Natural Treatments for Osteoarthritis

by Alan R. Gaby, MD

Abstract

Osteoarthritis (OA) is the most common form of joint disease. Although OA was previously thought to be a progressive, degenerative disorder, it is now known that spontaneous arrest or reversal of the disease can occur. Conventional medications are often effective for symptom relief, but they can also cause significant side effects and do not slow the progression of the disease. Several natural substances have been shown to be at least as effective as nonsteroidal anti-inflammatory drugs at relieving the symptoms of OA, and preliminary evidence suggests some of these compounds may exert a favorable influence on the course of the disease.
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Introduction

Osteoarthritis (OA), the most common form of joint disease, is characterized by erosion of articular cartilage. The joints most often affected by OA are the knees, hips, spine, and hands, although other joints may be involved. OA is usually classified either as primary (idiopathic) or secondary. In the former, no obvious predisposing factor can be identified; in the latter, the arthritis appears be the result of trauma, repetitive joint use, congenital or developmental defects, metabolic or endocrine disorders, or other factors. Clinical manifestations of OA include pain, stiffness, and decreased range of motion of affected joints. In more-advanced cases, significant disability may occur. It is estimated that 100,000 people in the United States are unable to walk because of severe OA of the hip or knee.

In the past, OA was considered a degenerative disorder, in which the joint gradually "wears out." However, more-recent evidence has resulted in a change of thinking concerning the pathogenesis and natural history of OA. It is now known that the joint cartilage of individuals with OA is highly metabolically active, engaging (at least early in the course of the disease) in a process of remodeling and repair of damaged tissue. Arrest or reversal of the disease, once thought to be impossible, has now been shown to occur spontaneously in some individuals with OA.[1]

Conventional pharmacological treatment of OA consists primarily of nonsteroidal anti-inflammatory drugs (NSAIDs) and analgesics. While these medications often relieve symptoms, they are far from ideal therapeutic agents. NSAIDs, in particular, can cause serious side effects, including peptic ulcer and (less commonly) hepatic or renal failure. And neither of these classes of medications prevents or delays the progression of OA. In fact, there is evidence, both in animals with experimental OA[2] and in humans,[3] that administration of NSAIDs may actually accelerate joint destruction. New approaches are therefore needed, both to increase the safety and efficacy of symptomatic treatment and to exert a favorable influence on the course of the disease.

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A number of substances that occur naturally in the body may have value for the prevention and/or treatment of OA. Some of these compounds have been shown to provide symptomatic relief, and preliminary evidence suggests some may positively affect the progression of the disease. Although much of the research is in its early stages, the possibility that natural substances can be used to prevent the degradation, or enhance the repair, of joint cartilage is intriguing.

Niacinamide

A half-century ago, William Kaufman, MD, described his results using niacinamide in the treatment of several hundred patients with OA.[4-7] The dosage was 900 to 4,000 mg per day, depending on the degree of impairment of range of motion of the joints. Treatment with niacinamide usually resulted in an increase in joint mobility (measured objectively), as well as subjective improvements in joint discomfort, inflammation, and pain. Improvement with niacinamide therapy usually did not occur until after three to four weeks of treatment. Thereafter, progressive improvement occurred for one to three years if niacinamide was continued. Some patients receiving long-term niacinamide treatment maintained improved joint function (as demonstrated by an increase in joint range index) for as long as twenty years. However, patients who stopped taking the vitamin gradually reverted to their pre-treatment status.

Because Kaufman's study lacked a control group, the possibility of a placebo effect cannot be ruled out. However, a recent double-blind study by Jonas et al[8] tended to support Kaufman's observations. In that study, 72 patients with OA of at least five years' duration were randomly assigned to receive niacinamide (500 mg six times per day) or a placebo for 12 weeks. Outcome measures included global arthritis impact, pain, joint mobility, and erythrocyte sedimentation rate (ESR). Global arthritis impact improved by 29 percent in patients receiving niacinamide and worsened by 10 percent in patients given placebo ($p = 0.04$ for difference between groups). Although pain levels were no different in the two groups, patients on niacinamide reduced their anti-inflammatory medication by 13 percent, compared with a slight increase in medication in the placebo group ($p = 0.014$ for difference between groups). Niacinamide reduced the ESR by 22 percent compared with placebo ($p < 0.005$) and increased joint mobility (as measured by the joint range index) by 8.0 degrees, compared with 3.5 degrees in the placebo group ($p = 0.04$).

