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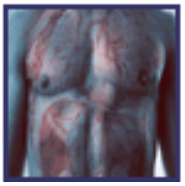
EDITORIAL

Noni Juice -- The Passing of Another Panacea?

Are you as amazed as I am about how popular some nutritional supplements and herbal products can become on the flimsiest of evidence? Over the years we have witnessed countless "miracle cures" and panaceas. One of my all time favorites to cite as an example is noni juice. Is this once immensely popular product about to drift away? Apparently so, based upon waning interest. [read the article]

By Michael T. Murray, N.D.

FEATURE ARTICLE



N-acetylcysteine, a Review of Efficacy in Pulmonary Disease

N-acetylcysteine (NAC) has an extensive and sometimes controversial history of use as a mucolytic in the treatment of bronchopulmonary disease. It has been recommended for both acute and chronic conditions including emphysema with bronchitis, chronic asthmatic bronchitis, tuberculosis, bronchiectasis and primary amyloidosis of the lung. NAC has also been used as a chelator for heavy metals including mercury, copper and zinc, and it has theoretical application in HIV infection (by inhibiting replication in vitro,) cancer, and heart disease. Since NAC and/or its metabolites serve as precursors for glutathione (GSH,) its recommended use is biochemically supported when high pulmonary and/or hepatic oxidant stress is present or suspected. Empirical as well as biochemical evidence also supports the use of NAC in pulmonary conditions. This experimental evidence is reviewed and information specific to NAC use in pulmonary disease is presented. [read the article]

By Kasra Pournadeali, N.D.

CLINICAL COMMENTARY

Does NAC offer any greater benefit than vitamin C?

As I read the review by Dr. Pournadeali on the possible clinical applications of N-acetylcysteine, I cannot help but wonder if NAC is really all that more effective than plain old vitamin C. Along with vitamin E, vitamin C is one the body's premier antioxidants. Because physicians and the public are constantly being bombarded by new substances being claimed to be more potent in antioxidant protection than these nutrients, there is a growing tendency to forget just how effective these simple nutritional agents are in protecting the body from oxidative and free radical damage. [read the article]

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Noni Juice -- The Passing of Another Panacea?

editorial

By Michael T. Murray, ND

Introduction
Are you as amazed as I am about how popular some nutritional supplements and herbal products can become on the flimsiest of evidence? Over the years we have witnessed countless "miracle cures" and panaceas. One of my all time favorites to cite as an example is noni juice. Is this once immensely popular product about to drift away? Apparently so, based upon waning interest.

What is noni?
Noni (*Morinda citrifolia*) or Indian mulberry is a small tree native to Polynesia that usually grows to a height of 10 feet high. The 4 inch sized fruit is the portion of the plant used. It starts out green then turns yellow.

What was noni used for historically?
Traditional Polynesian healers have used the fruit of the noni plant for just about everything from a tonic drink to mending broken bones, but it is said that a person won't take it until they are too sick and desperate because of its strong unpleasant odor and bitter taste. The bark yields a red dye while the root yields a yellow one. Both colors were used in the ceremonial outfits of Hawaiian chiefs.

Are there any clinical studies with noni juice?
No. In the early 1990s, noni juice became heavily marketed in the United States primarily through network marketing companies. However, despite tremendous claims and testimonials, there is little scientific documentation for noni. Yet, even to this day It remains a very popular Item even In health food stores.

What are the key constituents?
The key components in noni appear to be polysaccharides and a compound known as damnacanthal.[1-3] An alkaloid, given the name "xeronine," has been claimed to be an important constituent by the developer of a commercial product, but there has been no confirmation by independent researchers. Animal and in vitro studies have shown some anticancer and immune enhancing activity and an earlier animal study seemed to indicate the fruit exerts a mild sedative effect.[4-6] Specifically, the polysaccharide component has been shown to increase the release of immune enhancing compounds that activate white blood cells to destroy tumor cells while damnacanthal is thought to be responsible for producing sedative effects in animal studies.

How is noni usually taken?
The usual recommendation is the equivalent of four ounces of noni juice one half hour before breakfast (effectiveness is thought to be best on an empty stomach). Commercial products are now available that have either eliminated the odor, altered the taste, or made it available as an extract in tablets or capsules to increase palatability. For liquid concentrates the typical recommendation is two tablespoons daily. For powdered extracts the typical recommendation is 500 to 1,000 mg daily.

