in turn to overall **gains in fat mass.**
- Being overweight puts **an extra burden on weight-bearing joints** when they are already damaged or under strain.

Energy

- **TEE** is significantly lower in RA patients.
- Energy requirements should be adjusted according to:
  - the **weight** and
  - **activity level** of the individual.
- People with RA should:
  - **consume nutrient-rich diets**
  - incorporate **physical activity** throughout the day to boost their total energy expenditure (This helps maintain a healthy weight).

Protein

- Protein requirements for individuals who are:
  - poorly nourished or
  - in the inflammatory phase of the disease
    - are 1.5 to 2.0 g protein/kg body weight.
- Well-nourished individuals do not have increased requirements.

Fat

- **less than 30%** of the total energy intake
- The **type of fat** included in the diet is important:
  - an increase in the amount of **omega-3** fatty acids reduce inflammation in RA
    - **fish oils**
    - **alpha-linolenic acid** found in:
      - flaxseed, soybean oils, and green leaves
- The antiinflammatory diet with higher omega-3 fatty acids for RA patients can:
  - reduce inflammatory activity
  - increase physical function
  - improve vitality

Fat

- There is evidence of a fairly consistent, but modest, benefit of **marine omega-3 PUFAs** on:
  - joint swelling
  - pain and duration of morning stiffness.
- **Fish oil** at a high dose (**3.5 g/day**) has been shown to have additional benefits.
- Some other oils of marine origin and a range of vegetable oils (**olive** and **evening primrose** oil) have indirect antiinflammatory actions probably mediated via **PGE1**.
- The beneficial effects are generally delayed for up to 12 weeks after they are started, but last up to 6 weeks after discontinuing therapy.

Minerals, Vitamins, and Antioxidants

- Several vitamins and minerals function as **antioxidants** and therefore affect inflammation.
- **Vitamin E** is just such a vitamin, and along with **omega-3** and **omega-6**, may affect cytokine and eicosanoid production by decreasing **proinflammatory cytokines**.
- **Synovial fluid and plasma trace element concentrations, excluding Zn**, change in inflammatory RA. Altered trace element concentrations may result from changes of the immunoregulatory **cytokines**.

Minerals, Vitamins, and Antioxidants

- RA patients often have nutritional intakes below the **DRIs** for **folic acid, Ca, vitamin D, E, B, zinc,** and **Se.**
- In addition, the commonly used drug **MTX** is known to decrease serum **folate** levels with the result of elevated homocysteine levels. Low serum level of pyridoxal-5-phosphate correlates with increased markers of inflammation and continuous use of **NSAID** (**Naproxen**) also impairs **pyridoxine** metabolism by a mechanism related to COX inhibition.
- Thus, in these patients, adequate intakes of **folate** and vitamins **B6** and **B12** should be encouraged.

Minerals, Vitamins, and Antioxidants

- **Calcium** and **vitamin D malabsorption** and bone demineralization are characteristic of advanced stages of the disease, leading to osteoporosis or fractures.
- Prolonged use of **glucocorticoids** also can lead to **osteoporosis.**
- Therefore **supplementation with calcium and vitamin D** should be considered.

Vitamin D

- Vitamin D deficiency:
  - autoimmune diseases including DM1, MS, Crohn, dementia, CVDs, rheumatic disorders (SLE, RA).
- Vitamin D:
  - suppressing Th17 and Th1 cells (proinflammatory)
  - giving way to Th2 and Treg cells (antiinflammatory)
- As most studies indicate that vitamin D **insufficiency** is associated with **high disease activity of RA**, it would be logical to supplement RA patients with vitamin D.

Vitamin D

- Supplementation of **500 IU calcitriol daily** given to previously DMARD patients with early RA along with triple DMARD therapy resulted in significant higher **pain relief** (50% vs. 30%) at the end of 3 months, compared with patients treated with triple DMARD and calcium.
- Most clinicians agree that with the increasing adverse health outcomes associated with hypovitaminosis D, **supplementation** should be performed routinely in RA patients.

