Rheumatology Nursing Society
Patient Education Series

HANDLING THE
HARD QUESTIONS:

What Our Patients Are Asking Us About Rheumatoid Arthritis

The Purpose of this Document

Each day, rheumatology nurses field dozens of questions from their patients with rheumatoid arthritis (RA), and they need to be able to properly and effectively communicate appropriate responses. This pocket guide includes a brief summary of evidence surrounding some of the most common—and challenging—questions today's patients with RA are asking about. We hope you find this guide useful for your professional development and that it assists you with your day-to-day patient management.

CONTENTS

Why did I get rheumatoid arthritis?

The exact cause of RA is unknown. RA appears to arise from a mix of genetic risk factors, environmental exposures, and chance. This means that RA may develop for different reasons in different patients. One patient may have a strong genetic predisposition to RA, while another patient may develop RA after environmental exposures trigger an autoimmune response. Some researchers believe that these multiple pathways of development also lead to different disease phenotypes. This may explain why some patients with RA test positive for rheumatoid factor (RF) or anti-citrullinated peptide antibodies (anti-CCP) while others do not. Compared with patients who test negative for these autoantibodies, seropositive patients tend to have a more aggressive disease course.[1]

In general, patients have little control over whether they will develop RA. The one notable exception involves cigarette smoking, which appears to amplify the risk of RA in individuals with a genetic predisposition to the disease.[2] In patients who harbor certain genetic risk factors, smoking can increase the risk of developing RA more than 20-fold compared with nonsmoking in patients without these genetic risk factors for RA.[3] Patients who smoke also have a more aggressive disease course and more difficulty responding to RA therapy compared with nonsmokers.[4] Therefore, any discussion with current smokers around the origins of RA should include education on the importance of smoking cessation.

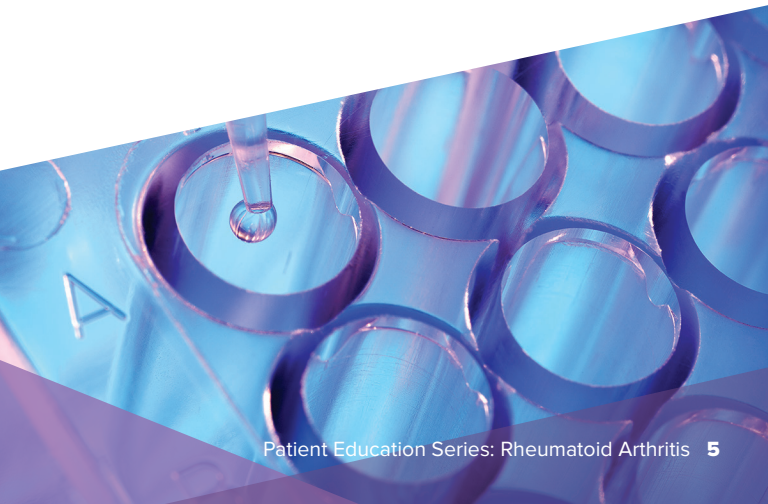
References

1. Smolen JS, Redlich K. Rheumatoid arthritis. In: The Autoimmune Diseases. Fifth Edition. Rose N, Mackay I, Eds. Elsevier, Inc; 2014.
2. Gibofsky A. Epidemiology, pathophysiology, and diagnosis of rheumatoid arthritis: a synopsis. *Am J Manag Care*. 2014;20:S128-135.
3. Sparks JA, Costenbader KH. Genetics, environment, and gene-environment interactions in the development of systemic rheumatic diseases. *Rheum Dis Clin North Am*. 2014;40(4):637-657.
4. Daïen CI, Morel J. Predictive factors of response to biological disease modifying antirheumatic drugs: towards personalized medicine. *Mediators Inflamm*. 2014;2014:386148.

Is there is a cure for rheumatoid arthritis?

