The use of Niacinamide and Solanaceae (Nightshade) Elimination in the Treatment of Osteoarthritis

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Abstract *The most common arthritic disease in our modern society is osteoarthritis (OA). It primarily involves the weight-bearing joints of the body, such as the lumbosacral spine, hips, knees, and feet, and less frequently, the cervical spine, and proximal and distal interphalangeal joints. It is a disease of old age, with more than 50% of all individuals over the age of 65 having radiographic features of the disease in the knees, and practically all individuals over the age of 75 will have OA changes in one joint of the body. Two novel treatment options for OA are described: niacinamide and the elimination of solanine-containing foods. These options are rarely considered by most clinicians when managing patients that have OA despite their clinical utility. Clinicians ought to review this information carefully and consider empirical treatment with one or both treatment options when clinically indicated.*

Introduction

The most common arthritic disease in our modern society is osteoarthritis (OA). It primarily involves the weight-bearing joints of the body, such as the lumbosacral spine, hips, knees, and feet, and less frequently, the cervical spine, and proximal and distal interphalangeal joints. It is a disease of old age, with more than 50% of all individuals over the age of 65 having radiographic features of the disease in the knees, and practically all individuals over the age of 75 will have OA changes in one joint of the body. However, OA is not a normal function of aging as the changes found in older "asymptomatic" individuals are much different than those osteoarthritic cartilage changes that occur in "symptomatic" individuals.

Aside from age, other factors predispose to OA such as certain occupations (e.g., a baseball pitcher who succumbs to OA of the shoulders), gender (e.g., women over the age of 55 have more OA than men), race (e.g., knee OA is more prevalent in African-American women than in white women), and obesity (e.g., OA of the knees). Some other factors that predispose to OA include trauma, rare inherited genetic mutations of collagen, and a history of inflammatory arthritis.

In OA, the cartilage is the site where the defect occurs. Cartilage is composed of water, proteoglycans (that contain glycosaminoglycans), and collagens, all of which contribute to the strength and stability of cartilage. Why does the cartilage get degraded in OA? The thought is that some event, due to mechanical stress, leads to al-

tered chondrocyte metabolism, proteolytic enzymes (e.g., matrix metalloproteinase; MMPs), and disrupted matrix properties. Eventually, this leads to decay and gradual loss of the articular cartilage, modification of the joint architecture, and osteophyte formation. Basically, when altered chondrocyte metabolism occurs there is increased production of other inflammatory mediators such as interleukin-1 (IL-1), tumour necrosis factor-alpha (TNF-alpha), and MMPs. The MMPs are normally inhibited by naturally occurring small proteins, known as tissue inhibitors of metalloproteinases (TIMPS). In OA, however, the disease progression is marked by too many MMPs and not enough TIMPS, leading to the osteoarthritic changes that were described above.

Diagnostics

History and physical exam are the most important, but the clinical diagnosis of OA is achieved from radiographic evidence of the affected joint.[2] The typical radiographic features include bony proliferation (osteophyte formation or spurs) at the joint margin, asymmetric joint space narrowing, and subchondral bone sclerosis.[2]

Orthomolecular Treatment

In this article, two novel treatment options for OA will be described: niacinamide and the elimination of solanine-containing foods. These options are rarely considered by most clinicians when managing patients having OA despite their clinical utility. It is the hope of the author that clinicians will review this information carefully and consider empirical treatment with one or both options when warranted.

1. Niacinamide Therapy

The use of niacinamide therapy for rheumatic diseases was developed by orthomolecular pioneer, Dr. William Kaufman. He reported that after 1943 the multisystem aniacinamidosis syndrome was virtually eliminated due to the compulsory enrichment of bread with thiamin, niacin, riboflavin, and iron.[3] The mandatory enrichment of bread ameliorated many of the following signs and symptoms that characterized the multisystem aniacinamidosis syndrome: anxiety, depression, personality changes, startle reaction to unexpected sounds, excessive fear of being hurt, paresthesias of the soles of the feet, metabolic edema, gastrointestinal symptoms, liver tenderness and enlargement, periosteal and cartilaginous tenderness to distal pressure, callusing in response to minor pressure, prolonged retention of sun-tan pigment, and vaginal redness, swelling and tenderness.

