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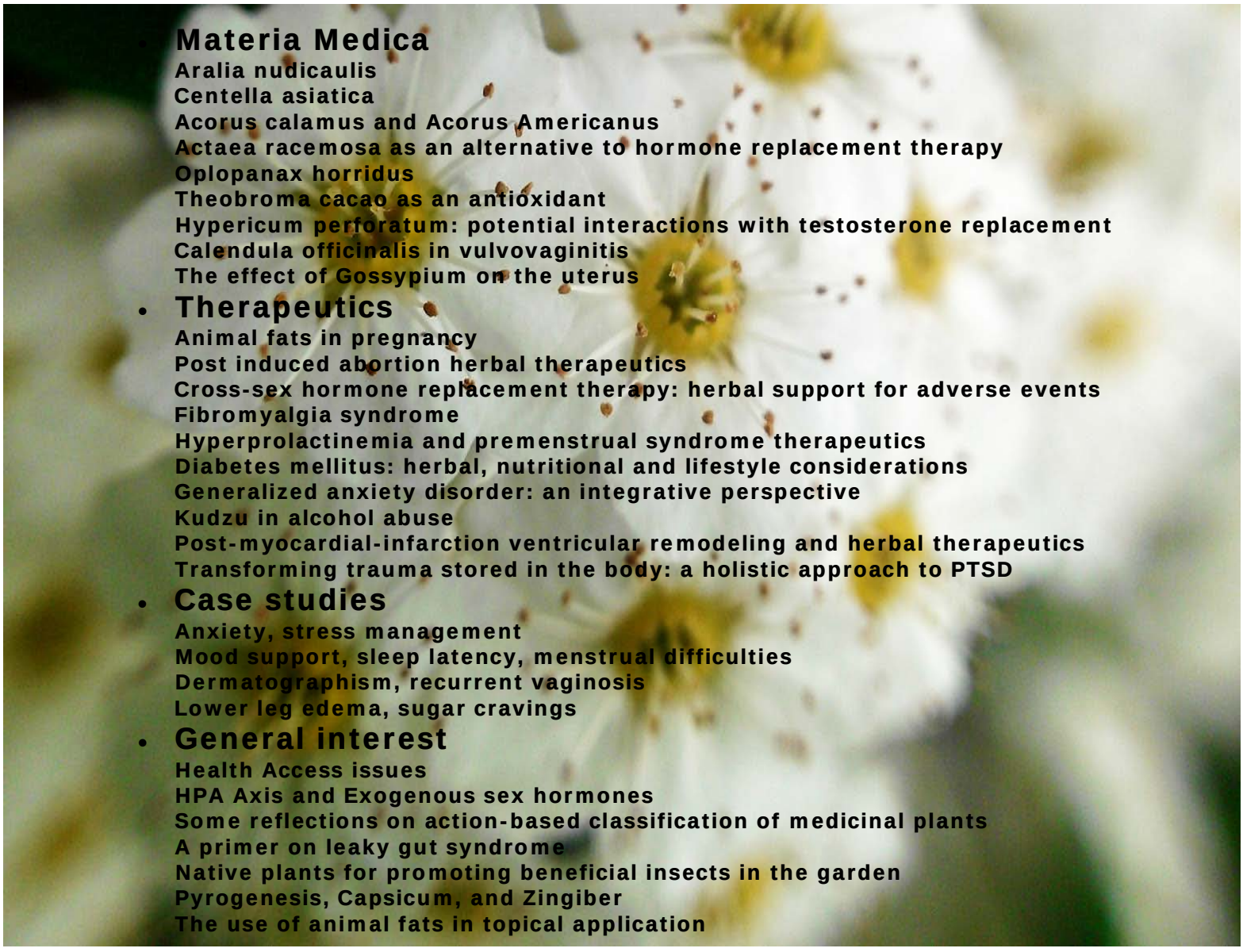

Volume 2, Number 1 (June 2014)

Welcome to another (much belated) issue of *Integrative Herbalism*. We are hoping you will find much that is of interest in this volume, arranged by area of focus: articles on botanicals, either comprehensive or focused on a specific area of applicability; detailed papers on integrative therapeutics for a wide diversity of imbalances; comprehensive case reports with follow-up data; and some fascinating pieces of general interest (including, for example, a study of which medicinal species might be best suited to attracting beneficial insects to your garden).

In this issue you will find that a good portion of the work has been dedicated to exploring the effects of herbs on transgender people. For instance, articles explore potential effects, side effects, and herbal interactions with exogenous hormones. I also recommend the article on herbal therapeutics following induced abortion – a detailed, thorough, and practical guide to navigating this difficult case.

We really feel that the caliber of work presented in this volume of Integrative Herbalism is outstanding, and reflects the caliber of students with whom we have the honor of working. As always, *Integrative Herbalism* is brought to you with full open access, in line with our mission of supporting an open, diverse conversation about herbal medicine in the twenty-first century.

For the plants!

Guido Masé, Director of Research
Vermont Center for Integrative Herbalism

Aralia nudicaulis

Jonathan Shapiro

Aralia nudicaulis is a common understory plant (McDonald) in the Araliaceae family whose range stretches across most of the northern United States and Canada (USDA, NRCS). It is a North American native (Felter and Lloyd 261) which likes to grow in rich, moist woods (Harding 352). It has a long history of medical use by indigenous peoples and settlers in North America (Horton 699). The Eclectics and others included it in their material medicae, and several notable contemporary herbalists publish or speak about its properties.

A perennial plant, the aboveground parts of A. nudicaulis grow up two feet high (Foster and Duke 63) although may be found as short as eight inches (Alpers and Murray 184). It sends up a petiole and scape in early spring; the petiole at full growth has three divisions, each

bearing a compound leaf of three to five leaflets (Alpers and Murray 184). The plant flowers in May through July (Cook 80) (Harding 353). “During the summer a dark purple, nearly black, drupe develops about one-fourth of an inch in diameter. This fruit is probably a welcome food for birds, as it disappears soon after ripening and can only seldom be found on the ground under the leaves. It does not seem to serve for the propagation of the plant, the creeping root-stock performing this function” (Alpers and Murray 184).

The root is the part used medicinally, and has variously been described as “perennial, brown, yellowish, creeping, twisted, sometimes many feet long, the thickness of a finger” (Howard 696); and “large, fleshy, horizontal, creeping, tortuous… very long, often many feet in length, about six lines in diameter, and yellowish or brownish externally” (Felter and Lloyd 261). Examples of the root up to 29 feet in length have been described (Alpers and Murray 183). William Alpers records impressions of the root being more potent in the fall (Alpers and Murray 189), and Jim McDonald recommends harvesting only the upright portion of the root connecting to the horizontal rhizomes because it has a stronger taste (McDonald).

A. nudicaulis has numerous common names, the most common of which are false sarsaparilla, wild sarsaparilla, and spikenard (sometimes referred to as little spikenard). (Alpers and Murray 183) (Cook 80) (Felter and Lloyd 261) (Foster and Duke 63) (Harding 352) (Howard 696) (McDonald) (Moore) (Rafinesque 53) (Sayre 252). Plants in the Aralia genus tend to have either woody stems, acrid aromatics, and spines, or herbaceous annual growth with somewhat succulent roots. The latter category often has a soothing balsamic taste and is generally higher in aralosides, ginsenosides, and saponins. “The four Aralia that can be loosely lumped together as ‘Spikenards’ are of this latter type.” (Moore).

The Eclectics and other 19th- and early 20th-century figures wrote about the plant’s uses and indicated the plant for a wide range of acute and chronic complaints, but most mention its alterative action and its effects on the pulmonary system in acute situations. Cook said it “expend[s] its properties chiefly upon the skin and kidneys…. It is seldom employed in pulmonary difficulties; yet is good whenever the lungs need a mild stimulant” (Cook 80). Horton described it is a pectoral (Horton 80). Lloyd and Felter, in *King’s American Dispensatory*, call it an alterative and mention its usefulness in pulmonary diseases (Lloyd and Felter 262). Sayre calls it an alterative (Sayre 252). Howard says that “all the spikenards are popular medical plants throughout the United States” and that they are all

pectorals (Howard 699). Scudder includes it in his list of alteratives and notes that “it is regarded by many as a valuable alterative agent” (Scudder 484).

Contemporary herbalists understand the plant’s effects in varying ways. Michael Moore considers it a long-term tonic, writing that it has the “Ginseng-like effects of modifying metabolic and emotional stresses” (Moore). Jim McDonald also views the plant as a metabolic normalizer, but says the term adaptogen is “problematic” and categorizes A. nudicaulis as an alterative with a specific action on the adrenal glands. McDonald elaborates that it has a soothing, tonifying effect on the adrenals and can balance a person out when in a state of sympathetic dominance, moving the body’s metabolism forward (McDonald).

At least one recent study has been performed on A. nudicaulis. A team of Canadian researchers performed *in vitro* studies on hexane fractions of the root and fruit and noted that they were cytotoxic against WiDr human colon adenocarcinoma cells, Molt human T-cell leukemia, and HELA human cervix epitheloid carcinoma cells (Wang et al 2157). Additionally, these two fractions were found to not differ significantly from the negative control in minimum survival rates of non-cancer HUV-EC cells (Wang et al 2160).

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Gotu Kola

Elise Walsh

Botanical Nomenclature: *Centella asiatica* (L.)
Family: *Apiaceae*

Common Names:

United States: [i] Gotu Kola, Indian pennywort
India:[ii] Brammi (Sanskrit)
Tamil: Vallarai.
Tel: Babassa, Mandukbrammi
Ben: Thulkurhi
Hindi: kula kudi
Mandukaparni ("frog-leaved")

China:[iii] Ji xue cao ("accumulation of snow herb")
Japan: sekisetsuso
Korea: jeokseolcho

Part used / Definition: Fresh or dried aerial parts

Botanical Identification:

Gotu Kola is a perennial herb with a low, spreading habit. Leaves are reniform, 1-4.5 x 1.5-5 cm with a crenate margin, and born on 0.5-10 cm petioles. Surfaces are glaborous or finely pubescent with 5-7 palmate veins. [iv] Inflorescence in a single umbel bearing 2-5 white star shaped flowers attached directly to the base without a stalk.[v] Flowers are sessile with 2 or 3 brachts that bloom April to October. [vi] They produce small, 2-3mm long fruit.[vii] Stolon (base of stem) is reddish in color.

Commercial Sources and Handling:

Gotu Kola is cultivated in India, Sri Lanka, and in the United States in Hawaii and Texas. High quality material should be green in color [viii] and organically sourced whenever possible. It is widely available as a dietary supplement under the Dietary Supplement Health Education Act of 1944 (DSHEA). Adulteration of *Centella* is not common though it has occurred due to a shared common name, *Brahmi*, which also refers to *Bacopa monnieri*.[ix]

Growing and harvesting information: *Centella* is distributed to nations in the southern hemisphere including India, Sri Lanka, Africa, Madagascar, Australia, Cambodia, Thailand, Lao, Vietnam, China, Indonesia, the Pacific Islands, Central and South America, and the southern United States.[x]

Centella grows wild in moist, drainage areas, though it is amenable to cultivation.[xi] Propagated most readily by root division, it grows as a perennial in tropical and sub-tropical climates and as an annual in temperate climates. To cultivate in temperate climates it should be transferred indoors to greenhouse during the cold season. [xii]

Plant material should be dried in a dark ventilated space and stored immediately to maintain potency. Gotu Kola should be stored in sealed container to protect from light, air and moisture.

Taste / Odor:[xiii]

Rasa (taste): bitter, pungent,
Virya (action): cooling
Vipaka (post-digestive effect): pungent

Energetics: [xiv] Cooling to neutral and mildly astringent, balancing to all doshas.

Physiological Actions: [xv] [xvi]

- Nervine relaxant/ Anxiolytic
- Connective tissue tonic
- Veinous tonic

- Vulnerary
- Anti-inflammatory
- Anti-oxidant

Specific Indications/patterns:

1. Wound healing and collagen modulation.[xvii]
2. Increases circulation to periphery, including both the brain and the extremities.[xviii] Used in the Ayurvedic tradition to promote intellect, improve concentration, and enhance meditative focus. [xix]

Doctrine of Signatures: leaf shape resembles the cerebellum, suggestive of its affinity for cognition.

Channels entered: [xx] Liver, Spleen, Kidney
Tissue States: [xxi] depression, relaxation and atrophy

Clinical Use:

Nervous System:

Generalized Anxiety Disorder [xxii] [xxiii]

Anxietous Depression [xxiv]

To improve concentration, memory and alertness[xxv]

- ADD/ADHD[xxvi]

Anti-aging tonic[xxvii]

Cognitive decline [xxviii] [xxix] (incl. dementia and Alzheimer's disease) and memory loss [xxx]

Epilepsy[xxxi]

Stroke[xxxii]

Integumentary:

Scleroderma[xxxiii]

Psoriasis and eczema [xxxiv] [xxxv] [xxxvi]

Wounds, Scars, Burns[xxxvii] [xxxviii]

Dry Skin Conditions[xxxix] (incl. Ichthyosis[xl])

Cellulite [xli]

Keloid[xlii]

Pre and Post Surgical healing [xliii] [xliv] (incl. oral surgery and cancer)

Ulcers[xlv] [xlvi] (internal and topical use)

- diabetic ulcers
- bed sores
- syphilitic ulcers[xlvii]

Weak nails/ hair[xlviii] esp. in hypothyroidism

Leprosy [xlix] [l]

Musculoskeletal:

Soft tissue injury to ligament or tendon [li] [lii]

Osteoarthritis[liii]

Rheumatism[liv]

Joint injury and inflammation[lv]

Cardiovascular: [lvi]

Varicose Veins[lvii]

Chronic venous insufficiency

Venous Hypertension

Stasis Ulcers (From varicose veins)

Many side affects of diabetes involving weakened micro-circulatory capillary flow:

- Neurapthy
- nephropathy

Gastrointestinal:

- Hemorrhoids[lviii]
- Inflammatory disease of bowel[lix] [lx] (including Ulcerative Colitis, Crohn's disease)
- Gastric ulceration and inflammation[lxi] [lxii] including Leaky gut
- GERD and other digestive tissue destruction to prevent scar tissue from forming
- Gingevitis, periodontal disease[lxiii]

Key Constituents: [lxiv] [lxv]

- Triterpene Saponins:

    - Asiatic acid, madecassic acid, centellic acid, centoic acid, asiaticentoic acid
    - Asiaticoside, madecassoside, centelloside , brahmoside, brahminoside, thankuniside, iso-thankuniside, brahmic acid, isobrahmic acid

- Glycosides: 3-glucosilquercetin, 3-glucosylkaempferol, 7-glucosylkaempferol
- Flavonoids: quercetin, kaempferol
- Volatile Oil:
    - Camphor, cineole
    - Sesquiterpenes: beta-caryophyllene, beta-farnesene, germacrene D
- Alkaloid: hydrocotyline
- Bitter principle: vellarine
- Fatty acid: oleic acid, linoleic acid, palmitic acid, stearic acid and lignoceric acid.
- Phytosterols: campesterol, sitosterol, and stigmasterol
- Oligosaccharide: Centellose
- Vitamin C
- Beta-carotene[lxvi]
- Potassium[lxvii]
- Sodium
- Amino acids

Pharmacology:

Centella's primary constituents, the triterpene saponins (asiaticoside, madecassoside and their aglycones asiatic acid, madecassic acid and others) have been most heavily researched in isolation, are often administered at dose higher than what may be practical in human subjects. Many of the plant actions are attributed to the Triterpene Fraction (TTF). Overall, pharmacological research on whole and TTF extracts support *Centella*'s traditional uses for venous pathologies, cognitive decline, anxiety, mood enhancement, epilepsy, cancer, wounds and skin ailments.

Triterpenes are comprised of a 30-carbon skeleton with pentacyclic structure of 5-carbon isoprene unit building blocks. [lxviii] Saponins are fat soluble, a portion of them are absorbed into the small intestine while the remaining quantity will pass to the large intestine where

they are transformed by microbes into their aglycone, allowing them to be absorbed into the blood stream.[lxix]

A pharmacokinetic examination by Grimaldi *et al*, found isolated asiaticoside underwent conversion to asiatic acid in the digestive system. Blood plasma levels of asiatic acid (AA) peaked five hours after administration (at 30 mg or 60mg dose) in healthy volunteers. Peak plasma concentrations and half-life values were significantly higher after repeated administration than those found following single dose. This study found that doses of 60mg AA had a half-life of 3.40 hours following single dose, compared to 10.28 hours during seven-day treatment.[lxx]

Cardiovascular Effects:

The Isolated triterpene saponins of *Centella* (or Total Triterpene Fraction (TTF)) have been researched for their use in microangiopathy. Their activity within the vasculature involves modulation of fluid balance and modulation of collagen synthesis. The triterpenes have been found to be effective for relieving the signs and symptoms of venous hypertension and venous insufficiency including edema, pain, cramping, heaviness in the lower extremities and tiredness. [lxxi] [lxxii] In addition, TTF caused reversal of alterations in fluid movement, as seen in the objective assessment of microcirculatory parameters such as capillary permeability (using laser doppler flowmetry) and transcutaneous oxygen (PO2) and carbon dioxide (PCO2).[lxxiii] [lxxiv] Air travel can cause complications for individuals with microangiopahty due to high pressure and stasis, which may increase the risk of thrombosis. TTF has been studied in subjects with venous diseases during air travel, where it attenuated microcirculatory alterations and edema.[lxxv]

The secondary mechanism through which we see TTF working is to heal the lining of the vasculature. TTF stimulates collagen synthesis and remodeling in and around the venous wall, leading to stabilization of hypoechoic and low-density carotid plaques.[lxxvi] These findings are relevant for reducing the risk of thrombosis and atherosclerosis, as well as for improvement of microcirculatory parameters in diabetic microangiopathy (including retinopathy, nephropathy and neuropathy). [lxxvii] [lxxviii]

Research involving whole plant extracts (alcoholic and aqueous) of *Centella asiatica* (CA) was shown to prevent ischemic reperfusion injury following myocardial infarction in rats. [lxxix]

Through preventing oxidation of lipids, protein and DNA, CA exerted a cardioprotective effect in animal models.[lxxx] While administration was though oral route in these studies, it exceed the dose ranges relevant for human consumption at 200mg/kg body weight of aqueous extract and100-1000mg/kg alcoholic extract, respectively.

Nervous System Effects:

Research in animal models suggests whole CA extracts and dried leaf powder confer neuroprotective effects through modulation of neurotransmitter status (ie GABA), improvement in free radical scavenging activity and enhancement of neuronal branching. Subsequent improvements in learning and memory are also seen.

