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Blue Cohosh: A Word of Caution

By Beth Irikura and Edward J. Kennelly, PhD

BLUE COHOSH (*CAULOPHYLLUM THALICTROIDES* [L.] MICHX., [Berberidaceae family]) has a long history of use, especially for gynecological conditions. The roots and rhizomes of blue cohosh were reportedly used by Native Americans for inducing labor or abortion. Other uses included colic, epilepsy, rheumatism, hiccups, and sore throat.[1]

In North America, blue cohosh is available in various forms, including herbal mixtures specifically targeted at women. The herb is oxytocic and has a reputation as an abortifacient. Because of the activity and questionable safety of some of its constituents, blue cohosh should be used only with extreme caution. Many herbalists advise against using it in early pregnancy and it may be best avoided even in late pregnancy.

Background

Blue cohosh is often used in conjunction with black cohosh (*Cimicifuga racemosa*), an unrelated plant with similar applications but different chemical constituents and an undetermined mechanism of action.[2] Both herbs received the Algonquin name cohosh, meaning rough, for the appearance of their roots. From 1882 to 1905, blue cohosh was listed (for labor induction) in the U.S. Pharmacopoeia. Although a casual survey of contemporary herbals reveals a variety of uses, the most common application is still for inducing labor.

A recent survey of 172 certified nurse-midwives (CNMs) found that, of the 90 CNMs who prescribed or encouraged use of labor-stimulating herbal preparations, 64% used blue cohosh and 45% used black cohosh.[3] Twenty-one percent of the CNMs reported complications when herbs were used. Those associated with use of blue and black cohosh were nausea, meconium-stained fluid, and transient fetal tachycardia. Two glycosides long known to be present in blue cohosh—caulosaponin and caulophyllosaponin—may be responsible for its reported oxytocic effects. Both saponins have vasoconstrictor activity, uterine-stimulating activity, and cardiotoxic effects.[4]

Suspected Teratogens

A recent study suggests that blue cohosh contains possible

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teratogenic compounds.[5] Several potentially hazardous alkaloids were identified in extracts of blue cohosh: anagyrine, *N*-methylcytisine, and taspine. Anagyrine is a known teratogen in grazing animals. When pregnant cattle consume lupine (*Lupinus* species), containing high levels of anagyrine, offspring often manifest crooked calf disease, a syndrome that includes bowed or twisted limbs, permanently rigid joints, spinal curvature and occasionally cleft palate.[6] In the 1970s, statistical correlation and controlled feeding experiments established that anagyrine caused the deformations.[7]

Anagyrine is one of the main alkaloid constituents of blue cohosh. It has been reported in blue cohosh rhizomes at a concentration of 290 ppm.[8] Effects on humans have not been proven, but the severity and frequency of teratogenicity in range animals are reasons for caution. Anagyrine may have very different effects in humans and ruminants because of differences in intestinal flora; it is thought that anagyrine may require metabolism by rumen microflora to exhibit teratogenic effects. One reported case suggests a link between maternal consumption of anagyrine-containing goat milk and birth defects in which the infant displayed vascular anomaly, skeletal dysplasia, and malformation of red blood cells.[9] FDA researchers have suggested that women may be wise to avoid ingesting anagyrine at any level, until more is known about its activity in humans.[8]

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A recent study using rat embryo cultures (REC) indicated that anagyrine had teratogenic potential only at the highest concentrations tested, when overall morphogenesis was also impaired.[5] The REC is an in vitro method to detect changes in development of an embryo over the 45-hour culture period. Teratogenicity is determined by the presence of malformations that cannot be explained by an overall, non-specific inhibition of growth and morphogenesis. The presence or absence of malformations is assessed separately from the determination of developmental status. The REC can identify potential neurotoxic effects. The relative inactivity of anagyrine at lower concentrations may be explained by the fact that the REC cannot detect musculoskeletal deformations.

N-Methylcytisine is a second potentially hazardous compound identified from blue cohosh. In cultured rat embryos, *N*-methylcytisine from blue cohosh caused major malformations.[5] At a concentration of 20 ppm the effects included open anterior neural tube, poor or absent eye development, and twisted tail. Higher concentrations of *N*-methylcytisine inhibited overall growth and morphogenesis, in addition to producing similar malformations.