Niacinamide's delayed onset of action, its capacity to induce progressive improvement, and its gradual (as opposed to abrupt) loss of effect after treatment is discontinued, suggest this vitamin somehow helps control OA, rather than merely relieving symptoms. Although its mechanism of action is not known, niacinamide does not appear to act merely as an anti-inflammatory agent or analgesic.

Kaufman observed niacinamide was most effective when taken in frequent, divided doses. Thus, 250 mg taken six times per day was more effective than 500 mg taken three times per day. The need for frequent dosing is presumably related to the short half-life of the vitamin. Sustained-release forms of niacinamide are commercially available, and some practitioners have found them to be an acceptable alternative to more-frequent dosing with regular niacinamide. It is not known whether niacin (nicotinic acid) or inositol hexanicotinate (the other available form of vitamin B3) can be used as substitutes for niacinamide in the treatment of OA. It should be noted, however, that niacin, particularly in the sustained-release form, is more hepatotoxic than niacinamide.

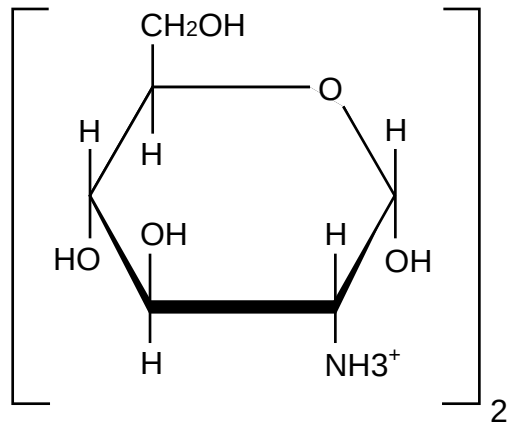
Niacinamide is generally well tolerated and appears to be relatively safe for long-term use. During several thousand patient-years of clinical experience with this vitamin, Kaufman did not observe any serious adverse reactions. In the study by Jonas et al, the mean SGOT (AST) level increased by 20 percent over baseline in the niacinamide group, but none of the values rose to a level considered dangerous or of concern. However, there are occasional reports of large doses of niacinamide causing clinically significant elevations of liver enzymes (serum transaminases) and, rarely, chemical hepatitis. Patients taking large amounts of this vitamin (such as 1,500 mg per day or more) should therefore have periodic tests to monitor liver function. Therapeutic doses of niacinamide should be used with caution, if at all, in individuals who have or are at risk of developing liver disease.

Glucosamine Sulfate

Articular cartilage contains a group of large protein molecules called proteoglycans. These proteins make up the ground substance of cartilage – the material that gives joints strength and resilience. Glucosamine, which is produced in the body from glucose, is a precursor molecule in the synthesis of proteoglycans. Glucosamine has been reported to stimulate proteoglycan synthesis *in vitro*, to inhibit its degradation, and to rebuild experimentally damaged cartilage; effects which might be useful for the prevention and treatment of OA.

The efficacy of glucosamine—as glucosamine sulfate (GS; see Figure 1)—in the treatment of OA has been investigated in a number of clinical trials, at least three of which have been double-blind and placebo-controlled. In one such study, eighty patients with OA affecting various parts of the body were randomly assigned to receive GS (500 mg three times per day) or a placebo for thirty days.[9] Articular pain, joint tenderness and swelling, and range of motion improved to a significantly greater extent in the GS group than in the placebo group. Samples of articular cartilage were obtained from two patients in each group at the end of the treatment period. Scanning electron microscopy performed on the cartilage obtained from patients receiving placebo showed a typical picture of established OA, whereas the tissue from those who received GS showed a picture more similar to healthy cartilage.