Gamma-Aminobutyric acid (GABA) is an inhibitory neurotransmitter in the central nervous system, increases in GABA availability bring about relaxation and reductions in anxiety.[lxxxi] Whole plant extracts of *Centella* exhibit dose dependent increases in GABA levels in the rat brain.[lxxxii] An *invitro* study found the mechanism by which brain GABA levels were affected was though CA stimulating Glutamic Acid Decarboxylase (GAD), thereby increasing GABA concentrations and neurotransmission.[lxxxiii]

Centella research has focused on possible mechanisms for attenuating age related neurological changes. Aging causes progressive changes in the antioxidant defense system. Oral administration of Centella extract to mice caused reductions in oxidative damage as indicated by lower levels of lipid peroxidation and protein carbonyl oxidation.[lxxxiv] A 2008 study in animals found dried leaf powder of *Centella* administered to mice over a four-week period caused significant reduction in malondialdehyde, reactive oxygen species and hydroperoxide levels, as well as higher levels of anti-oxidant enzymes throughout the brain.[lxxxv]

In addition to modulating neurochemical parameters aqueous CA extracts (dose of 200 and 300mg/kg) have been found to improve learning and memory as indicated by improvement in proficiency at shuttle box, step through, step down and elevated plus maze paradigms.[lxxxvi]

An Indian study found that fresh juice of *Centella* caused enhanced neuronal growth (indicated by dendrite length and arborization) in the rat amygdala, as compared to untreated cohorts, suggesting applicability for neurodegenerative disorders. The dosing

regime was 2,4 and 6mL/kg body weight of juice, this study serves to validate traditional preparation methods of the plant despite high dose levels.[lxxxvii]

Neurodegenerative Conditions:

Alzheimer's Disease (AD) is a neurodegenerative disease characterized by beta-amyloid (β-amyloid) protein plaque accumulation, with numerous factors that may contribute to pathogenesis.[lxxxviii] Given its traditional use to enhance cognitive function, *Centella* has been examined in animal and *invitro* studies which suggest it relevance for this condition. Preliminary research in this field has focused on animal and *invitro* examinations of the anti-oxidant defense system and β-amyloid plaque processing primarily, with a few studies examining cholinergic system.

In animal models of Alzheimers Disease, administration of aqueous and powdered (2.5 g/kg/day) *Centella* extracts attenuate behavioral abnormalities associated with beta-amyloid plaque, and improve learning.[lxxxix] [xc] In addition, post-mortem examinations demonstrated reduction in oxidative stress parameters (glutathione and catalase levels) and significantly reduced β-amyloid plaque levels. [xci] [xcii] Many of these actions can be attributed at least in part to the TTF, which improved cognitive function and increased superoxide dismutase (SOD) activity in the hippocampus and cortex when administered to animals orally. [xciii]

Isolated Asiatic acid applied invitro reduced production of β-amyloid protein, prevented cellular apoptosis in tissue where high levels of beta-amyloid plaque were present, reduced free radical concentrations and minimized glutamate-induced toxicity. [xciv] [xcv] [xcvi] Researchers found a reduction in levels of β-secretase, which is involved in the production of β-amyloid proteins and increased ADAM10 (an enzyme involved in processing β-amyloid protein), suggesting this possible mechanism of action for these changes in brain chemistry.[xcvii]

Acetylcholinesterase (AcHE) inhibition is the mechanism of action for the majority of the pharmaceuticals used to manage Alzheimer's disease. This mechanism is not curative but does help to manage cholinergic deficit, which is thought to contribute to the pathology. Isolated triterpene fractions of *Centella*, (administered via Injection) demonstrated inhibition of AcHE and improvements in learning and memory as indicated by passive avoidance testing.[xcviii] Hydroalcoholic extracts of *Centella* applied invitro showed 50% inhibition of AchE activity concentration of 100-150microg/mL, suggesting possible relevance for managing nervous system changes in AD.[xcix]

Another mechanism by which *Centella* may be beneficial to neurodegenerative conditions is by activity on cyclic AMP response element binding protein (CREB). CREB activity is relevant to long term memory formation, and is the currently being researched as possible axis for AD intervention.[c] Application of *Centella* extract invitro enhanced phosporylation of CREB in both healthy cortical cells and neuroblastoma cell lines expressing β-amyloid. The study suggests that increasing availability of CREB may be one mechanism of action by which *Centella* extracts modulate cell signal transmission.[ci]

Epilepsy

Research in Epilepsy has been restricted to animal and invitro studies, further studies are needed to determine the usefulness of *Centella* in seizure disorders at appropriate dose ranges for human subjects. In rat models of epilepsy, animals treated with *Centella* orally (at 200mg/kg and 300mg/kg body weight) showed increased levels of AcH and a reduction in AcHesterase after the seizures compared to placebo. These studies suggest that AcH sparing cholinergic modulation may contribute to the anti-convulsant activity of this plant, supporting its use in minimizing cognitive damage caused by epileptic seizure.[cii] [ciii]

Integumentary Effects:

Wounds, burns and keloids

Oral and topical applications of TTF of *Centella* (as cream) advance speed and quality of wound healing and reduce both scaring and keloid formation.[civ] Centella's wound healing effects have been demonstrated in human subjects, a randomized placebo-controlled study of diabetic wound patients (N=200) orally administered 100 mg asiaticoside three times per day showed improved granulation tissue formation and reduced scaring.[cv] This activity is attributed to isolated asiaticoside and madecassoside which stimulate Type I collagen production in human cell cultures.[cvi] Animal research using Oral and topical administration routes for whole *Centella* alcoholic extracts in both healthy and delayed healing (diabetic) models saw faster rates of wound epithelialisation, wound contraction and increases in tensile strength compared to controls. [cvii] [cviii] Isolated TTF used in animal burn wounds showed similar benefit with improvement of anti-oxidant activity, promotion of collagen synthesis and angiogenesis.[cix] [cx] Finally, *Centella* has relevance for fibroproliferative disorders such as keloid. An *invitro* study found it to prevent keloid formation through

inhibition of Transforming Growth Factor-β1 (TGF-β1) induced collagen synthesis and Plasminogen activator inhibitor-1 (PAI-1) expression in keloid fibroblasts.[cxi]

Scleroderma

Isolated madecassol used orally and topically as ointment over 6 months reduced the symptoms of scleroderma as indicated by reduction in hyperpigmentation, vascular trophic disorder and number of lesions.[cxii]

Allergies and Pruritis

Animal modeling has shown high doses of CA extracts exhibit anti-allergic and anti-pruritic activity to the skin. CA extracts (alcoholic and aqueous) inhibited mast cell degranulation, reduced itching and exhibited anti-inflammatory activity comparable to ibuprophen. Mechanisms are thought to involve the inhibition of enzymes such as protein kinase C, protein tyrosine kinase and phospholipase A2, as well as lipoxogenase and cyclooxygenase, which contribute to pro-inflammatory signaling pathways seen in prostaglandin biosynthesis.[cxiii]

Musculoskeletal Effects:

Animal models using TTF found benefit for degenerative joint conditions including Rheumatoid and Osteoarthritis.[cxiv] One study demonstrated the inhibition of Nitric Oxide (NO) production, and subsequent reduction in osteoarthritic cartilage degradation.[cxv] Researchers have found modulation of both humoral and cellular-mediated immunity by Madecassoside to contribute to this anti-inflammatory activity.[cxvi] Madecassoside administered orally to mice in arthritic model demonstrated reduced levels of infiltration of inflammatory cells to the joint, reduced synovial hyperplasia, and lowered IgG levels, thereby preventing joint destruction.[cxvii]

Immune System effects:

Preliminary research invitro and invivo has shown *Centella* extracts are anti-proliferative and chemoprotective. *Centella* extracts demonstrate dose dependent inhibition of cancerous human cell lines of the breast, colon and skin (melanoma). [cxviii] [cxix] [cxx] Modulation of certain apoptosis regulating enzymes including Bax, Bcl-2 and caspase-3, as well as generation of Reactive Oxygen Species are suggested mechanisms for these actions. [cxxi] In addition, administration of CA extracts orally to animals exposed to radiation caused a reduction in radiation-induced damage to DNA[cxxii], suggesting further relevance of this plant during

radiation therapy.

Furthermore, *invitro* research has highlighted TTF's anti-inflammatory activity through chemical signaling. These isolated extracts inhibit production of nitric oxide (NO), Prostaglandin E(2) (PGE(2)), tumor necrosis factor-alpha (TNF-alpha), interleukin-1 beta (IL-1beta) , IL-6 and cyclooxygenase-2 (COX-2). TTF inhibited activation of Nuclear Factor-Kappa B (NF-kappaB), and caused subsequent blocking of p65 protein translocation to the cell nucleus.[cxxiii] These studies suggest its relevance for cancer therapy and to support healthy tissue.

Clinical Trials:

This section includes research using whole plant extracts of *Centella*, excluding studies using it in combination or using the isolated triterpene fraction (TTF). Clinical research using whole *Centella* extracts are limited, and the available trials all have small sample size. Research in human subjects involving cancer and venous pathologies has not been performed, but given the usefulness of this plant in *invitro* and *invivo* applications, this area should be explored.

Anxiolytic Effects:

Two small clinical trials support *Centella*'s use to attenuate anxiety. Given the positive results from the research more large-scale studies are warranted. A small clinical trial of 33 subjects with Generalized Anxiety Disorder (GAD) were administered 500mg capsules 2x/day (of 70% hydro-ethanolic extract) of *Centella asiatica* (CA). They found significant improvement in anxiety levels (indicated by standard questionnaires), as well as reductions in stress and depression, and improved cognition.[cxxiv] *Centella*'s anxiolytic activity was examined in a double, blind placebo controlled study which assessed of forty healthy subjects. Subjects received 12g/day of Gotu Kola daily and found reduced anxiety levels as demonstrated by acoustic startle response compared to placebo.[cxxv]

Cognitive effects:

In a small, randomized double-blind, placebo controlled clinical trial, the effect of *Centella*

was examined on age-related decline in 28 health elderly subjects over the course of 2 months. Use of the highest doses of 750 mg *Centella*/day was found to be useful for both improving working memory and improving mood.[cxxvi]

Cognitive effects in neurodegenerative models:

Clinical trials for use of CA in Alzheimer's disease or other existing neurodegenerative conditions are presently lacking. The only such study had a limited sample size of 6 individuals, ages 65 and above, with mild cognitive impairment (MCI). Subjects used 1000mg/day of *Centella* powder over a 6-month period and experienced improvement in mental well-being, sleep quality, and reductions in depression.[cxxvii]

Safety Issues: [cxxviii]

Centella's long held traditional use as a vegetable, and the current scientific literature both attest to the safety of this herb. [cxxix] [cxxx] [cxxxi] [cxxxii] Side affects may include irritation topically on open wounds or inflamed mucus membranes within the digestive system (ie. GERD). In particular, topical application has the potential to cause rare cases of allergic contact dermatitis in sensitive individuals,[cxxxiii] the reaction is believed to be due to Medecassol.[cxxxiv] The isolated triterpene fraction is generally well tolerated, though it may cause skin irritation or gastric upset. [cxxxv][cxxxvi] High doses are well tolerated in animals, in mice receiving 2.5g/kg dried plant orally showed no adverse affects. [cxxxvii]

Centella is categorized by the FDA as Pregnancy Category B1, meaning limited applications in women have shown no harmful effects on the fetus, and animal studies show similar results. It is compatible with breastfeeding (Lactation Category C). *Centella* is considered safe for use in children.

Preparation and Dosage:

Herbalist Todd Caldecott:

Fresh Juice (*svarasa*): 25mL BID-TID

Powder (*Curna*): 3-10g BID-TID

Tincture : fresh 1:2 95% ETOH; dry 1:3 50% ETOH

1-5mL BID-TID

Infusion (*Phanta*): 30-120mL BID-TID

Nasya oil: 2gtt for nervous system disorders

Herbalist Larken Bunce:[cxxxviii]

Tincture: Dry 1:2 30-35% ETOH

3-4mL of a 15mL Formula

For surgical healing, up to three-weeks pre and post-operatively: 7.5mL/day

Tea for soft injury of anxiety: 2-5g/day

Topical: Salve containing *Centella* infused oil or 3mL tincture in cream preparation

Herbalists Simon Mills and Kerry Bone:[cxxxix]

Tincture: 1:2, 30% ETOH , 3-6 mL/day

Tea Infusion: 1.8 g/day dried aerial parts

Triterpene Fraction (TTF):

60-180mg/day (approximately equivalent to 2.5-7 g/day of dried herb)

Herbalist Donald Yance: [cxl]

Tincture: 1:1 fluid extract, 3-8mL/day

Tea infusion: 3-8g/day

Standardized Triterpene Extract: 150-1,000mg/day powder

KPS Khalsa and Michael Tierra:[cxli]

Fresh juice: 1-4 tsp, taken in the morning

Whole fresh herb cooked or raw in salad

Tea infusion:

For acute skin disease: 1-2 oz dried herb daily
For inflammatory skin disorders 1-3 oz dried herb daily

Capsule: 1 capsule/day as rejuvenating tonic

Nasya preparation with ghee:
For Nervous system disorders: 2 drops in each nostril several times daily

Chinese Herbal Medicine: Materia Medica:[cxlii]

Dried herb: 12-30 g

Combinations and Similar Herbs:

For eczema: viola, Oregon grape root and gotu kola.[cxliii]
For poorly healing wounds: calendula, echinacea and gotu kola.

For dry thinning hair with He Shou Wu.[cxliv]

Miscellaneous Facts:

First recorded writing about *Centella* was in the *Divine Husbandman's Classic Materia Medica,* the earliest Chinese pharmacopeia dated to 2698 BC.[cxlv]

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Acorus calamus and Acorus americanus

Rebecca Dalgin

NOMENCLATURE

Botanical Nomenclature

Acorus calamus, Acorus americanus

Botanical Family

Acoraceae

Taxonomic Distinctions of North American Population

Among botanists there has been debate as to the taxonomical and botanical differentiation among *Acorus* species in North America. Generally *Acorus calamus* (more specifically differentiated as *A. calamus var. americanus,* the diploid (2n) phenotype) or *A. americanus* have been used to refer to all *Acorus* species in North America. While there is still confusion perpetuated in much of the literature, the *Flora of North America*, published in 2000, is the first flora to clearly define two separate morphologically distinct *Acorus* species: *A. calamus,* an introduced sterile triploid and *A. americanus*, a native fertile diploid. Both species exist in North America (Thompson 2000:125).

Sub Species of *A. Calamus*

The species *A. calamus* is divided into phenotypes identified by the number of chromosomal pairs contained in the DNA. Subspecies generally occur in different distribution areas. Much of the literature refers to the diploid (2n) variety as *A. calamus var. americanus.* At present, *A. calamus var. americanus* is often considered synonymous with *A. americanus.* The latter is a distinctly diploid species (Thompson 2000:124) while *A. calamus,* found in North America, Europe, temperate India and the Himalayan region, is triploid (Motley 1994). The issues is further complicated as the *A. calamus* population in Asia does have a diploid phenotype morphologically distinct from the North American diploid *A. americanus*

(Thompson 2000:124). This diploid is still classified as *A. calamus.* There is also a tetraploid (4n) variety, *A. calamus var. angustatus,* found in Eastern and Tropical Southern Asia (Motley 1994) as well as the hexaploid (6n) variety from the Kashmir area (Small and Catling 1999:15). The differences among varieties are significant as they are associated with different levels of chemical constituents resulting in variance of medicinal actions and safety profile.

Species, Phenotype, and Distribution			
Species	**Phenotype**	**Sub Species Name**	**Distribution**
A. calamus	Diploid (2n)	Formerly *A. calamus var. americanus*	Asia
	Triploid (3n)	*A. calamus var. calamus*	North America (introduced from Europe), Europe, temperate India, Himalayan region
	Tetraploid (4n)	*A. calamus var. angustus*	Eastern and tropical Southern Asia
	Hexaploid (6n)		Kashmir area
A. americanus	Diploid (2n)	Formerly *A. calamus var. americanus*	North America (native), potentially Siberia

Common Names

English: Sweet Flag, Calamus

Cheyenne: *wi'ukh is e'evo* (bitter medicine)

Hudson Bay Cree: *pow-e-men-arctic* (fire or bitter pepper root)

Penobscot and Nanticoke: muskrat root

Pawnee: *kahtsha itu* (medicine lying in water)

Ayurvedic Tradition: Vacha

Chinese Medicine: shui chang pú (watery flourishing weed)

Definition/Part Used

Calamus consists of dried or fresh unpeeled rhizomes and roots of *Acorus calamus* or *Acorus americanus.*

HABITAT

Habitat

Both *A. calamus* and *A. americanus* species grow in wet open areas, marshes, swales, and along edges of quite water (Thompson 2000:126-127).

Companions and Role in Ecology

Calamus is often found growing among Blue Flag, Cattails and other wetland plants. Muskrats are often found to be in the vicinity. It has even been speculated that the musk scent they produce is due to the large quantities of Acorus they ingest. In her dissertation, Thompson discusses the relationship between Acorus, muskrats, and humans. There seems to be "a closely linked ecological relationship between Native Americans trapping muskrats and using Acorus, muskrats eating Acorus, and Acorus. Muskrat feeding habits may in part be responsible for the dispersal of Acorus, and Native American may have intentionally planted Acorus both for their own medicinal use and to ensure food for the muskrat, which was economically valuable to them (Morgan 1980). Thus, the many Native American names for Acorus, which involve muskrat as a root word, may reflect an important economic and ecological relationship among man, plants, and other wildlife" (Thompson 1995, 450).

Distribution and Range

There is some debate as to the origin of Acorus. Originally *A. calamus* was most likely native to southern Asia spreading widely through the continent and then introduced and naturalized in Europe and North America. Thompson proposes that *A. americanus*, the distinctly fertile diploid species of North America, is related to *A. calamus* but has a long portion of its history where it evolved separately (Thompson 1995; 385). The *A. calamus* species in North America stem from a few initially introduced individuals (Thompson 1995; 414).

***A. americanus* range:** Alta., B.C., Manitoba, N.B., Newfoundland, Labrador, N.W.T., N.S., Ontario, P.E.L., Quebec, Saskatchewan, Alaska, Connecticut, D.C., Idaho, Illinois, Indiana,

Iowa, Maine, Massachusetts, Michigan, Minnesota, Montana, Nebraska, New Hampshire, New Jersey, New York, North Dakota, Ohio, Pennsylvania, Rhode Island, South Dakota, Vermont, Virginia, Washington, Wisconsin, potentially central Siberia (Thompson 2000; 127).

***A. calamus* range:** N.B., N.S., Ontario, Quebec, Alaska, Arkansas, California, Colorado, Connecticut, Delaware, C.D., Georgia, Illinois, Indiana, Iowa, Kansas, Kentucky, Louisiana, Maine, Maryland, Massachusetts, Michigan, Minnesota, Missouri, Nebraska, New Hampshire, Hew Jersey, New York, North Carolina, Ohio, Oklahoma, Oregon, Pennsylvania, Rhode Island, South Carolina, South Dakota, Tennessee, Texas, Vermont, Virginia, West Virginia, Wisconsin, Europe, Asia, Africa, Indian Ocean Islands, Pacific Islands (Thompson 2000; 126).

BOTANICAL IDENTIFICATION

Botanical Identification: Shared Characteristics for *A. calamus* L. and *A. americanus*
General: Herbaceous, perennial, rhizomatous.
Leaves: Basal, not differentiated into petiole and blade, bases are sheathed, white and colored with pink or red, remainder of leaf bright green, sword shaped, parallel venation.
Sympodial leaves (structure functioning as flower stem and bract [Haines 2000]): sheathed base, develops inflorescence, sympodial leaf extends beyond inflorescence
Inflorescence: spadix, semi-erect, nearly cylindrical, tapering, obtuse apex
Flowers: arranged to cover the spadix in a diamond pattern, bisexual, six light brown tepals, 6 stamen, yellow anthers, 1 green ovary (Thompson 2000: 124, 126; Hilty 2011).
Rhizome: stout, shallow branching, knobby with a brown exterior and white interior (Hilty 2011).