Not yet. However, many patients with RA are able to achieve clinical remission with appropriate treatment. Clinical remission is defined as the absence of signs and symptoms of significant inflammatory disease activity.[1] Therefore, although patients in clinical remission will still have RA, they should be asymptomatic and experience minimal functional impairment associated with their disease.[2]

The Treat to Target (T2T) approach to RA management provides a clear framework for achieving clinical remission. Patients are likely familiar with the concept of targeting a certain blood pressure or cholesterol level to manage hypertension or hypercholesterolemia. Beginning with a defined target, the T2T algorithm guides clinicians through RA treatment initiation, monitoring, and adjust-



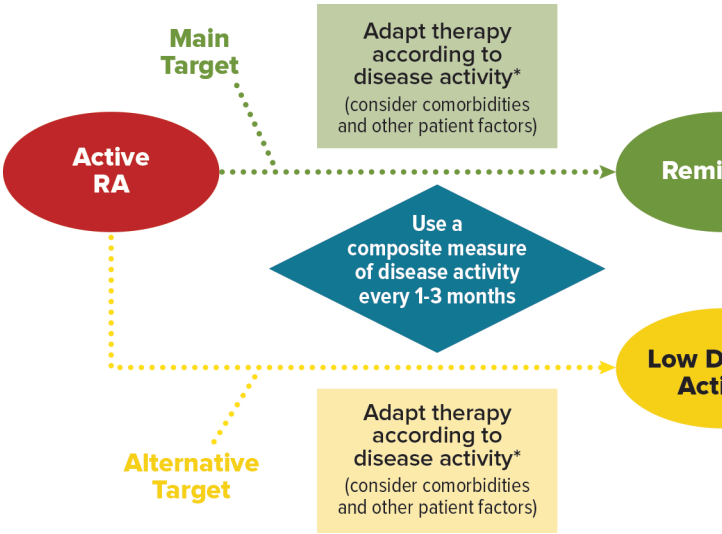


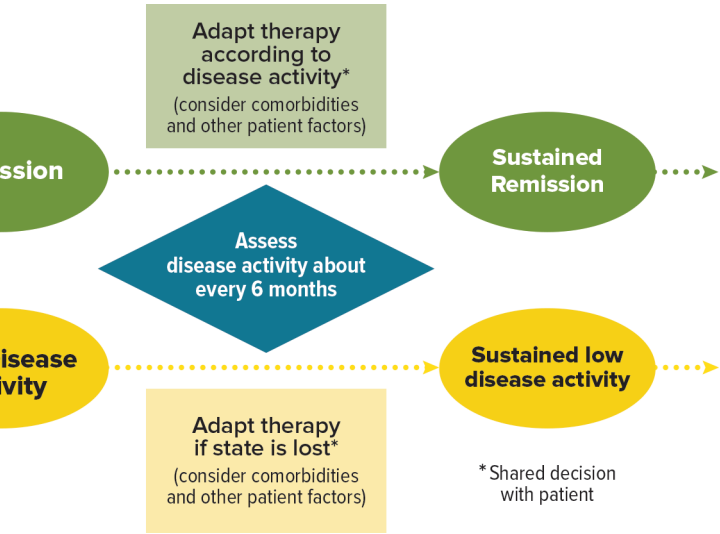
Figure. Treat-to-Target Algorithm for Active RA[1]

ment with the goal of achieving timely control of disease activity (Figure).[1] Importantly, each RA management choice should be based on a shared decision between the patient and the provider.

The T2T approach includes several key steps:

- **Determine the goal of therapy.** Ideally, most patients with early RA should aim to achieve clinical remission. For patients with advanced disease, low disease activity is an appropriate alternate goal to reduce many symptoms of RA and improve functional status and quality of life.

- **Select a composite measure of disease activity.** Clinicians can select from several validated measures of RA disease activity that include



joint counts, including the clinical disease activity index (CDAI) and the disease activity score with 28 joint counts (DAS28). Each measure has a specific threshold for defining clinical remission and low disease activity. For example, using the DAS28 score, clinical remission is defined as a DAS28 score <2.6.

- **Measure disease activity.** After starting RA treatment or switching to a new regimen, disease activity should be measured every 1 to 3 months. Patients who are responding to treatment should see a decrease in their disease activity scores.
- **Adjust treatment.** If necessary, the treatment regimen should be adjusted every 3 to 6 months until the goal of clinical remission is reached.

- **Maintain the treatment goal.** Once the patient has achieved remission, the goal is to maintain this state to prevent further joint damage, control symptoms, and maintain physical functioning.