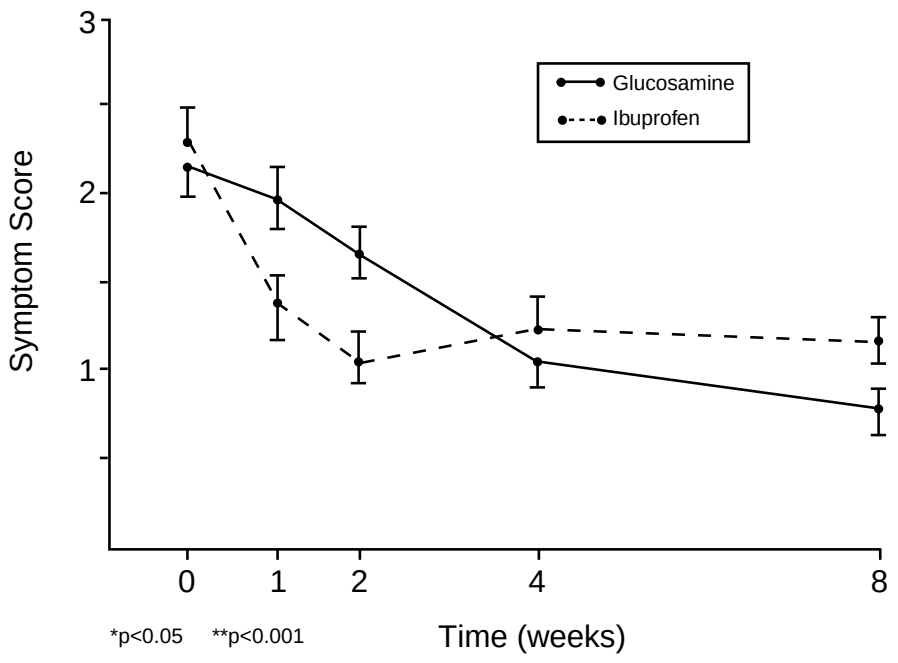
Figure 1. Glucosamine Sulfate



$2Na(or\ K) + SO_4 = 2Cl^-$

In another double-blind investigation, twenty patients with OA of the knee received GS (500 mg, three times per day) or a placebo for six to eight weeks. GS was significantly superior to placebo, as determined by improvements in pain, joint tenderness, and swelling. The results were rated as "excellent" in all ten patients receiving GS, whereas all ten patients receiving the placebo rated the results as "fair" or "poor."[10]

Figure 2. Changes in pain score during comparison study of glucosamine sulfate vs. ibuprofen. Adapted



In a separate investigation, 252 patients with osteoarthritis of the knee were randomly assigned to receive placebo or GS (500 mg three times per day) for four weeks.[11] A positive response to treatment was defined as a reduction of at least three points in the Lequesne's index, plus a positive overall assessment by the investigator. The mean Lequesne's index, which was 10.6 points in both groups at the start of the study, decreased by 3.2 points in the GS group, compared with 2.2 points in the placebo group ($p < 0.05$). On intention-to-treat analysis, the responder rate was 52 percent in the glucosamine sulfate group and 37 percent in the placebo group ($p = 0.016$).

Several studies have compared the efficacy of GS with ibuprofen. Forty patients with unilateral osteoarthritis of the knee received GS (500 mg three times per day), or ibuprofen (1,200 mg per day), in double-blind fashion for eight weeks. Although the rate of improvement was slower in the GS group than in the ibuprofen group, the improvement became progressively more pronounced in the former as the study progressed. By the eighth week, pain relief was significantly more pronounced in the GS group than in the ibuprofen group (see Figure 2).[12]

The same doses of GS and ibuprofen were compared in a four-week study involving a similar group of patients.[13] Again, clinical improvement tended to occur sooner in the ibuprofen group than in the GS group, but there was no difference between groups from the second week onward.

In a four-week study of 178 patients with osteoarthritis of the knee, both GS and ibuprofen significantly reduced OA symptoms, with a trend of GS being more effective.[14] The beneficial effects of both GS and ibuprofen persisted for at least two weeks after the treatments were discontinued; however, there was a trend toward a greater residual effect in the GS group.

GS has also been compared with piroxicam, another NSAID. Some 329 patients with osteoarthritis of the knee were randomly assigned to one of four daily treatments for 90 days: 1,500 mg of GS; 20 mg of piroxicam; GS plus piroxicam; or placebo.[15] Patients were followed an additional sixty days after treatment was discontinued. After ninety days of

treatment, GS was significantly more effective than placebo (as determined by the Lequesne index) and more effective than piroxicam (statistical comparison not presented). The beneficial effect of GS was maintained during the 60 days after treatment was discontinued, whereas the effects of piroxicam progressively decreased after withdrawal of treatment.