Are there any side effects or interactions?
There are no known side effects, although it has been suggested that noni be taken on an empty stomach and that it not be taken with coffee, tobacco or alcohol. Since the use of noni during pregnancy and lactation has not been adequately studied, it is recommended that it not be used during these times.

Conclusions
Noni may prove to be a phenomenal plant-based medicinal agent. Unfortunately, until it is tested more thoroughly soon it will probably fall by the wayside of many other possibly effective natural products. After all, experience tells us that unless a natural product stands up to scientific scrutiny it has little staying power. With all the money made on noni juice sales, certainly there is enough for some preliminary human trials at the very least.

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N-acetylcysteine, a Review of Efficacy in Pulmonary Disease

feature article

By Kasra Pournadeali, N.D.

Introduction

N-acetylcysteine (NAC) has an extensive and sometimes controversial history of use as a mucolytic in the treatment of bronchopulmonary disease [1]. It has been recommended for both acute and chronic conditions including emphysema with bronchitis, chronic asthmatic bronchitis, tuberculosis, bronchiectasis and primary amyloidosis of the lung [2,3]. NAC has also been used as a chelator for heavy metals including mercury, copper and zinc [4], and it has theoretical application in HIV infection (by inhibiting replication in vitro,) cancer, and heart disease [5]. Since NAC and/or its metabolites serve as precursors for glutathione (GSH,) its recommended use is biochemically supported when high pulmonary and/or hepatic oxidant stress is present or suspected. Empirical as well as biochemical evidence also supports the use of NAC in pulmonary conditions. This experimental evidence is reviewed and information specific to NAC use in pulmonary disease is presented.

Actions NAC's has many effects at the cellular and tissue level. Its sulfhydryl (SH) groups directly split disulfide linkage of mucoproteins thereby reducing viscosity of pulmonary secretions [2,3], it has direct antioxidant effects [5], and it maintains and restores hepatic, pulmonary and RBC concentrations of glutathione (GSH) [2,6,7,8,9]. Through its mucolytic action NAC has also been demonstrated to increase pulmonary oxygenation and oxygen saturation [1,10].

Structure & Pharmacokinetics

NAC is the N-acetyl derivative of the naturally occurring amino acid, cysteine. It is a white crystalline powder with the molecular formula C5H9NO3S, a molecular weight of 163.2 and a chemical name of N-acetyl-L-cysteine. NAC is readily absorbed from the gastrointestinal tract following oral administration, where it undergoes rapid deacetylation to yield cysteine or oxidation to yield diacetylcystine. Its mucolytic activity increases with pH, with optimal pH range being 7-9 [2]. Peak plasma concentrations of NAC are achieved 1-2 hours following oral dose, with a demonstrated bioavailability of 9.1% and protein binding of approximately 50% after 4 hours [11]. NAC's terminal half-life is 6.25 h and 5.58 hours for oral and intravenous routes respectively [12].

Clinical Applications

Many studies support the use of NAC in pulmonary disease. In a study of eighteen patients with fibrosing alveolitis (a condition characterized by excessive oxidative stress) treated with oral NAC, objective parameters including pulmonary function tests (PFTs) glutathione status, and methionine sulfoxide content of bronchoalveolar lavage fluid (BALF) were measured. After 12 weeks of 600mg TID dosage, increases in total glutathione (from 3.43 +/-0.30 microM vs. 4.20 +/-0.66 microM, $p<0.05$), and reduced glutathione (2.58 +/-0.24 microM vs. 3.42 +/- 0.54 microM, $p<0.005$), were noted showing improved antioxidant capacity. Methionine sulfoxide (an indicator of alveolar oxidative stress) decreased from 6.83 +/-0.71 vs. 4.60 +/-0.40, $p<0.005$. NAC administration also significantly improved PFTs thereby improving patient clinical as well as antioxidant status [13].

A study by Meyer et al. found potential benefit of NAC use in idiopathic pulmonary fibrosis (IPF), a condition also characterized by an increased pulmonary oxidative burden and insufficient glutathione. Seventeen non-smoking patients with biopsy-confirmed IPF were given oral NAC 600mg TID for 5 days. BALF and epithelial lining fluid (ELF) specimens, and the glutathione levels in each were quantified spectrophotometrically pre and post therapy. Pretherapy levels of glutathione were found to be lower than normal, while post treatment levels of glutathione increased in both ELF and BALF, to a statistically significant level in BALF suggesting that NAC may provide improve antioxidant status through improved glutathione status in IPF [7].