Morphological Differentiation Between *A. calamus* and *A. americanus*

Characteristic	***Acorus calamus***	***Acorus americanus***
Vegetative Leaf Height	up to 1.75m	up to 1.45m

Leaf Veins	1 prominent	2-6 prominent
Leaf Cross Section	rhomboid	swollen in center and gradually tapering to ends
Sympodial Leaf	usually shorter than or equal to vegetative leaves	usually equal to or taller than vegetative leaves
Leaf margin	often crisped or undulate	usually entire
Spadix	usually 4.9-8.9 cm long at anthesis	usually 3.3-7.4 cm long at anthesis
Flowers	3.0-4.0mm long	2.0-3.0 mm long
Pollen grains	not staining in aniline blue	usually deeply staining in aniline blue
Fruits	not produced	obpyrimidal berries
Seeds	not produced	tan narrowly oblong to obovate; embedded in mucilage

While morphological differences appear to be quite clear, they are rather subtle and hard to differentiate in the flesh. The difference in number of prominent veins is difficult to assess as the prominence of the veins is relative to the mid-vein prominence. The secondary veins of *A. calamus* are no more than .5 times the width of the mid vein while in *A. americanus* one or more of the secondary veins is .75-1 times as wide as the mid-vein. The clearest difference is seen in the formation of fruit. In late summer, *A. calamus* will appear to have a shriveled ovary while *A. americanus* will show swelling ovaries (Haines 2000). Unfortunately, even though *A. americanus* does have the capability to flower and reproduce sexually via berries and seeds, some sources conclude it rarely does so (Fara, 2005).

Table after Haines (2000) with additional information from Thompson (2000)

Botanical Identification: Differentiation Between Acorus and Other Species

While it has the potential to be confused with plants such as Iris or Cattail that often grow alongside it and have similar sword shaped leaves, Acorus is differentiated by the unique characteristic aromaticity of the bruised leaves or rhizomes. Additionally, Acorus leaves are bright green/yellow green with a spadix borne midway up the sympodial leaf versus the blue-green leaves and showy flower of Iris (Thompson 2000:125).

CULTIVATION AND HARVEST

Propagation and Cultivation

Propagation and cultivation is generally through root division in the spring. Calamus particularly likes to grow on wetland edges, however it can be grown in a garden of field with deep rich soil and daily watering (Babineau, 2000). Jim McDonald describes growing the plants easily from root cuttings in a non-draining planter filled with wild soil. He maintains high moisture in the planter, though routinely dumps of standing water to reduce mosquito larvae growth.

Harvest

Pay particular attention as to the location from which it is harvested. Water should be clean and not near contaminants as some consider the plant to take up pollutants (McDonald). Harvest two to three year old roots and rhizomes in early Spring (once source specifies June) or late autumn, ensuring they are not hollow. If harvesting from the wild, take only part of the rootstock, replanting the rest to continue growing. Clean and dry whole or tincture the unpeeled roots (Babineau 2000).

Endangered Status

According to the USDA plants database, *A. americanus* is considered endangered in Pennsylvania (at the recommendation of Thompson). United Plant Savers recommends judicious harvest. Thompson reports that both species are secure globally and *A. americanus* is not at risk, but rare in regions at the edge of its range with seven political regions having assigned unofficial conservation status to *A. americanus*. Four of these states however are not in the natural range of *A. americanus*. More research is needed in the field to refine conservation status (Thompson 1995;464). In India, *A. calamus* is considered nearly endangered due to excessive wildcrafting (Verma et al., 2012).

COMERCIAL SOURCES AND HANDLING

Identify origin of plant and ploidy if possible to ascertain which constituents are present. McDonald is skeptical of the legitimacy of differentiation between species commercially. Dried root should not be kept for more than a year as it loses its aromaticity.

TRADITIONAL USE

First Nations of North America

As cataloged in Moerman's (1998) ethnobotanical dictionary, *A. calamus* is one of the top ten plants with the greatest number of uses among first nation peoples. While referred to almost exclusively as *A. calamus* in relation to use by indigenous peoples of North America, it is most likely *A. americanus* that was/is actually used (Thompson 1995, 444). Though extensive ethnobotanical reports of usage in indigenous North American cultures exist, much information has been lost due to the history of oppression and exploitation. For this reason, it is likely that there are more subtleties in its traditional use by First Nations peoples than documented in the literature.

Among different tribes, the plant has numerous names, indicators of its value and place in their medicine and culture. Names conveying medicinal action or taste include *enausa'pokotikun*(a simple penetrator) from the Menominee of Wisconsin, *pow-e-men-artic* (fire root, bitter pepper root) from the Cree, *wi'ukh is e'evo* (bitter medicine) from the Cheyenne of Montana and *kahtsha itu* (medicine lying in water) by the Pawnee. Other names refer to the roots physical appearance; Ojibwa called it *na'buguck* (something flat), Lenape named it *pepachkana* (place where it is broken again and again) and the Caughnawaga of Quebec identified it as *anon-no-ron ka-te-ra-ra-ken* (with white roots). Many other names refer to the plants faunal association with the muskrat-- the Micmac of Atlantic Canada called it *ig gig'wesukwul* (muskrat root) and the Abenaki of Quebec called it *mokwaswaskw* (muskrat plant) (Thompson 1995, 446).

In ethnobotanical literature, Sweet Flag is used for a vast variety of health concerns. Following are the most widely used applications (as collated by Thompson, 1995) with the number of tribes reported to consider Sweet Flag for that particular use:

Respiratory:

- colds, flu, and sore throat (28 tribes)
- anti-tussive (10 tribes)

General:

- Prophylactic/tonic (13 tribes)
- Panacea (13 tribes)
-

GI:

- intestinal pain/colic (11 tribes)
- carminative (9)

Nervous/Endocrine System:

- **“**antifatigue”/stimulant (8 tribes)
- diabetes (2 tribes; not widespread use for this purpose, however there is some pharmacological data, anecdotal evidence and contemporary application; also type II diabetes was not common before the arrival of industrialized food; account from 1950s of Dakotas using root successfully for diabetes Howard, 1953 p. 608-609 as cited in Kindscher, 1992)

Other:

- Toothache (12 tribes)
- Analgesic for muscle/cramps/spasm/pain and contractions (8)

Spiritual:

- amulet/protective charm (8)
- ceremonial bundles or religious rituals (8)

For some tribes in particular, Sweet Flag was especially important. In the early 1900s, one writer reported Sweet Flag as “perhaps the most important herb in Penobscot pharmacology” (Speck 1917; 305). To the Cheyenne, the plant was considered so sacred that only qualified Sundance priests could collect it along with the making of offerings (Thompson, 1995). Two stories, one from the Malecite and the other from the Penobscot demonstrate the cultural and medicinal importance of Sweet Flag. Herbert Milton Sylvester retells the Malecite tale:

> “Long time ago a great sickness fell upon the tribe and many people died. They died so fast that those who were left could not make graves quickly enough and many were put in one large hole. At last there appeared to one of the men in a dream a strange being as of a man covered with joints of brass. 'I am, said he, Ke-wis-wask (sweet flag) and can make you well. Dig me up and steep me in water and drink me and I will cure you. After saying this, he disappeared. The next day the man did as he was told. He dug up the flag root and steeped it and gave the water to the Indians and after drinking it, they soon recovered.”

Frank Speck (1917; 305) recounts the Penobscot story:

> "A plague of sickness was sweeping the Indians away. There was no one to cure the people. One night a man was visited by a Muskrat in a dream. The Muskrat told him that he was a root and where to find him. The man awoke, sought the muskrat root, made a medicine of it, and cured the people of the plague."

A. americanus is still used in traditional ways. The Dakota traditionally wore a paste of the plant on their faces during battle to instill fearlessness and provide stamina. It was thought that since muskrats eat Acorus as a major food source it would make the warriors fearless like the Muskrat. During the wars of the 20th century, Native American soldiers chewed on Acorus rhizomes to promote stamina and endurance (Thompson, 1995).

Chinese Medicine

Other Acorus species, namely *A. tatarinowii* and *A. graminaeus (*both known as Acori tatarinowii Rhizoma or shi chang pu), are more commonly used in Chinese medicine, however *A. calamus* does have a place in the materia medica. Referred to as Acori calami Rhizoma pharmaceutically, the Chinese name, shui chang pú , translates literally into "watery flourishing reed."

Characterized as acrid, warm and bitter, shui chang pu enters through the channels of the heart, liver and stomach. The rhizome is considered to transform phlegm, facilitate the removal and elimination of dampness, open the orifices, strengthen the Stomach (refers to Chinese concept of the Stomach), fortify the Spleen, kill parasites and stop itching.

As in nearly all of the traditional literature and cultures where *A. calamus* has been used for a long time, shui chang pu is indicated for discomfort in the digestion specifically characterized by dampness and turbidity obstructing the middle burner with epigastric pain, abdominal distention, a poor appetite, and a greasy tongue coating. In this circumstance shui chang pu acts to improve the movement of qi while strengthening the Stomach.

The other main indication for shui chang pu in the Chinese materia medica is tremors and loss of consciousness or seizures originating from phlegm-heat (Bensky et al., 2004).

(Phlegm-heat arises from “external evils” binds the chest and congests and obstructs the lungs [Wiseman and Ye 1998, 437]). Under these circumstances, Shui chang pu is useful by extinguishing the wind, opening the orifices, and transforming the phlegm. (It is recommended with specific other herbs for sudden loss of consciousness and for seizures).

Generally speaking the plant is used for epigastric distention due to damp stagnation, diarrhea, dysenteric disorders, wind-damp painful obstruction, epileptic seizures, palpitations and forgetfulness. External applications are for sores and scabies. Of interesting note is a statement in the Illustrated Classic of the Materia Medica from the eleventh century that it should not be used for medicine. The Chinese *Acorus* is considered to have up to 80% b-asarone and so use of the diploid *A. calamus var. americanus* (most likely actually referring to *A. americanus*) is suggested. The aroma of *A. calamus* is considered to be less intense than that of *A. tatarinowii* (Bensky and others 2004:958-959).

Ayurvedic

The name Vacha or Vaca for *A. calamus* in the Ayurvedic tradition means “voice” or “speaking,” a name indicative of the intelligence or self-expression the plant is considered to encourage (Frawley and Lad, 1989). Vacha is considered pungent, bitter, astringent/heating, reducing to Vata and Kapha and increasing Pitta. The rhizome acts in a stimulant, rejuvenative, expectorant, decongestant, nervine, antispasmodic, and/or emetic manner in the nervous, respiratory, digestive, circulatory and reproductive systems. It is indicated for colds, cough, asthma, sinus headaches, sinusitis, arthritis, epilepsy, shock, coma, loss of memory, deafness, hysteria and neuralgia (Frawley and Lad, 1989). As in many other cultures where it is used, its strength for digestion and its association with the mind/clarity/nervous system seem most emphasized. Sometimes used on its own but most often as part of formulas, the rhizome is used in dyspepsia, flatulence and diarrhea as a stomachic and carminative (Caldecott 284; Dutt 1877, 251)

Considered sattvic, Vacha is “one of the best herbs” for the mind. In conditions where the ability to speak is lost, as in *apasmara* (epilepsy), Vacha is considered to bring one back to normal consciousness with its strong aromatic quality. Also in *unmada*, or psychosis, Vacha is an important remedy. The applications for Vacha in Ayurveda are copious, however at a basic level, the plant rejuvenates the brain and nervous system through purification and revitalization while clearing out toxins and obstructions (Frawley and Lad, 1989).

European

Other than a mention in Dioscordes, the plant does not appear often in European literature. Maude Grieves gives the lengthiest account. She discusses its use as a strewing herb on the floors at church festivals and makes note of the peculiar ability of the spathe to give out enough heat that the temperature around it rises noticeably. She explains that Acorus come form the name given to the plant by Dioscordes, Acoron with Con meaning the pupil of the eye. Grieves reports that in the time of Dioscordes it was used to address such issues. Like all of the other accounts, Grieves classifies the plant as an aromatic bitter and carminative used to increase appetite and benefit digestion. Chewing the dried root is useful for dyspepsia. She notes the combination of calamus and Gentiana campestris in Stockton bitters. Aside from digestion, Grieve also makes note of the dried root chewed to clear the voice. For the immune system she discusses the root for ague, low fever, and as a mild stimulant in typhoid cases. Grieve quotes Waller's British Herbal which consider the plant to be “of great service in all nervous complaints” including vertigo, headache, and hypochondria. Waller also consider it in the digestive system for dysentery, “chronic catarrh,” and when mixed with some water and white wine, as a stomachic. In Grieves account the powder is used as a substitute for ginger, cinnamon and nutmeg. In the British Pharmaceutical codex, the plant is considered an aromatic bitter and carminative that works by “removing the discomfort caused by flatulence and checking the growth of the bacteria which give rise to it.” It is used, according to the codex, to increase appetite and improve digestion (1911).

Eclectic

A. calamus does appear in the Eclectic literature, though, as Scudder (1898) notes, it is “but little used by the profession.” It is generally considered carminative, aromatic, stimulating and mildly tonic with use in flatulent colic, atonic dyspepsia, and general weakness of digestions (Felter, 1922; Felter & Lloyd, 1898; Scudder,1898; Cook,1869). Cook writes it “warms the stomach, aids the expulsion of flatus, and relieves cramps and colic.” It is compared by Scudder to Angelica and Canella in its aromatic stimulant and mild tonic activity. Also mentioned as a sialagogue (Felter, Scudder) and “breath perfume” (Felter). Scudder elaborated on its use as a sialagogue considering it for rheumatic and paralytic infections of the mouth. Externally King considered it a “valuable application” for indolent ulcers. Felter & Lloyd (1898) and Scudder (1898) also consider it for “*intermittents.*” Scudder recommends it as a substitute for tobacco.

Regulars

A. calamus was included in the Dispensatory of the United States of America. It was classified as warm, “bitterish,” pungent and a “feeble aromatic.” Its main use was as an adjuvant to preparations where a carminative was needed. The dispensatory comments the plant may be “easily misused” if given too freely or in irritated stomach and bowel conditions. One suggested preparation is boiled in milk with pimento or ginger.

SCIENTIFIC RESEARCH: CONSTIUENTS

Constituent Variety Among Chemotypes

Chemical constituents vary significantly among chemotypes. The variation is most clearly documented for a and b-asarone, however other constituents are variable mostly in quantity but also in content. Because studies generally come from India, or secondarily Europe, the ploidy of the species is often not identified. However, due to the presence of b-asarone content, an assumption is made that while the following constituents have application to *A. calamus* and *A. americanus*, they are generally based on the former species.

Thompson notes in her dissertation on *A. americanus* that the essential oil profile of *A. americanus* is significantly different than the triploid and tetraploid *A. calamus* as evidenced by the work of Rost and Bos, 1979. Unfortunately this study is unavailable, but Thompson does note the work of Rost and Bos for showing the presence of geranyl acetate, the main constituent in the essential oil of the diploid North American species, a constituent absent in the triploid and tetraploid populations of *A. calamus.*

Thompson summarizes “the confusion between two chemically-distinct, yet medicinally-important, species means that much of the literature on Acorus chemistry and use is dubious in terms of species identification...With the realization that the name, *A. calamus*, has been misapplied to another species, *A. americanus*, which is chemically-distinct from true *A. calamus*, new studies to clarify the chemical composition and medicinal use of both species are warranted” (Thompson 1995; 424).

Essential Oil Fraction (2-9%): Phenlypropanoids, Sesquiterpenes, and Monoterpenes

Phenylpropanoids: b, a, and g asarone, asarylaldehyde, acoradine, acoramone, coramone, eugenol, methylisoeugenol, phenyl indene derivatives, and calamol (Raja 2009)

The phenlypropanoid fraction of the essential oil varies the most among chemotypes. The Indian chemotype contains up to 80 % of its volatile oil fraction in phenlypropanoids while the European is composed of 13% and the American has close to none (Wichtl 2004: 98). The primary phenlypropanoid for which Calamus gains the most attention is b-asarone, also called cis-isasarone. Levels of b-asarone found in the rhizome essential oil were not detected in the diploid *A. calamus var. americanus/A. americanus*, but were found in the triploid *A. calamus var. calamus/ A. calamus var. vulgaris* at levels of 9-19 % in fresh and .3% in dried. Levels in *A. calamus var. angustatus/ A. triqueter*, the tetraploid variety, were found at 85-95% in fresh rhizome and 4.4-8.3% in dried (European Medicines Agency 2005).

Sesquiterpenes: Dried rhizomes yield about .62% sesquiterpenes and roots yield 2.5% sesquiterpenes upon distillation. Sesquiterpenes include:

- **Monocyclic sesquiterpenes isolated from n-hexane extracted essential oil of rhizome:** shyobunone (forms from the heat sensitive acoragermacrone during steam distillation), epishyobunone, and 2,6-diepishyobunone (Raja 2009; Wichtl 2004:98)

- **Bicyclic sesquiterpenes isolated with pet-ether-diethyl-ether fraction:** iscoalamendiaol (Raja 2009)

- **Tricyclic sesquiterenes:** calamenone (Wichtl 2004: 98)

- **Spirocyclic sesquiterpenes:** acorone, isoacorone (Wichtl 2004:98)

- **Other sesquiterpenes:** asulene (Meena 210), volatile/bitter sesquiterpene diketone (Wichtl 2004:98), and dioxasarcogaiacol, recently discovered in this plant (Zuag et al. 2011)

Monoterpenes: Isolated by steam distillation from the volatile oil include a and b-pinenes, myrcene, Cymene-Para, Terpinen-a, Phellandrene-b, Terpinene-g, Terpinolene, Thujane and Limonene (Wichtl 2004, Raja 2009, Meena 2010).

Non-Volatile Constituents:

Xanthone Glycosides
Bitter Glycosides: acorine and acoretin
Lignan: acoradin
Steroids: b-sitosterol
Flavones: Galangin
Triterpanoid saponins
Fatty Acids: myristic (1.3%), palmitic (18.2%), palmitoleic (16.4%), stearic (7.3%), oleic (29.1%), linoleic (24.5%) and arachidonic (3.2%) (Asif 1984)
Sugars: maltose (0.2%), glucose (20.7%) and fructose (79.1%) (Asif 1984)
Inorganic Constituents: oxalate at 2% and calcium at .078% (Raja 2009)
Other: tannins (.6-1%), mucilage, choline and small grained starch (Raja; Wichtl)

Primary Constituents in Volatile Oil Fraction Among Varying Populations:
The following exemplifies the variety in primary volatile oil constituents. All of the plants below were identified as *A. calamus*. Plant material origin if known is noted as is ploidy where it is mentioned in the study.

