PASSIONFLOWER

(Passiflora)

An overview of the research and clinical indications

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BACKGROUND AND USES

The genus Passiflora L. comprises about 520 species of dicotyledonous plants in the family Passifloraceae. Passiflora incarnata and P. alata, also commonly known as Passionflower, are two species of a perennial climbing vine with beautiful exotic flowers and delicious fruit that grow worldwide, preferring subtropical, frost-free climates. They are native to the tropical and semi-tropical United States (Virginia to Florida and as far west as Texas), Mexico, Central American, and from Brazil to Paraguay through northern Argentina.[1] Native Americans, such as the Cherokee of the southern Allegheny mountains, cultivated Passionflower for its sweet and sour fruit, which incidentally contains a significant amount of lycopene.[2,3] Spanish conquistadors learned from the Aztecs that Passionflower was used as a sedative for nervousness and insomnia. The plant was introduced into European medicine and widely cultivated there. The unique appearance of the plant's flowers led Spanish missionaries to name the plant in reference to aspects of the *passion* of Jesus Christ: the crown of thorns was symbolized by the coronal threads, the cords of the whips by the curling tendrils, the wounds by the five stamens, the nails on the cross by the three stigmas, the hammer by the ovary, and the ten "true" apostles by the five petals and five sepals of the flower.[4] Passionflower is currently official in the national pharmacopeias of Egypt, France, Germany and Switzerland, and also mongraphed in the *British Herbal Pharmacopoeia* and the *British Herbal Compendium,* the ESCOP monographs, the Commission E, the German Standard Licenses, the *German Homeopathic Pharmacopoeia,* and the *Homeopathic Pharmacopoeia of the United States*.[5 6 7 8 9 10 11 12 13 14 15 16]

In Germany, Passionflower is used as a component in sedatives (combined with valerian root and lemon balm), a pediatric sedative tea (*Species nervinae pro infantibus*: with lemon balm, lavender flower, and St John's Wort) and cardiotonic formulations (with hawthorn).[5] It is also a German homeopathic medicine for pain, insomnia, and neurasthenia.[17] In the United States, Passionflower is commonly a component of herbal sleep aid formulations. It was official in the fourth (1916) and fifth (1926) US National Formularies and removed in 1936. It was also an approved OTC sedative until 1978.[18,19]

In preparing this review, evidence for efficacy and activity of Passiflora was obtained on the website of the American Botanical Council, which references 6 clinical trials, 3 observational case reports, 45 animal studies, 22 pharmacodynamic studies, 32 analytical chemistry papers, and 19 genetic and molecular biology studies.[20] In addition, for the purpose of this review, studies cited in PubMed since 2007 (the date of the latest systematic review of Passiflora), limited to the sedative properties of the plant, were considered for inclusion.

DESCRIPTION AND ACTIVE CONSTITUENTS

Passionflower (Passiflora incarnata) is considered the most bioactive of the species in the family Passifloraceae. Purple passionflower (P. edulis) has often been confused with P. incarnata; P. edulis is the source of passion fruit. Passiflora alata (fragrant granadilla; wing stemmed passionflower) is also called passionflower by some publications, including the U.S. Department of Agriculture's botanical database.

Chemical content of Passiflora species is also not well delineated. Investigators have differed on whether its sedative effects are due to indole alkaloids such as harmane, harmaline, and harmol; flavonoids such as apigenin, luteolin, and scopoletin; or an isolated trisubstituted benzoflavone.

In addition, recently it was determined that Passiflora contains more gamma-amino butyric acid (GABA) than 20 other plants examined.[21] One of six alkaloids isolated from P. incarnata has been called "passiflorine," and is believed by some to be the plant's active compound, although the Agricultural Research Service's web site describes passiflorine as inactive. The Chemical Abstract Service's database's only similar entry is "passiflorin," a steroid-like molecule found in P. edulis stems and leaves that is not an alkaloid.

Numerous publications on P. incarnata have come from Dhawan's research group at Punjab University of Pharmaceutical Sciences in India.[22] Focusing on animal experiments, the group has used painstakingly fractionated alcohol/water extracts of selected plant parts and has attempted to narrow the field of potential active constituents of P. incarnata. Dhawan et al. have not found any bioactivity from extracts of roots or flowers. Inclusion of the whole plant in an extract raises the dose needed for efficacy. Only extracts from P. incarnata leaves produced anxiolytic effects. The group has reported on Passiflora's ability to restore sexual interest in aging male rats and in rats that are habituated to tetrahydrocannabinol, as well as restoring fertility which has been reduced by alcohol or tobacco use, and reducing anxiety while withdrawing from alcohol.