Evidence also suggests that NAC may have a modulating effect on pulmonary alveolar macrophages (AMs) and polymorphonuclear leukocytes (PMNs). With the understanding that activated AMs and PMNs produce reactive oxygen species that can lead to self-lysis, an experiment was conducted to evaluate NAC's effects on this process. The in-vitro study by Oddera et al. found that NAC not only enhanced the bactericidal activity of AMs and PMNs against Staphylococcus aureus, but without increased proportions of dead phagocytes. Their study implied that NAC might improve the bactericidal activity of alveolar WBCs while preventing increased WBC lysis through its antioxidant effects [14].

Another area where NAC use has shown potential benefit is in reducing sputum retention following pulmonary surgery. In a single-blind crossover study by Gallon involving 10 post-thoractotomy patients, nebulized NAC or normal saline was administered. Following NAC administration, sputum viscosity and difficulty of expectoration decreased, while the amount of sputum expectorated, and oxygen saturation improved. No changes were noted after nebulization of normal saline, demonstrating NACs superiority in the parameters measured [1].

Perhaps the pulmonary application where NAC has been most studied and found most effective is in bronchitis, asthma and emphysema. Rasmussen, and Glennow studied the effects of oral administration of NAC (300mg BID,) or placebo in 91 chronic bronchitis patients from nine centers with a double-blind approach over six months. They found that parameters including number of exacerbation days and the number of sick leave days (due to exacerbations) were lower in the NAC vs. the placebo group. After four months, the number of sick leave days was NAC: 173 vs. Placebo: 456, while the number of exacerbation days was NAC: 204 vs. Placebo: 399. After six months, the NAC group again showed a lower number of sick leave days and exacerbation days (NAC 260/ Placebo 739; and NAC 378/ Placebo 557 respectively). Although the authors note that statistical significance was not reached in every parameter, perhaps due to a small study population, their results do demonstrate NACs potential efficacy in reducing the number of sick leave days and exacerbations in patients with chronic bronchitis [15].

In an open surveillance study, Gerards and Vits also evaluated the efficacy of NAC in patients with bronchitis. Oral NAC 600mg in a single daily dose was used in 3076 patients from 744 centers. The treatment was not only well tolerated, but the treatment group experienced considerable improvement in a symptom complex consisting of cough, amount and quality of sputum, expectoration, and dyspnea [16].

Volkl and Schneider also reviewed the efficacy of NAC in the treatment of patients with bronchitis. Their observational study of 2510 patients with either chronic or acute bronchitis, some with concurrent asthma and emphysema, reviewed the efficacy of 200mg oral NAC TID for 4 weeks. Objective and subjective parameters including forced expiratory volume (FEV1) cough, amount and nature of expectorant and mucolytic effect all showed clear improvement in both chronic and acute bronchitis groups on NAC therapy [17]. Their data showing improvement in both subjective parameters and FEV1 provides further evidence for the use of NAC in bronchitis.

Dosage/ Toxicity

Recommended dosage of NAC has been variable. Oral dosage ranges from 200mg to 3000mg daily have been used in pulmonary conditions while a 140mg/kg-loading dose followed by fourteen 70mg/kg-maintenance doses every 4 hours have been used for acetaminophen toxicity. Typical dosing for pulmonary conditions, and preventing hepatic glutathione depletion in conditions of increased oxidant stress is 500mg 2-3 times a day between meals [2,3,18]. Nebulized NAC, is dosed at 1-10ml (20% solution) or 2-20mls (10% solution) 1-4 times a day depending on desired effect and patient response [2,3]. The nebulized tissue-specific delivery method is understandably preferred in the treatment of acute and chronic pulmonary conditions.

NAC appears to have extremely low toxicity, as animal studies using doses of 1000mg/kg/day provided no evidence of oncogenic activity [2]. NAC's LD50 is 7888mg/kg in mice and over 6000mg/kg in rats [5], however patients with advanced liver cirrhosis have decreased clearance of intravenously administered NAC, and optimal dosing in these patients needs to be determined individually [19].

NAC is not mutagenic in the Ames test, both with and without metabolic activation, and no adverse effects on fertility or teratogenic effects have been noted in animal studies at oral doses of 1000 and 500mg/kg/day respectively [3].