Place of Plant Origin	**Korea**	**China (origin unclear)**	**Finland**	**Czech Republic**
Primary Constituents of Essential Oil	41.5% methyl isoeugenol 21.3% cyclohexanone 6.87% IH-3A,7-methanoazulene 4.94% bezenaminium 2.37% cis-Asarone/ b-asarone 2.28% cyclohexanol 1.5%, calarene (1.5%) 1.29% a-gurjunene	74.6 % b-asarone 22.2% methylisoeugenol 2.9% isoelemicin .3% methyleugenol n.d. a-asarone n.d. elemecin	.75-1.87% total e.o. 10.24% b-asarone 11.07% solavetivone	1.2-2.92% total e.o. 16.11% b-asarone 18.65% g-asarone

	1.11% 2, 5-cyclohexadiene 1.08% (+)-cuparene			
ID of Plant	"Korean A. calamus"	"Rhizoma Acori calami"/A. calamus	A. calamus	A. calamus
Part	Part not specified; most likely rhizome however	Rhizome	Clean, cut and dried rhizome	Cleaned, cut and dried rhizome
Ploidy	Ploidy not specified	Ploidy not specified	Triploid	Triploid
Study	Kim et al (2011)	Wichtl (2011; 783)	Dusek et al (2007)	Dusek et al (2007)

Comparison of Diploid and Triploid Alcoholic Extracts:

Data on the chemistry of *A. calamus* dipoloid species and *A. americanus* is scarce. The most recent study that included a thorough examination of diploids *A. calamus* is elucidated below. It is unclear whether the species being examined is indeed *A. calamus* of the diploid varietal or actually *A. americanus.* The plants used in the study were grown in Botanical Gardens at the University of Turin. As this is in Italy, it is unlikely that the plants are actually *A. americanus.*

Comparison of Primary Constituents	
Triploid Alcoholic Extract	Diploid Alcoholic Extract
11% beta-asarone 5.02% a-selinene 3.28% E-b-ocimene 2.27% camphene 2.00% s-cadinol	26.33% acorone 22.81 preiso calamendiaol 8.62% iso- shyobunone 3.28% b-sesquiphellandrene 0% b-asarone.

1.54% camphor 1.42% calarene	
Data from Cinzia et al 2005	

Full Comparison of Diploid and Triploid Chemical Constituents from Cinzia et al 2005:

Compound	**KI**	**Diploid (%)**	**Triploid (%)**
α-Thujene[a]	433	0.0	*tr*
α-Pinene[a]	440	*tr*	0.4
Camphene[a]	456	*tr*	2.3
Sabinene[a]	481	0.0	0.2
β-Pinene[a]	485	0.1	0.1
Myrcene[a]	499	*tr*	*tr*
γ-Terpinene[a]	531	0.0	*tr*
p-Cymene[a]	541	0.0	*tr*
Limonene[a]	547	*tr*	0.3
E-β-Ocimene[a]	559	0.0	3.3
Z-β-Ocimene[a]	572	0.0	0.3
γ-Terpinene[a]	588	0.0	*tr*
Terpinolene[a]	630	0.0	0.1
Linalool[a]	646	0.0	0.5
Camphor[a]	715	0.0	1.5
Terpinene-4-ol[a]	768	0.0	0.1
α-Terpineol[a]	789	0.0	*tr*
Bornyl acetate[a]	948	0.2	0.1

Compound	**KI**	**Diploid (%)**	**Triploid (%)**
Nonanyl acetate	992	*tr*	0.0
α-Copaene	1097	*tr*	*tr*
α-Funebrene	1105	*tr*	0.1
α-Cedrene	1106	0.2	0.0
β-Elemene	1124	0.1	0.0
Dihydrocarveyl acetate[a]	1129	0.1	0.0
α-Cedrene	1138	*tr*	0.0
Z-*Iso*eugenol[a]	1149	*tr*	0.0
β-Funebrene	1160	1.6	0.9
β-Cedrene	1169	0.8	1.6
Calarene[a]	1187	0.0	1.4
α-*E*-Bergamotene[a]	1197	0.9	0.5
Prezizaene	1210	0.7	0.4
Caryophyllene[a]	1220	1.2	0.9
α-Humulene[a]	1221	0.0	0.6
Z-β-Farnesene	123	1.7	1.2

Compound	**KI**	**Diploid (%)**	**Triploid (%)**
	1		
β-Copaene	1238	0.6	0.0
β-Acoradiene[a]	1244	0.3	0.2
β-Curcumene	1252	0.1	*tr*
α-Neocallitropsene	1256	0.3	0.4
γ-Amorphene	1266	0.3	0.9
ar-Curcumene	1271	0.3	*tr*
β-Selinene	1273	0.0	0.2
allo-Aromadendr-9-ene	1277	0.2	0.4
α-Selinene	1289	1.5	5.0
Bicyclogermacrene[a]	1292	0.2	0.0
α-Muurolene	1297	0.0	0.1
α-Amophene	1298	0.1	0.0
Germacrene A[a]	1304	0.3	0.3
γ-Guaiene	1308	0.0	0.4
iso-Shyobunone	132	8.6	6.9

Compound	**KI**	**Diploid (%)**	**Triploid (%)**
	4		
β-Sesquiphellandrene	1336	3.3	2.7
E-Nerolidol	1405	1.3	0.4
4-α-Hydroxygermacra-1(10),5-diol	1415	1.6	1.8
Cedrol[a]	1431	0.5	0.5
Pre*iso*calamendiol[a]	1470	22.8	7.8
β-Asarone[a]	1488	0.0	11.2
Dehydroxy-*iso*calamendiol[a]	1497	0.3	0.0
τ-Cadinol	1514	0.3	2.0
α-Cadinol	1532	0.7	0.0
4-*epi*-Acorenone	1562	0.4	0.2
Khusiol	1573	0.1	5.9
Acorenone[a]	1585	5.3	9.3
Torilenol	1607	0.1	1.8
Acora-3,7(11)-dien-8-one	1630	0.5	0.0
Squamulosone	168	0.1	0.0

Compound	KI	Diploid (%)	Triploid (%)
	6		
Acorone[a]	1750	26.3	8.4
iso-Acorone	1779	1.3	0.1
n-Hexadecanoic acid	1954	0.2	0.0
Ethyl hexadecanoate	1989	0.0	2.4
9,12-Octadecadienoic acid (*Z Z*)-	2164	0.4	0.0
Methyl linoleate[a]	2191	0.5	4.6
9.12.15-Octadecatrienoic acid, ethyl ester. (*Z.Z.Z*)-	2197	0.1	0.8
γ-Sitosterol[a]	3269	0.1	2.6

Values are the mean of at least three injections. KI = Kovats Index.

[a] These compounds were identified by direct comparison with pure standards; *tr* = traces.

SCIENTIFIC RESEARCH: PHARMACOLOGY (Mukherjee, 2007 unless otherwise noted)

Constituent	Activity
a-asarone	Antispasmodic, genotoxicity and mutagenicity, sedative and hypnotic, CNS depressant, anticonvulsant, “behavioral changes,” acetylcholinesterase inhbitory/memory enhancing effect, CV actions, hypolipidemic effect
Essential oil	Antispasmodic, antibacterial, antifungal, anthelmintic, sedative and hypnotic, CNS depressant, anticonvulsant, “behavioral changes,”

	acetylcholinesterase inhibitroy/memor enhancing effect, anti-inflammatory, CV actions
Asaraldehyde	Antifungal
Acoradin	Antifungal
b-asarone alcohol extract	Antifungal
b-assarone	Insecticidal, genotoxicity and mutagenicity, sedative and hypnotic, CNS depressant, acetylcholinesterase inhibtory/memory enhancing effect, CV actions
Acetone	Insecticidal
Lectins	Anti-cancer effect
Acorine	Bitter and orexogenic (Bunce, 2011)

SCIENTIFIC RESEARCH: SELECTED INVITRO AND INVIVO STDIES

Gastrointestinal System

Spasmolytic Activity: In vivo, the oil of *Acorus calamus* rhizome inhibited excessive peristaltic movement of rabbit and dog intestines exhibiting an antispasmodic activity on involuntary muscle tissue (Chopra 1954 as cited in Mukherjee 2007). Alcoholic extracts have shown relaxation of isolated rat intestines (Agarwal et al. 1956), isolated guinea pig illeum (Bhakuni 1988 as cited in Mukherjee) and isolated rabbit jejunum (Gilani 2006). a-Asarone and the essential oil fraction to a lesser extent contribute to this spasmolytic activity through direct musculotropic action (Das 1962 as cited in Mukherjee 2007). Gilani et al. (2006) further elucidated the antispasmodic mechanism with the finding that the crude extract inhibits both spontaneous and high K+ induced contractions. Based on this, Gilani et al. hypothesis the spasmolytic activity is mediated through calcium channel blockade (CCB) due to CCB-like constituents present particularly in the n-hexane fraction of the plant. Gilani (2006) concludes this “provides a strong mechanistic base...for its traditional use in gastrointestinal disorder such as colic pain and diarrhea.”

Antidiarrheal Activity: In addition to the in vitro study Gilani et al. performed demonstrating a mechanistic explanation for antidiarrheal effects through anti-spasmodic action, an in vivo experiment has also been conducted. In mice with castor-oil induced

diarrhea, both aqueous (at room temperature) and methanolic extracts of dry *A. calamus* rhizome, harvested in India, were assessed for antidiarrheal properties. Extracts were administered at doses of 3, 7.5 and 15mg thirty minutes prior to castor oil. A dose dependent effect was seen for both extracts though to a greater extent with the methanolic extract with which the induction time of diarrhea and total weight of the feces was reduced significantly (Shoba and Thomas, 2001).

Antiulcerogenic Activity: The ability of *A. calamus* rhizome ethnolic extract to inhibit gastric secretions and to protect gastroduodenal mucosa against injury from pylori ligation and other ulcer inducing agents was examined in rats. *A. calamus* rhizomes used in this study were “procured from the local market” presumably where the study took place in Saudi Arabia. An oral dose containing 500mg of the plant per killogram of weight administered thirty minutes prior to ulcer inducing agents showed significant antisecretory, antiulcerogenic and cytoprotective activity in the rats. Volume and acidity of basal gastric secretions were significantly decreased, as was severity of duodenal ulcers. Cytoprotectivity against gastric lesions found in this study suggests a protective effect on gastric mucosa potentially through “adaptive cytoprotection” wherein cytoprotective activity results from prostaglandins generated via mild irritation. In *A. calamus,* this activity may be attributed to sesquiterpenes (Rafatullah et al. 1994). a-Asarone may also be implicated as it has shone antiulcer activity when administered intra-abdominally (at 310mg/kg) or internally (417.6mg/kg) to mice (Belova 1985). However, Belova's study involves a fairly high dose of an isolated constituent applied in a manner not clinically applicable. Rafatullah et al (1994) did conclude that their findings supported the traditional use of *A. calamus* for gastropathy, however it should be noted the dose given in this study would be equivalent to a 55kg person taking a 27.5g dose-an excessively large amount to consume.

Nervous System

Pharmacologically, *A. calamus* is considered to have significant effect on the central nervous system as an anticonvulsant, sedative, hypnotic, and memory enhancer.

Anticonvulsant Activity: Hazra et al. (2007) concluded that *Acorus calamus* prevents the development of ferric chloride induced epileptogenesis in rats by modulating antioxidant enzymes. Results were based on pretreatment with 200mg/kg of *A. calamus* for 14 days prior to induction along with diazepam.

In a 2009 mouse study (Yende et al) a hydro-alcoholic extract of *A. calamus* rhizome (most likely procured in India) was studied along with antiepileptic pharmaceuticals (phenytoin and phenobarbital) in induced seizures. 185mg/kg of the plant significantly potentiate the anticonvulsant action of pharmaceuticals reducing the median effective dose of phenytoin from 13.5mg/kg to 9.25mg/kg and phenobarbital from 8mg/kg to 5mg/kg. On its own, *A. calamus* was mild to moderately effective at 250mg/kg and ineffective at 150mg/kg. However, sub-effective doses of phenytoin (10 mg/kg) and phenobarbital (2mg/kg) had significant effects even when administered with the ineffective 150mg/kg *A. calamus* dose. The authors concluded that *A. calamus* shows synergistic anticonvulsant effects reducing the dose of phenytoin and phenobarbital as well as the side effects. Based on a previous study in which a-asarone prevented Metrazol-induced convulsions and electroshock seizures (Dandiya, 1963) Yende et al. speculate a-asarone may also be responsible for the anticonvulsant and synergistic effects with pharmaceutical anticonvulsants in their study. Once again dosing in this study is rather excessive for practicality of human consumption.

More recently, Jayarman et al. (2010) studied a methanolic extract of *A. calamus* rhizomes (procured from local market in India) administered orally to mice at 100 or 200mg/kg one hour before injection of convulsion inducer PTZ. The extracts were accessed for constituents and were found to contain saponins, alkaloids, tannins, sugars, gums and mucilage. The extract increased the latency period and reduced seizure duration significantly in PTZ induced seizures in a dose dependent manner. Because PTZ is hypothesized to induce convulsions either through inhibiting the GABA pathway in the CNS or increasing central noradrenergic activity, the authors suggest the extract is working through involvement in these pathways either in a GABA-ergic or noradrenergic way (Jayarman 2010). While still on the higher side, 5.5g for a 55kg person is a somewhat more realistic dose (100mg/kg).

A 2012 (Bhat et al.) study looked at the efficacy of two preparations of calamus on induced seizures in rats as compared to control and the antiepileptic drug phenytoin sodium. Of the two groups given calamus, one was given calamus that had undergone the traditional shodhana (though not necessarily traditional for calamus) procedure of processing. During this process, calamus was decocted successively in "*Gomutra, Mundi kwatha* (decoction prepared from whole plant of *Sphaeranthus indicus* Linn.), *Panchapallava kwatha* (decoction prepared from a group of five leaves), and *Gandhodaka* (decoction prepared from a group of aromatic herbs)" followed by a 12 day drying period then ground into a powder. The unprocessed calamus was dried and powdered in the same fashion. The rats received

powdered calamus mixed with water orally an hour before electroshock at a dose of 11mg/kg of body. This dose was extrapolated from the human dose of 120md/day dose recommended by the Pharmacopoeia of India.

The study found that pretreatment of rats with both calamus preparations decreased duration of tonic extensor phase with greater protection against induced seizures in the shodhita (classically processed) group. Though not superior to phenytoin, which completely mitigated the tonic extension phase, the study was concluded to confirm the anticonvulsant activity of calamus, an effect enhanced by the shodhana process.

Neuroprotective: An investigation of a 1:1 ethanol water extract of *A. calamus* rhizome (obtained in India) was preformed in rats experiencing neurotoxicity from acrylamide. The *A. calamus* extract was dosed at 25mg/kg for ten days. In rats treated with the plants, hind limb paralysis was significantly reduced. Glutathione levels also increased significantly, but there was no effect on dopamine receptors. The explanation for this is not yet understood, however the increased glutathione levels may explain the overall neuroprotective effect observed. The authors concluded that “neurobehavioral changes produced by ACR (acrylamide) may be prevented following treatment with Acorus calamus rhizomes” (Shukla et al. 2002).

In another study (Shukla et al. 2006), the neuroprotective potential of a 1:1 ethanol water extract of *A. calamus* rhizome in rats with middle cerebral artery occlusion induced ischemia was examined. Treatment with the extract significantly improve neurobehavioral performance, decreased malonaldialdehyde levels in the cortex, increased reduced levels of glutathione and SOD activity, and improved neurologic function score.

In a 2013 review (Schroder et al.), calamus was one of several individual plant and traditional formulas investigated for ability to regenerate or protect neurons, specifically in treating chemotherapy-induced peripheral neuropathy. The review found three studies carried about by the same scientists in which rats were inflicted with neuropathy causing events (chronic constriction injury of sciatic nerve, tibial and sural nerve transection, and vincristine induced) and then treated with *Acorus calamus*, which attenuated nerve pain in all three trials (Muthurman & Sing, 2011a; Muthurman, Sing & Jaggi, 2011, Muthurman & Sing, 2011b).

Antidepressant Activity: In an animal model of depression, behavior and 5-HT receptor involvement were evaluated before and after the administration of *A. calamus* (from India) dosed orally at 18mg/kg over four weeks. Behavioral deficit was prevented, however the plant did not elicit significant change in 5-HT1A receptors. The authors concluded the plant has a clear antidepressant action, but because there was no significant change in 5-HT1A receptor sensitivity, an anxiolytic effect was not marked (Tripathi and Singh 2010).

Sedative/Relaxant Activity Activity: Both European and Indian *A. calamus* have been shown to potentiate sedative action of pentobarbitone (Dandiya 1959) and the steam volatile fractions have prolonged sleep time in mice treated with pentobarbital and hexobarbital with the highest sedation activity in the volatile fraction of the petroleum ether extract (Dandiya and Cullumbine 1959). Malhotra et al. (1962) speculate the potentiation effect may be mediated through serotonin and catecholamines. A direct sedative tranquilizing effect was found in rats, mice, cats and dogs (Dhall and Bhattacharya, 1968). Isolated b-asarone has been shown to sedate rats (Bose et al. 1960). Unfortunately, details and dosing on the studies was unavailable.

In a more recent study (Hazra and Guha, 2002), the effect of an ethanolic extract of *A. calamus* on rat brains was examined. In rats administered the extract at 200mg/kg and 300mg/kg for 14 days, electrographic recording showed an increase in activity and norepinephrine levels in the cerebral cortex but a decrease in the midbrain and cerebellum. Seretonin increased in the cerebral cortex but decreased in the midbrain. Dopamine increased in the caudate nuclea and midbrain but decreased in the cerebellum. The authors concluded *A. calamus* exerted a CNS depressive action by changing electrical activity and altering brain monamine level in various brain regions.

In 2011 Zuagg et al. further clarified the GABA-ergic properties of *A. calamus*. A petroleum ether extract of the rhizome (obtained in Switzerland) were screened on *Cenous* oocytes that transiently expresses GABA A receptors of the subunit combination a1, b2, and g 25. The extract was found to enhance GABA, the major inhibitory CNS neurotransmitter. Individual constituent activity was measured as well. b-Asarone, (+)-dioxasarcogaiacol (a sesquiterpene previously unidentified in *A. calamus*), (+)-shyobunone, and (+)-preisocalmendiol were the four most active principles in inducing GABA. Isoshyobunone and acorenone exhibited weak GABA modulating properties. All constituents enhanced GABA in a

concentration dependent manner. Because b-asarone induced highest potentiation of GABA the authors of the study speculate that the sedative and tranquilizing activities of the essential oil may be due to this constituent.

Acetylcholinesterase Inhibiton: In vitro tests seem to demonstrate acetylcholinesterase (AchE) inhibiton (Oh 2004, Houghton 2006, Mukherjee 2007). Hydroalcoholic extracts and essential oil of *A. calamus* rhizomes showed effectivity as a methanol extract (Oh et al 2004), hydroalcoholic extract, and essential oil fraction. Greater inhibition was seen using the essential oil than the hydroalcoholic extract (Houghton et al. 2006 as cited by Mukherjee). Of the constituents found in *A. calamus*, b-asarone is a more active inhibitor of AchE than a-asarone and is considered to be the constituent responsible for this activity (Mukherjee 2007).