In a separate study, *N*-methylcytisine was also found to stimulate the ganglion cells of the cardiac vagus in frogs, paralyze the ganglia of the cardiac vagus in dogs, and produce hyperglycemia in rabbits.[10]

Some of the actions of *N*-methylcytisine are similar to nicotine.[10] Exposure to nicotine via smoking in pregnant women has been linked with impaired fetal growth, neural dysmorphology, fetal death, and cognitive deficits in surviving offspring.[11] This epidemiological evidence has been supported by in vitro and in vivo studies.

N-Methylcytisine in blue cohosh-containing dietary supplements has been measured at concentrations ranging from 5-850 ppm.[8] No research has been conducted on the pharmacokinetics or pharmacodynamics of blue cohosh or its constituents; therefore, the clinical significance of the experiments discussed above remains unknown. However, women anticipating a pregnancy may want to avoid using blue cohosh-containing dietary supplements until the potential in vivo teratogenic effects of this botanical are understood.

Taspine, another constituent of caulophyllum, is found only in low yield (0.00013% by weight) in blue cohosh rhizomes.[5] Taspine is cytotoxic at concentrations of 0.5 ppm.[12] In rat embryo cultures, taspine was lethal at concentrations of 5 ppm.[5] Taspine is related to other phenanthrene alkaloids such as morphine, heroin, and codeine, which are highly cytotoxic and suspected human teratogens.[13]

Adverse Fetal Effects in Humans

There are several reported cases of adverse birth

outcomes associated with ingestion of blue cohosh. In the best-documented case, the infant suffered profound congestive heart failure associated with acute anterolateral myocardial infarction from maternal ingestion of three times the recommended dose for three to four weeks prior to parturition.[14] The infant required mechanical ventilation 20 min after parturition, and displayed poor perfusion, mitral regurgitation murmur, cardiomegaly, and pulmonary edema. Hepatomegaly and abnormal liver function, consistent with cardiogenic shock, subsided gradually. The infant was hospitalized initially for 31 days and remained on the respirator for three weeks. Two years later the child was developing normally, but still required digoxin treatment for persistent cardiomegaly and reduced left ventricular function. The researchers ruled out other possible causes of myocardial infarction, such as coronary artery anomalies.

In another case, the mother was advised by a midwife to take a mixture that included blue cohosh and black cohosh to induce labor.[15] The researchers in this case suggest that the myocardial toxicity (known to be associated with caulosaponin and caulophyllosaponin found in blue cohosh) could explain the severe hypoxic-ischemic symptoms observed in the newborn.[16] This work has been criticized for not providing specific information on dosage, frequency or timing of usage, and type of herbal preparation ingested by the patient.[17]

In addition, two cases related to consumption of blue cohosh are listed in the FDA Special Nutritionals Adverse Event Monitoring System database.[18] The first involved stroke in an infant, following ingestion of blue cohosh with grain alcohol. In the second case, the infant developed aplastic anemia following maternal exposure to blue cohosh and unspecified product(s).

Conclusion

Several adverse birth outcomes may be explained by fetal exposure to blue cohosh. Additionally, recent work indicates that constituents of blue cohosh cause teratogenic effects in rat embryo cultures. Future studies may be able to correlate additional epidemiological evidence with animal studies. Practitioners are advised that blue cohosh is a powerful pharmacological agent and may be contraindicated during the course of pregnancy. Women of childbearing age are urged to consult knowledgeable health professionals before using this herb. ❖

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Another View on REC

By Anthony Scialli, MD

ALL COMPOUNDS ARE HARMLESS AT ONE DOSE AND toxic at another, and the same is true for so-called teratogens. Teratogenicity, like toxicity, is a relative concept. Just as all compounds are poisons at a high enough dose, all compounds should be expected to produce developmental toxicity at a high enough exposure level.