NAC is rated as pregnancy category B, and since it has been shown to cross the placenta with increased levels in newborn circulation following delivery, use in pregnancy is either not recommended or recommended only in cases of acetaminophen toxicity [3,20].

Adverse Effects/ Contraindications

NAC is extremely well tolerated at the doses recommended, with adverse reactions occurring in just 1.5% of patients [16]. Adverse reactions for nebulized NAC, when occurring, have included bad taste, stomatitis, nausea, vomiting, fever, rhinorrhea, drowsiness, clamminess, chest tightness, bronchoconstriction and headache [3]. Clinically overt acetylasthmatic bronchospasm can infrequently and unpredictably occur during use of the nebulized NAC, and therefore use in asthmatics should be closely monitored [3].

Anaphylactoid reactions have been reported with intravenous delivery of NAC, although in most cases no treatment or treatment with diphenhydramine alone allowed resumption of NAC therapy [21,22].

Oral administration of N-acetylcysteine, especially in the large doses needed to treat acetaminophen toxicity, may result in headache, nausea, vomiting and other gastrointestinal symptoms. Rash with or without mild fever has been observed rarely, but a low occurrence of side effects occurs with oral dosage patterns as listed for pulmonary conditions [3].

It is not clear if NAC is passed in breast milk, but it does cross the placenta resulting in therapeutic levels in the fetal bloodstream the effects of which are unascertained. The use of NAC during pregnancy and in nursing mothers therefore should be avoided unless addressing acetaminophen toxicity [3,20]. Even in such cases, NAC administration should take place in an inpatient setting with adequate support for untoward anaphylactoid reactions.

Use of therapeutic levels of NAC is not recommended for patients with liver cirrhosis as they have been shown to have decreased clearance and resultantly may have an increased tendency for anaphylaxis compared with controls [19].

Long term use of NAC can lead to depletion of Cu and Zn through its chelating effects, which in turn can cause impaired immune function or frequent infections, delayed wound healing and poor collagen integrity, anemia or poor glucose tolerance, undesirable lipid status, decreased appetite, etc [4,23,24,25]. Therefore it is important to supplement the diet with these minerals or foods rich in these minerals if employing long term use of NAC.

Conclusion

Although NAC has possible application in many conditions including HIV, influenza, cancer, and heart disease, it has documented

efficacy in acetaminophen and heavy metal toxicity. Likewise, its use in pulmonary disease is empirically well established. Studies document NAC's efficacy in not only reducing viscosity of sputum and sputum retention, but also in potentially modulating alveolar macrophage and polymorphonuclear leukocyte activity thereby providing benefit in pulmonary infection, and/or conditions of increased pulmonary oxidant stress. NAC is well tolerated with a low incidence of side effects and toxicity, although its use in pregnancy, nursing mothers or patients with liver cirrhosis is not advised. It has documented efficacy in idiopathic pulmonary fibrosis, and fibrosing alveolitis, typically found following radiation therapy. NAC has been shown to improve subjective as well as objective parameters in chronic and acute bronchitis, as well as asthma, and emphysema, and therefore may be of benefit in the management of patients with these acute or chronic pulmonary conditions.

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Does NAC offer any greater benefit than vitamin C?

commentary

As I read the review by Dr. Pournadeali on the possible clinical applications of N-acetylcysteine, I cannot help but wonder if NAC is really all that more effective than plain old vitamin C. Along with vitamin E, vitamin C is one the body's premier antioxidants. Because physicians and the public are constantly being bombarded by new substances being claimed to be more potent in antioxidant protection than these nutrients, there is a growing tendency to forget just how effective these simple nutritional agents are in protecting the body from oxidative and free radical damage.

Vitamin C: An overview
Vitamin C works as antioxidant in aqueous environments in the body - both outside and inside human cells. It is the first line of antioxidant protection in the body and is the body's most important nutritional antioxidant. Vitamin C's primary antioxidant partners are vitamin E and carotenes as these antioxidants are fat-soluble. Vitamin C also works along with antioxidant enzymes such as glutathione peroxidase, catalase, and superoxide dismutase. Vitamin C is also responsible for regenerating oxidized vitamin E in the body, thus potentiating the antioxidant benefits of vitamin E. Vitamin C is the premier antioxidant in the lung.