After 1943, only 5 to 10% of the patients Kaufman treated had the complete syndrome of aniacinamidosis. However, even with the enrichment of bread many of the signs and symptoms that were part of the multisystem aniacinamidosis syndrome remained. These were changes in the lingual membrane, impaired muscle strength, impaired maximum muscle working capacity, impaired joint mobility, impaired balance sense, and in persons over 55, a mental syndrome consisting of mild depression or agitation and hyperkinesis. According to Kaufman, this proved conclusively that much more niacinamide was required than what was provided through diet, in addition to the amount of niacinamide obtained from the enrichment of cereal products and grains.

In 1949, Kaufman published his second book, *The Common Form of Joint Dysfunction*.[4] This book details the effects of niacinamide therapy on 455 Caucasian patients of both sexes ranging from 4 to 78 years of age having osteoarthritis or rheumatoid arthritis, treated between March 1945 and February 1947. In order to objectively measure the effects of niacinamide on joint dysfunction, Kaufman devised or adapted goniometers with which to measure maximum joint movement before and during treatment. He selected 20 separate joint ranges which could be easily calculated to give a single number called the Joint range Index or JRI. The 20 joint ranges he routinely measured for the incorporation into the JRI were the following: lateral rotation of the neck to the right and left; extension and flexion of the wrist joint of each hand; extension of the meta-

carpophalangeal joints of each of the four fingers of both hands; circumduction of each shoulder joint; abduction of each hip joint with the thigh maintained in a plane perpendicular to the trunk; and extension of the right and left knee when the thigh is maintained perpendicular to the trunk.

The JRI is the "weighted" average of the 20 ranges of joint movement chosen for measurement. Kaufman made sure that the entire procedure was accurate, reproducible on any given visit with no more varying of the JRI than plus or minus 0.3 of a JRI unit. The entire procedure took less than three minutes to perform. Table 1 (p.5) illustrates the clinical classification of joint dysfunction in terms of numerical values derived from the JRI.

For each clinical grade of joint dysfunction, patients were given a certain amount of niacinamide therapy. In subsequent journal articles, Kaufman standardized the dosages of niacinamide therapy for each category of joint dysfunction (will be discussed later). Regarding the 455 patients treated with niacinamide, Kaufman discovered that long term therapy usually increased joint mobility (an increase in the JRI) and decreased subjective complaints of articular pain, stiffness or subjective limitation of joint movement, crepitus, articular discomfort attributed by the patient to changes in weather, articular swelling, and articular deformities. When niacinamide therapy was discontinued there was a gradual worsening of symptoms and an eventual return to the pre-treatment state. The improvement with niacinamide therapy typically did not occur until after 3 to 4 weeks of treatment. Thereafter, continued improvement occurred for 1 to 3 years if niacinamide was continued. See Tables 2-4 (p.6) for a summary of the clinical data from Kaufman's 1949 book.

The data presented in **Tables 1-4** (p.17,18) does not exclude those patients who took the niacinamide in dosages less than the prescribed amount, or who did not follow the dosage schedule properly. It would have been possible to demonstrate greater improvement in the JRIs if these individuals were left out of the calculations.

In Kaufman's 1949 book he also described four complicating syndromes that frequently coexist with joint dysfunction, which were the following: delayed post-traumatic articular syndrome; the chronic allergic syndromes; the sodium retention syndrome; and the syndrome of psychogenically induced, sustained hypertonia of somatic muscle. It is beyond the scope of this paper to address these syndromes. For those interested in learning about these syndromes please refer to the text.

In a 1953 study published in the Connecticut State Medical Journal, Kaufman measured the JRI of 758 male and female patients (4-78 years of age).[5] Six hundred and six of these patients accepted therapy with niacinamide. A new dosing schedule of niacinamide was adopted for this particular study (**Table 5,** p.19). This standardized way of administering the niacinamide was continually used by Kaufman in subsequent clinical studies.