These studies indicate that GS can relieve the symptoms of OA at least as effectively as some commonly used NSAIDs. Although circumstantial evidence, i.e., residual effects after withdrawal of treatment and positive changes seen in biopsy samples, suggests GS may also have a favorable influence on the disease process, long-term studies are needed to confirm this possibility.

No serious side effects have been reported with the use of GS and the compound is generally well tolerated, particularly when compared with NSAIDs. Recently, concern has been expressed about the potential for GS to induce insulin resistance.[16] This effect has been demonstrated with continuous intravenous infusions of GS in normoglycemic[17] but not hyperglycemic[18] animals, using the euglycemic hyperinsulinemic clamp method. Although insulin resistance was detectable with GS doses as low as 0.1 mg per kg body weight per minute, the relevance of these findings to humans is not clear. No abnormalities of glucose metabolism have been reported in individuals taking GS. However, until long-term studies are done, the potential for GS to promote insulin resistance should be kept in mind.

Although GS is not widely accepted by conventional physicians in the United States, it is a mainstay of treatment in Germany and Russia.[19] Doctors in these countries usually recommend 500 mg three times per day for six to eight weeks, then twice a day thereafter. For flare-ups, low doses of NSAIDs are prescribed, along with 500 mg of glucosamine once a day. The use of glucosamine reportedly makes it possible to cut the effective dosage of NSAIDs in half.

Chondroitin Sulfate

Chondroitin sulfate (CS) is a term used to denote a group of structurally similar polysaccharides, typically comprised of sulfated and unsulfated residues of glucuronic acid and N-acetylglucosamine. CS is one of the components of proteoglycans, the macromolecules that contribute to the structural and functional properties of joint cartilage. There is evidence that CS stimulates the synthesis of proteoglycans by chondrocytes.

Forty-two patients (aged 35-78 years) with symptomatic OA of the knee were randomly assigned to receive, in double-blind fashion, 800 mg of a proprietary product containing chondroitin 4- and 6-sulfate (CS; Condrosulf, IBSA, Lugano, Switzerland) per day or a placebo for one year.[20] After three months, joint pain was reduced to a significantly greater extent in the CS group than in the placebo group. The difference in pain reduction between groups became even more pronounced after twelve months (63% vs. 26%; $p < 0.01$). The increase in overall mobility capacity (assessed by a visual analogue scale) was significantly greater at six and 12 months in the CS group than in the placebo group (69% increase vs. 19% increase; $p < 0.01$). After one year, the mean width of the medial femoro-tibial joint was unchanged from baseline in the CS group, but had decreased significantly in the placebo group. Although no statistical comparison was presented for the change in joint width between the CS and placebo groups, these findings are consistent with the possibility that CS treatment slowed the progression of OA.

In another study, 85 patients with OA of the knee were randomly assigned to receive, in double-blind fashion,

Chondrosulf (400 mg twice a day) or a placebo for six months.[21] Lequesne's Index, spontaneous joint pain (visual analogue scale) and walking time (defined as the minimum time to perform a 20-meter walk) all decreased progressively in the CS group. There was a significant difference in favor of the CS group for each of these parameters (after three months for Lequesne's Index and spontaneous joint pain; after six months for walking time).

In a randomized, three-year, double-blind trial, 34 patients with OA of finger joints received Chondrosulf (400 mg three times per day) and 85 patients received a placebo.[22] During the study, a comparable number of patients in each group developed OA in previously non-affected finger joints. However, in the CS group compared with the placebo group there was a significant decrease in the number of patients with new erosive OA of finger joints. The authors concluded administration of CS may protect against the development of erosive changes in patients with finger joint OA.

In another study, 146 patients with OA of the knee were divided into two groups.[23] One group received diclofenac sodium (Voltaren; 50 mg three times per day) for one month, followed by a placebo for two months. The other group received CS (400 mg three times per day) for three months. Both groups then received a placebo for an additional three months. Clinical efficacy was evaluated by assessing the Lequesne's Index, spontaneous pain, pain on load, and acetaminophen use. CS was significantly more effective than placebo in all parameters measured, and the effect of CS became more pronounced with time. The beneficial effects of CS diminished gradually during the three months after treatment was discontinued.