Passionflower extracts consist of fresh or dried aerial parts of P. incarnata or P. alata, collected during the flowering and fruiting period. Botanical identity is confirmed by thin-layer chromatography, microscopic and macroscopic examination, and organoleptic evaluation. Extracts contain 0.825% apigenin and luteolin glycosides, vitexin, isovitexin and their *C*-glycosides, kaempferol, quercetin, and rutin; indole alkaloids (0.01%), mainly harman, harmaline, harmine; coumarin derivatives; cyanogenic glycosides (gynocardin); amino acids (including GABA); fatty acids (linoleic and linolenic); gum; maltol; phytosterols (stigmasterol); sugars (sucrose); and a trace of volatile oil. [7 8 10 13 16 23]

The content of harman alkaloids varies; it must not exceed 0.01 percent.[24] The *British Herbal Pharmacopoeia* requires not less than 15% water-soluble extractive, among other quantitative standards.[6] The *French Pharmacopoeia* requires not less than 0.8% total flavonoids calculated as vitexin by measuring the absorbence after reaction.7 The ESCOP monograph requires that the material comply with the French, German, or Swiss pharmacopeias.[10,14]

MECHANISM OF ACTION

Numerous pharmacological effects of Passiflora incarnata are mediated via modulation of the GABA system including affinity to GABA(A) and GABA(B) receptors, and effects on GABA uptake. Although the active ingredients have not been conclusively delineated, most available data suggests flavonoids and GABA account for the reported effects.[22,25]

Dhawan's group conducted at least 3 rodent studies with what they described as a newly isolated benzoflavone (BZF) moiety from P. incarnata.[22] An Austrian group subsequently performed a study to isolate the compound for analytical purposes, in P. incarnata from 3 different origins (India, Italy and France).[26] A compound with the published thin layer chromatograph was detected in trace amounts only in the Italian product, and none was found in the herbs from France and India. In a commercial extract, two compounds with the expected TLC characteristics were detected, one as a phytol isomer and the other in such small amounts

that its structure could not be elucidated. The Austrian investigators concluded: "The detection of only trace amounts of a BZF-like compound in one of three commercial samples of Passiflorae herba and in an extract suggests for the first time that BZF is not the active principle in this drug and should not serve as an active marker."

Studies in animal models show efficacy of Passiflora extracts and flavonoid fractions against pentylenetetrazol (PTZ) induced seizures.[27] This effect of Passiflora can be inhibited by the benzodiazepine site antagonist Ro 15-1788, suggesting the involvement of GABA(A) receptors.[28] Flavonoids bind with high affinity to the benzodiazepine site of the GABA(A) receptor[29], but appear to modulate GABA(A) and also GABA(C) receptor currents by a different mechanism than benzodiazepines.[30]

In 2007, researchers at the US Army Graduate Program in Anesthesia Nursing in Georgia studied the effects of chrysin, a Passiflora extract, and modulation of the benzodiazepine receptor on the GABA(A) receptor in rats.[31] Chrysin significantly decreased anxiety to the same degree as midazolam.

A study of GABA-mediated anxiolytic activity of P. incarnata was conducted in mice at the University of Florida using the elevated plus maze test.[32] Following oral administration, the P. incarnata extract exerted an anxiolytic effect that was comparable to diazepam (1.5 mg/kg) at a dose of 375 mg/kg and exhibited a U-shaped dose-response curve. In addition, antagonism studies using the GABA (A)/benzodiazepine receptor antagonist flumazenil and the 5-HT (1A)-receptor antagonist WAY-100 635 were conducted. The active dose was effectively antagonized by flumazenil, but not by WAY-100 635.

In 2010, researchers at the Oregon Health Sciences University and the Helfgott Research Institute at the National College of Natural Medicine in Portland, Oregon explored the potential mechanisms of P. incarnata extracts and the effects of extraction methods on ingredients and biological effects.[25] Total flavonoid yields increased substantially with hot vs. cold extraction methods. Whole Passiflora extract induced prominent, dose-dependent direct GABA(A) currents in hippocampal slices, but the expected modulation of synaptic GABA(A) currents was not seen. GABA was found to be a prominent ingredient of Passiflora extract, and GABA currents were absent when amino acids were removed from the extract.

