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Who Needs Benzos? Lavender and Insomnia

By David Kiefer, MD

Dr. Kiefer is a Clinical Instructor, Family Medicine, at the University of Washington in Seattle; Clinical Assistant Professor of Medicine at the University of Arizona in Tucson, and Adjunct Faculty at Bastyr University in Seattle; he reports no consultant, stockholder, speaker's bureau, research, or other financial relationships with companies having ties to this field of study.

GORDON LIGHTFOOT SANG ABOUT IT, AND YOU HAVE SURELY SEEN its beautiful purple flower in gardens or its oil on the shelves of health food stores. Perhaps you have even enjoyed lavender as a part of the flavoring in a gourmet chocolate bar. But does it put you to sleep? This article will attempt to shed light on the topic of lavender and sleep, and comment on whether a simple eye pillow impregnated with lavender is sufficient or, if not, what formulations and doses might be most useful. Read on for the details about a plant whose traditional use is making its way into the modern herbal era.

History and Traditional Use

Lavender, both the whole plant and its isolated essential oil, has a long history of traditional use for a variety of medical conditions: externally for rheumatic conditions; internally for anxiety, insomnia, and gastrointestinal problems; and via aromatherapy for insomnia.[1-3] Other, vaguely described uses are for migraines, menstrual cramps, and asthma.[3] In addition, there are many properties listed for lavender associated with its essential oils. For example, some sources mention antibacterial, antifungal, and insecticidal actions for lavender oil.[4,5]

Botany and Pharmacology

Lavender is a member of the mint (Lamiaceae) family, and the most common species used medicinally is *Lavandula angustifolia*, often referred to as English lavender. Many other names exist, such as common lavender, French lavender, and garden lavender,[3] and other scientific names mentioned in the herbal and medical literature include *L. officinalis*, *L. vera*, *L. spica*, *L. dentata*, *L. latifolia*, *L. pubescens*, and *L. stoechas*.

L. angustifolia is native to the Mediterranean, Russia, Africa, and the Arabian peninsula,[1] but is cultivated extensively throughout Europe and the rest of the world. It is a shrub that grows to 2-3 feet tall, and has characteristically fragrant, purple flowers. Lavender oil is produced by steam distilling the fresh flowering tops of *L. angustifolia*, yielding two main compounds: linalool (20-50% of the essential oil) and linalyl acetate (30-40% of the essential oil), together with numerous other minor oils such as cis-ocimene, terpinene-4-ol, beta-caryophyllene, and lavandulyl acetate.[3,6] Other phytochemicals found in lavender include flavonoids, triterpenoids, and coumarins.[2]

Mechanism of Action

Some animal trials of lavender have begun to elucidate its mechanism of action. Regarding potential gastrointestinal effects, an in vitro study on guinea pig smooth muscle found that linalool acted as a spasmolytic, probably via a rise in intracellular cAMP, and that isolated linalool seemed to act similarly to the whole lavender oil.[7]

Other researchers have explored the effect of linalool on the excitatory neurotransmitter glutamate, documenting dose-related inhibition of glutamate binding in rat cerebral cortex, which the researchers claim as validation for the fact that linalool-containing plants are often used in traditional medicine as anticonvulsants and hypnotics.[8] Another trial tested lavender (diluted 1:60 in olive oil) orally in mice and found sedative effects on a battery of tests, as well as potentiation of the ability of pentobarbital to increase sleep time.[5] Other animal research has documented that orally administered lavender acts as a CNS depressant, as an anticonvulsant, and as a potentiator of the effect of chloral hydrate.[9]

One experiment on mice documented that aerosolized lavender oil (37% linalool, 42% linalyl acetate) caused a distinct decrease in observed motor activity as compared to untreated controls, and that lavender oil, linalool, and linalyl acetate were each able to inhibit the increase in motor activity that occurred when the mice were injected with caffeine.[9] The plasma concentration of linalool correlated directly with time after inhalation and was well below levels that the researchers listed from oral and dermal toxicity studies previously published.

Clinical Studies

There is a paucity of definitive clinical research for the use of lavender and, more specifically, lavender oil aromatherapy for sleep. One single-blind, crossover, randomized pilot study of 10 people with insomnia compared essential oil of lavender flowers to a sweet almond oil placebo for one week each, including a one-week washout period.[10] The oils were added to the cartridge of an aerosolizer that ran all night long, providing aromatherapy to the treatment and placebo groups. The lavender group showed a trend toward improvement ($P = 0.07$) in sleep quality as per the Pittsburgh Sleep Quality Index. Women, as well as men younger than 39 years old, had a slightly better response to lavender.

A small ($n = 4$) case series showed that aromatherapy with lavender oil significantly prolonged sleep when compared to baseline in three patients who recently stopped their pharmaceutical hypnotic medications, and even for one patient who wasn't taking any medication for sleep.[11]

The effects of lavender aromatherapy on humans were explored by one research team who focused on mood, relaxation, alertness, anxiety, EEG changes, and math computational skills in 40 adults randomized to either three minutes of lavender oil or rosemary oil aromatherapy.[12] Both groups had less anxiety and felt more relaxed ($P < 0.05$ and $P < 0.001$, respectively), but only the lavender group experienced improved mood ($P < 0.01$). Furthermore, both groups completed the math tasks faster, but only the lavender group improved in math accuracy ($P < 0.05$). EEG changes in the lavender group suggested increased drowsiness.