Antimicrobial Activity

A. calamus was tested in vitro against methicillin-resistant Staphylococcus. Extractions of *A. calamus* using ethyl acetate, acetone, and methanol demonstrated antibacterial activity as well as synergistic antibiotic activity with antibiotic pharmaceuticals. Flavanoids and phenols were considered to be the major active constituents in the plants antimicrobial activity (Agil 2006). A study specifically of Korean *A. calamus* showed the essential oil, hexane extract, and isolated methyl isoeugenol (the primary constituent in the rhizomes used) had strong antimicrobial activity as per the following table:

Antimicrobial Activities of the Essential Oil, Hexane Extract, and Major Components from Rhizome of Korean A. calamus (copied from Kim et al. 2011)

	Gram-negative		Gram-positive			Yeast
Compounds	*Escherichia coli*	*Salmonella typhimurium*	*Staphylococcus aureus*	*Bacillus subtilis*	*Propionibacterium acne*	*Candida albicans*
Essential oil	+	+++	+++	+++	+++	+++
Hexane extract	+	++	++	+++	+++	++
Methyl isoeugenol	+	++	++	−	++	++
Cyclohexanone	+	+	−	−	+	+
Tetracycline	+++	+++	+++	+++	+++	−

Notes: Inhibitory zone diameter: +, < 10 mm; ++, 10–20 mm; +++, > 20 mm; and −, not detected.

Kim et al. note specifically the anti-microbial property of the extract and essential oil against the bacteria, Propionibacterieum acne, involved in acne vulgaris and suggest its use in topical acne vulgaris remedies (Kim et al. 2011). While this study attribute antibacterial activity to isoeugenol, this property has also been attributed to alpha and beta asarone (Ganjewala 2011).

Wound Healing

A 2014 study (Shi et. al) assessed *A. calamus* in vitro for its anti-inflammatory effects and in vivo for its ability to heal wounds. In vitro, they found that applied to RAW 264.7 cells, *A. calamus* inhibited mRNA expression of inflammatory mediators. They found aqueous extracts applied topically to animal wounds BID to TID "enhanced significantly the rate of skin wound-healing."

Respiratory System

Bronchodilatory: In an examination of the crude extract of *A. calamus applied* to guinea pig tracheal segments, a relaxing effect on high K+ contractions was observed. The n-hexane fraction was equally potent against both carbachol and K+ contractions while the ethylacetate fraction was more potent against carbachol precontractions and was only negligible in its dilatory effects against K+ contractions. The authors concluded that the crude extract of *A. calamus* exhibits a papaverine-like dual inhibition of calcium channels and phosphodiesterase in their n-hexane fraction demonstrating pharmacological basis for the use of the plant in airway disorders (Shah and Gilani, 2010).

Use in Diabetes

Increase of Insulin Excretion and Decrease in Carbohydrate Break Down: The ethyl acetate fraction of *A. calamus* was found to increase insulin secretion in HIT-T15 cells and inhibited a-glucosidase activity in vitro. (In type II Diabetes, a-glucosidase inhibitors are used pharmaceutically to prevent digestion of carbohydrates into sugars absorbable through the intestine thus reducing a potential spike in blood sugar). In vivo, doses of *A. calamus* (rhizome harvested in Indonesia) ethyl acetate fraction (200,400 and 800mg/kg) significantly decreased fasting serum glucose and reduced the increase of blood glucose

levels after loading with amylum (starch). The authors of the study concluded the ethyl acetate fraction of *A. calamus* may have hypoglycemic activity by promoting release of insulin while inhibiting a-glucosidase effectively improving postprandial hyperglycemia. The control of postprandial hyperglycemia is considered especially crucial in maintaining cardiovascular health in those with diabetes (Si et al. 2010).

Decrease in Serum Glucose and Increase in Adiponectin: An earlier study found that *A. calamus* ethyl acetate fraction in vitro increased glucose consumption mediated by insulin in L6 cells. In vivo, the fraction administered to mice at a dose of 100mg/kg reduced significantly serum glucose, triglyceride, and free fatty acid levels while increasing adiponectin levels. (Adiponectin is an insulin sensitizing hormone secreted by adipocytes. Low levels of adiponectin are often found prior to development of type II diabetes ([Linh et al., 2005]). Serum insulin was not significantly decreased. Whereas the insulin sensitizing diabetes drug, rosiglitazone increases body weight, *A. calamus* decreased overall intake of food and water and did not result in any weight gain. The authors concluded that due to its insulin sensitizing ability, *A. calamus* has potential uses in diabetes and associated cardiovascular complications (Wu, 2009).

Activation of PPAR Receptors: A third study analyzed a variety of plants in their relation to the peroxisome proliferator-activated receptors (PPAR) which are considered central in metazoan lipid and glucose homeostasis. In dislipidemia, fibrate drugs are used to synthetically activate PPAR-a (thus lowering cholesterol) while in diabetes glitazones are used to activate PPAR-g (thus increa
extracts studied, *Acorus calamus* (most likely sourced from Germany) was among the most active in terms of PPAR activation providing, as the study concludes, additional pharmacological support for traditional use of *A. calamus* in diabetes (Rau et al. 2006).

Clinical Trials:

Premedicant Prior to Anesthesia: Forty healthy patients were divided into two groups. Group I (control) received injected Glycopyrrolate (a pre-anesthetic that acts as an anticholinergic to reduce salivation, bronchial secretions and minimize bradycardia; it is also used to reduce GI secretions in ulcer treatment) and Phenergan (CNS depressant potentiator). Group II (Trial) were premedicated with Glycopyrrolate and 100mg of *A. calamus* (most likely sourced in India) orally ninety minutes prior to anesthesia. *A. calamus*

was found to raise body temperature and produce good sedation reducing anesthesia induction time and reducing the post operative recovery time from anesthesia.. No cardiovascular or respiratory depression was produced. The authors concluded that *A. calamus* was useful in ensuring quick and smooth anesthetic induction. They found it preferable to Phenergan for allaying apprehension and reducing complications during anesthesia (Pande and Mishra, 2009).

Anxiety Disorder: A clinical trail with thirty-three participants was undertaken to determine the effects of *A. calamus* in generalized anxiety disorder. *A. calamus* was given as a 70% hydro-ethnolic extract inside gelatin capsules at a dose of 500mg two times a day after meals. The regimen was undergone for sixty days with assessment at days 0, 30 and 60. *A. calamus* (most likely obtained from West Bengal area) was found to significantly attenuate anxiety related disorders, reduced stress phenomenon as well as correlated depression and improve significantly the willingness for adjustment. Patients did not report any adverse events nor were there any observed by physicians. Self-perceive stress improved by 23.3%, depression scale by 22.5%, and adjustment score by 22.8%. All were considered statistically significant. In summary the authors of the study considered *A. calamus* to reduces stress, attenuate anxiety, negate depression, and enhance adjustment, attention and ability to cope with mental overwork (Bhattacharyya et al. 2011).

MODERN THERAPEUTIC USE

The following section takes into account contemporary use and traditional modalities, pharmacological research, and the experience of modern herbal practitioners to summarize the use of *A. calamus* and *A. americanus* in current clinical applications. Note that chemical differences between species and ploidys does effect their medicinal application.

Note that Ayurvedic uses and Chinese uses are most likely referring to 3n, 4n, or 6n phenotypes (though there is a 2n phenotype in Asia). Traditional uses from indigenous peoples of North America may be referring to either species (*A. americanus* or *A. calamus* 3n). McDonald is most in depth about *A. americanus* specifically.

Energetics:

- warm, dry, astringent
- **Ayurveda** (Frawley and Lad, 1986): astringent/heating/pungent; decreases Vata and Kapha, increases Pitta

- **Chinese Medicine** (Benseky et al., 1994):
  - Enters heart, liver and stomach meridians
  - Transforms phlegm, facilitates removal and elimination of dampness
  - Opens the orifices
  - Strengthens the stomach
  - Fortifies the spleen

Taste: bitter, pungent, acrid, aromatic, astringent, resinous (Wood, 2009; Frawley and Lad, 1986; Bensky et al., 1994)

Taste Differentiations Among Phenotypes: Following from different chemistries, species and phenotypes vary in taste. McDonald (2012) describes the higher volatile oil strains (*A. calamus* 3n &4n) as heavy, oily and aromatic while he writes the native North American variety (*A. americanus*) is more well rounded in its action and has a taste profile that is more bitter, spicy and "zingy."

Summary of Actions (Skenderi, 2003 ;Tierra, 2008; McDonald ,2012; Frawley and Lad, 1986; Benskey et al., 2004):

- **Antispasmodic (particularly smooth muscle)**
- **Anticatarrhal**
- **Carminative**
- **Orexigenic**
- **Aromatic Bitter**
- **Anti-bacterial**
- **Insecticidal**
- **Fungicidal**
- **Emetic (in higher doses)**
- **Expectorant**
- **Nervine**
- **Opens Orifices**
- **Transforms phlegm and removes dampness**
- **Stimulant/Energizer/Relaxant**
- **Mental Rejuvinative**

Actions Separated Out According to Eclectic, Traditional, and Modern Herbalists:
Attributed actions vary depending on source. For the most part, everyone agrees calamus works on the digestive tract, use as a nervine and expectorant is less uniform.

- **PDR for Herbal Medicines 4th Ed (Thomson Healthcare, 2007):** stomachic, carminative, digestant, sedative, rubefacient, neurotherapeutic
- **Skenderi (2003):** aromatic, bitter tonic, demulcent, antispasmodic, carminative, astringent, mild neurocirculatory stimulant
- **Tierra (2008):** stimulant, carminative, expectorant, emetic
- **Smith (1999)**aromatic bitter, carminative, tonic to mm of membrane of mouth and throat
- **Ayurveda:** stimulant, rejuvenative, expectorant, decongestant, nervine, antispasmodic, emetic (Frawley and Lad, 1986); energizer, antispasmodic, nervine (Khalsa & Tierra, 2008)
- **Culbreth (1927):** Stimulant, carminative, tonic bitter, aromatic; dyspepsia, colic, flatulency, coughs

Action Differentials Among Species: McDonald (2012) writes Indian Acorus (*A. calamus*) was favored among the eclectics after a passage in King's American Dispensatory reading "Persian and East Indian calamus is said to be of better quality than that of other parts of the world". This species, McDonald writes, acts more strongly on the digestion containing higher volatile oil content. While the eclectics preferred *A. calamus*, McDonald prefers *A. americanus*, which he describes as "more balanced" in action. Depending on what activity or qualities one is looking for, they both have their place, though they are slightly different places.

Summary of Uses: (Wood, 2009; Smith,1999; Hoffman, 2003; Skenderi,2003; Frawley and Lad, Bensky et al., 2004; McDonald, 2012)

Gastrointestinal: atonic dyspepsia/unease in stomach/bowels, putrid flatulence, hypertonic digestion (Snow as cited by Bunce, 2011), deficient or excessive digestive secretions (Treben; Cabreara as cited in Tierra & Khalsa, 2008, p. 113), increases peristalsis, spastic gastrointestinal tract, loss of appetite, anorexia due to convalescence, anorexia due to stomach cancer, minor diarrhea, constipation, gastric ulcers, colicky pain, digestive discomfort with bacterial involvement, dysentery, bad breath

Gastrointestinal / Nervous System: IBS, reflux if also scattered, anorexia nervosa

Nervous System: failing memory due to old age/drug abuse, improves mental focus/sharpens memory, lack of comprehension, inability to grasp words, impaired consciousness, restore power of speech after stroke, comma, shock, insanity, depression, anxiety, neuralgia, headache, anxiety from tobaccos cessation, epilepsy, neuropathy

- Added to baths to reduce nerves and offer a "state of tranquility" (Tierra, 1998, p.108).
- Aid to quit smoking tobacco or marijuana (Tierra,1998; Treben)—Tierra cites its ability to help with lack of mental focus for its use as an antidote for marijuana; Tierra also writes that chewing calamus root and then smoking creates a "mild feeling of nausea" p.108

Respiratory: spastic/irritable dry coughs, spastic bronchitis, tracheitis, chest cold, common cold, asthma, chronic catarrh, sinus headache, toothache, inflammation of mouth and throat, throat cold, head cold, rhinitis with reduced thinking, catarrh in head, congested sinuses, laryngitis/horse voice worse from over use/sing in atmosphere with tobacco smoke, toothache

- McDonald (2012) writes calamus "excels in addressing throat colds, sore throats, irritable coughs, chest colds, and head colds," particularly he goes on to explain, when one has the feeling of head congestion, a completely stuffy nose along with a "hazy dullness." Also "strongly antimicrobial," McDonald chews Calamus when been exposed to contagions.
- Oil nasya: an "exceptionally decongestant" (McDonald, 2012) preparation for using calamus to clear up stuffy sinuses and a stuffy mind. (See preparation and dose for recipe.)

Musketal Skeletal: rheumatic pains, arthritis, cold achy joints

Endocrine: increased energy, allays hunger, relaxant despite stimulant qualities, potentially useful in diabetes

Reproductive: Henriette Kress (2012) writes of chewing on calamus root for endometriosis and of using it for menstrual cramps.

Cardiovascular/Circulatory:

- Treben uses warm calamus baths for chilblains by steeping roots overnight in water, bringing to a boil the next day, infusing for five minutes, then soaking the area affected in the preperation; also uses for cold hands and feet

Specific Indications:

Gastro-Intestinal

- Atonic dyspepsia-slow, sluggish, deficient digestion (Bunce, 2011; McDonald, 2012) especially associated "with tension, and perhaps infection or purification" (McDonald, 2012), flatulent digestions
- Hypertonic digestion (Snow as cited by Bunce, 2011))-clamped down, spasmodic, cramping digestion
- Digestive discomfort from dampness and turbidity causing epigastric pain, abdominal distention, poor appetite and a greasy tongue (Bensky et al., 2004)
- Dizziness and nausea, McDonald considers a "primary indication" (2012)
- GERD, especially if scattered and anxious (McDonald, 2012)
- First choice her for nausea associated with panic attacks (McDonald, 2012)

Nervous System

- Anxiety
  - Of anxiety and calamus, McDonald writes, "it is of this virtue of the plant that has really stood out to me, and set it wholly apart from any other remedy I might consider to ease anxiety" (2012). He describes its action as "unscattering energy."
  - "Invaluable" for anorexia nervosa because of bitter/anxiolytic combination (McDonald, 2012)
  - Nausea associated with panic attack (McDonald, 2012)
- Trauma: post trauma that was handled well in the moment but flashback result in nausea/dizzyness-Acorus brings back to reality (McDonald, 2012)

- Epilepsy
  - Tremors and loss of consciousness or seizures originating from phlegm-heat (Bensky et al., 1994)
- Mental Clarity
  - Satvic, considered one of the best herbs for the mind and congested thinking
  - Lack of mental focus—Ayurvedic traditional use, Tierra speculates coming out of its ability for “focusing the digestive power” (Tierra.1998; p.108)

Other:

- Laryngitis caused/aggravated by speaking, yelling and/or singing (McDonald)

Stimulating and Relaxing

Calamus is considered both simulating and relaxing. While this may vary some with phenotype and species, both qualities do come up across the board. McDonald (2012) explains this seeming contradiction well:

> “"Stimulant" and "relaxant" shouldn't be seen a contradictory uses occupying opposite ends of a polarity: stimulating herbs increase the expression of the vital force, while relaxants relax *resistance* to that expression. They work together in a complimentary fashion to achieve the same end: better flow of energy and vitality. Calamus isn't a sedative (it won't put you to sleep), but can be incredibly effective in treating anxiety. *Incredibly* effective”

Calamus and the Voice: Cross-culturally, calamus has a traditional association with the voice. Herbalist Joyce Wardwell explains, as quoted by McDonald (2012), the use of Calamus during the Inipi Ceremony. During hours of singing on end at a Pow-Wow, singers may chew on a bit of calamus root to help numb the vocal chords and continue singing. Wardwell reports that calamus “increase(s) saliva, increase(s) range…and center(s) ones energy.” In Ayurveda, calamus is called vacha, meaning “to speak” or “speech.” Khalsa writes this “refers to its action on the fifth chakra and its propensity to help you speak from your highest consciousness” explaining that it is considered “to stimulate the power of self-expression.” On a more literal level, Khalsa (2009) discusses the use of Vacha with autistic clients to promote self-expression connecting the brain and the mouth, promoting verbalization, communication, articulation, speech and self expression. Wood (2009) points out the rhizome looks very

much like the trachea.

Calamus and Autism: Khalsa (2009) discusses the use of calamus in autism as both a plant to promote self-expression and for its use in epilepsy, a condition often occurring in conjunction with autism.

Calamus as “Teacher Plant:”

This section affords a whole other write up about calamus but I would feel remiss in not including some mention of it. Those who have a relationship with calamus, and in many of the traditional uses, calamus is considered a sacred plant. Jim McDonald has a very deep relationship with this plant, considering calamus a “Teacher Plant.” He writes (2012):

> “ Calamus is best understood as a plant whose spirit teaches those who make relationship with it how to live in a good way upon the Earth; to live gently, lucidly, perceptively. She is subtle, and teaches a subtlety of perception, a subtlety of awareness”

SAFETY PROFILE

Concerns: There is a broad range of suggestions regarding the safety of calamus ranging from not fit for human consumption to safe. The concern is due mainly to one constituent, b-asarone. The concern stems mostly from numerous studies where rats were fed large quantities of b-asarone. A document published by the Belgian Scientific Committee on Food (2002) details the numerous experiments (many of which are unpublished) done. The document can be accessed hear in full for a thorough review of the studies:

Despite long standing traditional use even of the high b-asarone content containing phenotypes, based on these studies, it is generally recommended that these varieties only be used short term or that varieties and species with lower b-asarone content are used instead. The FDA prohibits use of calamus, calamus oil, or extract of oil in food (2013). Mills and Bone (2005) suggest use only of Acorus with low levels of b-asarone and that the isolated volatile oil not be ingested. In their more recent edition of their book (2012), Mills and Bone still list *A. calamus* in an appendix of “toxic or potentially toxic herbs” that should not be taken during pregnancy or lactation and that are “not recommended for internal use under any circumstances” (p.968). The Herbal PDR 4th ed, summarizes that with proper

dosage, there are no known health hazards or side effects but to avoid long term use. The AHP gives calamus a safety rating of 1, meaning “herbs that can safely be consumed appropriately” (1043). In an earlier publication, McGuffin writes, “the potential hazard of low doses of… b-asarone…is very minimal” (2007,134). However, he goes on to write that long term use of b-asarone containing herbs is not advisable. In accordance with the caution to avoid large dose (which would result in emesis) or long term use, calamus was traditionally used in some cultures only in small amounts and for shorter periods of time (Bensky et al., 1994; Thompson 1995).

Other herbalists (McDonald) do not feel the concern over the use of the rhizome (versus large isolated quantities of b-asarone) is warranted writing he does not “worry at all” about the use of the whole plant as potentially carcinogenic (2012). Indeed, in every experiment with negative effect cited in the Belgian Scientific Committee on Food Publication (2002), doses of b-asarone where far higher than suggested doses of the whole plant in terms of mg/kg of body weight.

Additionally, many of the studies deeming b-asarone problematic in that publication were done in the 1960s and 70s. More recent studies (Muthuraman and Singh, 2012; Shah et al. 2012) challenge the conclusions made by previous studies regarding the safety of Acorus. Of additional consideration, is the effect various preparations of *A. calamus* may have on the presence of b-asarone (see section in dose and prep below).