- **Adjust treatment again as needed.** If disease activity ever increases, suggesting a loss of treatment effect, RA therapy should again be adjusted until the patient returns to a state of clinical remission.

For additional details on the T2T strategy, refer to the Core Curriculum for Rheumatology Nursing available on the Rheumatology Nurses Society website.[3]

References

1. Smolen JS, Breedveld FC, Burmester GR, et al. Treating rheumatoid arthritis to target: 2014 update of the recommendations of an international task force. *Ann Rheum Dis*. 2015 May 12. [Epub ahead of print]
2. Singh J, Furst D, Bharat A, et al. 2012 Update of the 2008 American College of Rheumatology Recommendations for the Use of Disease-Modifying Antirheumatic Drugs and Biologic Agents in the Treatment of Rheumatoid Arthritis. *Arthritis Care & Research*. 2012;64(5):625-639.
3. Carter SC, Patty-Resk C, Ruffing V, Hicks D, eds. Core Curriculum for Rheumatology Nursing. First Ed. Rheumatology Nurses Society;2015.

Is rheumatoid arthritis going to affect my life expectancy?

It is difficult to predict the impact of RA on life expectancy in individual patients. However, based on large population-based studies, RA appears to increase the risk of death by up to 40% compared with individuals of the same age and sex in the general population.[1,2]

Most patients with RA do not die from RA itself, but from other diseases that share the same underlying pathophysiology of chronic inflammation and immune system dysfunction.[2,3] Much of the excess mortality seen in patients with RA is attributable to an increased risk of cardiovascular disease (CVD). In one recent study, patients with seropositive RA

had a 39% increase in the risk of death from any cause. This included a 31% increase in the risk of cardiovascular death.[2] Other leading causes of death in patients with RA include malignancies, infections, and respiratory diseases.[3]

The good news is that there are effective strategies to reduce the negative effect of chronic inflammation and CVD on life expectancy in RA. In 2010, the European League Against Rheumatism (EULAR) developed guidelines for managing cardiovascular risk in patients with RA and other forms of inflammatory arthritis. The EULAR guidelines include the following recommendations for risk-factor screening and management:[4]

- All patients with RA should be evaluated for cardiovascular risk factors every year
- Screening should be repeated whenever a change is made in RA therapy
- Interventions to manage hypertension, dyslipidemia, and other risk factors should follow national guidelines, such as those from American Heart Association

Patient education regarding physical activity, smoking cessation, and adherence to prescribed interventions are also essential to reducing the risk of CVD and improving the long-term prognosis of patients with RA.[4]

References

1. Humphreys JH, Warner A, Chipping J, et al. Mortality trends in patients with early rheumatoid arthritis over 20 years: results from the Norfolk Arthritis Register. *Arthritis Care Res (Hoboken)*. 2014;66(9):1296-1301.
2. Radovits BJ, Fransen J, Al Shamma S, et al. Excess mortality emerges after 10 years in an inception cohort of early rheumatoid arthritis. *Arthritis Care Res (Hoboken)*. 2010;62(3):362-370.
3. Choy E, Ganeshalingam K, Semb AG, Szekanecz Z, Nurmohamed M. Cardiovascular risk in rheumatoid arthritis: recent advances in the understanding of the pivotal role of inflammation, risk predictors and the impact of treatment. *Rheumatology (Oxford)*. 2014;53(12):2143-2154.
4. Peters MJ, Symmons DP, McCarey D et al. EULAR evidence-based recommendations for cardiovascular risk management in patients with rheumatoid arthritis and other forms of inflammatory arthritis. *Ann Rheum Dis*. 2010;69:32531.

Are my children going to get rheumatoid arthritis?

Because there is a genetic component to RA, it is possible that children and other relatives of patients with RA have an increased risk for developing the disease.[1] In addition to sharing genes, family members also share environmental risk factors for RA. In one study of multiple sets of twins with RA, 12% of the risk of RA was attributed to shared genetic risk factors, while 50% was attributed to shared environmental effects.[2]

Environmental exposures during childhood contribute heavily to the lifetime risk of RA. In contrast to siblings, spouses show almost no overlap in the risk of RA, suggesting that shared environments later in life have minimal effect on RA risk.[2] Smoking is the leading environmental

risk factor for RA, contributing up to 25% of all RA risk, and up to 35% of the risk for seropositive RA.[1] Avoiding smoking is especially important for individuals with a family history of RA.