Vitamin C vs. NAC: A direct comparison
For our first step in comparison, let's compare the benefits of vitamin C versus N-acetyl-cysteine in the ability raise tissue glutathione levels. Along with vitamin C and E, glutathione assumes a critical role in defense against free radical damage. Individuals with a hereditary deficiency of glutathione due to a defect in synthesis, have markedly increased cell damage.

In an effort to increase antioxidant status in individuals with impaired glutathione synthesis, a variety of antioxidants have been used including: glutathione, 2-mercaptopropionyl-glycine, vitamin E, vitamin C, and N-acetylcysteine (NAC). Of these agents, only vitamin C and NAC have been able to offer some possible benefit. To determine the relative effectiveness of vitamin C vs. NAC, a 45-month-old girl with an inherited deficiency of glutathione synthesis was recently followed before and during treatment with vitamin C or NAC. High doses of vitamin C (500 mg or 3 g per day) or NAC (800 mg per day) were given for one to two weeks. Measurements of glutathione (GSH) levels indicated that 3 g per day of vitamin C increased white blood cell GSH four-fold and plasma GSH levels 8-fold. NAC also increased white blood cell (3.5-fold) and plasma (2- to 5-fold). Based on these results, it was decided that vitamin C would be given for one year at the 3 g per day dosage. At the end of a year glutathione levels remained elevated and the hematocrit increased from a baseline 25.4% to 32.6% and the number of immature red blood cells (reticulocyte count) decreased from 11% to 4%. These results indicate that vitamin C can decrease cellular damage in patients with hereditary glutathione deficiency and is more effective and less expensive than NAC.[1]

The significance of these results to the general population is that vitamin C may offer the benefits being attributed to NAC at only a slightly reduced cost. To put this in perspective, a daily dosage of 3 g of vitamin C costs about $10.00 per month while a dosage of 1 g of NAC would cost about $20.00.

There is more to this story. Over the past 5 to 10 years the use of NAC as an antioxidant has become increasingly popular among nutritionally-oriented physicians and the public, but is this use valid?

There is a biochemical rationale for this practice. It is thought that NAC acts as a precursor for glutathione and, therefore, should raise tissue glutathione levels. While supplementing the diet with high doses of NAC may be beneficial in cases of extreme oxidative stress (e.g., AIDS), it may be an unwise practice in healthy individuals. The reason? One study indicated that when NAC was given orally to 6 health volunteers at a doage of 1.2 grams per day for 4 weeks, followed by 2.4 grams per day for an additional two weeks, it actually increased oxidative damage by acting as a pro-oxidant.[2]

Compared with controls the concentration of glutathione in NAC treated subjects was reduced by 48% and the concentration of oxidized (inactive) glutathione was 80% higher. Oxidative stress actually increased by 83% in those receiving NAC. And there was no sign of antioxidant effects. From the results of this study it can be concluded that at high doses NAC may act as a pro-oxidant in healthy subjects. Unfortunately, 1.2 grams per day is not an unreasonable dosage to attain from commercially available sources of NAC.

Conclusion
Clearly NAC has been shown to exert clinical usefulness. However, what is not clear is how much greater of an effect it really has compared to vitamin C as well as is appropriateness of use in healthy individuals. Hopefully, further research will answer these questions.

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Vitamin E not effective in severe heart disease

Vitamin E intake is associated with a lower risk of coronary heart disease and atherosclerosis, but its therapeutic effect in severe heart disease has not been adequately studied. In a double-blind study, a total of 2545 women and 6996 men 55 years of age or older who were at high risk for cardiovascular events because they had cardiovascular disease or diabetes in addition to one other risk factor were enrolled. These patients were randomly assigned to receive either 400 IU of vitamin E daily or matching placebo and either an angiotensin- converting-enzyme inhibitor (ramipril) or matching placebo for a mean of 4.5 years (the results of the comparison of ramipril and placebo are reported in a companion article). The primary outcome was a composite of myocardial infarction, stroke, and death from cardiovascular causes. The secondary outcomes included unstable angina, congestive heart failure, revascularization or amputation, death from any cause, complications of diabetes, and cancer. A total of 772 of the 4761 patients assigned to vitamin E (16.2 percent) and 739 of the 4780 assigned to placebo (15.5 percent) had a primary outcome event (relative risk, 1.05; P=0.33). There were no significant differences in the numbers of deaths from cardiovascular causes (342 of those assigned to vitamin E vs. 328 of those assigned to placebo; relative risk, 1.05), myocardial infarction (532 vs. 524; relative risk, 1.02), or stroke (209 vs. 180; relative risk, 1.17). There were also no significant differences in the incidence of secondary cardiovascular outcomes or in death from any cause. The conclusion of this study was that in patients at high risk for cardiovascular events, treatment with vitamin E for a mean of 4.5 years had no apparent effect on cardiovascular outcomes. Although these results were largely publicized, it must be kept in mind that vitamin E, like many nutritional supplements and antioxidants, is probably more important in prevention than treatment.