Kaufman calculated that the JRI rises from 6 to 12 units during the first month of therapy. Thereafter, the JRI continued to rise from 0.5 to 1.0 each month as long as the patient continued with the niacinamide. With adequate niacinamide therapy, most patients benefited in the following ways: increased alertness; decreased fatigability and irritability; a sense of well-being; the loss of certain digestive complaints, such as bloating and constipation; improvement in appetite; fewer aches in the joints, and less subjective awareness of the stiffness; the ability of the joints to withstand mechanical micro- and macrotrauma more efficiently than previously; some improvements in skin morphology and lingual mucous membrane pattern; no more liver enlargement and tenderness; no more tenderness on palpation over the abdominal aorta and iliac vessels; improvement in muscle strength; improvement in joint mobility; and occasionally joint deformities lessen in severity. Even though some of the 606 patients receiving the niacinamide therapy did not experience any subjective benefits, all of the treated patients demonstrated

an increase in JRI with continued therapy.

Kaufman published another paper in 1955, "The Use of Vitamin Therapy to Reverse Certain Concomitants of Aging" in the *Journal of the American Geriatric Society*.[6] In this study, 663 patients (aged 4-80) were treated with 900-4,000 mg/day of niacinamide in divided doses according to the criteria set forth in Table 5. The findings confirmed the results of the 1953 study. There was a rise in the JRI from 6 to 12 units during the first month of therapy, and a 0.5 to 1.0 unit increase each month the therapy was continued. Some patients reached a JRI value of 96.0, while other patients stabilized at 86.0 or better. The addition of vitamins C, D, A, or other B vitamins did not enhance the improvement in joint mobility, as achieved by the niacinamide. Nor did the addition of these vitamins retard the gains in JRI induced by niacinamide therapy.

Not all patients on niacinamide experienced improvement in muscle function. However, when niacinamide was combined with thiamin and riboflavin (and in some instances 2.4 grams of choline dihydrogen citrate in divided doses) improvement in muscle function was attained. Choline improves muscle power by supplying labile methyl groups for enzymatic and metabolic processes. In 70% of patients niacinamide treatment (alone or in combination with other vitamins) improved muscle function significantly. In 30% of patients no improvement in muscle function was demonstrated regardless of the combination of vitamins employed. Improvements in agitation, depression, and balance sense were improved with niacinamide alone, or in combination with vitamins B_1, B_6, and B_{12}. In 150 of the 663 patients on niacinamide therapy, vitamin C (in doses of 1,500 to 2,000 mg/day in divided doses) was added to the program, and capillary fragility progressively became less marked over a period of three to six months of treatment. According to Kaufman, this study demonstrated that vitamin therapy can improve joint mobility, muscular working capacity and strength, dysequilibrium, capillary strength, and mental decline, which are concomitants of aging.

A 1983 article by Kaufman in the *Journal of the International Academy of Preventive Medicine* reviewed his extensive clinical experience and findings with niacinamide therapy.[7] He cited many cases of patients on niacinamide therapy for as long as twenty years. In all his years of using niacinamide (amounting to several thousand patient-years of experience) he observed no untoward effects with this therapy. Kaufman noted that the half-life of niacinamide is relatively short and its renal clearance is relatively high. For this reason, it must be given at frequent, but regular intervals. For example, 250 mg taken every three hours for six daily doses is more effective than taking 500 mg three times per day even though the total dose (1,500 mg/day) is the same. Niacinamide repairs articular cartilage by inducing metabolic changes in articular cartilage cells (i.e., the chondrocytes) thus enhancing the ability of cartilage to repair itself. Continuous therapy with niacinamide is necessary to sustain improvement in joint mobility. In fact, as the JRI rises there will be a decrease in the patient's sedimentation rate (i.e., a general marker of inflammation). According to Kaufman, it is best not to reduce the frequency and dose of niacinamide as the JRI improves. It is important to maintain the patient on the dosing schedule based on the pre-treatment JRI. If niacinamide therapy is stopped, the gains are lost and the patient returns to the pre-treatment condition in a few weeks.

A double-blind, placebo controlled study on the effect of niacinamide on osteoarthritis was published in 1996 in the journal *Inflammatory Research*.[8] This study was developed with the assistance of Kaufman and the results confirmed many of his previous clinical observations. The study involved 72 patients with OA who were randomized to treatment with either niacinamide or placebo. The patients took niacinamide (500 mg) or placebo tablets 6 times each day for three months. After three months, the global arthritis impact improved by 29% in the niacinamide group and worsened by 10% in the placebo group. There was no change in pain levels,

Table 1. Clinical Classification of Joint Dysfunction

Degrees of Joint Dysfunction	Joint Range Index
No joint dysfunction	96-100
Slight joint dysfunction	86-95
Moderate joint dysfunction	71-85
Severe joint dysfunction	56-70
Extremely severe joint dysfunction	55 or less

Adapted from: Kaufman W: The common form of joint dysfunction: its incidence and treatment. Brattelboro, VT. E.L. Hildreth & Company. 1949; p.21.