The in vitro effects of a dry extract of P. incarnata (sole active ingredient in Pascoflair® 425 mg) on the GABA system were investigated by a German team.[33] The extract inhibited [(3) H]-GABA uptake into rat cortical synaptosomes but had no effect on GABA release and GABA transaminase activity. P. incarnata inhibited the binding of [(3) H]- SR95531 to GABA(A) -receptors and of [(3) H]-CGP 54626 to GABA(B) -receptors. Using the [(35) S]-GTPγS binding assay Passiflora could be classified as an antagonist of the GABA(B) receptor. In contrast, the ethanol- and the benzodiazepine-site of the GABA(A) -receptor were not affected by this extract.

SAFETY DATA

Passionflower extract is classified as "generally regarded as safe" (GRAS) by the Food and Drug Administration. There is one case report of occupational allergic asthma and rhinitis to Passiflora in a pharmacy technician who prepared extracts of P. alata.[34] Mild adverse effects were reported in only one other study, including dizziness, drowsiness and confusion.[35] There are no other confirmed reports of adverse effects or toxicity, no known adverse interactions, and no known contraindications. Beneficial interactions have been reported in vitro and in a mice model, showing a synergistic anti-depressive effect of P. incarnata with Hypericum.[36] Oral administration of extracts of P. incarnata given for 15 days have also shown hypoglycemic and hypolipidemic effects in streptozotocin-induced diabetic mice, suggesting the possibility of additive or synergistic effects when Passiflora is given to humans who are taking anti-diabetic or lipid-lowering medications.[37] A randomized controlled study of 60 patients undergoing spinal anesthesia showed that oral P. incarnata extract had no effect on hemodynamics during their operations.[38] Pregnant women are advised to avoid high doses.

RECENT RESEARCH

Anxiety

Mice treated orally with P. incarnata at 3 different doses revealed activity in the elevated plus-maze (EPM) test at 375mg/kg, but none at 150 and 600 mg/kg respectively, indicating a U-shaped dose response curve.[39]

A Chinese study of the anxiolytic effects of P. edulis and P. edulis flavicarpa in the EPM test of mice revealed activity at equivalent oral doses but significant differences in flavonoid compounds between the two species.[40]

A Brazilian team evaluated the effects of P. alata and P. edulis on anxiety and memory in rats injected intraperitoneally with aqueous extracts prior to the EPM test, inhibitory avoidance test, and habituation to an open-field apparatus.[41] Treatment with both extracts induced anxiolytic-like effects comparable to diazepam. Memory was not affected with any dose of P. alata or P. edulis, but diazepam disrupted memory process. P. edulis was active at half the dose of P. alata, which could be explained by the differences in flavonoid content.

In 2010, a study at the University of Florida College of Pharmacy characterized the anxiolytic activity of fractions from a hydroethanol extract of P. incarnata, again using the EPM test in mice.[42] The results suggested that the active principle of Passionflower was in the chloroform fraction and to a lower extent in the butanol fraction, but the petroleum ether fraction did not show any effects.

In 2001, an Iranian group performed a controlled study of P. incarnata with oxazepam in the treatment of generalized anxiety disorder (GAD).[35] 36 outpatients with GAD were randomized to Passiflora extract plus placebo tablet or oxazepam 30 mg/day plus placebo drops, for a 4 week trial. The two treatments were equally effective. Oxazepam had a more rapid onset of action, but caused greater impairment of job performance.

In 2008, another Iranian group randomly administered oral P. incarnata or placebo as a premedication to 60 patients undergoing inguinal herniorrhaphy and found that it reduced anxiety without inducing sedation.[43] An unusual (possibly not standardized) numerical rating scale was used for patients to assess anxiety and sedation before, and 10, 30, 60 and 90 minutes after premedication. Psychomotor function was also assessed. There were no significant differences in psychological variables in the post-anesthesia care unit and recovery of psychomotor function was comparable in both groups.