Other Uses

Lavender aromatherapy has been studied as a treatment for other maladies, including mood disorders, and

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to promote overall well-being, sometimes in conjunction with other therapies such as massage or in combination with other essential oils.[13,14] For example, 80 healthy women were given either grapeseed oil or lavender oil in a daily bath for two weeks, and were asked to fill out surveys quantifying mood and psychological well-being.[14] There was a significant effect on many parameters simply due to the experience of the daily bath, but the lavender group showed improvement in the anger-frustration subset ($P < 0.01$) and in reduced negative thinking about the future ($P < 0.01$). Other trials have documented benefits of lavender oil aromatherapy for agitation in dementia patients,[15,16] in hospice,[17] or in conjunction with bergamot and cedarwood oils to reduce anxiety associated with chemotherapy.[18]

In a clinical trial of lavender for treatment of depression, lavender tincture showed less of an effect than imipramine tablets, but the combination of lavender tincture and imipramine was more effective than imipramine alone.[19] In another study, lavender oil was added to bath water for perineal discomfort after childbirth, though no statistically significant difference was noted as compared to the control group.[20]

Dosage and Formulation

For insomnia and anxiety, most research has focused on lavender oil administered as aromatherapy, with the exact dose depending on the specific aerosolizer used. One easy and commonly used aromatherapy dosing regimen is 2-4 drops of lavender oil in 2-3 cups of boiling water.[1] Most experts recommend against the oral ingestion of essential oils due to potential contact irritation and hepatotoxicity; an infusion of lavender flowers or leaves can be safely ingested orally, however, because such compounds are dilute and presumed safe when not separated and concentrated as purified essential oils. The oral dose as an infusion is 1-2 teaspoons lavender flowers or leaves per cup of boiling water, steeped for 5-10 minutes, strained, and consumed when sufficiently cool.

Adverse Effects, Contraindications, and Drug Interactions

As mentioned above, a significant interaction with the barbiturate pentobarbital was observed in mice.[5] There are only rare reports in the literature of central nervous depression with the use of lavender oil aromatherapy in humans.[1] Furthermore, one well-respected source lists lavender as a Class I herb, that is, "Herbs which can be safely consumed when used appropriately."[21]

The acute oral toxicity (LD_{50}) in rats for lavender oil and linalool are 5 g/kg and 2.8 g/kg, respectively, and the acute dermal toxicity in rabbits (LD_{50}) is 5 g/kg and 5.6 g/kg, respectively.[9] This would translate into about 350 g of lavender oil, or about 1.5 cups.

Case reports warn of gynecomastia with exposure to lavender oil topically.[22] This occurred in a 4-year-old boy with regular use of a lavender herbal balm, a 10-year-old boy using a lavender and tea tree oil shampoo, and a 7-year-old boy with a history of using lavender-scented skin lotion and soap. A possible mechanism for these results was demonstrated by in vitro testing that revealed both estrogenic and anti-androgenic effects of lavender oil on several different cell lines.[22] Whether similar concerns will be raised for lavender aromatherapy remains to be seen.

In vitro testing on human skin fibroblasts and endothelial cells showed a dose-dependent toxicity of lavender oil, which the authors postulate may be the mechanism behind the irritation and inflammation (including contact dermatitis) occasionally encountered with many topically applied essential oils, including lavender.[23] In Japan, over a nine-year period, the rate of contact dermatitis with exposure to topical lavender oil was 3.7%.[24] Interestingly, at the end of the test period, there was a spike in reactivity to lavender oil (13.9% in the last year), which the researchers ascribe to increasing popularity of topical lavender oil. With increased exposure an increased incidence of sensitization among the population may have occurred.

Coumarins are present in the phytochemical analyses of lavender. This class of compounds is relatively common in the plant kingdom and, though chemically distinct from coumadin (warfarin), may theoretically have blood-thinning activity. Caution is advised in people who are concomitantly taking antiplatelet or anticoagulant medications.

Conclusion

Lavender, whole-plant and oil, has a long history of traditional use for a variety of medical conditions. For insomnia, there are in vitro and animal research data that suggest effects on neurotransmitter systems and behavior that could signify increased drowsiness and relaxation. These effects have been most significantly documented using lavender oil and its two main constituents, linalool and linalyl acetate. In humans, however, lavender oil aromatherapy has been researched for its hypnotic effect in only three low-quality trials; while initial results are promising, there is a clear need for further, methodologically sound investigation into the potential benefits and risks of lavender. One significant problem associated with lavender aromatherapy

research is devising a placebo arm for a fragrant plant like lavender.

Potential adverse reactions should be noted, specifically with respect to topical applications where dermatitis may occur (people may add lavender oil to their pillow, for instance) and, theoretically, when lavender is used concomitantly with other central nervous system depressants. For the former reason, aromatherapy using an aerosolizer is preferred over an eye pillow impregnated with lavender oil; lavender flowers and leaves in an eye pillow would be safe (very unlikely to cause dermatitis because of minimal spread of the oil) but less likely to volatilize the oils enough to lead to clinical effect.

Recommendation

The short-term use of lavender oil aromatherapy can be considered as a treatment for insomnia, provided that other pharmaceutical or dietary supplement central nervous depressants are not also in use. Pay attention to the development of adverse skin reactions and practice caution in people taking blood-thinning medications. There are convincing mechanistic data, in vitro evidence, and animal research for the use of lavender for insomnia, but a paucity of human data exist in this regard. An improvement on the few clinical trials investigating lavender for the management of insomnia will help to refine this recommendation. ❖

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The Role of Diet and Nutrition in Attention-Deficit/Hyperactivity Disorder

By Richard G. Petty, MD

Dr. Petty is Scientific Director, Promedica Research Center, Loganville, GA, and Adjunct Professor, Georgia State University, Atlanta, GA. Dr. Petty discloses that he is retained as a consultant by Astra Zeneca Pharmaceuticals and Janssen Pharmaceuticals and serves on the speaker's bureau for Astra Zeneca Pharmaceuticals, Janssen Pharmaceuticals, Abbott Pharmaceuticals, and Avanir Pharmaceuticals.

ATTENTION-DEFICIT/HYPERACTIVITY DISORDER (ADHD) is a common disorder of childhood, and most studies have shown that it is rarely something that a child grows out of. Most young people who have it will continue to have some symptoms—and some of the complications—of ADHD throughout life. This is sometimes missed for two reasons. First, the clinical features of ADHD are usually entirely different in girls and boys; and second, the symptoms and complications metamorphose and migrate as boys and girls become older.

There is a great debate in psychiatry and many allied professions concerning the nature of illness. The issue can be summarized very quickly: Are we medicalizing normal human variations? Fifty years ago unruly inattentive children were given detention or some other punishment. But now they receive a medical diagnosis and treatment with medication.