Table 2. The Means of the JRIs for Each Successive Five-Year Age Group of Patients in Series 1-4

Ages	Series 1	Series 2	Series 3	Series 4
1-5	91.2	97.1	97.1	99.9
6-10	88.3	93.2	95.9	96.9
11-15	83.5	90.9	92.2	94.1
16-20	84.1	90.1	91.1	93.8
21-25	81.1	87.6	89.6	89.7
26-30	79.1	85.5	88.3	89.4
31-35	78.1	84.5	86.9	89.3
36-40	77.3	85.6	87.2	89.6
41-45	74.6	83.2	85.5	88.9
46-50	71.6	81.5	84.0	86.9
51-55	70.5	80.5	83.8	86.9
56-60	68.6	79.4	83.5	86.1
61-65	65.7	78.0	81.0	83.5
66-70	61.9	74.0	77.9	80.3
71-75	67.7	76.8	81.3	82.5
76-80	59.3	71.7	78.2	83.1

Series 1: Before niacinamide therapy (455 male and female patients).
Series 2: With less than two months of niacinamide therapy (266 male and female patients).
Series 3: With various periods of niacinamide therapy, maximum JRIs, as of September 1, 1947 (298 male and female patients).
Series 4: With various periods of niacinamide therapy, maximum JRIs, as of September 1, 1948 (367 male and female patients).

*Adapted from: Kaufman W: The common form of joint dysfunction: its incidence and treatment. Brattelboro, VT. E.L. Hildreth & Company. 1949; p.188.

Table 3. Mean JRIs Patients in Series 1-4

	Series 1	Series 2	Series 3	Series 4
Total Population (Males and Females)	74.50	83.11	85.58	87.95
Males only	72.53	80.88	83.47	86.41
Females Only	75.76	84.33	86.93	88.92

Series 1: Before niacinamide therapy (455 male and female patients).
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*Adapted from: Kaufman W: The common form of joint dysfunction: its incidence and treatment. Brattelboro, VT. E.L. Hildreth & Company. 1949; p.188.

Table 4. Severity of Joint Dysfunction

Ages	Before Niacinamide (455 patients)	After Niacinamide (367 patients)
1 to 10	slight	none
11 to 50	moderate	slight
51 to 60	severe	slight
61 to 80	severe	moderate

*Adapted from: Kaufman W: The common form of joint dysfunction: its incidence and treatment. Brattelboro, VT. E.L. Hildreth & Company. 1949; pp.183-187.

but the group on niacinamide was able to reduce their usage of anti-inflammatory medications. Those on niacinamide had a reduction in erythrocyte sedimentation rate by 22% and experienced an increase in joint mobility by 4.5 degrees over controls. The mean aspartate aminotransferase levels in the niacinamide group rose by 20% over baseline, but never increased to dangerous or concerning levels. Forty percent of the patients in the niacinamide group experienced mild gastrointestinal disturbances (i.e., eructations, nausea, or loose stools) compared to 28% of the patients in the placebo group, but these side effects were well managed by having them take the tablets with food or extra fluids. The mechanism explaining the therapeutic effectiveness of the vitamin was related to the raising of coenzymes NAD and NADP in the synovial fluid and within the cartilage matrix itself. This would provide energy and nucleic acids through non-oxidative processes, which is vital for cartilage repair in the deeper layers of the matrix and might have the net effect of increasing cartilage repair rates.