A. americanus and the 2n phenotype of *A. calamus* are considered safe as they do not contain b-asarone as long as they are positively identified (tricky to do both in commerce and in the field).

Pregnancy and Lactation:
Herbs containing b-asarone should be avoided in pregnancy and lactation. McDonald, though not concerned with the b-asarone content, still advises against using calamus during pregnancy feeling it is more “overtly medicinal plant” than is advisable to use during pregnancy.

Other Concerns:

- Emetic in high doses
- More suited to Vata and Kapha then Pitta

- Not in deficient yin (Bunce, 2011)
- Antagonizes ephedra (Bunce, 2011)
- “It can easily be misused by giving it too freely, or in irritable conditions of the stomach and bowels “ (Cook, 1869). Use gently if going to use in a condition like IBS.

Interactions:

- No clinical trials or case reports of drug interactions.
- Animal studies show potentiation of sleep time induced by pento-barbitone (Gardner, 2013).
- Moderate CYP450 interaction potential (Pandit et al, 2010)

DOSAGE AND PREPERATION

Notes on Preparation and b-asarone:

A 2009 study (Chen et. al) concluded decoction of the root, as is done in Chinese Medicine, reduces the b-asarone. (This preparation is most likely lacking in other essential oils attributed to the carminative, antispasmodic effect of Acorus).

Two studies out of India, one published in 2013 (Gholkare et al.) and in 2012 (Bhat et al.), discuss and evaluate the traditional process of *Shodhana or sodhana prakriya (Gholkare et al, 203)*, translated as “purificatory procedures” (Baht et al, 2012) or “detoxification process” (Ghokare, 2013) or just simply defined as “processing” (Ilanchezhian et al., 2006). Bhat et al. (2012) explains shodhana is designed “to overcome the undesired effects from various poisonous and nonpoisonous drugs.” Oddly, this particular study does not once mention b-asarone, explaining the reason for shodhana of calamus is not elucidated in any of the Ayurvedic texts the study refres to, but speculating the reason it is to mitigate the emetic effects of calamus.

The later study (Gholkare et al., 2013),however is specifically geared at assessing the effects of shodhana on b-asarone. While shodhana prakriya is a traditional way of processing certain plants, it not entirely clear in either of the studies, nor with further research, if shodhana prakriya for calamus specifically is a traditional preparation. It seems, that most likely, this is a modern use of a traditional process. Gholkare et al. explain that it

is due to the presence of b-asarone that calamus undergoes shodhana in the Ayurvedic system. According to Bhat (2012), the Ayurvedic pharmacopoeia of India as recommending sodhana be applied specifically to calamus. (Bhat, 2012).

Various versions of sodhana were carried out in both studies essentially involving three three hour session of boiling in various substances, including water in some of the trial, drying, washing, and then drying again. Sodhana also may involve steaming.

Gohlkar et. al. concluded that b-asarone was reduced in the three different shodhana process tried (one traditional, the other two modified), attributing the decrease of b-asarone to volatilization. Additionally, other substances used during the shodhana process may contribute to decrease in b-asarone content (Laddha et al., 2009).

While I did not come across any mention of calamus being prepared traditionally in this way, decoction in milk (Tierra and Khalsa, 2008; Frawley and Ladd, 1986) or water (Frawley and Ladd) does appear to be traditional. This would also serve to volatize some of the essential oils (though not as much as nine hours of boiling done in the shodhana processes).

Summary of Dosage and Preparation Recommendations:

Tincture Prep:

- British Herbal Pharmacopeia: 1:5 60% alcohol (3n variety)
- Galen's Way Herb Company: 1: 1.5 40-45% alcohol

Ayurvedic Tradition (Frawley and Ladd, 1986):

- 250-500mg of powder decocted in either milk or water or made into a paste
- Paste applied externally to forehead for headaches or on painful arthritis joints
- Nasal herb "perhaps the best" for administration in nasal congestion and nasal polyps

Chinese Tradition: (Bensky et al., 2004): 3-6g QD

Culbreth (1927): 1-4g dried rhizome

Smith (1999): 30-40 drops BID-QID

Tierra (2008):

- 3-9g in infusion
- tincture: 10-30 drops
- 1tsp dried root infused

McDonald (2012): Is a big advocate of simply chewing on the dried rhizome, 1-2 tbsp. While this is his preferred method, if one is to take an aqueous extract, he prefers a cold infusion steeped overnight. Maria Treben concurs writing "the calamus root tea is only prepared as a cold infusion" at a dose of 1tsp per ¼ litre of cold water. Treben suggest cold infusing overnight then lightly warming with a waterbath in the morning.

British Herbal Pharmacopoeia (1983):

- 1-3g dried herb (3n variety) or 1-3g in infusion TID
- 2-4ml of 1:5 tincture as a dose

Candied: Historically, the root was often candied.

Topical Preparations:

- Ayurveda-Paste applied exteranlly to forehead for headches or on painful arthritis joints (Frawley and Ladd, 1986):
- Ayurveda-Nasal herb "perhaps the best" for administration in nasal congestion and nasal polyps (Frawley and Ladd, 1986)
- Oil Nasya for Decongesting Sinuses and a "Foggy Mind" (McDonald):
    1. Infuse root in oil and water over a double boiler, heating until all water evaporates.
    2. Strain root.
    3. Laying down, with head tilted back to prevent dripping, apply a little oil to nostril and snuff into the sinuses.
- Calamus Bath: "About 200 gm. of Calamus roots are soaked in 5 litres of cold water overnight, brought to the boil the next day, allowed to infuse and added to the bath water" (Treben).
- Poultice (Bunce, 2011)

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Black Cohosh (*Cimicifuga racemosa*) as an Alternative to Hormone Replacement Therapy for Menopausal Symptom Control

Leilani Courtney

Menopause is a change that occurs during the fourth and fifth decades of a woman's life, when natural ovarian senescence occurs, resulting in the gradually change of hormonal levels of estrogens and androgens. Sometimes, this process is induced by surgical menopause or ovarian failure as a cause of cancer treatment (Al-Azzawi, 2009). Under all circumstances, the declining, and often widely fluctuating, levels of estrogen and androgens can produce a variety of symptoms in many tissues of the body. The signs of this change can include vasomotor symptoms, (hot flashes, night sweats), decreased bone density, changes in mood and energy (psychological problems, insomnia, poor concentration & cognition, depression), loss of pubic hair and changes in the genital tissues (vaginal atrophy, vaginal dryness, endometrium), and sexual desires and functions (Laakmann, 2012).

All women at some point will go through this change, and as there is an estimated 50 million women in the United States (80% showing symptoms) that have reached menopause to date ("Menopause," 2013). It is no wonder that treating these symptoms is a primary concern for health care practitioners and women alike. Hormone replacement

therapy (HRT) use synthetic hormones, that may or may not be identical to those made in the human body, but are believed to act similarly enough in order to relieve moderate to severe symptoms (Hendrix, 2007). HRT has also shown some beneficial effects on bone health and osteoporosis (Low Dog, 2003), although as more women undergo this treatment, many are finding that the risks are outweighing the benefits – showing a recent decline of 50% in hormone replacement therapy (Ross, 2012). Since this synthetic form of therapy was initiated before there was sufficient understanding of the molecular mechanisms of estrogen (and their similar looking synthetic replicas), several years of this on-going therapy has resulted in known side effects of bloating, breast tenderness, cramping, irritability, depression, breakthrough bleeding, or a return to monthly periods (Low Dog, 2003). More potentially serious side effects include increased risk of endometrial, ovarian and breast cancers (Rossouw, 2002) from hormone induced estrogen receptor-positive cell proliferation (Low Dog, 2003). The risks for these serious health concerns, in particular the cancers associated with HRT, are higher in women over the age of 55 years and for those that have used HRT for more than 5 years (Lieberman, 1998). What was once believed to be a healthy alternative, the Women's Health Initiative found enough associated risks with HRT that in 2002, the US Preventative Services Task Force voiced a published recommendation against the routine use of HRT for prevention of chronic conditions of menopause ("Menopausal hormone, et al," 2012).

For women that do not wish to experience the symptoms of menopause, but also do not wish to undergo HRT, there are natural and effective alternative therapies that have long traditional specifications for treatment of these symptoms. There is a general skepticism of the efficacy and safety of medicinal herbs that have not undergone clinical testing, and now with the ever-growing interest in natural medicine, this field is gaining the interest of many scientific evaluations. Although there are many herbs that aid in the menopause treatment, the rhizome of black cohosh (*Cimicifuga racemosa*) is the most widely studied (Lieberman, 1998).

Black cohosh has a long history of clinical use and has been used in Europe for almost 50 years to manage menopausal symptoms (Low Dog, 2003). This herb was previously described as a phytoestrogen, but with recent evidence, it is indicated that black cohosh may act more like estrogen in only a few parts of the body. The estrogenic effects of this plant are understood to reduce hot flashes via the brain, potentially help to prevent or treat osteoporosis in the bones, and possibly alleviate dryness and thinning of the vagina

(Seidlová-Wuttke, 2003). Interestingly though, unlike current estrogens in HRT, that act non selectively as an agonist in all tissues that contain estrogen receptors, it does not appear to act like estrogen in the breast or the uterus, and therefore reduces the possibilities for estrogenic cancer to form in those tissues (Bodiet, 2002). Another proposed mechanism of action for black cohosh is through serotonergic pathways. This was suggested after it was discovered that women on anti depressants experienced less hot flashes and night sweats, therefore it is possible that black cohosh works by also inhibiting the reuptake of serotonin (Oktem, 2007).

Although it is recognized that a large, long-term study using rigorous methodology is needed to fully understand the mechanisms of treatment and effects, after reviewing much of the data regarding safety and efficacy of black cohosh, it should be reassured to be a safe option for women who wish to take it for relief of menopausal symptoms. Even if not immediately as effective as HRT, there are considerably less side effects, and seen to be as effective over time as hormone replacement therapy (Low Dog, 2003).

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Ethnobotanical Uses of Oplopanax horridus

Suzyanna Mapachi

From the Cascades and north along the Pacific coast grows Oplopanax, an important medicinal plant that is still used by various indigenous groups of the region in which it grows. Oplopanax is an unmistakable spiny shrub that can grow up to ten feet tall in the right conditions. (2) I encountered Oplopanax growing in the temperate rainforests of the Olympic Peninsula, where its quite moist year round with a shady forest canopy. The leaves are maple-shaped and large to be able to catch the bits of sunshine that make its way through the canopy of sitka spruce, western hemlock and douglas fir. The most striking feature of this plant to me are the spines that cover the entirety of its stem. Its been used extensively as a physical medicine and as a spiritual medicine, and these two types of medicine often overlap in traditional coastal cultures. (3)

Oplopanax horridus's common name is devil's club – most likely due to its diabolical-seeming spiky stems. There are 13-15 known etymons for Oplopanax in over 25 different languages. (2) While there is very little pharmacological research, we can gain some insight into its medicinal uses from the traditional cultures that have a relationship with Oplopanax.

The first ethnographic record of the use of devil's club is from 1843, when the chief physician for the Russian American company, Eduardo Blaschke, reported the use of devils club ash as a treatment used by the Tlingit for sores. (1) Listed in the following paragraphs are specific uses by different tribes of people.

The Tlinget, of southern Alaska and coastal British Columbia, used seal fat to infuse the inner bark and this preparation was drunk as an emetic and cathartic. The inner bark was chewed and poulticed onto wounds to relieve pain as an emergency analgesic. The infusion of the bark and root were drunk for general strength, colds, chest pains, arthritis, ulcers, constipation and tuberculosis. It was mixed with pine pitch for skin wounds and abrasions. The dried inner bark was laid on cavities for pain relief. (3)

The Tanaina, of southern Alaska, made a decoction of the stem for fever, used the inner bark

decoction for tuberculosis, stomach upset, coughs, cold, swollen glands, boils and sore throats and other infections. (3)

The Haida, of northern British Columbia, used the decoction of the inner bark in a sea water solution that was drunk for 9 days for rheumatism and arthritis. The stems were also used like a whip as a counter-irritant for arthritis. The bark was chewed and the juice swallowed for colds. The berries were used on childrens heads to treat lice and dandruff. (3)

The Gitksan, of the Skeena river in British Columbia, used a decoction of Oplopanax stems as a purgative in treating gonorrhoea, and with Viburnam as a diuretic. The bark was also used mashed with Abies bark, Pinus gum and Lysichitum root and applied warm to boils, ulcers and rheumatism. (3)

Oplopanax has documented use as an appetite stimulant, for arthritis and rheumatism, as birth control, as a blood purifier, for broken bones, for cancer, during childbirth and menstruation, as an emetic and purgative, for fever, flu, gall stones, haemorrhaging and blood disorders, for heart disease, for internal and external infections, as a laxative, for lice and dandruff, for measles, as an analgesia, for pneumonia, various respiratory ailments, for coughs, colds, as a skin wash, for various sores, stomach trouble, as a tonic, for venereal disease and vision problems. The most widespread traditional uses has been for the treatment of external and internal infections, from cuts and scrapes to tuberculosis. The most common preparation is a decoction of the stem bark. (1)

There are many ethnographic accounts of spiritual applications of Oplopanax. It was used by many tribes as a protective plant. Various groups used the bark and stems as an amulet, bathed in the infusion for protection, and the ash was used in protective face paint for ceremonial dancers who were vulnerable to evil influences during rituals. (1) The Straits Salish and the Ditidaht combined devils club charcoal with bear grease for making a blue tattoo ink. Devils club was used in homes as a wash to purify the space after a sickness or death, and placed in the home to protect against bad influences. (1)

Devils club has also been used in everyday utilitarian life. The woody stems were carved into fish lures, since the wood is light weight. The Hesquit scraped the spines off the bark and boiled them down with Vaccinium and Lonicera berries to make a paint and basket dye. (4)

Acknowledging that ethnobotanical research is often a form of colonization is important to say in this paper. I believe that the only real primary sources on Oplopanax are the people indigenous to the region it grows, and have a long history of co-existence with this plant.

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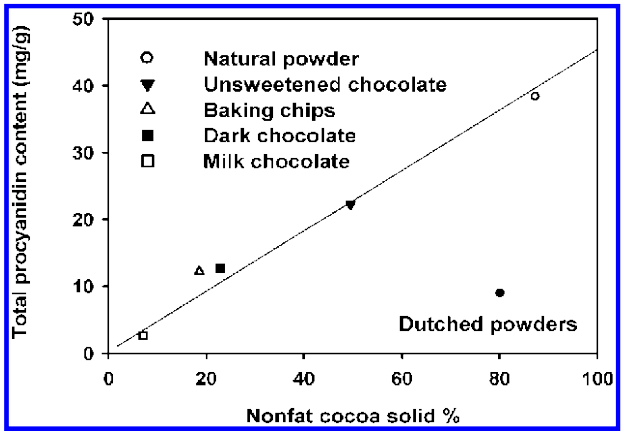
Chocolate and its Antioxidant Content

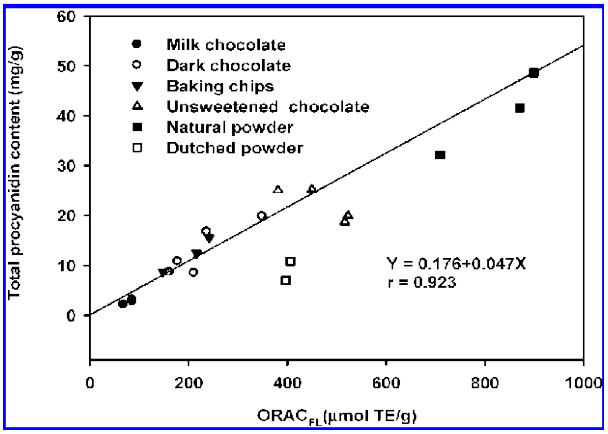
Rachel Davey

The health benefits gained from consuming chocolate come from its significant antioxidant content. Procyanidins, an antioxidant in cacao, is found in higher concentrations in cacao than cranberries and blueberries.(Gu 2002) When compared to other chocolate products it was determined that the amount of cocoa solids has a direct correlation to the amount of procyanidins and the therefor the amount of antioxidant capacity. (Gu 2006, Figure 3 & 5) The antioxidants found in cacao are responsible for suppressing low-density lipoproteins (LDL) oxidation and the formation of atherosclerosis (Kurosawa, 2005); inducing vasodilation and reducing hypertension in men (Fisher, 2003); increasing insulin sensitivity (Grassi, 2005); and inhibiting the growth of breast cancer cells (Ramljak, 2005).

In response to studies indicating that dark chocolate had a significant antioxidant content, (Miller, 2006) more and more "dark chocolate" products began showing up on shelves. A chocolate bar with a cocoa solid content of 40-100% is now referred to as "dark chocolate". Attempts to test the antioxidant content of cocoa or chocolate and its subsequent products have illuminated many variables (McShea, 2008) which make it difficult to assess: genetic variability, processing, product recipes and bioavailability of the antioxidant constituents.

High variability of polyphenol content in different cacao varieties; epicatechin concentrations among freshly harvested seeds of verified genetic origin ranged from 21.89 – 43.27mg/g. (Richelle,1999; Thomas-Berberan, 2007) Similar variation has been found between batches and growing regions. While cacao has remarkable health benefits it is safe to say that a





majority of chocolate consumers today do not eat, raw, unfermented, cacao as the Olmec/Mayan/Aztec people did nearly 1000 years ago. The processing of the cacao takes place to remove the bitterness and astringency lent by theobromine, caffeine, l-leucine, procyanidins, and catechin flavonoids.(Stark, 2006), therefore removing the bitters will inevitably involve the breakdown or removal of some of these constituents.

Processing on the simplest level involves, fermentation of the fresh seeds, drying & roasting and finally winnowing. Fermentation involves spontaneous inoculation of the seeds over the course of seven days, primarily from yeasts and later bacteria. Fermentation occurs unevenly within a batch allowing for much variety in the outcome. It is during this first step that some of the bitter antioxidants are destroyed. This may be because of the temperature reached during fermentation 50^{o}C (122^{o}F) or exposure to the by products of fermentation: lactic acid, ethanol and acetic acid. Under-fermented cacao seeds contain more antioxidant than fully fermented seeds; a technique utilized by producers interested in a "raw" product. The seeds are then dried until much of the moisture is removed. The seeds are washed off and then roasted. Roasting serves to loosen the husk from the fermented cotyledon as well as to bring out the favorable flavors of the chocolate. Temperatures at this point reach as high as 150^{o}C (302^{o}F). While catechins have been proven to be fairly stable at high temperature (Wang, R, 2006) epichatechins may require the stabilizing influence of other present molecules to remain intact.