No serious side effects were reported in any of the studies with CS. Thus, CS appears to be safe and effective for the symptomatic treatment of OA. Preliminary evidence suggests CS may also have a favorable influence on the course of the disease but definitive studies are lacking.

Glucosamine Sulfate vs. Chondroitin

There has been an ongoing debate concerning whether GS or CS is the preferable therapeutic agent or whether these compounds should be used in combination. Orally administered GS has been shown to be well absorbed.[24] On the other hand, CS is a relatively large molecule and is presumably hydrolyzed in the intestinal tract prior to being absorbed. While some CS does appear to be absorbed intact,[25] the proportion of an oral dose that is absorbed is said to be small.[26] Some have argued that CS is largely broken down in the gastrointestinal tract and then reassembled after being absorbed. If that is true, administering CS is merely an expensive way to obtain precursor molecules. Since GS serves as a precursor to, and appears to promote the synthesis of, CS, administering GS may be a less-expensive method of increasing the CS content of joint cartilage. On the other hand, it is conceivable that the small amount of CS that does get absorbed (or perhaps one of its byproducts of partial digestion) exerts beneficial effects that cannot be duplicated by giving GS.

To date, there have been no studies comparing the efficacy of GS and CS, or comparing the combination to either compound by itself. Until such studies are done the choice of which regimen to use remains a matter of individual preference.

Vitamin E

Twenty-nine patients with OA at various sites were randomly assigned to receive (single blind) 600 mg of vitamin E (type not specified) per day or a placebo for ten days,

and then the alternate treatment for an additional ten days.[27] Fifty-two percent of the patients reported a reduction in pain while receiving vitamin E, compared with only 4 percent receiving placebo ($p < 0.01$).

In another study, 53 patients with OA of the hip or knee were treated for three weeks with vitamin E (d-alpha-tocopheryl acetate 400 mg three times per day; equivalent to approximately 600 IU three times per day) or diclofenac (50 mg three times per day).[28] Both treatments appeared to be equally effective in reducing the circumference of knee joints and walking time, and in increasing joint mobility.

Although the mechanism of action of vitamin E against OA is not known, this vitamin has been reported to have anti-inflammatory activity [29] and may also inhibit prostaglandin synthesis. In addition, vitamin E may help stabilize lysosomal membranes, thereby inhibiting the release of enzymes believed to play a role in the pathogenesis of osteoarthritic joint damage.

Boron

Boron has long been recognized as an essential trace element for plants, but has only recently been considered to be possibly essential for humans. Boron appears to participate in hydroxylation reactions, which play a role in the synthesis of steroid hormones and vitamin D. In Australia, where much of the food is grown on soil deficient in this mineral, boron supplements were popular as a treatment for OA, and were reportedly selling at a rate of 10,000 bottles per month before the Australian government removed the product from the market.[30]

In a double-blind study, 20 Australians with OA were randomly assigned to receive boron (6 mg per day as sodium tetraborate decahydrate) or a placebo for eight weeks.[31] Of those receiving boron, 50 percent improved, compared with 10 percent of those given placebo. Because of the small sample size, this difference was not statistically significant. When the five subjects (25%) who dropped out of the study (mostly because of clinical deterioration) were excluded from the analysis, 71 percent of those in the boron group improved, compared with 12.5 percent of those in the placebo group ($p < 0.05$). No side effects were seen and there were no significant changes in common laboratory parameters.

These results suggest boron supplementation may be helpful for individuals with OA whose diets are likely to be low in boron. Further research is needed to confirm this preliminary study and to determine whether individuals with a higher dietary intake of boron can benefit from supplementation.

The average American diet provides approximately 1-2 mg of boron per day, primarily from fruits, vegetables, and nuts; however, according to German research, intake can vary from 0.3 to 41 mg per day. While the capacity of boron to increase estrogen levels[32] might raise concerns about possible cancer risks with boron supplementation, there is no evidence that populations with a high intake of boron (such as the French) have an increased incidence of hormone-related cancers.