Minerals, Vitamins, and Antioxidants

- Elevated levels of **copper** and **ceruloplasmin** in serum and joint fluid are seen in RA.
- Plasma copper levels correlate with the degree of **joint inflammation**, decreasing as the inflammation is diminished.
- Elevated plasma levels of **ceruloplasmin**, the carrier protein for copper, may have a **protective role because of its antioxidant activity.**

ANTIINFLAMMATORY DIET

- The **antiinflammatory diet**, a diet resembling the Mediterranean diet, has been useful for the treatment of inflammatory diseases, including RA.
- The diet aims for the inclusion of:
  - as much fresh food as possible
  - the least amount of processed foods and fast food
  - minimal amounts of sugar, particularly fructose and sucrose
  - an abundance of: fruits (especially berries) and vegetables, lean proteins, from animal sources such as chicken and fish, and vegetarian sources such as legumes and nuts, essential FAs, and dietary fiber.

ANTIINFLAMMATORY DIET

- Although this diet is not intended for weight loss, people often **lose weight** on it.
- Changing the dietary habits to a Mediterranean diet reduces the **inflammatory** status in **healthy persons**, as well as in patients with obesity, cardiovascular disease, and Crohn's disease.
- The antiinflammatory diet works by reducing the expression of genes involved in the inflammatory process such as **IL-1**, **IL-6**, and **TNF-a**.

COMPLEMENTARY OR INTEGRATIVE THERAPIES

- Because of:
  - the **chronic** nature of arthritic diseases,
  - their effects on quality of life,
  - and the fact that most treatments result only in **modest improvement** in symptoms and function,
  - patients commonly try alternative methods of treatment.
- Favorable effects of self-help treatments are often reported **anecdotally**, but usually no **cause-and-effect** relationships are documented.
- with unlimited access to the Internet, patients have greater exposure to remedies and controversial treatments. In a recent survey, rheumatologists (US) showed a favorable opinion toward **CAM** for patients with rheumatic diseases.

COMPLEMENTARY OR INTEGRATIVE THERAPIES

- The terms CAM refer to health-related products and practices that **are not** generally considered part of **conventional** medicine.
- **Complementary** medicine is used together with conventional medicine, and **alternative** medicine is used in place of conventional medicine.
- Therapies vary from aromatherapy to reflexology, herbal, natural products and supplements, physical activities, body work, and many more.

COMPLEMENTARY OR INTEGRATIVE THERAPIES

- Natural products encompass herbs (botanicals), vitamins and minerals, and probiotics.
- They are widely marketed, available to consumers, and often sold as "dietary supplements," including:
  - fish oil, extra virgin olive oil
  - curcumin, turmeric
  - boswellia, comfrey
  - alfalfa, cat's claw, thunder god vine, CoQ-10

- **Capsaicin**, the compound responsible for the burning sensation produced by chili peppers, is used as a rubbing topical gel to relieve pain, particularly in joints of OA and RA patients.

Elimination Diets

- Many people have the belief that certain foods may contain harmful substances that can worsen their arthritis symptoms. One theory is that they are having an allergic reaction to the food.
- According to the NIH, the **nightshade elimination diet** for arthritis management is considered CIM.
- It is believed that nightshade plants aggravate the **inflammation** that causes pain, swelling, and stiffness in the joints of some patients with arthritis.

Elimination Diets

- Nightshades are a diverse group of foods, herbs, shrubs, and trees that include more than 2800 species of plants of the *Solanaceae* family, such as **potatoes, tomatoes, sweet and hot peppers,** and **eggplants.**
- They contain a group of chemicals termed alkaloids, like **solanine** and **chaconine,** which are believed to cause damage to the joints and increase the loss of calcium from the bones.
- The nightshade elimination diet is believed to be safe, but there is always the risk that when eliminating certain foods from their diet, arthritis patients may not get enough of the necessary nutrients (vitamins, minerals, antioxidants).