Table 5. Dosage Schedule of Niacinamide Based on the Joint Range Index

Joint Range Index	Clinical Status	Oral Dosage Schedules of Niacinamide Per Day	Range of Total Mg of Niacinamide taken per 24 hours
96-100	No joint dysfunction	–––	–––
86-95	Slight joint dysfunction	150-250 mg every 3 hours for 6 doses	900-1,500
71-85	Moderate joint dysfunction	250 mg every 3 hours for 6 doses; or 250 mg every 2 hours for 8 doses	1,500-2,000
56-70	Severe joint dysfunction	250 mg every 2 hours for 8 doses; or 250 mg every 1.5 hours for 10 doses	2,000-2,500
55 or less	Extremely severe joint dysfunction	50 mg every 1.5 hours for 10 doses; or 250 mg every hour for 16 doses	2,500-4,000

*Adapted from: Kaufman W: Niacinamide therapy for joint mobility. Conn State Med J, 1953;17: p.586.

The benefits of niacinamide therapy can be summed up by Kaufman himself. In a correspondence with him in 1998 concerning the systemic effects of niacinamide, he responded with the following remarks:[9]

"The 250 mg dose of niacinamide is the dose to use. The 500 mg dose is from 40 to 50% less effective than the 250 mg dose. Don't use niacinamide which is manufactured in hard gelatin capsules; they do not release niacinamide at the rate required. Some brands of niacinamide have excipients in them which act as preservatives and increase the shelf life; but some people have severe adverse reactions from taking these brands.

Niacinamide has ungated access to the brain. When it enters the brain, it has a strong affinity for the benzodiazepine receptors and causes a desirable calmative effect which you have observed. But it also improves other functions of the central nervous system.

Starting on page three of my 1943 book describing aniacinamidosis, you will see the psychological patterning of central nervous system impairments in function which give rise to a myriad of psychological symptoms.

Please keep in mind that it is a systemic therapeutic agent. It helps improve joint mobility in arthritis, it reduces and often completely eliminates arthritic pain, it improves balance sense, muscle strength and maximal muscle working capacity, it heals broken DNA strands and does many other therapeutic tasks."

Kaufman's research indicates that the

ideal dose should be 250 mg in tablet form, taken anywhere from 3-16 times each day, depending on the severity of OA. The severe cases demand more frequent dosing and higher daily dosages. Unfortunately, my clinical experience with this method of dosing has not proven to be one that facilitates compliance. The majority of my patients have found it to be too cumbersome and difficult to follow. For the past decade, I have relied upon the dosing schedule that was used in the study by Jonas et al,[8] which involves taking 500 mg niacinamide tablets six times each day. Since mild gastrointestinal side effects can occur, it is best to advise patients to take each dose of niacinamide with some food.

Since most clinicians would not be determining the JRI scores of patients, how should clinicians track the impact from niacinamide treatment? There are various psychometrically validated OA questionnaires (see Veenhof et al[10] for a systematic review of the various questionnaires available to clinicians). It is recommended that clinicians have patients' complete an appropriate questionnaire at baseline and then at predetermined intervals over the course of treatment.

2. Elimination of Solanine-Containing Foods

Dr. Abram Hoffer reported that about 10% of arthritics have allergic reactions to the solanine family of plants, which include potatoes, tomatoes, peppers, eggplant, and tobacco.[11] The percentage of arthritic patients who are sensitive to the solanine family of plants might be significantly greater than 10%. A 1982 study published in the *Journal of the International Academy of Preventive Medicine* demonstrated significant improvements in over 70% of 5,000 (> 3,500) arthritic patients after having eliminated solanine-containing foods from their diets.[12] The study duration was seven years and data was obtained through questionnaires. More than 70% of the patients reported gradually increasing relief from aches and pains while some patients even noted improvements in the disfigurement associated with their arthritis as well.

I recommend that all patients with OA eliminate the solanine family of plants for a period of 4-6 weeks to see if their symptoms improve. Sometimes patients obtain considerable relief from their OA symptoms by simply eliminating these items. An alternative strategy is to instruct the patient to follow a hypoallergenic diet for at least one to two weeks. After the elimination period, the patient would consume solanine-containing foods during all main meals for at least 1-2 days. Symptoms need to be recorded during the day that foods are reintroduced. If the OA symptoms worsen from the reintroduction of solanine-containing foods, then the patient is sensitive to these foods and should avoid them on an ongoing basis.

Conclusion

It is doubtful that rigorous controlled trials will be ever conducted on these interventions. Given the safety, putative efficacy, and low cost of these interventions, they should be offered to patients that want complementary and/or alternative options for OA.

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Competing Interests

The author declares that he has no competing interests.

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