Vitamin D

In addition to its effects on calcium metabolism, vitamin D plays a role in the normal turnover of articular cartilage. In a prospective study of 556 participants in the Framingham study, low dietary intake of vitamin D and low serum levels of the vitamin were each associated with increased radiographic progression of OA of the knee, but not with the incidence of newly diagnosed OA.[33] In an eight-year prospective study of 237 individuals (aged 65 years or older), low serum levels of 25-hydroxyvitamin D were associated with an increased risk of developing OA of the hip, as defined by joint-space narrowing.[34] These studies suggest adequate intake

of vitamin D (or, presumably, adequate sunlight exposure) may slow the progression and possibly help prevent the development of OA.

Ascorbic Acid

Vitamin C is required for the synthesis of collagen, an important structural protein of joint cartilage. In guinea pigs, supplementation with vitamin C had a slight protective effect on experimentally-induced cartilage degeneration of the knee.[35] In a study of participants in the Framingham Osteoarthritis Cohort Study, a higher intake of vitamin C (upper tertile) was associated with a reduced risk of cartilage loss and disease progression in individuals with OA of the knee.[36] In an uncontrolled clinical trial, 59 patients with petechiae and either rheumatoid or osteoarthritis received ascorbic acid (300-1,000 mg per day) plus hesperidin (a flavonoid). Abnormal capillary fragility improved and clinical improvement also frequently occurred.[37] These observations suggest adequate intake of vitamin C may help prevent progression of OA. It is not known whether supplementation with pharma-cological doses of vitamin C would provide additional benefit.

Manganese

Animal studies have shown that manganese plays a role in the synthesis of chondroitin sulfate,[38] an important component of articular cartilage. Manganese deficiency has been found to cause a cartilage metabolism disorder in farm animals. This condition is said to resemble Mseleni joint disease, an OA-like disease endemic to a remote part of Zululand, where dietary intake of manganese is believed to be low.[39]

It is not known whether manganese deficiency plays a significant role in the pathogenesis of OA; however, one cannot rule out the possibility of subtle manganese deficiency in Western societies. According to Pfeiffer, modern farming techniques deplete manganese from the soil, resulting in lower concentrations of manganese in food.[40] In addition, individuals who consume refined grains (such as white bread) obtain only half as much manganese in their diet as those who eat whole grains.

S-Adenosylmethionine

A metabolite of the essential amino acid methionine, S-adenosylmethionine (SAMe) functions as a methyl donor in many biochemical reactions. *In vitro* studies have provided evidence that SAMe stimulates the synthesis of proteoglycans by human articular chondrocytes.[41] During clinical trials of SAMe as a treatment for depression, some patients reported marked improvement in their OA. Subsequently, extensive clinical trials, which enrolled approximately 22,000 patients, suggest SAMe is as effective as NSAIDs in the treatment of OA, but is better tolerated.

In one such study, 734 patients with OA were randomly assigned to receive, in double-blind fashion, placebo, SAMe (1,200 mg per day), or naproxen (750 mg per day) for 30 days.[42] The reduction in pain and improvement in function were similar in the SAMe and naproxen groups, and both active treatments were significantly more effective than placebo. For most parameters measured, naproxen was significantly more effective than placebo by day 15, whereas statistical significance was not seen with SAMe until day 30. SAMe was better tolerated than naproxen, both in terms of physicians' ($p < 0.025$) and patients' ($p < 0.01$) assessments, and in terms of the number of patients with side effects ($p < 0.05$). There was no difference between SAMe and placebo in the number of side effects.

Other short-term (three to four weeks) double-blind trials have produced similar results. In a study involving 76 patients, SAMe (1,200 mg per day) was significantly more effective than placebo at relieving pain.[43] The same dose of SAMe was also as effective as

indomethacin (150 mg per day)[44] and ibuprofen (1,200 mg per day),[45] as assessed by standard scoring systems for various clinical parameters.

In a 12-week double-blind study of 48 patients with OA of the knee, SAMe (1,200 mg per day) was as effective as piroxicam (20 mg per day), as determined by improvements in pain, mobility, morning stiffness, and pain-free walking distance.[46] Moreover, the improvement in pain score was maintained for at least eight weeks after SAMe was discontinued, whereas a significant worsening was seen in the piroxicam group 28 days after the end of treatment.