This is an important discussion that extends into some of the farthest reaches of human behavior: The argument goes that a person is not allowed to be shy, but is instead socially phobic and in need of a medicine. Another person is not bad, but has a personality disorder and needs hospital treatment rather than incarceration. We all have our own biases in answering those questions. However, in the case of ADHD we can apply a number of commonsense principles to show that it is a real clinical entity. First is good evidence from different types of brain imaging; there are predictable differences in the brains of most people with ADHD. Second is that if left untreated ADHD can cause suffering either to the individual or to other people, and suffering is an important criterion for calling something an illness. Third, untreated or inadequately treated ADHD gives rise to a number of complications.[1,2] *(See Table 1.)*

Thus, the recognition and treatment of ADHD is not a device to make children and adults more tractable, or to help people get better grades in school or have better job evaluations. If untreated, ADHD can have many devastating consequences. Unfortunately, few treatment studies—including medication studies—have examined these consequences, and clinicians need to be aware of these complications when recommending a course of action.

This is particularly important when faced with a question about nonpharmacological approaches instead of using medications. Research has shown that at least 50% of American families who receive treatment for ADHD in specialty clinics also use complementary or alternative medical therapies excluding diet.[3] Yet only 12% report this use to their clinicians. This article was prompted by a recent national survey that indicated that 92% of pediatricians had been asked by parents about complementary therapies for ADHD.

Prevalence

ADHD is common: 5-8% of all U.S. school-age children are estimated to have the disorder and in 36.3% of cases it persists into adulthood,[1] though some of the clinical manifestations may change over time. There is marked geographical variation in diagnosis, with the

Table 1

Complications of inadequately treated ADHD

- Academic problems
- Low self-esteem
- Relationship difficulties
- Increased rates of expulsion and dropping out from school
- Increased rates of substance abuse; earlier onset and less likely to quit in adulthood
- Increased rates of bicycle accidents
- Increased numbers of visits to the emergency room
- Increased rates of motor vehicle accidents
- Increased rates of driving under the influence
- Increased rates of sexually transmitted diseases
- Increased rates of unwanted pregnancy
- Increased rates of divorce
- Occupational and vocational difficulties; and ultimately achieve a lower occupational status than predicted
- Legal problems
- Supporting the young person through the bicycle accidents, emergency room visits, and substance abuse
- The personal and economic costs of continuing substance abuse
- Increased rates of parental divorce or separation
- Increase in sibling fights
- Increased parental absenteeism
- Reduced parental productivity

highest rates of diagnosis being in the northeastern United States.[4] Nearly 4.5% of American adults fulfill criteria for ADHD,[5] though some studies have found high rates of partial forms of ADHD (i.e., many adults learn to compensate for their difficulties).

First described more than 150 years ago, both the incidence and prevalence appear to be increasing. This increase does not appear to be a reflection of increasing awareness or changing diagnostic criteria, and there are a number of theories that attempt to explain it:

- Increasing environmental stress affecting the neuroplasticity of the growing brain
- Less and poorer quality sleep
- Increasing need to multi-task in different modalities
- Increasing demand to participate in more activities, which exceeds the attentional capacity of the brain
- Substance use and abuse, including stimulants
- Degradation of the food supply

Table 2 details the current DSM-IV-TR criteria for ADHD. The most important point is that the problem has to have been present before the age of 7, and has to be persistent. An additional problem in diagnosis is that ADHD is highly comorbid: Worry and anxiety disorders, conduct disorder, learning disabilities, oppositional defiant disorder, depression, bipolar disorder, and tics and Tourette's are all more common.

Table 2

DSM-IV-TR criteria for attention-deficit/hyperactivity disorder

Inattentiveness

Has a minimum of six symptoms regularly for the past six months:

- Symptoms are present at abnormal levels for stage of development
- Lacks attention to detail; makes careless mistakes
- Has difficulty sustaining attention
- Doesn't seem to listen
- Fails to follow through/fails to finish projects
- Has difficulty organizing tasks
- Avoids tasks requiring mental effort
- Often loses items necessary for completing a task
- Easily distracted
- Is forgetful in daily activities

Hyperactivity/Impulsivity

Has a minimum of six symptoms regularly for the past six months

- Fidgets or squirms excessively
- Leaves seat when inappropriate
- Runs about/climbs extensively when inappropriate
- Has difficulty playing quietly
- Often "on the go" or "driven by a motor"
- Talks excessively
- Blurts out answers before question is finished
- Cannot await turn
- Interrupts or intrudes on others

Additional Criteria

- Symptoms causing impairment present before age 7
- Impairment from symptoms occurs in two or more settings
- Clear evidence of significant impairment (social, academic)
- Symptoms not better accounted for by another mental disorder

Dietary Manipulations in ADHD

Dietary interventions are the most common type of complementary approach to the treatment of ADHD.

The three main dietary therapies for ADHD are: the Feingold diet, sugar restriction, and avoiding allergens and toxins in food. There are others, but these are the most widely used and also the ones that have been most widely studied. These diets are sometimes used in combination.

The Feingold Diet

The Feingold diet is both the best known and most studied dietary intervention for ADHD. It aims to eliminate three groups of synthetic food additives and one class of synthetic sweeteners:

- Petroleum-based synthetic colorings, FD&C and D&C;
- Synthetic flavorings; the phenolic compounds butylated hydroxyanisole (BHA), the related compound butylated hydroxytoluene (BHT), and tert-Butylhydroquinone (TBHQ)
- The artificial sweeteners aspartame, neotame, and alitame

During the initial weeks of the Feingold program, foods containing salicylates, which include almonds, apples, apricots, blackberries, cherries, cucumbers, grapes, gooseberries, oranges, strawberries, and tomatoes, are removed and are later reintroduced one at a time so that the child can be tested for tolerance. In this phase of the diet, foods like pears, cashews, and bananas are used instead of salicylate-containing fruits.