MICROBIOTA AND ARTHRITIS

- **Increased levels of antibodies** directed against antigens of certain species of gut bacteria point to the relationship between bacteria and arthritis.
- **Mechanisms** through which the microbiota may be involved in the pathogenesis of rheumatic diseases include:
  - altered epithelial and mucosal permeability
  - loss of immune tolerance to components of the indigenous microbiota
  - trafficking of activated immune cells and antigenic material to the joints

MICROBIOTA AND ARTHRITIS

- In 2007, *Prevotella copri* was recovered from human feces.
- *P. copri* is an obligate anaerobic, nonmotile, gram-negative rod.
- The importance of this discovery lies in that *P. copri* has been recovered in:
  - 75% of patients with new-onset untreated RA (NORA)
  - only in 21.4% of healthy individuals

MICROBIOTA AND ARTHRITIS

- Potential future therapeutic **approaches** may include **modification of the microbiota** through the use of **probiotics** or **prebiotics.**

Complementary and Integrative Therapies

- The increasing popularity of the use of **complementary** treatments appears to be particularly evident with people afflicted with RA. **Herbal therapy** is popular; however, concerns of **toxicity** must also be addressed because the Food and Drug Administration (FDA) provides relatively little regulation of herbal therapies.

Complementary and Integrative Therapies

- **Gamma-linolenic acid (GLA)** is an omega-6 fatty acid found in the oils of:
  - black currant, borage, and evening primrose
  - that can be converted into the antiinflammatory PGE1.
- This antiinflammatory PGE1 may relieve:
  - pain, morning stiffness, and joint tenderness
  - with no serious side effects.

BOX 39-2 The Antiinflammatory Diet

General principles: Aims for variety, with plenty of fresh food, the least amount of processed foods and "fast foods," and abundant fruits and vegetables

Includes plenty of fruits and vegetables, except onions and potatoes, which contain the alkaloid solanine

Low in saturated fat and devoid of trans fats

Low in omega-6 fats, such as vegetable oils and animal fat

High in omega-3 PUFAs such as those found in olive oil, flax, walnuts, pumpkin seeds, and fatty cold-water oily fish such as salmon, sardines, mackerel, and herring. Other healthy oils include grapeseed, walnut, and canola.

Low in refined carbohydrates such as pasta, white bread, white rice, and other refined grains, and sucrose (table sugar) and sucrose-containing products such as pastries, cookies, cakes, energy bars, and candy

Favors intake of whole grains such as brown rice, bulgur wheat, and other unrefined grains such as amaranth, quinoa, and spelt.

Includes lean protein sources such as chicken and fish

Low in eggs, red meat, butter, and other full-fat dairy products

Low in refined and processed foods

Includes spices such as ginger, curry, turmeric, and rosemary, which have antiinflammatory effects

Includes good sources of phytonutrients: fruits and vegetables of all bright and dark colors, especially berries, tomatoes, orange and yellow fruits, and dark leafy greens; cruciferous vegetables (cabbage, broccoli, brussels sprouts, cauliflower); soy foods, tea (especially white, green or oolong), dark plain chocolate in moderation

Additionally, weight should be maintained within healthy parameters, and exercise should be included.

Sjögren Syndrome

Sjögren Syndrome

- SS is a chronic autoimmune inflammatory disease that affects the exocrine glands, particularly:
  - the salivary (dryness of the mouth, xerostomia)
  - the lacrimal glands (dryness of the eyes, xerophthalmia)
- SS can present:
  - alone (primary SS) or
  - secondary as a result of a previous rheumatic disorder (commonly RA or SLE).
- It mainly affects middle-age women, with a female/male ratio of 9:1.