The long-term effects of SAMe have been evaluated in an open trial involving 108 patients with OA of the knee, hip, or spine.[47] Each patient received 600 mg of SAMe per day for two weeks, followed by 400 mg per day for a total of two years. Clinical improvement, as determined by symptom score, was seen after two weeks, and there was further continuous improvement up to the sixth month and beyond. Eighteen (19%) of 97 patients who completed two years of treatment experienced total remission of symptoms by the end of the study. More than 90 percent of the physicians and more than 85 percent of the patients assessed the effects of treatment as "very good" or "good." Non-specific side effects (mostly gastrointestinal) occurred in 20 patients, but in no case did the treatment have to be discontinued. Most side effects disappeared during the course of therapy.

Although SAMe has been used in Europe for many years it has only recently become commercially available in the United States. The product is rather expensive compared with other nutritional supplements. In addition, concerns have been raised about the stability of most of the SAMe being sold in this country. Crystalline SAMe degrades rapidly upon exposure to heat and/or moisture, and some of the imported raw material has been said to be partially decomposed upon arrival.[48] Therefore, it may be preferable to use the enteric-coated, pharmaceutical-grade tablets imported from Europe, rather than other preparations currently being sold in the United States.

Avocado/Soybean Extract

An extract of unsaponifiable fractions of avocado and soybean oil (AS) has been shown to stimulate collagen synthesis in articular chondrocyte cultures. In a recent study, 164 patients with OA of the hip or knee were randomly assigned to receive, in double-blind fashion, 300 mg per day of this extract (Piascledine 300; Pharmascience Laboratories, Courbevoie, France; containing unsaponifiable fractions of avocado oil [one-third] and soybean oil [two-thirds]) or a placebo, for six months.[49] The mean Lequesne's index score and the mean pain score improved to a significantly greater extent in the AS group than in the placebo group ($p < 0.001$ and $p = 0.003$, respectively). Thirty-nine percent of the patients in the AS group were considered clinical successes, compared with 18 percent in the placebo group ($p < 0.01$). A residual effect of the avocado/soybean extract was evident two months after treatment was discontinued. No severe side effects were reported.

Herbal Remedy for OA

Forty-two patients with OA were randomly assigned to receive, in double-blind fashion, an Ayurvedic preparation (Articulin-F) or a placebo for three months, and then the alternate treatment for an additional three months. Articulin-F contains (per capsule) 450 mg of *Withania somnifera* root, 100 mg *of Boswellia serrata* stem, 50 mg of *Curcuma longa* rhizome, and 50 mg of a zinc complex. The dosage was two capsules three times per day, after meals. Compared with placebo, Articulin-F significantly reduced the severity of pain and the disability score. Side effects

included nausea (n = 2), dermatitis (n = 3), and abdominal pain (n = 3), none of which required discontinuation of treatment.[50]

Dietary Factors

Some clinicians have observed that identification and avoidance of allergenic foods will relieve the symptoms of OA in some cases. In addition, one report has implicated foods from the genus Solanaceae (Nightshades: tomato, potato, eggplant, bell pepper, and tobacco) as possible triggering agents for OA.[51] The reaction to Nightshade foods is believed to be due to solanum glycoalkaloids present in these foods. Although no controlled studies have been done on the relation between diet and OA, some patients appear to benefit from individualized dietary modifications. Avoiding allergenic foods typically produces results within several weeks or less, whereas it may take a number of months on a Nightshade-free diet before improvement is seen.

Discussion and Conclusion

This article has reviewed a number of promising alternatives for preventing and treating OA. Although additional research needs to be done, some of the treatments discussed appear to be as effective as, and better tolerated than, conventional drug therapy. In addition, preliminary evidence (such as the persistence of improvement after treatment is discontinued, and positive radiographic and biopsy findings) suggests some of these treatments may help arrest or reverse the disease process.

There are few, if any, data on whether the various agents described in this article would have an additive or synergistic effect if used in combination. However, it is not likely that each of these compounds has the same mechanism of action. Therefore, it would be worth comparing the effects of various combinations (such GS plus niacinamide, or GS plus niacinamide plus trace minerals) with the effects of each treatment by itself.

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