There has been a great deal of debate about the efficacy of the Feingold and related diets. In a double-blind crossover study, 40 of 55 children with ADHD had significant improvements in behavior after a six-week trial of the Feingold diet.[6] An interesting feature was that over 3-6 months, 26 of the children (47.3%) maintained their improvement following liberalization of the diet. In another study, 19 of 26 children responded favorably to

an elimination diet.[7] It is particularly interesting that when the children were gradually put back on to a regular diet, all 19 reacted to many foods, dyes, and/or preservatives.

A recent meta-analysis identified 15 studies that met predefined criteria of being double-blind and placebo-controlled.[8] The authors focused on artificial food colorings, and looked at whether ADHD symptoms worsened in children with ADHD when challenged with a food coloring. There was a common finding: Parents' rating of worsening of symptoms was much higher than that of teachers and health professionals. Parents reported a significant improvement off the food colorings; the teacher and health workers did not. Parental expectation may have been a factor, or the parents and professionals were assessing different aspects of the children's behavior.

There does appear to be an effect of food colorings, but it is small and unstable. There may well be a subset of children who are allergic to food additives and there is increasing evidence that some allergies are more common in children with ADHD.[9] There is no credible published research on the use of the Feingold diet or of food additives and/or allergy in adults with ADHD. This author and another analysis of the literature both came to the same conclusion: More research is needed.[10]

There is an impression that clinicians may be more interested in elimination diets in Europe than they are in the United States. In 2004, a large (n = 1,873) randomized, blinded, crossover trial of 3-year-old children was published.[11] Of the original 1,873 children, 1,246 had skin prick tests to identify atopy. After baseline assessment, children were given a diet eliminating artificial colorings and benzoate preservatives for one week. During the next three weeks, the children participated in a within-subject double-blind crossover study, during which they received, in random order, periods of dietary challenge with a drink containing artificial colorings (20 mg daily) and sodium benzoate (45 mg daily)—the active period—or a placebo mixture, as a supplement to their diets. The results showed consistent, significant improvements in the children's hyperactive behavior when they were on a diet free of benzoate preservatives and artificial colorings. They had worsening behavior during the weeks when these items were reintroduced. But once again the improvement was only detected by parents and not by a simple clinic assessment. On the basis of this and other studies, in 2004 schools in Wales mandated the withdrawal of foods containing additives from school lunches. There are not yet any published data on the long-term effects of this change.

The biggest problem with the Feingold and other elimination diets is that they are expensive and hard to follow. Whatever the final results of the controlled studies, those barriers will always limit their utility. It is also essential to ensure that children on any kind of diet maintain adequate nutrition.

Sugar Restriction

Most clinicians will be familiar with the notion that sugar can make children hyperactive. Happily it is not true. At least 10 double-blind studies have failed to show a link between sugar and hyperactivity.[10,12,13]

Food Allergies

The evidence that allergies may be more common in children with ADHD lead to the question whether children with ADHD could be allergic not only to additives, but also to certain foods themselves. When speaking to patients and their families, it is useful to differentiate allergies—the result of abnormal reactivity of the immune system to proteins in food—from sensitivities that are the direct result of substances in food. The notion was strengthened by the observation that celiac disease may be linked to an increased risk of ADHD and other symptoms.[14]

In an open study of 78 children with ADHD, 90% of whom had previously noticed a reaction to certain foods and who were referred to a diet clinic in London, 59 improved during a "few foods" elimination diet trial that eliminated foods to which children are commonly sensitive.[15] There was a huge range of offending foods and additives, but the most commonly observed were cow's milk, wheat, corn, chocolate, and eggs. Nineteen of the children were able to participate in a second phase. This was a double-blind crossover trial of suspected foods or additives that could be disguised by mixing them with food the children could tolerate. The provoking foods produced a significant worsening of behavior and psychological test performance. On this occasion, both raters and parents picked up the effect, and one conclusion of the study was that clinicians should give weight to the observations of parents and teachers.

At one time it was popular to try and identify allergies using the radioallergosorbent test (RAST). Although technically easy to perform, the RAST is now little used because of problems with sensitivity and specificity. In an allergy testing study of 43 food extracts, 52% of children with ADHD (n = 90) had an allergy to one or more of the foods tested.[16] Over the next few years several researchers carried out open-label studies in which children with ADHD and food allergies were treated with sodium cromoglycate. However, although some authors claimed benefit, the studies were extremely small, not well designed, and have never been replicated.

Clinicians may well be asked about practitioners and commercial entities that claim to be able to identify food sensitivities with methods ranging from blood and muscle testing to electrical and energetic techniques. Some may be helpful, but a detailed search of the literature has not found any to be of proven efficacy.

Many clinicians recommend that parents keep a diet diary for 1-2 weeks to see if any obvious associations between diet and behavior emerge. They will then try an additive-free diet, low in sugar and avoiding foods that are suspected of exacerbating symptoms.

Conclusion

Good nutrition is a fundamental component of any form of treatment or health maintenance program. But the converse: that food—or constituents or adulterants of food—can cause disease is not so clear. Despite the beliefs of many patients, their families, and the media, the evidence remains far from clear. However, there is enough evidence to warrant further research and to recommend a diet diary and a nutritionally sound elimination diet in selected individuals.

It is also essential for the clinician to emphasize that dietary management is but one aspect of treatment: We must also deal with the psychological effects of ADHD, and its impact on relationships, study, and work habits.