The risk of birth defects in humans for a given exposure cannot be extrapolated from embryo culture tests without considerable information on the exposure level anticipated in humans, and without an understanding of the kinetics of the compound in humans and in the test system. In vivo (whole animal) tests provide some predictive information on the risk of birth defects, because the dose level at which maternal toxicity is present serves as an intraspecies comparator of the sensitivity of the embryo.

Opioids are an example of the importance of exposure level in evaluating developmental toxicity potential. Although opioids increase developmental toxicity in whole animal teratology studies at a sufficiently high dose, these agents have not been associated with an increased incidence of congenital anomalies under conditions of clinical use for pain relief.

Rat whole embryo culture by itself cannot be used as a screening test for risks to human reproduction, although the technique can be designed to investigate mechanisms of toxicity when such toxicity has been identified in whole animal testing. Whole embryo culture is not recognized as an appropriate screen for human reproductive risk by the National Institute of Environmental Health Sciences (NIEHS), the National Center for Evaluating Health Risks to Reproduction (NCEHR), or the Environmental Protection Agency (EPA).

The finding that components of blue cohosh produce abnormal development of the rat embryo in culture cannot be interpreted without additional information:

- How do the concentrations in culture compare to human plasma concentrations under usual conditions of use of this drug?
- How rapidly is the agent being studied metabolized or excreted by the mother? Remember, in whole embryo culture, there is no maternal organism to get rid of the drug, so exposures tend to be longer and at higher levels than in real life.
- Is the developmental toxicity unique, or (more likely) a nonspecific consequence of general toxicity? High enough concentrations of any compound can be expected to prevent normal embryo flexion and neural tube closure, for example, because these events are the biggest things happening during the culture period. ❖

Dr. Scialli is the Editor of Reproductive Toxicology *and is on the editorial advisory board.*

Good News About Chocolate

By Adriane Fugh-Berman, MD

IN THE MOVIE *SLEEPER*, WOODY ALLEN WAKES UP IN THE 21st century to find many things changed, including the news that hot fudge sundaes are good for you. The data are not in on the ice cream, but chocolate may well be a health food in the next century. It turns out that chocolate is extremely high in antioxidants, with dark chocolate besting milk chocolate (which contains milk and more sugar than dark chocolate).

The Origins of Chocolate

Chocolate comes from the beans of the cacao tree (*Theobroma cacao*, family Sterculiaceae), a 20-foot evergreen that originated in South America and was brought into Mexico by the Mayas before the 7th century AD. Its species name, *Theobroma*, is Greek for "food of the Gods." The words chocolate and cocoa are derived from the Aztec; chocolate is a version of *xocoatl*, which means "bitter water" in Nahua. "Cocoa" is an 18th century corruption of the tree's name "cacao." Cacao beans were used as currency in Mexico until 1887 (100 beans could buy a slave or a canoe). This currency was even counterfeited by scooping out the pulp and replacing it with wax or dirt; such was punishable by death.[1]

Several anthropologists believe that they have discovered the "Cradle of Chocolate" in the Ulua river valley in northwestern Honduras, an area known to be one of the first places where cocoa was cultivated. John S. Henderson, PhD, and Rosemary A. Joyce, PhD, uncovered pieces of pottery dating back to 1600 BC that are thought to be vessels for chocolate.

Originally used only as an unsweetened beverage flavored with chili peppers, Europeans invented the sweet form of hot chocolate. The use of chocolate in confectionery dates only from 1828, when the use of a screw press to separate cocoa butter from chocolate became popular. Defatted cocoa powder was one result of this innovation, and the other was cocoa butter, which could now be added to chocolate to create the smooth candies that are so popular today.[2]

Most chocolate today comes from Africa, although

South America is also a source, and Hawaii has recently entered the market with a unique variety.

Active Compounds

Chocolate contains the methylxanthines theobromine, theophylline, and caffeine. The caffeine content of chocolate is much lower than that in tea or coffee; by weight, the caffeine content of cocoa is 0.009%; coffee 0.04%; black tea 0.06%; and green tea 0.01%.[3] Chocolate also contains three unsaturated *N*-acylethanolamines that may act as cannabinoid mimics (see *Alternative Therapies in Women's Health*, March 1999, pp. 28-29) and flavonoid polyphenols. It is these flavonoids that are antioxidants, and their presence in chocolate makes it unnecessary to add preservatives. One of the flavonoid polyphenolics in chocolate is (-)-epicatechin. Because catechins are the primary flavonoids in tea, sometimes they are called tea flavonoids. Catechins (as well as other flavonoids) are strong antioxidants and are thought to have a protective effect against cardiovascular diseases.