In 2009, Australian researchers performed an analysis of plant material cultivated in Australia that revealed two chemically distinct groups; hence an investigation was carried out to determine whether distinct intraspecific chemotypes exist in this species. Eleven *P. incarnata* samples were analyzed by HPLC, LC-MS and two different TLC methods. The samples fell into two distinct groups with respect to their C-glycosyl flavone profile, with little within-group variation. One chemotype was dominated by isovitexin and schaftoside/isoschaftoside, as is most widely reported in the literature for this species. The other chemotype was characterized by a high level of swertisin, with low levels of schaftoside/isoschaftoside. The two chemotypes are readily identified by both HPLC and TLC. The researchers concluded that although the compounds responsible for the therapeutic activity of *P. incarnata* are yet to be definitively identified, phytomedicines should be made with the accepted isovitexin chemotype until the pharmacological implications of chemotypical differences are understood.[44]

Aslanargun et al.'s 2012 publication of P. incarnata extract as an anxiolytic before spinal anesthesia found significantly less anxiety in the treatment vs. control group without changing psychomotor function test results, sedation level, or hemodynamics.[38]

Anxiogenic effects

In the Oregon study by Elsas referenced earlier[25], five different extracts, prepared from a single batch of P. incarnata, were administered to mice for one week in their drinking water before evaluating their behavioral effects. Anticonvulsant effects against pentylenetetrazol-induced seizures were seen in mice that received two of the five Passiflora extracts. Instead of the anxiolytic effects described by others, anxiogenic effects in the elevated plus maze were seen in mice receiving any of the five Passiflora extracts. Comparing ingredient and biological activity from all extracts, there was no correlation between total flavonoid or GABA content with anxiogenic or anticonvulsant effects. The two extracts with the highest GABA content did not show anticonvulsant effects, and the two extracts with anticonvulsant effects differed widely in their total flavonoid content. Similarly, there was no correlation between GABA content and anxiogenic effects. This is the first study administering P. incarnata extracts in drinking water for 1 week prior to behavioral testing in mice, thus closely mimicking regular intake using the oral route in humans. Surprisingly, the data showed an increase in anxiety measures. The activity of the mice was not affected, excluding the possibility that potential alterations in activity contributed to the increase in anxiety measures. Consistent with this, the investigators also found that intraperitoneal (i.p.) injections of wild-type mice or mice deficient in apolipoprotein E once 1 hour prior to behavioral testing increased measures of anxiety.

In contrast to these findings by Elsas et all, other studies have reported anxiety-reducing effects of Passiflora extracts following single i.p. injections or oral (p.o.) administration. These

divergent findings may be related to differences between Passiflora species tested, geographical source of plants, extraction methods, dose, method and duration of administration, vehicle used, test animal species and strain, baseline anxiety levels and potential effects of the extracts on activity levels. In the Oregon study, the baseline anxiety levels in the vehicle-treated animals were such that both increases and decreases in measures of anxiety could be detected. However, for all previous animal studies on effects of P. incarnata on anxiety, baseline anxiety levels were very high, with animals spending 5% or less time spent in open arms of the plus maze. It would clearly be difficult if not impossible to detect a potential anxiogenic effect under these experimental conditions. In addition, potential effects on activity levels could contribute to group differences in performance in anxiety mazes. Therefore, it is essential to assess activity levels to exclude these potential confounds but this is not actually done in all studies. This is particularly important for anxiety-modulating compounds that often affect activity levels at higher doses. Future studies are warranted to determine the mechanisms underlying these differential effects. However, together these data support the concept that the effects of Passiflora incarnata extracts on measures of anxiety probably depend on the baseline anxiety state of the animals. In turn, this suggests that the extracts might function as modulators of one or several neurotransmitter systems, similar to positive and negative allosteric modulators whose action critically depends on the presence or absence of endogenous neurotransmitters whose levels would vary with baseline anxiety states. The anxiogenic results observed by Elsas et al., *in vivo,* also contradict reported clinical anxiolytic effects of Passiflora incarnata*.* However, the clinical results are specific to the Passiflora product tested. Plant extracts containing so many different constituents can exhibit a variety of effects, sometimes contradictory, depending on the chemical composition, route of administration and dosage of the individual extracts used. Furthermore *in vitro* and *in vivo* effects of pharmacological agents do not always correlate directly to clinical studies, due to issues of bioavailability, metabolism, and species differences. Additionally, previous clinical data is not without limitations. Two of the published clinical studies of Passiflora extracts did not have a placebo control[43,45], and as previously mentioned, the third[44] measured anxiety by an unusual, possibly not standardized scale.