Practice Points

- ADHD is a genuine clinical problem that can devastate individuals and their families.
- Food intolerance remains a complex and poorly defined issue, with no uniform international consensus about the best ways to monitor and challenge people who may be food sensitive.
- The possible food sensitivities associated with ADHD are relatively indolent and therefore different from the acute idiosyncratic anaphylactic reactions found in certain individuals. Nonetheless, avoidance remains the cornerstone of treatment.
- Patients and their families will have ready access to hundreds of web sites and popular publications that claim that the link between diet and ADHD is firmly established, and that dietary manipulation is therefore the one answer to dealing with the problem. Neither is true, and it is valuable to point out that many of these publications mix the Feingold recommendations with those pertaining to sugar and food sensitivities.
- It has been claimed that food cravings are a sign of an "offending" food. However, there is no robust evidence that patients are more likely to be sensitive to foods that they particularly like.
- In ADHD, the evidence for elimination of foods is marginally stronger than the evidence for a role for food additives.
- Research studies have consistently shown that parents may be better at recognizing the impact of food on symptoms than either health care providers or teachers; therefore, listen to the reports of parents but then evaluate them.
- Immunological testing has not been proven to be of value in the ADHD, though the possibility remains that there is a subgroup in whom it might be helpful, particularly if they have other atopic symptoms.
- Evaluation is best done with a short (2-6 week) nutritionally sound elimination diet.
- Before beginning, ensure that there are no other symptoms that could be attributable to food, for example skin allergies, rhinitis, or asthma.
- Begin with a bland diet, with emphasis on the elimination of salicylate-containing fruits and vegetables, together with chocolate, cow's milk, and carbonated beverages. After one week, reintroduce each food at a rate of one every three days. If no change is noted by the time that the individual is back on a regular diet, it is unlikely that food is responsible for the ADHD.

[Editor's Note: Dr. Petty is the author of Healing, Meaning and Purpose *and has lectured to more than a quarter of a million people in 45 countries. His newsletter, reports, blogs, and podcasts on health, personal growth, and integration are available at www.richardgpettymd.com or call (770) 554-8812.]* ❖

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CME Questions

CME Instructions: Physicians participate in this continuing medical education program by reading the articles, using the provided references for further research, and studying the CME questions. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material.

After completing this activity, participants must complete the evaluation form provided at the end of each semester (June and December) and return it in the reply envelope provided to receive a credit letter. When an evaluation form is received, a credit letter will be mailed to the participant.

After completing the program, physicians will be able to:

a. present evidence-based clinical analyses of commonly used alternative therapies;
b. make informed, evidence-based recommendations to clinicians about whether to consider using such therapies in practice; and
c. describe and critique the objectives, methods, results and conclusions of useful, current, peer-reviewed clinical studies in alternative medicine as published in the scientific literature.

28. Lavender oil aromatherapy as a treatment for insomnia should be used cautiously, if at all, in:
a. patients with atopic sensitivities.
b. patients taking blood-thinning medications.
c. patients taking other CNS depressants.
d. All of the above

29. Research has shown that at least 50% of American families who receive treatment for ADHD in specialty clinics also use complementary or alternative medical therapies; however, only 12% report this use to their clinicians.
a. True
b. False

30. At least 10 double-blind studies have failed to show a link between sugar and hyperactivity.
a. True
b. False

31. Research studies have shown which group to be better at recognizing the impact of food on ADHD symptoms?
a. Siblings
b. Parents
c. Health care professionals
d. Teachers

Answers: 28. d, 29. a, 30. a, 31. b.

Clinical Briefs

With Comments from Russell H. Greenfield, MD

Dr. Greenfield is Clinical Assistant Professor, School of Medicine, University of North Carolina, Chapel Hill, NC; and Visiting Assistant Professor, University of Arizona, College of Medicine, Tucson, AZ.

Pretzel Logic—Yoga for Migraines

Source: John PJ, et al. Effectiveness of yoga therapy in the treatment of migraine without aura: A randomized controlled trial. *Headache* 2007;47:654-661.

Goal: To assess effectiveness of a specific program of yoga therapy vs. self-care in the treatment of migraine.

Study design: Randomized, controlled clinical trial over three months.

Subjects: People with migraine without aura (n = 72).

Methods: Subjects were recruited from a multispecialty headache clinic with the understanding that a study was being performed to evaluate treatment for migraines "intended to reduce the negative effect on their personal, family, and social lives." A headache diary was maintained for four weeks prior to randomization to either yoga therapy or self-care. The yoga group was taught a set of practices by a trained yoga therapist, and given handouts on techniques to use during the prodromal stage of migraine. Yoga therapeutics included specific postures emphasizing neck, back, and shoulder stretching followed by relaxation, strengthening, and

flexibility exercises. Vigorous bending exercises were excluded. Breathing exercises, relaxation, and meditation techniques were also employed. Yoga was to be practiced five days a week for one hour at a time. Deep cleansing (called kriya) was performed once a week in association with relaxation, and involved nasal douching followed by forced exhalations. Subjects were instructed not to perform the specific yoga practices during an acute headache, or during resolution or the postdromal stage. Those in the self-care group received an educational session once a month that addressed migraine types and triggers, as well as self-care preventive strategies. Primary outcomes of interest were headache frequency as determined through a headache diary, migraine severity as reported on a 0-10 numeric scale, and pain as measured via the McGill Pain Questionnaire. Secondary outcome measures included levels of anxiety and depression as determined via the Hospital Anxiety and Depression Scale, and medication usage.

Results: Participants in the yoga group reported significant improvements in headache intensity, frequency, duration and pain, and in symptoms of anxiety and depression as compared with the self-care group. In addition, subjects who used yoga required less medication over the study period than did people in the self-care group.

Conclusion: Specific yoga practices performed over three months can result in improvements in a variety of migraine parameters.

Study strengths: Diagnosis of migraine without aura was confirmed by a neurologist; three-month duration of trial.

Study weaknesses: Minimal and unclear description of exclusion criteria; no sham yoga group; unique and "special" attention paid to subjects in the yoga group; lack of objective assessment; and as an aside, there were multiple spelling errors present in the text.

Of note: A 2002 survey found that 85% of patients at an outpatient head/neck pain clinic had used some form of alternative medicine to address their headaches, and 60% felt they had received benefit from these therapies; generally positive results have been reported on specific types of yoga therapy against a number of clinical entities that include anxiety, depression, stress, asthma, and musculoskeletal disorders; participants in this trial were charged a registration fee for entrance into the study, and were asked to obtain their own yoga mat and a neti pot (for sinus irrigation); subjects were permitted to use rescue medications as prescribed by their neurologist but no other agents, including over the counter remedies; the study was performed under the auspices of the Department of Zoology (!), University of Rajasthan, India.