A recent study compared the catechin content of dark chocolate, milk chocolate, and black tea.[4] Dark chocolate contained the most catechins (53.5 mg/100 g); milk chocolate contained 15.9 mg/100 g, and an infusion of black tea (1 g/100 ml water) contained only 13.9 mg/100 ml. The type of catechins present was also found to differ. For example, chocolate contained only (+)-catechin and (-)-epicatechin. Whereas tea contained only low concentrations of those catechins, tea contained other types of catechins, including high concentrations of (-)-epigallocatechin gallate (EGCG) and (-)-epicatechin gallate, as well as low concentrations of (-)-epigallocatechin and (+)-gallocatechin.

EGCG is the major tea polyphenol, and its levels in green tea are three to eight times higher than in black tea.[5] It would have been useful for the researchers to compare green tea and black tea; both come from the *Camellia sinensis*, but are processed differently, which results in different levels of catechins. It is unclear whether different catechins have different effects.

Chocolate may also have immunoregulatory effects; cacao liquor polyphenols inhibited reactive oxygen species (hydrogen peroxide and superoxide anion in activated granulocytes and human peripheral blood lymphocytes).[6]

Because people tend to consume larger amounts of tea than chocolate on a daily basis, the researchers also estimated the total intake of catechins from chocolate and tea in a representative sample of the Dutch population. Tea was the most important source of catechins, supplying 55% of the total daily intake, but chocolate was an important source as well, contributing 20% of total intake.[3]

For those concerned about fat intake, cocoa contains almost no fat but retains antioxidant effects. Cocoa powder has more phenols by weight than bakers (unsweetened) chocolate, which in turn has more phenols than milk chocolate. A 41 g (1.5 oz) piece of milk chocolate contains 205 mg phenol, equivalent to the 210 mg phenol found in a 140 ml (5 oz) serving of red wine. An in vitro experiment found that cocoa phenols inhibited human LDL oxidation by 75%.[7] The authors suggest that the pairing of red wine and chocolate may have cardiovascular as well as gustatory benefits.

The antioxidant effect of cocoa has been confirmed in vivo. LDL oxidation lag time was measured in the blood of 12 male volunteers, who then consumed 35 g of cocoa.[3] Prior to cocoa ingestion, LDL oxidation lag time was 61.2 min. Two hours after cocoa intake, oxidation lag time was prolonged to 70.3 min; four hours after ingestion, oxidation lag time had returned to 64.4 min. This is significant because there is evidence that the susceptibility of LDL cholesterol to oxidation is a factor in the development of atherosclerosis.

Chocolate Innocent in Migraines

Although chocolate is thought to trigger headaches, especially migraines, a double-blind study has cast doubt on this belief. Sixty-three women with chronic headache (50% migraine, 37.5% tension-type, and 12.5% mixed) followed a diet restricted in vasoactive amine-rich foods for two weeks before undergoing provocative trials with two samples of chocolate and two samples of carob presented in random order.[8] Subjects maintained diaries throughout the study, recording diet and headache. Chocolate was no more likely to provoke headache than was carob in any of the headache groups. Subjects' belief in whether chocolate ingestion was related to headache did not correlate with the results.

Potential Adverse Effects

If you're trying to increase your daily ingestion of chocolate—for medicinal use, of course—you may want to steer clear of hot chocolate vending machines. When a number of employees in a Minneapolis manufacturing plant became ill after drinking hot chocolate from a machine, significant amounts of *Bacillus cereus* were isolated from dispensed beverages.[9] Citywide testing of vending machines dispensing hot chocolate found that seven of 39 licensed machines tested were contaminated; two machines had levels high enough to cause illness.