Winnowing removes the woody husk of the cacao seed by shaking it violently; during this process the cotyledon is broken into 2-mm fragments called *nibs*. Sometimes the nibs are roasted again to further enhance the flavor. The nibs, which are comprised of 50% fat (cocoa butter) can be melted down into liquor or they can then be pressed to separate the cocoa butter from the cocoa solids, which are then ground into a fine cocoa powder. Cocoa powder contains approximately 87% cocoa solids and is where the antioxidants are found. (Gu 2006)

Processing of the chocolate until it reaches the stores follows a series of steps that involve secret "tricks of the trade" that are largely unknown to people outside the industry. Pressing and grinding further expose the cocoa to high temperatures. Conching is a multi-day heat treatment step, which applies gentle grinding to improve the flavor characteristics and reduce the concentration of free acids and other volatile by-products from the cacao bean. (McShea, 2008). "Dutching" is an alternative process to conching and utilizes a mild base to neutralize the free acids. Antioxidants are significantly reduced during the Dutching process, making this process undesirable for maintaining the antioxidant activity in chocolate products. (Miller, 2006)

The next step is typically to add, dairy fats, sweeteners and emulsifiers, whose purpose is to improve on the flavor or mask remaining bitterness. The addition of fats, sugars, emulsifiers etc. present their own risks as well as further dilute the overall presence of cocoa solids and therefore antioxidants in the resulting product. When does chocolate cease to be chocolate?

The U.S.D.A has established reference amounts for commonly eaten cocoa and chocolate products: natural/Dutched powders: 5g, baking chips and unsweetened chocolate: 15 g and milk/ dark chocolate: 40 g. A serving, based on the reference amount, of milk chocolate, dark chocolate, or unsweetened chocolate would provide on average 108, 517, and 312 mg of procyanidins; and 3200, 9100, and 6950 Trolox equivalents (TE) of antioxidant capacity (AOC), respectively. These amounts exceed those in most foods on a per-serving basis. The consumption of one serving of these chocolates would provide more procyanidins and antioxidant capacity than the average daily amount consumed in the United States (Gu 2004, Wu 2004).

The **bio-availability** and net benefit of the resulting antioxidants when combined with the added ingredients of the chocolate product being consumed needs to be properly weighed. Are the health risks of consuming sugar and fats offset by the presence of antioxidants? Current data on bioavailability suggests that the presence of other ingredients does not seem to prevent the uptake of epicatechin. (Keough, 2007; Roura, 2007)

In the end I believe that chocolate in its darkest and or rawest forms can truly be a benefit to someone's health as long as the goal is to ingest antioxidants and not dessert. You have to separate the chocolate from the bar.

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Hypericum and HRT Testosterone's Interactions with CYP450 System

Danielle Rissin- Rosenfeld

I decided to do my research paper on the ways in which *Hypericum perforatum* (St. John's wort) and Testosterone used in Hormone Replacement Therapy (HRT) are metabolized, in order to determine whether or not they are contraindicated.

There has not been thorough evaluation of risks of testosterone administration for Female to Male transgendered people, this includes a lack of studies on its effects and interactions with other drugs and herbs. [8,14] Therefore this paper is a comparison of the research I have found on both subjects and in particular attempts to evaluate whether Hypericum is contraindicated alongside Testosterone administration.

Hypericum has been popularly used for depression. [5,6,12] Because of its wide use for depression many studies have evaluated drug-herb interactions of Hypericum and pharmaceutical drugs. [12]. Paul Bergner talks about the metabolism of Hypericum in Phase I detoxification in the liver and the intestinal and kidney cells. [1] He says, "Many drugs are mainly metabolized by the CYP3A enzymes, as are many fat soluble hormones, including estrogens and cortisol and testosterone." [1]

A study based on the interactions between Hypericum and oral contraceptives found that "Hypericum is a potent inducer of the hepatic cytochrome P450 (CYP) 3A4 enzyme and the P-glycoprotein drug transporter, both of which can result in lower blood concentrations of drugs that are substrates for these pathways." [12] Another review also stated that Hypericum "has been implicated in lowering serum concentrations and the efficacy of several drugs, including oral contraceptives. Several reports have also documented breakthrough bleeding and unwanted pregnancies with concomitant use of Hypericum and oral contraceptives." [13]

Oral contraceptives and other steroid hormones including testosterone are metabolized by the Cytochrome P-450 enzyme system in the liver (specifically CYP3A.)[11,14] The enzyme that converts testosterone to a 5a- dihydrotestosterone (DHT) is mainly found in androgen responsive tissue: brain, pituitary, skin, bone and liver. After testosterone is metabolized in the liver 90% is excreted in the urine. [11] CYP2C19 was also found to oxidize testosterone enzymes and in human liver microsomes.[15]

HRT testosterone is often taken by Female to Male (F to M) transgendered people, as well as people of many different genders, to develop characteristics such as increased body hair, deepened voice, cessation of menses, clitoral growth, and increased muscle mass. [8, 13] Testosterone is administered by either injectable or transdermal preparations. Injectable formulations are most commonly used. [4,8,13].

The research of a transgendered male MD Nick Morton and Jamie Booth found that: "There are numerous drugs that increase or decrease the activity of this enzyme. This change in P-450 activity may cause increased or decreased levels of sex steroids as well as other drugs metabolized by this system. Cytochrome P-450 Inducers may cause decreased levels of testosterone."[1, 12]

In an ex vivo study based on Hypericum's induction of hepatic drug metabolism through activation of the pregnane X receptor, a 48 hour isolation study took place. The CYP3A4 enzymes were induced with crude ethanol extracts of St. John's wort, rifampicin (10 μM), or hyperforin (1 μM) and were added to the culture medium as 1,000× stocks in either ethanol or dimethyl sulfoxide. This resulted in the binding of PXR which switched the enzymes. [9] The study concluded that Hypericum activates the orphan nuclear receptor PXR and consequently induced the expression of CYP3A4, meaning that Hypericum is likely to interact with the many drugs that are metabolized by CYP3A4. [9]

CYP3A4 is shown to be one of the major P450 forms involved in catalyzing Testosterone.[15] The most common form of interaction is when a foreign chemical (i.e. Hypericum) acts as an inhibitor or inducer for the CYP enzyme metabolizing a drug. This can result in slow or fast clearance of the drug and therefore reduce its effects.[13] Paul Bergner says that, “Depending on whether the drugs are metabolized to their active form or inactive forms by the enzymes, simultaneous consumption of Hypericum extracts may either increase or decrease blood levels. Consequences could range from innocuous to fatal depending on the nature of the drug and how critical the drug dose is to the patient's health.” [1]

In conclusion Hypericum has been found to be contraindicated with many pharmaceutical drugs, including HRT Testosterone.[1] Both Hypericum and HRT Testosterone are processed through the CYP450 pathway and specifically by CYP3A4. [9,11, 13, 15]. Since Hypericum has been shown to be an inducer of CYP3A4, it may decrease the desired effect of HRT Testosterone. [9,13,15] It may also result in other side effects [1], but there has been little research to determine what results may occur.[8,14] Based upon my research I believe people receiving HRT Testosterone should be cautious or avoid taking Hypericum concurrently. Taking HRT testosterone and Hypericum at the same time may decrease the desired effects of testosterone, by speeding up its metabolism through Hypericum’s inducing effects on the CYP450 pathway. Unfortunately in reviewing the literature no case reports were found.

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Treating Vaginitis with Calendula

Emily Peters

Vaginitis or vulvovaginitis includes various inflammatory conditions in the vagina, usually accompanied by an infection, itching, irritation, and abnormal vaginal discharge. Almost all vaginitis symptoms are caused by bacterial vaginosis, vulvovaginal candidiasis (VVC or a yeast infection), and trichomoniasis. This report will focus on treatment for VVC or undiagnosed vulvovaginitis. VVC is usually caused by an overgrowth of *Candida albican*s, but can also be caused by *C. tropicalis* and *C. glabrata.* Many women self diagnose and treat themselves for vulvovaginal candidiasis without having a microscopically confirmed diagnosis. VVC is especially difficult to diagnose because 50% of all women have Candida organisms as a part of their normal vaginal flora. Specific symptoms of VVC include mild to severe vaginal itching and irritation, possibly with a characteristic white curd-like vaginal discharge with a mild yeast-like odor. The vulva can be inflamed, swollen, red and raw (Romm 256-259). The Merck Manual calls VVC Candidal vaginitis. Specific symptoms they include are vulvar burning or irritation, pruritus, dyspareunia (pain during vaginal penetration), dysuria (painful or uncomfortable urination), and a white cottage cheese-like discharge that adheres to the vaginal walls. Symptoms may increase the week before menses. A healthy vaginal flora is kept in balance by *Lactobacillus spp*, preventing overgrowth of pathogenic bacteria (Merck). Eclectic herbal physicians Harvey Wickes Felter and John Uri Lloyd did not use distinct terms for different types of infection; they used the term leucorrhoea or vaginitis for any white discharges of the vagina (Felter, Felter and Lloyd).

In treating these infections, our goal is to both find the root cause and to relieve the symptoms. Possible causes include: fungi or bacterial pathogen overgrowth, intestinal microorganism imbalance, pregnancy, change in pH, hygiene products, allergies, antibiotic use, antifungal treatments, oral contraceptives, hormonal replacement therapy, hormonal imbalances, spermicide, lubricants, latex, frequent intercourse, multiple sexual partners, sexual transmission, stress, diabetes, hyperglycemia, and immunosuppression such as HIV/AIDS (Romm 258). Aviva Romm suggests that one common cause of chronic vaginitis could be intestinal dysbiosis and permeability (leaky gut syndrome)(265). It is important for

a person who has symptoms of VVC to explore their own risk factors and seek assistance in treating the possible root cause if the symptoms persist for a long time or are recurring.

Using Calendula

Eclectic physicians Felter and Lloyd pronounce that “in vaginitis, endometritis, all uterine and vaginal abrasions, and non-malignant ulcerations, leucorrhoea, and as an intra-uterine wash, calendula has received strong endorsement.” (Felter and Lloyd). It is a non-irritating, non-poisonous vulnerary that promotes the healing of wounds and stimulates healthy tissue regeneration (Felter, Romm 263). A wash of calendula flowers was used for vaginitis, cervicitis, endometritis, vaginal abrasions, gonorrhea and leucorrhea. It has also been effective to reduce discharges (Felter). It is recommended to be used internally to aid the local action (Felter and Lloyd).

The essential oil of calendula flowers have shown anti-bacterial and anti-fungal activity in-vitro specifically against *Candida albicans* (Jannsen). Matthew Wood connects the immune tonic properties of calendula to its lymphatic action, as the lymphatics are a location of much immune activity. Wood also states that “calendula is used for conditions where there is dampness in the wound or in the tissues: thrush, swollen lymphatics, and vaginal discharge” (Wood 1997, 181-184). He explains an antiseptic action of calendula as due to stimulating the body, and sending impurities to the surface. He indicated it as an immune tonic, for use on all wounds, abrasions, and infections. He describes calendula as being suited for people with a history of candida and depression, as well as specifically for leucorrhea and a dry, irritated vaginal wall. calendula's bright yellow flowers are indicated for “places where the sun doesn't shine” including the groin (Wood 2008, 154-158). Calendula as a vulnerary is soothing to digestive mucosa and other mucous membranes (Wood 2004, 243-244). David Winston describes calendula as an antifungal, antibacterial, styptic for conditions including bacterial vaginosis (Winston 68).

Aviva Romm indicates calendula as an important ingredient in topical vulvovaginitis preparations, as an antimicrobial vulnerary that promotes tissue regeneration (263). Topical treatments she recommends for vulvovaginitis are a sitz bath or a peri-rinse. A sitz bath involves making a strong tea diluted with water, putting it in a basin and sitting in it for 5-20 minutes. To make a peri-rinse you take the same tea and wash the area as needed, using a squeeze water bottle if preferred. The tea is anti-microbial and anti-inflammatory. A recommended combination is: calendula, thyme, lavender and uva-ursi in equal parts. As an anti-microbial, calendula can directly reduce infections. As a vulnerary, calendula heals and

strengthens the sensitive epithelial tissues of the vagina and vulva, and tones the mucous membrane in the gastrointestinal tract (263-264). If one cause of VVC is intestinal permeability as Romm suggested, an important aspect of recovery is to improve the integrity of the intestinal mucosa and restore healthy gut flora. The former can be reduced by taking herbs internally similar as we use externally: vulnerary, anti-microbial, anti-inflammatory herbs including calendula. The balancing of intestinal flora can be helped with probiotic foods or supplements (265). The Eclectics also point out the importance of using calendula internally to aid the local action (Felter, Felter and Lloyd). Although they don't specify the mechanism of action internally, we can expect that the effectiveness is due to its actions as an anti-microbial, vulnerary and anti-inflammatory, and that these actions are exerted in the GI tract.

Calendula's long history of use for different kinds of vaginitis should encourage us to try it. I began writing this article while having persistent low-grade symptoms of a yeast infection for 2 weeks. I had a diagnosis of VVC in the past, though not recently. I decided to try using calendula in the ways suggested through the texts. I made a tea with calendula, lavender and oregano, and used it as an external wash 3 x/day, with a tincture of calendula taken internally 3-5x/day. My symptoms cleared completely within 2 days, I continued the treatment for one more day, and I haven't had any recurring symptoms since.

Many women suffer from vaginitis and either don't go to a clinic for a diagnosis, or are not properly diagnosed because of difficulties in establishing the cause (Romm 256-259). Calendula is a safe herb to use internally and externally with no known toxicity or contraindications except for people with known allergies to members of the Asteraceae (Hoffman 534-535). Its wide range of therapeutic actions and safety suggests that calendula is an excellent remedy for vulvovaginal candidiasis and for undiagnosed symptoms of vaginitis. It helps bring symptomatic relief, heal the tissues, and potentially provide support in treating underlying causes especially if consumed internally as Felter and Lloyd suggest.

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The effect of Gossypium sp. on the Uterus

Allison Dellner

Gossypium species have an ethnobotanical history of use as an abortificatient, emmenagouge, and parturient[1]. A decoction of the fresh inner root bark of Gossypium was used by African American slaves in the cotton districts of the southern United States particularly to abort early pregnancies[2]. Eclectic doctors corroborate this use; saying Gossypium herbaceum was known to "procure abortion without injury to general health", noting its relative non-toxicity in the context of abortifacient herbs, but had little confidence in the efficacy of Gossypium to act on the uterus[3]. This paper attempts to find more a specific action for Gossypium and to substantiate or disprove its claims as an abortifacient.

William Cook says that although it is claimed to be abortifacient, he did not think it exerted any powerful influence on the uterus, but that its action was "rather good". Cook thought of Gossypium as a "feeble medicine" to be used as a relaxant, a mild uterine tonic when the nervous system is irritable in labor, and to slowly promote menstruation in "nervous persons". In over ten years experience, Cook "did not get a strong article in experiments".[4]

Harvey Felter blamed the freshness of the bark, saying that the "old root is valuless as medicine", noting that the fresh root was not available to many. In his materia medica, Felter echoed Cooks experience, using Gossypium root as an emmenagouge in late menstruation, and claiming it was not as effective in improving uterine inertia during labor as it was touted. Felter also noted that Gossypium root was "non-toxic"[5].

In present day, Gossypium species are still used by women and midwives as an abortifacient and emmenagouge[6]. The language used to describe it's action has changed to describe the synergystic effect it has with oxytocin. "Cotton root" does not contain or mimic

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[2] Wood, George B. "A Treatise On Therapeutics, And Pharmacology Or Materia Medica Vol2". (1867)
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oxytocin, but only will potentiate the effects of oxytocin already present in the body[7]. Michael Moore has some opinions on this plant, having used the species of cotton native to Mexico and parts of the southwest, Gossypium thurberi. Moore uses this plant as a "reliable oxytocin synergist" that increases the tone and contractability of the uterus, and also of the seminal vessicles, prostate and the myoendothelial tissues of the breast. He points to the interest in research that Gossypium periodically receives, and then claims that researchers find nothing "patentable" in the plant and then moves on[8].

It is interesting to me that the modern uses Moore describes for Gossypium are related to male reproductive anatomy because there are many studies concerned with an isolated constituent called gossypol, which causes a decrease in sperm density and motility and eventually sterility in men[9]. The other body of research on Gossypium is done on cows and lambs. One cow study focused on the effects that eating cottonseed products had on the reproductive quality and ability of ruminant animals. The outcome was largely that the amount of gossypol is dangerously high in cotton seed and negatively affects the ovulation, pregnancies and fertility of the cows[10].

Another interesting animal study was done on a patent that was being created for a blend of herbs to help with encouragement of the "let down" reflex of milch animals such as sheep and cows. The animals were being treated with synthetic oxytocin to ease in the let down. The trial blends contained varying amounts of Asparagus Racemosa, Gossypium arboreum, Foeniculum vulgare, Lepidium sativum, Cholophytum boivilianum, Ipomoea digitata, Withania somnerifa, and Leptadenia reticulata. The outcome of using this formula was that the animals had an easier time in "letting down" their milk, and also the quantity of milk that they produced was greater, even up to 3 days after administration of herbs. The use of the herbal formula also had none of the same side effects that farmers were accustomed to noticing from the oxytocin shots. A "synergystic effect" was reported to occur when these herbs were used in combination with each other. [11] Although this study was done on ruminants, I find it interesting that Gossypium is used in this formula as a substitute for oxytocin. It somewhat substantiates the claim that Gossypium is an oxytocin potentiator. The body of research I did find on modern Gossypium actions and constituents

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merely underscored the fact that it is under-researched in the female population and there is much more we could know about its use as an emmenagouge[12]. It was my personal experience with a deficit of information on Gossypium that led me to ask what it actually does and what its mechanisms were.

All of the reading have done now on Gossypium informs my own personal experience with using the root bark tincture. Knowing the actions of this herb and learning about the reputation it had to be safe with little side effects, I tried using Gossypium as an abortifacient in the second and third weeks of pregnancy. After doing this paper, I decided my dose of 10 drops every 2 hours was too low, as this is a very high dose plant. Now I also can understand why my combination was unsuccessful. There was probably not sufficient levels of oxytocin in my body to work together with the herbs to potentiate an abortion.

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Fats in the Prenatal Diet: Healthy, Hazardous, or Just Plain Confusing?

Kate Westdijk

My mother recently disclosed that, when she was pregnant with me, she would try to be "good" and make it through her full time work day on a diet of carrot sticks, raisins, and saltine crackers. Then, at the end of her work day- and her guilt as she admitted this was palpable- she would scramble to the nearest convenience store, buy a pint of Ben & Jerry's vanilla ice cream, pull off the top, and lick it as she drove home. Clearly, she was experiencing some confusion about what she was "supposed to" eat, and what her body was craving.

Dietary fat plays a key role in the development of healthy tissue and function during pregnancy and in the growing fetus (Larque, E., 2012; Jordan, R. G., 2010; Innis, S. M., 1993). Recent reviews of the scientific literature do not substantiate historical claims that a diet high in fat will lead to cardiovascular disease and obesity (Ajala, O. et al., 2013; Hite, A. H., 2011; Siri-Tarino, P. W., et al., 2010). Despite these facts, concerns about dietary fat intake are held by, and conveyed to, pregnant women (BabyCenter, 2013; USDA, 2013; Kleinman, R. E., 2009; Root, R., & Browner, C., 2001), and common prenatal nutrition resources are quick to warn of the danger of excessive weight gain in pregnancy (Murkoff, H. & Mazel, S., 2008; Brewer, G. S., & Brewer, T. H., 1983). However, sufficient fat intake is essential for a safe pregnancy and delivery (Jordan, R. G., 2010; Odent, M., 2006; Brewer, G. S., & Brewer, T. H., 1983), as well as healthy fetal development (Larque, E., 2012; Kleinman, R. E., 2009).