Reference:
Yusuf S, Dagenais G, Pogue J, Bosch J, Sleight P. Vitamin E supplementation and cardiovascular events in high-risk patients. The Heart Outcomes Prevention Evaluation Study Investigators. N Engl J Med 2000;342(3):154-60.

The effect of dietary fiber on PSA

To assess whether high fiber diets influence serum prostate specific antigen (PSA) a randomized crossover controlled trial was performed on 14 healthy men with hyperlipidemia on diets containing foods high in soluble or insoluble fiber and approximately 25 to 30 gm. dietary fiber per 1,000 kilocalories. Serum PSA, free testosterone and estradiol, and fecal bile acid and neutral sterol excretion were evaluated at the beginning and end of the four-month trial. Mean serum PSA was lower with the soluble than the insoluble fiber diet (0.07+/-0.03 ng./ml., P=0.035). No treatment difference was seen in free testosterone or estradiol, although the latter decreased significantly with the insoluble fiber diet (9+/-3 pmol./l., P=0.004). After 16 weeks total fecal bile acid output was greater with the soluble (341+/-56 mg. daily) compared to the insoluble (203+/-35, p = 0.001) fiber diet but no differences were seen in fecal neutral sterol elimination. The treatment difference in fecal lithocholic acid output related to the difference in serum PSA (R=0.57, P=0.035).

These results indicate a small but statistically significantly lower serum PSA was seen in healthy men consuming soluble fiber, which was not related to changes in serum sex hormones but was related to the increased lithocholic acid output as a possible marker of increased fecal steroid elimination. The effect of soluble fiber on prostatic disease may warrant further investigation.

Reference:
Tariq N, Jenkins DJ, Vidgen E, et al. Effect of soluble and insoluble fiber diets on serum prostate specific antigen in men. J Urol 2000;163(1):114-8.

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Coumarin in arm lymphedema following breast cancer surgery

Coumarin (1,2-benzopyrone) is a component of several medicinal plants historically used in cancer including dong quai (Angelica sinensis), sweet clover (Meliotus officinalis) and red clover (Trifolium pratense). Coumarin, which possesses no anticoagulant activity, should not be confused with Coumadin (warfarin). Numerous studies over the past twenty years indicate that coumarin and other benzopyrones such as flavonoids are quite helpful in improving lymphedema. A recent double-blind study indicated that coumarin may reduce the development of arm lymphedema following breast cancer surgery.

In a randomized, parallel group study, the clinical efficacy of coumarin 90 mg/day (Group A) was compared with 135 mg/day (Group B) in 77 women age 35-65 years with lymphedema of the upper limb secondary to surgery and irradiation for treatment of breast cancer. During 12 months of coumarin therapy, the arm volume of lymphedema and a clinical score (degree of arm edema, heaviness, hardness, and neuralgia/dysesthesia) were determined. In both groups, the volume of arm lymphedema decreased, the overall clinical score improved, and the overall efficacy of coumarin was similarly good or excellent; the differences between Groups A and B were non-significant. Only mild to moderate side effects of drug therapy were recorded. Coumarin prevents a spontaneous trend toward an increase in arm lymphedema after treatment of breast cancer, decreases the severity of local symptoms, and overall improves the quality of life with no significant difference between the 2 dosages

As coumarin is not commercially available in the U.S., butcher's broom (Ruscus aculeatus) can be used instead. The standard dosage of butcher's broom is based on the ruscogenin content. For best results the dosage of any butcher's broom preparation should supply 16.5 to 33 mg of ruscogenins 3 times daily.

Reference:
Burgos A, Alcaide A, Alcoba C, et al. Comparative study of the clinical efficacy of two different coumarin dosages in the management of arm lymphedema after treatment for breast cancer. Lymphology 2(1):3-10, 1999.