Also, it is possible that chocolate could contribute to renal stone formation in susceptible individuals, although no cases have been reported. One study found that a single chocolate bar caused less pancreatic islet cell stimulation than sucrose (as determined by glucose,

insulin, and C-peptide levels) but caused a significant increase in triglyceridemia, calciuria, and oxaluria.[10] ❖

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CME Questions

16. Catechins are highest in:
a. dark chocolate.
b. milk chocolate.
c. black tea.

17. Which of the following bacteria has been isolated from vending machine hot chocolate?
a. *E. coli*
b. *B. cereus*
c. *S. aureus*
d. *S. typhi*

18. Compared to oral administration of progesterone, transdermal administration:
a. results in equivalent serum levels.
b. results in higher serum levels.
c. results in lower serum levels.

19. A recent study found that perinatal mortality for babies born in water, compared to babies born conventionally, was:
a. lower.
b. higher.
c. about the same.

20. A recent study found an association between uterine myomas and high intake of which of the following?
a. Meat
b. Dairy products
c. Meat and dairy products
d. Vegetables and fruits

Clinical Abstracts

With Comments from Adriane Fugh-Berman, MD

Progesterone Cream Does Not Benefit Bone

Source: Leonetti HB, et al. Transdermal progesterone cream for vasomotor symptoms and postmenopausal bone loss. *Obstet Gynecol* 1999;94:225-228.

Design and Setting: Randomized, double-blind, placebo-controlled trial in Bethlehem, PA.

Subjects: 102 healthy postmenopausal women who were within five years of menopause and had not taken hormonal therapy for at least a year. Twelve women dropped out (two for rashes—one in each group; two for unrelated hospitalizations; and eight failed to keep appointments or missed more than 15% of medication doses). Final analysis was done on 90 women (43 in the treatment group and 47 in the placebo group).

Treatment: Transdermal progesterone cream.

Dose/Route/Duration: One quarter teaspoon of cream (containing 20 mg progesterone or placebo) was applied to the skin daily; all women received both multivitamins and 1,200 mg calcium daily. The study lasted one year and patients were seen every four months.

Outcome Measures: Bone mineral density (measured in the lumbar spine and hip by dual energy x-ray absorptiometry), symptomology, and lipid profiles.

Results: There were no significant differences between the treatment and control groups in terms of change from initial bone mineral density (of the lumbar spine, femoral neck, or total hip) nor in the number of subjects in each group who showed an increase of bone mineral density of over 1.2%. Eight women in the treatment group experienced vaginal spotting. Biopsies found proliferative endometrium in one woman, the other seven had tissue insufficient for diagnosis. Controls and women in the treatment group had vasomotor symptoms. Of those who reported vasomotor symptoms initially, 5/26 women in the control group reported improved vasomotor symptoms compared to 25/30 women in the treatment group. No significant changes were seen in lipids or mood ratings between groups or from baseline.

Funding: "In part" by Astraea, Inc. (Portland, OR); donations from June Pearson, MD.

Comments: This study should lay to rest the notion that progesterone cream increases bone density. (See *Alternative Therapies in Women's Health*, April 1999, pp. 33-36.) It has been known for some time that progestins can decrease hot

flashes, but it is news that transdermal creams can have this effect (a previous study[1] found that serum levels of progesterone from transdermal creams were only a third of those obtained from oral micronized progesterone). ❖

Reference

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Red Meat Intake and Uterine Fibroids

Source: Chiaffarino F, et al. Diet and uterine myomas. *Obstet Gynecol* 1999;94:395-398.

Design/Setting/Subjects: Data from a case-control study on risk factors for uterine myomas conducted in Italy between 1986 and 1997. The study compared 843 cases of women hospitalized for fibroid surgery (and whose myomas were diagnosed within the past two years) to 1,557 women age 55 and younger who had no history of fibroids or hysterectomy and were admitted to the hospital for acute non-gynecological, non-oncological conditions.