We knew that: The most commonly employed CAM therapies for headache include massage therapy, medicinal herbs, acupuncture, and chiropractic care; yoga breathing exercises (also referred to as pranayama) are often promoted as a means of balancing the autonomic nervous system and contributing to a sense of calm.

Comments: Few would argue that yoga, meditation, and other mind/body therapies can help ameliorate anxiety, stress, and perhaps even depression. In addition, specific yoga therapy may contribute to an improvement in symptoms with certain maladies, if not an improvement in the experience of those symptoms. The present study is intriguing but significantly flawed. Did migraineurs benefit due to the special attention paid them in the yoga group, or was it the specific interventions that created clinical improvement? Was there a sinus component to the headaches? Would an hour of relaxation and gentle stretching on most days of the week be as effective as a specific yoga practice? Many questions remain unanswered, and little from this trial's results can be applied clinically with any degree of authority. Yoga therapy may well be of help to some people with migraine headaches, but a paucity of sound clinical research assessing risks and benefits of yoga in this setting remains a serious obstacle to acceptance.

What to do with this article: Remember that you read the abstract. ❖

Move the Cheese: Dairy and Prostate Cancer

Source: Mitrou PN, et al. A prospective study of dietary calcium, dairy products and prostate cancer risk (Finland). *Int J Cancer* 2007;120:2466-2473.

Goal: To examine the association between dietary intakes of calcium and dairy products in relation to the risk of prostate cancer.

Study design: Prospective, randomized, double-blind, 2 × 2 factorial design, primary prevention trial.

Subjects: Cohort of 27,028 male smokers aged 50-69 years who smoked five or more cigarettes a day at study entry (data taken from the Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study, also called ATBC).

Methods: Dietary intake was assessed at baseline using a validated 276-item food questionnaire that addressed food preferences over the prior year, and nutrient intakes were calculated using an accepted food composition database. Participants provided detailed information regarding medical history and demographics, as well as data on smoking. Cases of prostate cancer were identified through the Finnish Cancer Registry. Medical records were reviewed by two study oncologists, and cytology specimens were reviewed by 1-2 pathologists to confirm presence of cancer and histologic type.

Results: Over 17 years of follow-up, a total of 1,267 incident cases of prostate cancer were identified. A graded positive association was readily evident between calcium intake and total risk of prostate cancer. Across increasing categories of calcium intake there was an associated increase in the relative risk (RR) of prostate cancer as follows (mg/d, RR): < 1000 mg/d, 1.00; 1000-1,499 mg/d, 1.28; 1,500-1,999 mg/d, 1.38; and > 2,000 mg/d, 1.63. Similar results were found after analyzing data by quintiles of calcium intake. No association was found between stage or grade of prostate cancer and calcium

from total dairy intake, and no clear association was found for any individual dairy food and prostate cancer risk. When dietary calcium or calcium from dairy products was controlled for, the association between total dairy intake and increased risk of prostate cancer became statistically nonsignificant.

Conclusion: High intakes of dietary calcium or some related component contained in dairy foods is associated with an increased risk for prostate cancer.

Study strengths: Long duration of follow-up (up to 17 years); detailed examination of total risk of prostate cancer and risk by stage and grade of disease; essentially complete identification of cases of prostate cancer; multivariate model that accounted for a multiplicity of potential confounding variables.

Study weaknesses: Problems inherent in the use of a food frequency questionnaire; issues of generalizability since population was limited to older male smokers (but other trials that included nonsmokers have suggested similar risk).

Of note: In a prior ATBC report based on eight years' follow-up, researchers suggested that men with low calcium and higher phosphorus intakes may be at decreased risk of prostate cancer; numerous potential mechanisms have been posited for the apparent association between dairy product intake and increased risk of prostate cancer, including the potential for dairy products to increase serum levels of insulin growth factor I, and especially due to the propensity for calcium intake to suppress the active form of vitamin D; the ATBC study was performed in Finland, a country where dairy intake is generally high; the researchers focused on dietary calcium because calcium intake in the Finnish population results mainly from dietary and not supplement intake; variables like total fat, dietary vitamin D intake, lycopene, alcohol, and red meat intake did not alter study results appreciably; in general, men in the trial with higher dietary calcium intakes were slightly older with higher BMIs, were more likely to have diabetes, lived in urban areas, were less highly educated, and less physically active than men with low calcium intakes; a large proportion of the cases of prostate cancer were detected as a result of clinical symptoms (population-based PSA screening programs are not widespread in Finland).

We knew that: The ATBC trial tested whether daily supplementation with 20 mg beta-carotene and/or 50 mg alpha-tocopherol reduced the incidence of lung cancer in male smokers from southwestern Finland, with the trial ending in 1993 (notable findings were that beta-carotene increased the incidence of lung cancer as well as mortality, and that vitamin E appeared to have a chemopreventive effect against prostate cancer, but was also associated with an increased incidence of death due to hemorrhagic stroke); epidemiologic data regarding an association between calcium intake and prostate cancer risk have yielded inconsistent data (several case control studies, specifically, have shown an increased risk with high calcium intake either from food or from supplements, especially in regard to advanced cancers); total dietary vitamin D intake is a poor correlate of total vitamin D because humans mainly derive vitamin D from sunlight; smoking decreases intestinal calcium absorption; calcium appears to offer chemopreventive effects against colon cancer, and offers benefit against osteoporosis, and possibly against hypertension and insulin resistance.

Comments: Results of this well-done study bring added concerns about high dairy intake to the fore. Calcium has an important role in health for men as in women, but dosage becomes the critical question, and may ultimately become gender-specific. To quote the authors, "Our data suggest that the current recommended dietary allowance (RDA) of 1,200 mg/d of calcium for men aged 50 or over (equivalent to 2-3 glasses of milk per day) may exceed the optimal amount needed to achieve a balance between the apparent health benefit and risks of calcium." For now, men without specific clinical indications (like osteoporosis) should shun calcium supplementation, especially older men with a predisposition towards prostate cancer, and consider moderating their intake of dairy products. Foods like kale, spinach, fortified cereals, and salmon provide calcium at lower levels as well as offering additional nutrients. Unfortunately, studies like this one turn a milk mustache into a frown.