- Common oral signs include:
  - Thirst
  - Burning sensation in the oral mucosa
  - Inflammation of the tongue (glossitis) and lips (cheilitis)
  - Cracking of the corners of the lips (cheilosis)
  - Difficulties in chewing and swallowing (dysphagia)
  - Severe dental caries
  - Oral infections (candidiasis)
  - Progressive dental decay
  - Nocturnal oral discomfort

- Patients may also suffer from extraglandular disorders affecting:
  - Skin
  - Lung
  - Kidney
  - Nerve
  - Connective tissue
  - Digestive system
- SS patients also may develop:
  - Dysosmia
  - Dysgeusia

- Digestive involvement is frequent in Sjögren patients, mainly in the form of autoimmune disorders:
  - Chronic atrophic *gastritis*
  - *Esophageal* motility dysfunction
  - Lymphocytic co*litis*
  - Primary biliary *cholangitis*
  - Autoimmune *hepatitis*
  - *Pancreatic* involvement
  - Co*eliac* disease

Pathophysiology

- Although the pathogenesis of SS remains elusive, environmental, genetic, and hormonal contributors seem to be involved.
  - Most lymphocytes infiltrating the salivary glands are CD4+ T cells.
  - Increased levels of IL-1 and IL-6 in the saliva of SS patients suggest that a Th1 response participates in the pathogenesis of the disease.
  - A recent metaanalysis reported an increase in IL-17 in serum and saliva of primary SS (pSS) patients which also was associated with the severity of the symptoms.

- Although T cells were originally considered to play the initiating role in SS, whereas B cells were restricted to autoantibody production, it has been shown that B cells play a central role in the development of the disease.
- A distinctive hallmark of SS is:
  - the presence of ***anti-Ro/SSA*** and ***anti-La/SSB*** autoantibodies.
  - Interestingly, anti-Ro/SSA may be found either solely or concomitantly with anti-La/SSB antibodies, whereas exclusive anti-La/SSB positivity is rare.

- Recent studies indicate that dysbiosis may play a role in SS pathogenesis.
  - The cause-effect direction is not clear yet because the dysfunction of salivary glands induces alterations in the microbiome which is linked to worsening of symptoms and disease severity.
- Although this information is preliminary:
  - dietary patterns that support a **healthy microbiome** including
    - a **plant-based fiber-rich diet** may be a reasonable.

Medical Management

- The therapeutic management of SS is based on:
  - symptomatic treatment of glandular manifestations
  - the use of DMARD for systemic involvement

- Symptomatic treatment has beneficial effects on oral and ocular dryness prevents further complications such as:
  - oral candidiasis
  - periodontal disease
  - corneal ulceration and perforation

- Topical treatment of xerostomia:
  - avoiding irritants such as:
    - caffeine, alcohol, and tobacco, followed by appropriate hydration (small sips of water)
  - the use of saliva substitutes
  - lubricating gels
  - mouth rinses
  - chewing gum
  - Lozenges
  - oils

- Any of these treatments is effective in the short term, but none improves saliva production.
- After consumption of *sugary foods or beverages*, the teeth should be *brushed* and *rinsed* with water immediately to prevent dental caries.

- Topical treatment for xerophthalmia:
  - Avoiding:
    - dry, smoky, or windy environments
    - prolonged reading or computer use
  - Artificial tears can be used
- The muscarinic receptor agonists:
  - pilocarpine (Salagen)
  - cevimeline (Evoxac)
    - may be used for the treatment of dry mouth and dry eyes only in patients with residual gland function.

- B-cell depletion therapy with rituximab improves:
  - the stimulated whole saliva flow rate
  - lacrimal gland function
  - RF levels
  - extraglandular manifestations (arthritis, skin vasculitis)
  - Fatigue
  - quality of life
    - indicating a major role of B cells in the pathogenesis of SS.