Concerned parents may be reassured to know that early detection and treatment with modern therapies can minimize the long-term damage associated with RA.[3] Individuals with a family history of RA should be aware of the signs and symptoms of RA and seek prompt evaluation should any of these signs appear.

References

1. Sparks JA, Costenbader KH. Genetics, environment, and gene-environment interactions in the development of systemic rheumatic diseases. *Rheum Dis Clin North Am*. 2014;40(4):637-657.
2. Svendsen AJ, Kyvik KO, Houen G, et al. On the origin of rheumatoid arthritis: the impact of environment and genes--a population based twin study. *PLoS One*. 2013;8:e57304.
3. Singh J, Furst D, Bharat A, et al. 2012 Update of the 2008 American College of Rheumatology Recommendations for the Use of Disease-Modifying Antirheumatic Drugs and Biologic Agents in the Treatment of Rheumatoid Arthritis. *Arthritis Care & Research*. 2012;64(5):625-639.

What if I want to get pregnant?

With careful planning, rheumatoid arthritis (RA) should not interfere with a safe pregnancy. According to the American College of Rheumatology, all women who are interested in becoming pregnant should undergo counseling before conception to discuss their risk of complications and to develop a plan for managing their RA before, during, and after pregnancy. Ideally, a patient's RA should be under control for a period of at least 3-6 months before attempting conception. For women with uncontrolled RA, pregnancy should preferentially be postponed until they achieve remission or substantial improvement in disease activity.[1]

A patient's medications should be reviewed as part of the preconception counseling process. To assist with pregnancy-related decisions, the U.S. Food and Drug Administration requires all medications to be categorized by risk as follows:[2]

- **CATEGORY A:** Controlled clinical studies in humans have failed to demonstrate a risk to the fetus in the first trimester of pregnancy, and no evidence exists suggesting risk in later trimesters.
- **CATEGORY B:** Reproduction studies in animals have failed to demonstrate evidence of impaired fertility or harm to the fetus. However, no controlled clinical studies have been conducted in humans.
- **CATEGORY C:** Reproduction studies in animals have either not been performed or have demonstrated evidence of impaired fertility or harm to the fetus. However, the benefit of the drug may still outweigh its risk.
- **CATEGORY D:** Adverse reaction data in human investigational trials or marketing experience has been demonstrated. However, the benefit of the drug may still outweigh its risk, especially in emergency presentations.
- **CATEGORY X:** Studies in animals or humans have demonstrated fetal abnormalities or there is positive evidence of fetal risk based on adverse reaction reports from investigational or marketing experience, or both. The risk of using the drug clearly outweighs any possible benefit.

Prior to conception, treatment for RA should be adjusted from drugs that are incompatible with pregnancy to those that are compatible with pregnancy (Table). In particular, methotrexate and leflunomide are contraindicated in the setting of pregnancy (ie, category X). Once disease activity

Table. Antirheumatic drugs and risk during pregnancy[3,4]

Drug class	FDA pregnancy category	Clinical recommendations
NSAIDs	B	• First part of pregnancy
	C	• After 30 weeks of gestation • Increased risk of premature closure of the ductus arteriosus
Corticosteroids	C	• First-trimester use associated with increased risk of oral cleft • Increased risk of adrenal insufficiency
Nonbiologic DMARDs		
Sulfasalazine	B	• No increased risk of congenital malformations • Combine with folate supplements
Azathioprine	D	• Can be continued to maintain remission during pregnancy
Methotrexate	X	• Contraindicated in pregnancy • Discontinue 3–6 months before conception
Leflunomide	X	• Contraindicated during pregnancy • Discontinue use 2 years before pregnancy
Antimalarials	C	• HCQ is compatible with pregnancy • Risk for retinal toxicity and ototoxicity higher for chloroquine than for HCQ
Biologic DMARDs		
TNF inhibitors	B	• Anti-TNF antibodies are not transferred to the embryo/fetus in first trimester of pregnancy
Abatacept	C	• No human pregnancy data available • Discontinue 10 weeks before planned pregnancy
Rituximab	C	• Reversible B-cell depletion or lymphopenia in the neonate • Long half-life; discontinue 1 year before planned pregnancy
Tocilizumab	C	• No human pregnancy data available • Discontinue 10 weeks before planned pregnancy
Tofacitinib	C	• No human pregnancy data available