What to do with this article: Keep a hard copy in your file cabinet. ❖

Phooey on Folate? Colorectal Cancer Chemoprevention

Source: Cole BF, et al. Folic acid for the prevention of colorectal adenomas. *JAMA* 2007;297:2351-2359.

Goal: To evaluate safety and effectiveness of folic acid supplementation to prevent new colorectal adenomas in subjects with a history of same.

Study design: Double-blind, placebo-controlled, two-factor, phase 3, randomized clinical trial performed at nine clinical centers in 2004 (the Aspirin/Folate Polyp Prevention Study [AFPPS]).

Subjects: Patients with a history of colorectal adenomas but no invasive colorectal carcinoma (n = 1,021, aged 21-80 years).

Methods: Questionnaires were completed by participants that addressed personal characteristics, medical history, and lifestyle habits. Subjects were randomized to receive folic acid 1 mg/d (n = 516) or placebo (n = 505), and were separately randomized to receive aspirin 81 mg or 325 mg, or placebo. Development of adenomas was determined by colonoscopic evaluation and pathology review. Follow-up colonoscopy was scheduled to take place after three years, and then again after another three or five years (the latter being offered as an invitation to participate and without mandatory use of aspirin). During the first follow-up interval, subjects received questionnaires regarding multiple parameters including symptoms and adherence to study protocol. During the second follow-up interval, questionnaires were again mailed every four months to participants who continued to take study tablets, and annually to all other subjects. Plasma folate levels were determined at the end of the first follow-up

interval to gauge adherence to study protocol. The primary outcome measure of interest was development of at least one colorectal adenoma. Secondary outcome measures included occurrence of advanced lesions and presence of multiple adenomas.

Results: During the first surveillance cycle, the incidence of at least one colorectal adenoma was 44.1% in the folic acid group and 42.4% for the placebo group, while the incidence of at least a single advanced lesion was 11.4% and 8.6%, respectively. During the second surveillance cycle, incidence of at least one colorectal adenoma was 41.9% in the folic acid group and 37.2% for the placebo group, while the incidence of at least a single advanced lesion was 11.6% and 6.9%, respectively. For the 607 subjects with endpoint information on both follow-up intervals, the rate of any adenoma being identified in either interval was 71.3% in the folic acid group and 65.5% in the placebo group, and the overall rate of advanced lesions was 23.1% and 17.1%, respectively. Suggestion of an increased risk of colorectal adenomas with folic acid was confined to those subjects not randomized to receive aspirin. Use of folic acid was associated with a higher risk of multiple colorectal adenomas, as well as noncolorectal cancers (specifically due to an excess incidence of prostate cancer).

Conclusion: Folic acid taken at a daily dose of 1 mg/d for up to six years does not reduce the risk of colorectal adenomas in people with a history of adenomas, and may actually increase the risk of colorectal neoplasia.

Study strengths: Excellent follow-up for first colonoscopy cycle (96.7%); consideration of potential time constraints in showing a benefit for folate as compared with aspirin; excellent adherence to study protocol, especially during first follow-up cycle, including avoidance of nonstudy folic acid-containing supplements; intention-to-treat analysis.

Study weakness: Marked decrease in follow-up rate for second surveillance cycle.

Of note: The epidemiological data supporting the idea that a low-folate diet contributes to colorectal neoplasia is especially notable for people who regularly imbibe alcohol (which can antagonize folate metabolism); plasma vitamin B_{12} and, as necessary, methylmalonic acid levels, were obtained to avoid masking of vitamin B_{12} deficiency and to exclude those people with preexisting B_{12} deficiency; animal data exist suggesting a cancer-promoting effect of folate supplementation, and a protective effect of folate deficiency against experimental carcinogenesis (some data suggest a protective effect of folate on normal mucosa, but a cancer-promoting effect on early neoplasia); originally, the AFPPS was designed to investigate only aspirin, but it was quickly expanded to examine folic acid; participants had to have had removal of all known polyps within three months of study enrollment; allocation to the folic acid group resulted in a significant increase in plasma folate and a modest decrease in plasma homocysteine levels.

We knew that: Fortification of the food supply with folate began in 1996 and was made mandatory if 1998; adenomas are precursors of most colorectal cancers; folate is a derivative of folic acid; folic acid and its derivatives play an important role in nucleotide synthesis and methylation reactions; bench data and epidemiologic research both suggest chemopreventive effects for folic acid against colon cancer; folate supplementation lessens the risk for neural tube defects; folate deficiency leads to macrocytic anemia; the AFPPS data regarding aspirin showed that low-dose (81 mg/d) aspirin had a moderate chemopreventive effect against development of colorectal adenomas, whereas high-dose aspirin (325 mg/d) provided no clinical benefit; foods high in folic acid include fortified cereals, garbanzo beans, broccoli, lentils, and turkey.

Comments: A high-quality trial raising the specter of over-supplementation with specific nutrients leading to possible morbidity is important news, especially so in the case of folate, where mandatory fortification efforts have been in place since 1998. Results of this trial suggest an increased risk of colorectal adenomas in people with a history of adenomas even in subgroups where the benefits of folate supplementation would seem readily apparent (low folate levels or high alcohol intake, for example). There is also the suggestion of an increased risk of noncolorectal neoplasia. The trial does not, however, address primary prevention of colorectal adenomas.

Recent studies have caused some practitioners to question their enthusiasm for folate supplementation in the setting of cardiovascular disease. The findings of this trial further dampen hopes for folate being a panacea. Until further data become available, those with a history of colorectal adenomas should not use supplemental folic acid, but should continue to enjoy food sources of folate in moderation. Future studies need to focus on whether folate provides a chemoprotective effect for individuals with no history of colorectal adenomas.