- Both IL-1 and TNFα play a major role in the development of SS.
  - Blocking IL-1 (anakinra) is beneficial in the treatment of SS
  - whereas blocking TNF-α (etanercept) is ineffective
- Antimalarials (*hydroxychloroquine*), besides improving *salivary flow*, can help SS patients with arthromyalgia.
- The use of *corticosteroids* may be used in patients with *extraglandular* manifestations.

Medical Nutrition Therapy

- The first goal of dietary management for patients with SS is to help them:
  - relieve their oral symptoms
  - reduce the eating discomfort derived from the difficulty with chewing and swallowing.
- Very often SS patients modify their dietary habits on a "trial and error" basis to cope with their oral symptoms:
  - particularly to improve:
    - biting
    - chewing
    - swallowing

Medical Nutrition Therapy

- Biting
  - cutting fruits, vegetables, and meats in small pieces
- Chewing
  - making foods softer by preparing them as soups, broths, casseroles, or as tender cooked vegetables and meats
- Swallowing
  - moistening foods with sauces, gravies, yogurts, or salad dressings

Medical Nutrition Therapy

- Foods that worsen oral symptoms should be limited, such as:
  - citrus fruits
  - irritating, hot, or spicy foods
  - alcohol

- Malnutrition or weight loss are not common
- Deficiencies of several nutrients are common. These include:
  - vitamin D
  - vitamin B6
  - vitamin B12
  - Folate
  - iron
    - which can be corrected easily by proper nutritional counseling or supplementation.

- The beneficial effects of **probiotic** supplementation in RA patients might also benefit patients with SS and other autoimmune conditions:
  - to shift their microbiome away from a disease-promoting and proinflammatory pattern.
- Higher adherence to the **Mediterranean diet** was associated with a lower likelihood of developing SS
  - **fish intake** was the Mediterranean diet domain most strongly associated with lower likelihood of SS.

Systemic Lupus Erythematosus (SLE)

Systemic Lupus Erythematosus

- SLE is known commonly as *lupus*.
- SLE is a chronic autoimmune disease characterized by production of autoantibodies directed against nuclear (anti-dsDNA, anti-SM, anti-nRNP) and cytoplasmic antigens affecting several organs and tissues.

SYSTEMIC LUPUS ERYTHEMATOSUS

- SLE is most prevalent in women of childbearing age with a female to male ratio of 9:1
- SLE is more common in African Americans and women of Hispanic, Asian, and Native American descent than in Caucasians.
- In the United States, SLE is among the top 20 leading causes of death in females between 5 and 64 years of age.

Pathophysiology

- The cause of SLE is multifactorial and involves multiple genes and environmental factors such as infections, hormones, and drugs.
- In SLE, circulating antinuclear antibodies (anti-dsDNA, anti-Sm, anti-RNP) and others (anticardiolipin) can deposit in several tissues.
- The production of cytokines such as type 1 IFN *activates B and T* cells and propagates the signal to produce more IFN by DC. This up-regulation of the "type 1-IFN pathway" is critical in the severity and progression of SLE.

- Common symptoms include:
  - extreme fatigue
  - painful or swollen joints
  - muscle pain
  - sensitivity to the sun
  - unexplained fever
  - skin rashes most commonly on the face
  - mouth ulcers
  - pale or purple fingers or toes from cold or stress (Raynaud phenomenon)
  - kidney insufficiency

Medical Management

- General treatment of SLE includes:
  - sun protection
  - diet and nutrition
  - smoking cessation
  - Exercise
- whereas organ-specific treatments include use of:
  - steroids, NSAID, DMARD, and biologics.
- Pharmacologic treatment includes the *cytotoxic agents* cyclophosphamide and azathioprine; their combination with corticosteroids must be employed early if there is major organ involvement to prevent or minimize irreversible damage.

- The **hallmark of SLE** is B-cell activation and the production of harmful autoantibodies.
  - Therefore B-cell depletion
  - cytokine or Treg cells targeted therapies,
  - alone or combined with cytotoxic drugs, have been used to treat SLE.
- There are more than 20 biologic therapies used for SLE. They have different immune targets, such as B cells, T cells, and myeloid cells and their cytokines, known to contribute to lupus pathogenesis.