Opiate withdrawal

In 2001, Akhondzadeh's group also reported on a controlled trial of P. incarnata in the treatment of opiate withdrawal.[46] According to the authors, opiate detoxification is completed in 75% of inpatients and 15% of outpatients. The typical withdrawal syndrome consists of symptoms such as anxiety, convulsions, nausea and tremor. The drug clonidine is an accepted treatment in this context but only ameliorates the physical symptoms and not the psychological symptoms of withdrawal. The trial compared clonidine + Passiflora with clonidine + placebo in 65 male opiate addicts undergoing detoxification on an outpatient basis. 15 subjects in each of the two groups completed the 14 day trial. A psychiatrist interviewed each subject on days 0, 1, 2, 3, 4, 7 and 14 and asked for ratings of severity of 16 withdrawal symptoms on the modified Short Opiate Withdrawal Scale (SOWS). On day 14, the mean total SOWS score was significantly lower in the clonidine + Passiflora group. The mental withdrawal symptoms were also significantly lower in severity in the clonidine + Passiflora group.

Insomnia

Enhanced sleep in laboratory animals given Passiflora has been demonstrated in several controlled experiments.[47] The efficacy of P. incarnata tea on sleep quality was measured by an Australian team in a controlled fashion in a study of 41 healthy young adults, using sleep diaries validated by polysomnography plus an anxiety inventory.[48] Subjects with a history of sleep disorder were excluded. Participants were exposed to 1 cup of either Passionflower or placebo tea (Parsley) for one week, followed by a 1 week 'washout' period, and then crossed over. Sleep quality was significantly better ($p < 0.01$) for those who drank Passionflower tea, but Passionflower tea failed to influence the other outcomes measured. The authors addressed this by noting that the dose may have been too low (1 cup vs. the standard dose of 3 cups) as well as other design limitations.

Seizures

Current therapeutic treatment of epilepsy with antiepileptic drugs is associated with side effects, teratogenicity, and dose-related and chronic toxicity and approximately 30% of patients continue to have seizures with anti-epileptic therapy. Passiflora has been explored in at least two mice studies as a potentially safe and effective alternative to prescription anticonvulsants. An Iranian study investigated "Pasipay", a hydro-alcoholic extract prepared from the standardized extract of leaves, flower and fruit of P. incarnata, with a total flavonoid content of 4%.[27] In this pentylentetrazole (PTZ) model, Pasipay, diazepam and normal saline were injected peritoneally 30 minutes before PTZ. Flumazenil and naloxone were also injected 5 minutes before Pasipay. Compared to the saline group, Pasipay significantly prolonged the onset time of seizure and decreased the duration of seizures ($p < 0.001$). Seizure and mortality protection were 100%. Flumazenil and naloxone suppressed this anticonvulsant effect.

As the main endogenous inhibitory neurotransmitter, GABA might be expected to act as a natural anticonvulsant. Elsas et al.[25] found that extractions methods affected the anticonvulsant potential of Passiflora. They did not, however, find correlation between Passiflora extract's GABA content and its anticonvulsant activity in mice. Two of five extracts reduced the severity and frequency of PTZ-induced seizures.

CLINICAL INDICATIONS

- anxiety or nervousness
- Generalized Anxiety Disorder (GAD)
- pre-surgical anxiety
- insomnia
- symptoms of opiate withdrawal
- seizure disorder
- attention deficit hyperactivity disorder
- palpitations
- arrhythmia
- hypertension
- low libido

PRACTITIONER DOSING

Dried herb: 2 g, three to four times daily.
Infusion: 2 g in 150 ml water, three to four times daily.
Fluid extract 1:1 (g/ml): 2 ml, three to four times daily. Can be taken in liquid phyto-cap form, 2 capsules, three to four times daily.
Tincture 1:5 (g/ml): 10 ml, three to four times daily.

CONCLUSIONS

Preliminary positive evidence of anxiolytic activity exists for P. incarnata. RCTs examining the effectiveness of passiflora for anxiety are too few in number to permit any definite conclusions to be drawn. Caution should be taken when interpreting the results as many studies have not been replicated. RCTs are needed with larger samples that compare the effectiveness of Passiflora with placebo and other medications (e.g. Kava, prescription anxiolytics and hypnotics). Of the papers reviewed in this monograph, all but one show a positive direction of evidence for Passiflora as an anxiolytic without the risk of serious side effects. Several studies have found a U-shaped dose response curve for Passiflora, suggesting that extracts with a higher percentage of active ingredients may have an anxiogenic rather than anxiolytic effect.