DMARD = disease-modifying antirheumatic drug; FDA = Food and Drug Administration; HCQ = hydroxychloroquine; NSAID = nonsteroidal antiinflammatory drug; TNF = tumor necrosis factor.

is stable using medications that are compatible with pregnancy, women can attempt to conceive.[3]

Approximately two-thirds of women with RA will experience a decrease in disease activity during pregnancy due to hormonal and immunological changes.[4] Nonetheless, many pregnant women with RA will require some form of medication to control symptoms during pregnancy.[3] Treatment with tumor necrosis factor (TNF) inhibitors and other biologics should be discontinued as soon as pregnancy is recognized, given the lack of safety data on the long-term effects of exposure during pregnancy. Certain other agents—hydroxychloroquine, sulfasalazine, and azathioprine—can be used throughout pregnancy.[3]

References

1. American College of Rheumatology. Pregnancy and rheumatic disease. Revised May, 2013. http://www.rheumatology.org/Practice/Clinical/Patients/Diseases_And_Conditions/Pregnancy_and_Rheumatic_Disease/.
2. U.S. Food and Drug Administration. Code of Federal Regulations Title 21. April 1, 2014. http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfCFR/CFRSearch.cfm?fr=201.57.
3. Hazes JMW, Coulie PG, Geenen V, et al. Rheumatoid arthritis and pregnancy: evolution of disease activity and pathophysiological considerations for drug use. *Rheumatology*. 2011; 50(11):1955-1968.
4. Krause ML, Amin S, Makol A. Use of DMARDs and biologics during pregnancy and lactation in rheumatoid arthritis: what the rheumatologist needs to know. *Ther Adv Musculoskelet Dis*. 2014;6(5):169-184.

Will I need to take this medication for the rest of my life?

In the absence of a cure, rheumatoid arthritis (RA) will remain a chronic disease that requires lifelong management. The goal of RA treatment is to achieve clinical remission or low disease activity with a drug regimen that best suits the needs of individual patients. However, the process of finding the best regimen for each patient is complex. Many patients with RA will need to switch regimens—often multiple times—to find the right medication or combination of treatments.[1] Therefore, most patients are unlikely to take the same medication for the rest of their lives. A more likely scenario is that patients will require multiple treatment adjustments to achieve and then maintain the

treatment goals of remission or low disease activity. Patients may also experience a change in lifestyle that necessitates a treatment adjustment, such as a switch from weekly to monthly injections to accommodate a new work schedule.

In the future, patients with RA may have the option of taking long-term drug holidays under careful supervision. Researchers have been exploring the possibility of drug-free remissions in select patients with early RA who meet very specific selection criteria.[2,3] Until this option is ready for use outside of the research setting, patients should focus on maintaining control of their RA disease activity with approved therapies.

References

1. Smolen JS, Breedveld FC, Burmester GR, et al. Treating rheumatoid arthritis to target: 2014 update of the recommendations of an international task force. *Ann Rheum Dis*. 2015 May 12. [epub ahead of print]
2. Ajeganova S, van Steenbergen HW, van Nies JA, et al. Disease-modifying antirheumatic drug-free sustained remission in rheumatoid arthritis: an increasingly achievable outcome with subsidence of disease symptoms. *Ann Rheum Dis*. 2015. [Epub ahead of print]
3. Emery P, Burmester GR, Bykerk VP, et al. Evaluating drug-free remission with abatacept in early rheumatoid arthritis: results from the phase 3b, multicentre, randomised, active-controlled AVERT study of 24 months, with a 12-month, double-blind treatment period. *Ann Rheum Dis*. 2015;74(1):19-26.

Am I going to get cancer from my biologic?

This is an important question that has been studied in hundreds of randomized clinical trials (RCTs) and observational studies.[1,2] Like all individuals, patients with rheumatoid arthritis (RA) are susceptible to developing cancer irrespective of their treatment. To date, it does not appear that biologic therapy adds to the normal background risk of cancer in patients with RA.