- Hydroxychloroquine (Plaquenil) has potential benefits for dermatological manifestations
- some of the DMARDs used to control SLE manifestations are:
  - Steroid hormones
  - Azathioprine
  - MTX
  - mycophenolate mofetil

Medical Nutrition Therapy

- Because *kidney insufficiency* is common in SLE:
  - total **protein** intake may need to be reduced
- SLE patients tend to have:
  - higher consumption of carbohydrates
  - low intake of dietary fiber and ω-3 (EPA and DHA) and ω-6 fatty acids. The latter has been negatively associated with increased disease activity, altered serum lipid profiles, and increased carotid plaque presence.
- In addition, SLE patients often have:
  - inadequate intakes of calcium, fruits, and vegetables
  - high consumption of oils and fats

- low levels of vitamin D in SLE due to:
  - Photosensitivity, sunlight avoidance, the use of sun protection
  - low dietary intake
  - medications prescribed to treat the symptoms of the disease
  - Decreased conversion of 25-hydroxyvitamin D to its active form, 1,25-dihydroxyvitamin D (calcitriol), is possible because of renal impairment common in SLE

- **Vitamin D deficiency** has been associated with higher ANA levels in:
  - healthy subjects
  - treatment-naive SLE patients
    - suggesting it might be a trigger for autoantibody production.
- **Vitamin D supplementation** may be beneficial to patients with high anti-dsDNA positivity, possibly **reducing clinical flares.**

- Vitamin D supplementation in patients with SLE is recommended because increased vitamin D levels seem to:
  - ameliorate inflammatory and blood markers
  - show a tendency toward subsequent clinical improvement.
- General international recommendations have established that vitamin D supplementation with
  - 800 to 1000 IU/d
  - or 50,000 IU monthly
    - is safe for most individuals and can ensure levels of vitamin D within the optimal range.

- **Vitamin A** has beneficial effects, alone or in combination with low-dose immunosuppressive drugs, in lupus nephritis and cytokine modulation in SLE.
- The most likely mechanism of action for vitamin A in SLE is via:
  - IL-17 and TGF-β **cytokine regulation**, and possibly others like IL-6.

- An adequate intake of **dietary fiber** is recommended in SLE because of the beneficial effects of fiber in:
  - decreasing cardiovascular risk
  - promoting gut mobility
  - reducing serum levels of:
    - inflammation markers such as CRP, cytokines
    - homocysteine

Complementary Integrative Therapies

- More than an estimated 50% of patients with SLE have used CIM to reduce symptoms and manage their health.
- Supplements of N-acetyl cysteine and turmeric reduce SLE activity and,
- together with mindbody methods (cognitive-behavioral therapy and other counseling interventions), improve mood and quality of life of SLE patients.

- A small study showed short-term turmeric supplementation in patients suffering from lupus nephritis can decrease:
  - Proteinuria
  - Hematuria
  - Systolic blood pressure

Table 89.1 Arthritis Drug-Nutrient Interactions[10,21-23]

Drug	Affected Nutrient Status
Aspirin	↓folate, ↓iron, ↓vitamin C
Salicylate	↓folate
Nonsteroidal Anti-inflammatory Drugs	↓Iron, ↓folate
Sulfasalazine	↓folate
Methotrexate	↓folate
Corticosteroids	↓calcium, ↓vitamin D, ↓potassium, ↓zinc, ↓vitamin C, ↓magnesium, ↓folate, ↓selenium
Tetracycline	↓calcium, ↓magnesium, ↓iron
Colchicine	↓vitamin B_{12}, ↓sodium, ↓potassium
D-penicillamine	↓vitamin B_6, ↓magnesium, ↓zinc, ↓copper

Thanks for attention

Autumn
Silvana, Urmia, 1404.8.25