Different extraction methods of Passiflora influence the extract yield and total flavonoid and GABA content. While many plant extracts contain amino acids, Passiflora extracts were found to have the highest GABA content of 21 examined plants.[21] The study by Elsas et al.[25] show that Passiflora extracts not only contain a high amount of GABA, but are also able to induce direct GABA(A) currents in rat hippocampal pyramidal neurons. Since the extract with reduced amino acid levels induced no current, it is likely that the GABA content of the extract is sufficient to explain the observed GABA currents *in vitro*. However, no correlation was found between an extract's GABA content and its anticonvulsant activity *in vivo.* Large, well- designed clinical studies, possibly comparing a variety of Passiflora extracts, are needed for a more complete understanding of the range of clinical actions, mechanisms of action, therapeutic dosage range, drug/botanical agent interactions and to explore any unknown toxicity. A Canadian randomized trial of naturopathic care for anxiety was published in 2009 using Ashwaganda as an anxiolytic, along with dietary counseling, deep breathing relaxation techniques, and a standard multi-vitamin, in comparison to psychotherapy, relaxation techniques, and placebo.[49] A similar whole practice trial using Passiflora as the anxiolytic would be a valuable comparison study. Two randomized controlled clinical trials using Passiflora as the primary component of herbal formulas for insomnia are currently underway by the author.

ABOUT THE AUTHOR

Michael Traub, ND, DHANP, CCH, FABNO attended the University of California, Irvine where he received a B.S. in biological sciences, and graduated from National College of Naturopathic Medicine (NCNM) in 1981. He completed a residency program in Family Practice and Homeopathy at NCNM. Since 1986, Dr. Traub has been Medical Director of an integrative healthcare center (Lokahi Health Center) in Kailua Kona, Hawaii. He is board certified in naturopathic oncology as well as homeopathy.

Dr. Traub was the first naturopathic physician in contemporary times to be appointed to a hospital staff – North Hawaii Community Hospital (NHCH). He was Chairman of the Integrated Healing Committee from the opening of the hospital in 1996 until 2001 and succeeded in gaining approval for the natural medicine formulary in the hospital including botanical, nutritional and homeopathic medicines. In 1998 he developed the Hawaii Residency Training Program at NHCH and has continued to serve as the Residency Program Director. He conducted a pilot study at the hospital of integrated treatment programs for breast cancer. He currently is co-coordinator of the course for fourth-year medical students on Integrative Healing at NHCH in collaboration with the University of Minnesota Center for Healing and Spirituality.

From 2001-2003, Dr. Traub served as President of the American Association of Naturopathic Physicians (AANP). In 2006 he was honored by the AANP as "Physician of the Year." Dr. Traub has held numerous other leadership positions within the naturopathic profession throughout his career, from Chairman of the Hawaii Board of Naturopathic Medicine to President of the Homeopathic Academy of Naturopathic Physicians and President of the North American Board of Naturopathic Examiners.

Dr. Traub has conducted a wide range of research studies on homeopathic growth factors in HIV and AIDS, Attention Deficit and Hyperactivity Disorder, Paroxysmal Nocturnal Hemoglobinuria in Bone Marrow Failure Syndromes, Hypovitaminosis D, and Elderberry Extract for Prevention of Influenza.

He has been invited to make presentations at numerous medical conferences, including the 1999 International Conference on HIV/AIDS in Paris. He is the author of "Essentials of Dermatological Diagnosis and Natural Therapeutics" and "Essentials of Dermatological Diagnosis and Integrative Therapeutics." He has contributed to several textbooks including the Textbook of Natural Medicine. He serves on the editorial board of the Natural Medicine Journal, the International Journal of Naturopathic Medicine, and Holistic Primary Care. He is a member of the Board of Directors of the Integrated Healthcare Policy Consortium and was co-author of the "Final Report of the National Policy Dialogue to Advance Integrated Health Care: Finding Common Ground, 2001-2002, and co-editor of "The Affordable Care Act & Beyond: A Stakeholder Conference on Integrated Healthcare Reform," Sept. 2010. He currently serves on the Scientific Advisory Boards for Gaia Herbs, Inc. and Nordic Naturals.

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