Results: Consumption of beef and other red meat was associated with an increased risk of developing fibroids; consumption of green vegetables was associated with a protective effect. Consumption of fish was also associated with a protective effect. There was no association found for consumption of other index foods (milk, cheese, butter, eggs, oil, margarine, liver, carrots, whole-grain foods, coffee, or tea). Multivariate odds ratios in the upper tertile were 1.7 (95% confidence interval [CI] 1.4-2.2) for beef and other red meat; 1.3 (CI 1.0-1.6) for ham; 0.5 (CI 0.4-0.6) for green vegetables; 0.7 (CI 0.6-0.9) for fish; and 0.8 (CI 0.6-1.0) for fruit.

Funding: Not stated.

Comments: It is common lore among alternative medicine practitioners that consumption of animal products (meat, dairy products, and eggs) causes or worsens fibroids. However, the reasoning behind this is based on theory or speculation rather than data. This interesting study supports the possibility that frequent meat consumption may be associated with fibroids. (There was no association found for consumption of milk, cheese, or eggs.)

The distribution of cases and controls was not even; women with uterine myomas were more educated, more frequently premenopausal, and less frequently smokers than controls. One of the limitations of this study, as the researchers point out, is that although average weekly frequencies of consumption were noted, there was no attempt to quantify intake. Additionally, control subjects did not undergo sonograms, so the actual incidence of fibroids is unknown in the control group. The use of index foods can also be misleading. It would have made more sense to group dairy products (questions were asked about milk, cheese, and butter, but not yogurt or ice cream). Possible mechanisms for the association were not discussed. It is quite possible that high levels of meat intake is a marker for other health habits or socioeconomic factors. Another possibility is an effect on estrogen levels. Meat is high in fat, and in premenopausal women, low-fat diets reduce endogenous estrogen levels.[1] Also, estrogenic hormones are used as growth promoters in cattle and chickens and residues may be in meat (although they probably would be in milk as well). ❖

Reference

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Are Water Births Safe?

Source: Gilbert RE, Tookey PA. Perinatal mortality and morbidity among babies delivered in water: Surveillance study and postal survey. *BMJ* 1999;319:483-487.

Design/Setting/Subjects: Over a two-year period (1994-1996), all 1,500 consultant pediatricians in the British Isles were surveyed each month on whether they knew of any births that resulted in perinatal death or hospital admission within 48 hours following labor or delivery in water. Detailed information on cases was gathered. As checks on accuracy, regional coordinators were contacted to see whether they knew of additional unreported cases, and findings were compared to the confidential inquiry into stillbirths and neonatal death (a mandatory, regional notification system). Additionally, in 1995 and 1996 a mail questionnaire was sent to all National Health Service units in England and Wales.

Results: Reported water births constituted 0.6% of all deliveries. Of 4,032 water births during the study period, there were five perinatal deaths. Two of these were stillborns. The three postpartum deaths were associated with abnormal findings; a baby who died eight hours after birth had hypoplastic lungs; one baby died at three days of age of neonatal herpes; and the third died of intracranial hemorrhage 30 min after a precipitous delivery.

There were 32 other admissions for special care, resulting in a risk of 8.4/1,000 live births, including 15 with lower respiratory tract problems (of which one was diagnosed with freshwater drowning). Two babies had evidence of streptococcal pneumonia. Five cases of hypoxic ischemic encephalopathy or perinatal asphyxia were diagnosed; 15 had a variety of other diagnoses.

Compared with a large data set of low-risk women who delivered conventionally in the North West Thames region, there was no increased risk of perinatal mortality for babies delivered in water. Special care admission rates were significantly lower for babies delivered in water than those delivered conventionally.

Comments: As the researchers point out, the numbers of perinatal death or admission for special care were small and the confidence intervals wide. (Only a relative risk of 3.6 or greater would have been picked up by this study). Perinatal mortality estimates may be more accurate than estimates of special care admissions. Researchers were confident

that they missed no deaths (because no additional deaths were identified through the confidential inquiry into stillbirths and infant deaths), but note that the number of water births may well have been underestimated, so the actual denominator may well be larger. In other words, the figure for perinatal mortality is an upper estimate. However, there may well be underreporting of admissions for special care following water birth, so it is possible that this number is underestimated. Two admissions may have been due to water aspiration (although it is thought that babies don't breathe until exposed to air, animal experiments have shown that this inhibitory mechanism can be overridden by sustained hypoxia). Although this study clearly shows no substantially increased risk of perinatal mortality or morbidity among babies born in water, compared to those born conventionally, small increases in risk cannot be ruled out. ❖