In a systematic review of 63 RCTs, 29,423 patients with RA who were randomly assigned to 1 of 3 treatment groups: **a)** biologic therapy alone or in combination with methotrexate (MTX), **b)** nonbiologic disease-modifying antirheumatic drug (DMARD) therapy, or **c)** placebo. This study examined a range of available biologics, including

abatacept, adalimumab, anakinra, certolizumab pegol, etanercept, golimumab, infliximab, rituximab, and tocilizumab. During the first year of treatment, the incidence of cancer was very low across all treatment groups: 0.64% in the biologic monotherapy group, 0.77% for patients treated with a biologic plus MTX, and 0.66% in the nonbiologic DMARD group. Through 3 years of follow up, biologic therapy was not associated with an increased risk of any type of cancer compared with other treatment groups.[1]

In another meta-analysis of 33 RCTs and 9,555 patients with RA, there was no increase in the risk of malignancy during treatment with anti-tumor necrosis factor (TNF) therapy, and no differences in cancer risk between any of the biologic agents (infliximab, etanercept, adalimumab, golimumab, or certolizumab pegol).[2]

Researchers have also studied the interaction between anti-TNF therapy and cancer risk in patients with other inflammatory conditions. In a Danish nationwide study of 56,146 patients with inflammatory bowel disease, there was no difference in the risk of cancer between patients who were treated with infliximab, adalimumab, or certolizumab pegol and those who were not exposed to anti-TNF therapy.[3]

Taken as a whole, the growing body of evidence in patients with RA and related conditions should be reassuring for patients who are concerned about the risk of cancer associated with biologic therapy.

What about the use of biologic agents in patients with a history of cancer? In the 2012 RA guidelines,

the American College of Rheumatology (ACR) advised clinicians to wait for 5 years following a cancer diagnosis to treat patients with biologic therapies.[4] However, based on greater clinical experience with biologics and new data from long-term observational studies and RCTs, the ACR is in the process of updating these recommendations.[5] Draft guidelines suggest that the ACR will lift the 5-year waiting period and recommend a wider range of treatment options for patients with a history of cancer (Table). Watch for the finalized ACR guidelines later in 2015 to clarify the best options for managing RA across multiple patient groups, including patients with a history of cancer.

Figure. Considerations for RA Treatment in Patients with a History of Cancer[5]

Patient History	Treatment Considerations
Previously treated or untreated melanoma	TNF inhibitor is preferable to tofacitinib
Nonmelanoma skin cancer	Combination DMARD therapy or a non-TNF biologic is preferable to an anti-TNF agent
Previously treated lymphoproliferative disorder	Combination DMARD therapy or non-TNF biologic is preferable to an anti-TNF agent
Treated solid organ malignancies	No special adjustments to RA therapy are required

DMARD = disease-modifying antirheumatic drug; RA = rheumatoid arthritis; TNF = tumor necrosis factor.

References

1. Lopez-Olivo MA, Tayar JH, Martinez-Lopez JA, et al. Risk of malignancies in patients with rheumatoid arthritis treated with biologic therapy: a meta-analysis. *JAMA*. 2012;308:898-908.
2. Moulis G, Sommet A, Béné J, et al. Cancer risk of anti-TNF-α at recommended doses in adult rheumatoid arthritis: a meta-analysis with intention to treat and per protocol analyses. *PLoS One*. 2012;7(11):e48991.
3. Nyboe Andersen N, Pasternak B, Basit S, et al. Association between tumor necrosis factor-α antagonists and risk of cancer in patients with inflammatory bowel disease. JAMA. 2014;311(23):2406-2413.
4. Singh J, Furst D, Bharat A, et al. 2012 Update of the 2008 American College of Rheumatology Recommendations for the Use of Disease-Modifying Antirheumatic Drugs and Biologic Agents in the Treatment of Rheumatoid Arthritis. *Arthritis Care & Research*. 2012;64(5):625-639.
5. Singh JA. New ACR recommendations for the management of rheumatoid arthritis. Presented at the 2014 American College of Rheumatology Annual Meeting. November 14-19, 2014; Boston, MA.

Are there any herbal treatments or supplements I can take that will help?