Ginkgo protects against macular degeneration

Preliminary evidence indicate that flavonoid-rich extracts of bilberry (Vaccinium myrtillus), Ginkgo biloba, or grape seed (Vitis vinifera) offer significant benefits in the prevention and treatment of age-related macular degeneration (ARMD). In addition to possessing excellent antioxidant activity, all three extracts have been shown to exert positive effects on retinal blood flow and function. Small clinical studies in humans have demonstrated that all three are also capable of halting the progressive visual loss of dry ARMD and possibly even improving visual function.

To investigate the functional protective effect of Ginkgo biloba extract (GBE) against retinal degeneration, Wistar rats were exposed for 24 hours to 1700-lux light after treatment with GBE. In the untreated group, electroretinograms showed that light exposure caused collapse of the b-wave sensitivity curves. Bmax was reduced by 51% at day 1 without subsequent recovery. Retinal morphometric analyses revealed that the outer nuclear layer thickness decreased markedly in the superior retina. In the treated group, light exposure had a weaker effect on sensitivity curves. The values of Bmax were not significantly different from those in the unexposed-untreated group, although K increased temporarily. Retinal morphometry was preserved. Thus, this study demonstrates that GBE affords functional protection against light-induced retinal damage and offers further support for its use in the battle against ARMD.

Reference:
Ranchon I, Gorrand JM, Cluzel J, et al. Functional protection of photoreceptors from light-induced damage by dimethylthiourea and Ginkgo biloba extract. Invest Ophthalmol Vis Sci 40(6):1191-9, 1999.

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Acupressure in nausea due to chemotherapy

Nausea due to chemotherapy is an extremely common side effect. Recently a study sought to compare differences in nausea experience and intensity in women undergoing chemotherapy for breast cancer between those receiving usual care plus acupressure training and treatment and those receiving only usual care. The study was a single-cycle, randomized clinical trial conducted in an outpatient oncology clinic in a major teaching medical center and a private outpatient oncology practice. Seventeen women participated in the study. The intervention included finger acupressure bilaterally at P6 and ST36, acupressure points located on the forearm and by the knee. Baseline and post-study questionnaires plus a daily log were used to collect data. The results of the study indicated significant differences existed between the two groups in regard to nausea experience P<0.01) and nausea intensity (P<0.04) during the first 10 days of the chemotherapy cycle, with the acupressure group reporting less intensity and experience of nausea. These results suggest that simple finger acupressure may decrease nausea among women undergoing chemotherapy for breast cancer.

Reference:
Dibble SL, Chapman J, Mack KA, Shih AS: Acupressure for nausea: results of a pilot study. Oncol Nurs Forum 2000;27(1):41-7.

Stress management in hypertension

Stress management intervention (SMI) was one of seven non-pharmacological approaches evaluated in Phase I Trials of Hypertension Prevention (TOHP-I) for efficacy in lowering diastolic blood pressure (BP) in healthy men and women aged 30 to 54 years with diastolic BP 80- 89 mm Hg. In the study, a total of 242 and 320 participants were randomized to SMI or an "assessment only" SMI Control, respectively, at four clinical centers. The SMI consisted of 37 contact hours in 21 group and two individual meetings over 18 months and included: training in four relaxation methods, techniques to reduce stress reactions, cognitive approaches, communication skills, time management, and anger management within a general problem-solving format. Standardized protocols detailed methods and timing for collecting BP, psychosocial measures, and urinary samples from both SMI and SMI Control participants. The results of the trial based indicated significant baseline to termination BP reductions were observed in both groups, but the net differences between the SMI and SMI Control groups' BP changes were not significant: -0.82 mm Hg for diastolic BP, and -0.47 mm Hg for systolic BP. However, sub-group analyses found a significant 1.36 mm Hg (P=0.01) reduction in diastolic BP relative to SMI Controls at the end of the trial for SMI participants who completed 61% or more of intervention sessions. These results indicate that the more compliant a patient is to SMI, the more likely they are to experience a reduction in blood pressure.

Reference:
Batey DM, Kaufmann PG, Raczynski JM, et al. Stress management intervention for primary prevention of hypertension: detailed results from Phase I of Trials of Hypertension Prevention (TOHP-I). Ann Epidemiol 2000;10(1):45-58.

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