Label Review

Mango Passion Crisp

with St. John's Wort and Kava
Yogi Bhajan's Peace Cereal

Package Information

"Herbs have been used for thousands of years for humankind's healing. Every culture and nation has Grandma's special recipes. Humankind learned to reach out to herbs as their simple friends. Through a study of herbal use, it is true: 'You are what you eat.' We have experienced through this process that herbs are our friends, and they can serve us as they have in the past. With that hope we bring you Peace Cereals. Each Peace Cereal is enhanced with a specialized combination of the highest quality herb powders and extracts. We are not making any claims; we are just sharing what **St. John's Wort** and **Kava** are all about. You can make your own personal search and discover it for yourself."

Nutrition Facts

Serving size 1 cup (55 g)
Servings per container 5

Amount per serving	*Cereal*	*Cereal with 1/2 cup skim milk*
Calories	210	250
Calories from fat	45	45
	% Daily Value**	
Total fat 5.0 g*	8%	8%
Saturated fat 0.5 g	3%	3%
Cholesterol 0 mg	0%	0%
Sodium 170 mg	7%	10%
Total carbohydrates 37 g	12%	14%
Dietary fiber 3.0 g	10%	10%
Sugars 9 g		
Protein 5.0 g		
Vitamin A	2%	2%
Vitamin C	0%	0%
Calcium	2%	17%
Iron	8%	8%

*Amount in cereal. One half cup of skim milk contains an additional 40 calories, 65 mg sodium, 6 g total carbohydrate (8 g sugars), and 4 g protein.

**Percent Daily Values are based on a 2,000 calorie diet. Your daily values may be higher or lower depending on your calorie needs.

Manufactured by Golden Temple (a member of Khalsa International Industries and Trades), Eugene, OR

Ingredients: Oat clusters (OG* rolled oats, unsulfured molasses, water, Expeller pressed canola oil, crisp rice (milled rice, unsulfured molasses, salt and malt), brown rice syrup, whey powder, herbal tincture (vegetable glycerin, OG* St. John's wort leaf and flower tops, OG* fennel seed, OG* cinnamon bark, OG* spearmint leaf, fenugreek seed, OG* ginger root, cardamom seed, kava kava extract [10% kavalactones], lavender flowers, natural licorice flavor, OG* clove bud and OG* black pepper, natural passion fruit flavor, natural mango flavor, salt, lecithin) cornflakes (milled corn, crystallized evaporated cane juice, salt, malt), and mango.

*OG organically grown and processed in accordance with the California Organic Foods Act of 1990.

Price: $3.29, 11.5 oz (326 g)

Comments by Adriane Fugh-Berman, MD

This very busy cereal box has dense print on the outside and the inside of the box, and both sides of the box top as well. Also included are directions for a kundalini yoga stretch, an announcement for winners of the Golden Temple Peace Cereal grants, directions for nominating people for the award, a letter from a grant recipient, quotes from various people, and advertisements for other cereals, teas, body oils, and other natural products.

The cereal contains a 12-ingredient herbal tincture that includes St. John's wort leaf and flower tops and kava kava extract (10% kavalactones). Other tincture ingredients include fennel, cinnamon, spearmint, fenugreek, ginger, cardamom, lavender, licorice, clove, and black pepper.

Cereal ingredients are listed in order by weight, but one cannot tell by any cereal label how much of anything is in it. A label statement says, "Each cereal serving is enhanced with 110 mg of our unique St. John's Wort tincture." Assuming that the tincture referenced is the 12-ingredient mixture, there must be a depressingly small amount of St. John's wort in this cereal. Even if the tincture contained only St. John's wort, 110 mg is far from the usual dose of 900 mg/d of a standardized extract. The situation for kava is similar; a usual dose of kava is 210 mg/d. Also, the usual percentage of kavalactones is 70%, and the kava used in this cereal is only 10% kavalactones. There is no reason to think that this cereal would have any beneficial therapeutic effects other than enhancing the skills of a budding cereal box reader! ❖