Despite extensive research on the topic, there is no compelling evidence that herbal supplements are effective in treating rheumatoid arthritis (RA).[1,2] The U.S. Food and Drug Administration does not regulate the safety, efficacy, or quality of herbal supplements. For this reason, the American College of Rheumatology (ACR) recommends against the use of herbal remedies in patients with rheumatologic disease.[1] Of note, herbal supplements should not be confused with nutritional supplements such as folic acid, a type of B vitamin. Folic acid supplementation can reduce the risk of nausea and vomiting in patients who experience gastrointestinal side effects during treatment with methotrexate.[3]

Many patients turn to complementary and alternative medicine (CAM) to cope with chronic pain and debilitating disease. In a survey of CAM use among 1,063 rheumatology patients, 92% of patients reported using at least 1 form of CAM to manage their arthritis.[4] Patients who used CAM tended to use multiple CAM strategies. In total, 35% of patients reported using 3 or 4 strategies, and 28% reported using 5 or more forms of CAM to manage their arthritis.

In the CAM study, only 54% of CAM users reported ever discussing it with their rheumatologist.[4] Women and those who used more types of CAM were more likely than men and infrequent CAM users to volunteer information about their CAM use. In addition, patients who rated their rheumatology provider as having a more participatory style of decision-making were more likely to report their CAM use. This highlights the importance of the patient/provider relationship in facilitating an open dialogue. The Rheumatology Nurses Society (RNS) recommends that nurses ask patients about their use of herbal supplements at every visit. These treatments should be documented in the patient record.[5]

Keep in mind that patients may be asking about herbal supplements because they are interested in options for treating RA other than standard drug therapy. Exercise is an example of a non-pharmacologic intervention that can enhance the benefits of RA medication. The ACR recommends regular exercise for patients with RA to reduce joint damage and bone loss.[6] Ideally, an exercise program for patients with RA should

include moderate-intensity aerobic activity for 150 minutes per week, spread out over several days. Patients can meet this target in ways that work best for their lifestyle and comfort level, whether through multiple 10-minute bouts of exercise or with more sustained activity. Moderate-intensity exercise should pass the Talk Test, meaning that the patient can speak normally while exercising without getting short of breath or overheated.

Some patients with RA may be concerned that exercise will be painful. Exercise should not increase pain or disease activity (ie, no flares). Safe options for patients with RA include walking, aerobic dance, aquatic exercise, and exercising on gym equipment such as stationary bicycles or treadmills. Body awareness exercises such as Tai chi and yoga can improve balance, coordination, sense of joint position (proprioception), and relaxation. Leisure activities such as playing golf and walking the dog can also be considered exercise if performed at a moderate intensity level.[6]

References

1. American College of Rheumatology. Herbal remedies, supplements and acupuncture for arthritis. 2015a. Available at www.rheumatology.org/Practice/Clinical/Patients/Diseases_And_Conditions/Herbal_Remedies,_Supplements_and_Acupuncture_for_Arthritis/. Accessed June 26, 2015.
2. Cameron M, Gagnier JJ, Chrubasik S. Herbal therapy for treating rheumatoid arthritis. *Cochrane Database Syst Rev*. 2011;(2):CD002948.

3. Shea B, Swinden MV, Tanjong Ghogomu E, et al. Folic acid and folinic acid for reducing side effects in patients receiving methotrexate for rheumatoid arthritis. *Cochrane Database Syst Rev*. 2013;5:CD000951.
4. Sleath B, Callahan LF, Devellis RF, Beard A. Arthritis patients' perceptions of rheumatologists' participatory decision-making style and communication about complementary and alternative medicine. *Arthritis Rheum*. 2008;59(3):416-421.
5. Carter SC, Patty-Resk C, Ruffing V, Hicks D, eds. Core Curriculum for Rheumatology Nursing. First Ed. Rheumatology Nurses Society;2015.
6. American College of Rheumatology. Exercise and arthritis. 2015b. Available at www.rheumatology.org/Practice/Clinical/Patients/Diseases_And_Conditions/Exercise_and_Arthritis/. Accessed June 26, 2015.

Supported by an educational grant from **Pfizer**

BE THE RESOURCE THEY NEED.

RNSnurse.org

RHEUMATOLOGY NURSES SOCIETY™

8437 Tuttle Avenue - Suite 404 | Sarasota, FL 34243