What to do with this article: Keep a hard copy in your file cabinet. ❖

Supplement to *Alternative Medicine Alert*
July 2007

ALTERNATIVE MEDICINE ALERT™
A Clinician's Evidence-Based Guide to Alternative Therapies

Getting a Good Night's Sleep

MOST ADULTS NEED 7-8 HOURS OF SLEEP PER NIGHT AND SHOULD FEEL REFRESHED AFTER that much sleep. Unfortunately, Americans are sleeping less. Insufficient or disrupted sleep may contribute to decreased energy and fatigue, decreased alertness, and weight gain. Poor sleep increases the risk of developing depression and having a serious accident. This handout details good sleeping habits that may help improve both quantity and quality of sleep.

Get into a good routine

- Leave enough time for at least 7 hours of sleep per day.
- Go to bed and get up about the same times every day.
- Try to sleep as soon as you feel sleepy, but don't stay in bed staring at the clock if you don't fall asleep with in a half hour. Get up, read until you feel sleepy, then try to sleep again.
- Don't do anything in your bed except sleep or have sex. All other activities (reading, TV watching, eating, etc.) should be done elsewhere.
- Don't nap during the day—it makes it harder to sleep at night.

Understanding Sleep

UNTIL THE 1950S, MOST PEOPLE THOUGHT OF SLEEP AS A PASSIVE, DORMANT PART OF OUR daily lives. We now know that our brains are very active during sleep. Moreover, sleep affects our daily functioning and our physical and mental health in many ways that we are just beginning to understand.

At least 40 million Americans each year suffer from chronic, long-term sleep disorders, and an additional 20 million experience occasional sleeping problems. These disorders and the resulting sleep deprivation interfere with work, driving, and social activities. They also account for an estimated $16 billion in medical costs each year, while the indirect costs due to lost productivity and other factors are probably much greater.

Almost everyone occasionally suffers from short-term insomnia. This problem can result from stress, jet lag, diet, or many other factors. Insomnia almost always affects job performance and well-being the next day. About 60 million Americans a year have insomnia frequently or for extended periods of time, which leads to even more serious sleep deficits. Insomnia tends to increase with age and affects about 40% of women and 30% of men. It is often the major disabling symptom of an underlying medical disorder.

Mild insomnia often can be prevented or cured by practicing good sleep habits. For more serious cases of insomnia, researchers are experimenting with light therapy and other ways to alter circadian cycles.

Source: National Institute of Neurological Disorders and Stroke, National Institutes of Health. Available at: www.ninds.nih.gov/disorders/brain_basics/understanding_sleep.htm. Accessed on June 18, 2007.

- If you have to get up during the night, to go the bathroom for example, try not to turn on any bright lights. Return to bed and try to fall asleep quickly.

Prepare well for bed
- Don't start anything that wakes you up near bedtime.
- Don't exercise or increase physical activity near bedtime.
- Don't get into difficult or challenging conversations near bedtime.
- Don't do stimulating mental activities near bedtime.
- Limit smoking near bedtime.
- Consciously prepare for sleep with a set a regular activities beginning about 30-45 minutes before trying to sleep.
- Practice a short relaxation exercise.
- Use a set of calming yoga poses.
- Take a warm bath. Add lavender or other calming essential oils to water to enhance the relaxing effects.
- If you get anxious and have many intrusive thoughts or make lists when you lie down to sleep, consider drawing for 10-15 minutes before bed.
- Write down a short list of things that are bothering you, promise yourself you will address these issues in the morning and then put the list away—mentally and literally.
- Read something relaxing or even boring for a few minutes to distract you and allow you to get sleepy. No scary or stimulating books!
- Consider listening to a tape of relaxing music or tapes designed to help induce restful sleep.

Create an environment conducive to sleep
- Invest in a good bed with a comfortable mattress, but make sure it is firm enough to give adequate support to the back.
- Keep the room cool.
- Make the room as dark as possible.
- Decrease the sound in the room or wear ear plugs.
- Consider a white noise or relaxing noise machine.
- Use relaxing essential oils (lavender, geranium). You can put them on a tissue on your pillow or use a diffuser.

Eat to promote a good sleep
- Don't eat a heavy meal within two hours of bedtime.
- Limit the amount of caffeine—you may need to limit caffeine all day, not just after dinner. Try having no more than two caffeinated beverages during the day, even if you feel tired after a poor night's sleep.
- Alcohol may make you feel relaxed and seems to help you fall asleep, but it disrupts normal sleep architecture, leading to fracture or disrupted sleep. Alcohol is not recommended.
- Sugary or spicy foods before bed may be stimulating and interfere with sleep.
- Eating a small snack containing foods high in carbohydrates or tryptophan (like turkey, tuna, soy, whole wheat crackers, yogurt, banana, milk) may increase a neurotransmitter in the brain making it easier to sleep.

Treat other medical conditions that can interfere with sleep
- Maintain a normal weight. Obesity can contribute to sleep apnea.
- Treat chronic painful conditions so that pain does not interrupt sleep.

Supplements to aid sleep
- Valerian 600-900 mg nightly 30 minutes before bedtime. Take daily for at least 2-4 weeks to see benefit.
- Relaxing tea such as Celestial Seasons Sleepytime® tea (chamomile and spearmint).
- Melatonin can be very helpful for some people. Try 1 mg at bedtime to start. Do not use if this supplement stimulates you or if you have a seizure disorder.
- Hyland's Calms Forté™ is a homeopathic preparation that can help you fall asleep. Take 4-6 capsules into a clean mouth (nothing to eat or drink for 30 minutes before or after this dose). This medication does not make you groggy or hung over so it is a good thing to take if you wake up with only a few hours left in the night, if you are elderly, or if you are very sensitive to medications.

When to get medical follow up
- If you snore loudly or stop breathing briefly during sleep.
- If you fall asleep suddenly during the day, especially if you are doing activities like driving or other activities that should keep you awake.
- If you have abnormal movements in your legs at night.
- If you think one of your regular medications is interfering with sleep. Drugs that can interfere with sleep include: selective serotonin reuptake inhibitors like Prozac® (fluoxetine hydrochloride), decongestants, corticosteroids, and some asthma medications.
- If you are very anxious or depressed. These medical conditions can interfere with sleep.