There is general agreement in the medical community that the "good fats" - such as the omega-3 alpha linolenic acid- are important as part of a prenatal diet (BabyCenter, 2013; USDA, 2013; Kleinman, R. E., 2009). Other less mainstream nutrition sources also include saturated fatty acids as an important component of the human diet (Gedgaudas, N., 2011; Enig, M. G., & Fallon, S., 2006; Schmid, R. F., 1997). However, discriminating between "good fats" and "bad fats" is a confusing challenge for the average citizen (Taubes, G., 2008), and approximately three thousand women per year in the United States (5%)

receive inadequate to no prenatal care in order to receive information on healthy dietary choices (Child Trends Data Bank, 2012; US Census Bureau, 2012). Prenatal nutrition advice impacts women's dietary behavior while pregnant (Sweeney, C. et al., 1985), but women receiving this advice have reported that is often contradictory or vague (Root, R., & Browner, C., 2001). The Pediatric Nutrition handbook even goes so far as to suggest that any fat besides essential fatty acids could be eliminated from the diet and replaced by carbohydrates (Kleinman, R.E., 2009), but emerging evidence suggest that this would not be wise because of the connections between high carbohydrate diets and obesity (Hite, A. H., 2011). If a pregnant woman is able to access adequate prenatal care in the first place, and then persevere through conflicting societal and medical concerns about fats and links to obesity (not to mention their own body messages and cravings), the recommended sources and quality of the fats they consume may not be sufficient for delivering the types of fatty acids necessary for a healthy pregnancy (BabyCenter, 2013; Kleinman, R. E., 2009; Brewer, G. S., & Brewer, T. H., 1983).

Which Fats are Important for a Healthy Pregnancy?

Polyunsaturated Fatty Acids

There are two polyunsaturated fatty acids (PUFAs) recognized as essential dietary nutrients: linoleic acid (LA) and alpha linolenic acid (ALA). They are called "essential fatty acids" because they must be derived from the diet (Kleinman, R. E., 2009). Linoleic (an omega 6 fatty acid) and *α*-linolenic acid (omega 3) act as precursors for the synthesis of longer chained PUFAs (Simonpoulis, et al., 2000). Modern agriculture, due to its emphasis on production and not nutrition, has reduced the omega 3 fatty acid content in many foods (Simopoulos, A., 2002). Excess omega 6 and deficient omega 3 leads to an imbalanced immune response from inflammation as well as increased risk of cardiovascular disease (Fontani, G. et al., 2005) and poor bone growth and metabolism (Watkins, B. A. & Seifert, M. F., 1996).

Long Chain Polyunsaturated Fatty Acids (LCPUFAs) make up the majority of human brain tissue and the retina (Innis, S. M., 1993). The long chain fatty acids DHA and EPA, from fish oil, have been correlated with key pregnancy outcomes for both the mother and the developing fetus. Benefits to both may include reduced risk of eclampsia (Odent, M., 2006; Velzig-Aarts, F. V. et al., 1999; Imhoff-Kunsch, B., et al., 1991) and increased gestation length (Jordan, R. G., 2010). LCPUFAs consumed in pregnancy have been linked to improved cognitive development and reduced allergies postpartum (Larque, E., 2012).

DHA supplementation during pregnancy decreased the duration of infant illnesses and the occurrence of colds in children (Imhoff-Kunsch, B. et al., 2011).

The commonly recommended sources of PUFAs often include vegetable oils, fortified eggs, or dairy products (due to concerns about mercury contamination of marine sources) (BabyCenter, 2013; USDA, 2013; Kleinman, R. E., 2009), but these sources are likely to supply oxidized or rancid oils which do not impart the sought health benefits (Enig, M. G., & Fallon, S., 2006; Gallobart, J. et al., 2001). Instead, they are correlated with a variety of other problems such as cardiovascular disease or bone weakness (Gedgaudas, N., 2011; Ravnskov & McCully, 2009; Gallobart, J. et al., 2001; Parhami, F. et al., 1997). Whole food sources of PUFAs, such as sardines or avocados, typically contain natural antioxidants that protect fragile fatty acids in both the food and the tissue of the consumer. Sardines are also a low-mercury fish that would be an excellent choice for pregnant women (Abehlson, A. et al., 2011).

Saturated Fatty Acids

Saturated fat in the human diet is controversial and conventional nutrition sources generally associate it with increased risk for obesity and cardiovascular disease (Kleinman, R. E., 2009; Lees, R.S., 1990). However, a recent review of 21 studies, published in the American Journal of Clinical Nutrition, determined that there is no scientific basis for the widely held (and practiced) claim that saturated fats contribute to cardiovascular disease (Siri-Tarino, P. W., et al., 2010). Another recent review of high fat, low carbohydrate diets indicate that healthy levels of fat in the diet can actually prevent excessive weight gain by contributing to feelings of satiety and reducing cravings for sweets (Hite, A. H., 2011). There is simply insufficient evidence to justify restriction of saturated fats from our diets (Ajala, O., 2013). Conversely, proponents of traditional diets recognize that saturated fats from natural sources (as opposed to hydrogenated in a lab) are safe and even necessary in the human diet, especially in pregnancy, for several reasons (Gedgaudas, N., 2011; Enig, M. G., & Fallon, S., 2006). Saturated fats are important for the growth and maintenance of healthy bones (Watkins, B. A. & Seifert, M. F., 1996). They are the only stable fats to cook with because they are inherently resistant to oxidation, and, when consumed with essential fatty acids for example, they protect these fragile lipids from oxidation (also known as rancidity) so they can be used for healthy processes in the body (Enig, M. G., & Fallon, S., 2006; Feher, J. et al., 2007). Saturated fats are required for absorption and use of essential fatty acids (Garg et al, 1988) as well as essential vitamins such as A, D, and E (Romm, A. J.,

2011). In sum, saturated fats provide many benefits, and there is insufficient scientific evidence to suggest that they be restricted from our diets.

Ironically, my mother still lovingly blames me, not the ice cream, for the weight gain she experienced in her pregnancy. Perhaps if she had been encouraged to nourish her body with high quality fats and proteins throughout her day, her experience may have been less frustrating and confusing.

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Herbal Therapeutics Post-Induced Abortion

Lisa Weiss

Approximately one in four recognized pregnancies in the United States ends in an induced abortion (Rock and Jones 2008). According to the American Congress of Obstetricians and Gynecologists, more than 1.2 million abortions occur each year and one third of all women will have had either a medical or a surgical abortion by the age of 45 (ACOG 2010). Surgical abortions are the most common type performed in the U.S (ACOG 2011) –only 6% of all induced abortions are medical (Finer and Henshaw 2000). There are risks associated with both medical and surgical abortion, and women concerned about these risks or experiencing side effects from either procedure may consult an herbalist. This paper only considers the therapeutics post-*legally* performed abortion; those performed by an untrained practitioner, common in countries where abortions are illegal, come with additional risks (Faundes 2010).

During a typical medical abortion, two pharmaceuticals are given: Mifepristone and Misoprostol. Mifespristone binds to progesterone receptors but does not activate them, acting as an anti-progesterone (ACOG 2005). Without progesterone, the lining of the uterus thins, preventing the embryo from remaining implanted, and the cervix softens (ACOG 2011). Misoprostol, a prostaglandin E analogue, is administered either orally or vaginally 48 hours after administration of Mifespristone (ACOG 2005). Misoprostol causes the uterus to contract and expel the embryo (ACOG 2011). There is typically a follow up 14 days post-administration to confirm expulsion. After a medical abortion, bleeding is much heaver than from menses and cramping may be severe, though some degree of bleeding and uterine cramping is necessary for the abortion to complete (ACOG 2005). It is advised to seek emergency care if two full-sized sanitary napkins per hour for two hours in a row are saturated with blood (Creinin and Aubeny 1999). However, less than 1% of women will need emergent curettage because of excessive blood loss (Schaff et al. 1999). Over the counter pain medications are recommended after medical abortion for pain from uterine cramping (ACOG 2005). Five deaths have been reported associated with medical abortion and in three of five cases, the cause was infection with *Clostridium sordellii,* an anaerobic, gram-positive bacteria (CDCP 2005).

The most common type of surgical abortion is suction/vacuum curretage. Before the

procedure, local anesthesia is applied to the cervix and general anesthesia or sedatives may or may not be given. A speculum is inserted to hold the vagina open and a dilator is then inserted into the cervix to stretch the opening. After the cervix is dilated, a small tube is inserted into the uterus. The tube is attached to a vacuum pump, which removes the pregnancy. Antibiotics are given post-operatively to prevent infection. Soreness or cramping may occur for up to two weeks after the procedure. Bleeding may last up to six weeks, but is rarely heavy enough to require a blood transfusion (ACOG 2011). In cases of hemorrhage, most often due to uterine atony, uterotonic agents, such as Methergrine (Planned Parenthood 2005), are given along with manual compression of the uterus (Rock and Jones 2008).

Practitioners working with clients post-abortion should be aware of the symptoms that indicate necessary emergency care. Acute hematometra (post-abortal-syndrome) occurs in 1.1 per 100 abortions and the cause is unknown. Symptoms include a large, tender uterus; severe cramping; and less blood then expected within two hours after the procedure. Women with these symptoms should immediately return to their doctor or to the emergency room. Treatment is a repeat of the curretage followed by uterotonic agents. Retained tissue is also possible, occurring in 1 in 100 surgical abortions. Symptoms include fever, cramping, and excessive bleeding. Antibiotics and a second curretage may be needed—clients with these symptoms should be referred back to their doctor (Rock and Jones 2008). Gestational age is one of the key factors determining the likelihood of serious complications post-abortion; complications increase progressively with advancing gestational age (Rock and Jones 2008).

Lahteenmaki and Luukkainen (1978) studied the first menstrual cycle after an induced abortion in 18 women. They found that the plasma concentration of estradiol and progesterone declined rapidly, and was followed, in most women, by an increase in plasma estradiol levels from the seventh post-abortal day onward. The authors also report that progesterone was lower than normal during the luteal phase of the cycle, and that ovulation returned during the first cycle in 14 of the 18 women.

Feelings of relief, sadness, elation, or depression are common post-abortion and may be strong because of hormonal changes (Planned Parenthood 2005). In a mega-analysis of 22 peer reviewed studies, Coleman (2011) reports that women who have had an abortion experience 81% higher risk of mental health problems compared to women who have not, including alcohol misuse, anxiety, depression, and suicide. Nearly 10% of the 81% was shown to be attributable to abortion. Suliman et al. (2007) report that of 133 women choosing to have a surgical abortion, 18.2% had symptoms of PTSD at three months post-

procedure. Pre-procedural cortisol levels were correlated with these PTSD symptoms, indicating that women experiencing a stress response to the procedure were more likely to experience PTSD later. Likewise, Fergusson et al. (2009) demonstrate that the risks of developing mental health problems are dependent on the extent of distress or guilt reported by a woman pre-procedure.

Other than a handful of trials on Chinese herbal formulas for post-abortion hemorrhage, no pharmacological research or clinical trials have been conducted specifically on herbal therapeutics post-abortion. Below is a collection of plants used by both modern and historical herbalists/doctors for post-abortion recovery and some extrapolation from research conducted on related or more general subjects.

For a woman that connects her anxiety, depression, and/or PTSD to an abortion, nervines, anti-depressants, and adaptogens may be indicated, most likely as adjuvants to counseling with a mental health professional. Some of the following plants could also benefit the male partner, who may also be affected by the abortion. Because similar hormonal changes occur after pregnancy as after abortion (low estrogen and progesterone levels (Lahteenmaki and Luukkainen 1978)), herbs typically used in postpartum depression such as *Hypericum perforatum, Artemesia vulgaris* (Hoffmann 2003)*, Rosmarinus officinalis* (Wood 2004), and *Melissa officinalis* (Weed 1986) may be useful. Not only have multiple clinical trials demonstrated the use of *H. perforatum* as an anti-depressant, it also has antimicrobial, nervine, and astringent/vulnerary properties (Mills and Bone 2000). *Artemisia vulgaris,* which Matthew Wood recommends for women who have been through abortions or other "harshness" in the uterus, is a mild nervine and anti-depressant (Wood 2009). The antispasmodic (Khan 2009) and antibacterial (Chen 1989) properties of *A. vulgaris* also make it useful post-abortion. *R. officinalis*, a nervine and antispasmodic (Hoffmann 2003), was demonstrated by Machado et al. (2009) to have an anti-depressant effect in mice through an interaction with the monoaminergic system. *M. officinalis* has mild antidepressant properties, is useful for spasm and anxiety, and is possibly trophorestorative (Hoffmann 2003). In a double-blind placebo-controlled study conducted by Kennedy et al. (2003) mood was assessed in 20 healthy individuals given one capsule with 1000 or 1600 mg of powdered, dried *M. officinalis.* With either dose there was a significant increase in "calmness" compared to placebo. Another plant to consider is *Lavandula officinalis,* which Culpeper describes as having "...special good use for all the griefs of the head..." (Culpeper 1995) and which is indicated when there is grief around loss of a connection with someone or something (Bunce personal communication 2011). Not only does *L. angustifolia* have a history surrounding grief, a common emotion felt by women post-abortion, it is also a

nervine (Hoffmann 2003) and anti-depressant. Akhondzadah et al. (2003) investigated *L. angustifolia* alone (dose of 3mL of a 1:5 tincture), imipramine alone, and a combination of the two in the treatment of mild to moderate depression in a random, double-blind study. All three treatment groups showed significant improvement in depression scores. *Leonurus cardiaca* may also be indicated for its nervine and antispasmodic properties as well as for its potential to regulate the menstrual cycle (Hoffmann 2003).

Adaptogens may be useful post-abortion because of the increase in cortisol levels reported in some women pre- and post-procedure (Suliman et al. 2007). Specific adaptogens useful post-abortion include *Withania somnifera, Rehmannia glutinosa, Glycorrhiza glabra,* and *Asparagus racemosus*. *W. somnifera* is not only an adaptogen but also an immune stimulant (Yamada et al. 2007), a calming nervine, an antispasmodic, and contains Iron (Mills and Bone 2000). Bhattacharya et al. (2000) found support for the use of *W. somnifera* as a mood stabilizer in clinical anxiety and depression. *R. glutinosa* is an adrenal trophorestorative, has anti-hemorrhagic properties, is rich in iron, and balances hormones/regulates menstruation (Mills and Bone 2000). *G. glabra* inhibits glucocorticoid hepatic metabolism, slowing down the rate that cortisol is broken down by the body and thus sparing the adrenals from having to produce more (Whorwood 1993). Phytoestrogens present in *G. glabra* inhibit seratonin re-uptake (Ofir et al. 2003) as well as balance sex hormones by inhibiting progesterone dehydrogenases (Mills and Bone 2000). In addition, *G. glabra*, along with several other herbs, is part of a chinese formula called “Free and Easy Wanderer Plus” that is reported to be useful for PTSD (Wang et al. 2009). *A. racemosus* is a nourishing adaptogen and rejuvenating tonic that has been demonstrated to reduce corticosterone levels produced by stress (Kanwar 2010). The saponins in *A. racemosus* modulate levels of estrogen in the body (Rao 1981) and the plant is also reported to act as an antidepressant (Singh 2009), an immune stimulant (Sharma 2011), and an antibacterial against gram-positive bacteria (Mandal 2000).

Though the research demonstrates that in many women, hormone levels return to “normal” within the first menstrual cycle post-abortion, a percentage of women will still experience low levels of sex hormones, especially progesterone (Lahteenmaki and Luukkainen 1978). In such women, there are several herbal agents, other than the hormone modulating adaptogens mentions above, that may be useful. William LeSassier recommends, for both balancing the hormones and toning sexual organs and glands, equal parts *Smilax officinalis, Cnicus benedictus*, *Mitchella repens*, *Viburnum prunifolium*, and *Glycyrrhiza glabra* as a tea. He suggests drinking 4-5 cups a day during the first week after an abortion, 2-3 cups in the second week, 1-3 cups in the third, and tapering off to one cup

in the fourth week (Parvati 1978). Gail Edwards recommends *Vitex agnus-castus* after an abortion to restore hormone balance, normalize reproductive function, and act as an anti-inflammatory on the endometrium (Edwards 2000). Bergmann et al (2000) report that *V. agnus-castus* increases progesterone secretion in the luteal phase. If sex hormones are having trouble returning to normal, liver function should be examined. In addition, because Vitamin E protects pituitary and adrenal hormones from oxidation (follicle stimulating hormone, luteinizing hormone, etc.), supplementation or recommending more whole grains in the diet is a good idea (Haas 2006).

If a woman experiences unusually heavy bleeding after an abortion but not enough to warrant emergency care, uterine astringents should be employed to act as anti-hemorrhagic agents on the uterus. Many of the plants listed below will be of benefit post-abortion even if excess bleeding is not a concern because they also act as uterine tonics—strengthening and nourishing the tissue and function of the reproductive system. *Astragalus membranaceus* together with *Leonurus artemisia* have been extensively studied in China for prolonged bleeding after medical abortion (Qin 2005, Zha 2008). Matthew Wood recommends *Alchemilla vulgaris* not only for uterine hemorrhage but also for gently balancing the menstrual cycle after abortion. Elisabeth Brooke uses it when there is trauma to the uterus from abortion to "maintain feminine organs and psychological security" (Wood 1997). *Rubus spp*. have long been used to strengthen and tone the uterus as have *Viburnum spp*., which improve nutrition, circulation, and enervation to the uterus, as well as act as astringent tonics for hemorrhage and pain (Hoffmann 2003). The eclectic physicians had a few favorite plants for uterine hemorrhage. Fyfe (1908) recommends *Capsella bursa-pastoris* as a specific for hemorrhage after spontaneous abortion and writes that *Cinnamomum zeylandicum* "is one of the most certain remedies we have for uterine hemorrhage." *Caulophyllum thalictroides* was also employed after spontaneous abortion to relieve irritability of the reproductive system and prevent hemorrhage (Stephens 1930), as well as for its nervine property (Stephens 1936). *C. thalictroides* (Weed 1986) as well as *Achillea millefolium* (Tilgner 1999) tonify atonic uterine tissue and relax spastic uterine muscles, making them both useful in the case of uterine cramps as well. Scudder (1898) wrote that *Mitchella repens* "exerts a direct influence upon the reproductive apparatus of the female, giving tone and improving functional activity. It has been extensively used as a uterine tonic...". *M. repens* has the added benefit of being a moderately stimulating tonic nervine (Lyle 1932). More recently, Shipochliev (1981) tested the water extracts of several herbs and reported that *Matricaria recutitia, Calendula officinalis, Plantago sp., Symphytum officinalis,* and *Capsella bursa-pastoris* have a uterotonic effect on rabbit and guinea pig

uteri.

If uterine cramping is an issue post-abortion, antispasmodics are indicated. *Dioscorea villosa, Matricaria recutita*, *Zingiber officinale*, *Valeriana officinalis*, and/or *Viburnum spp.* all work to ameliorate pain from spasm in the uterus, specifically (Hoffmann 2003). After an abortion, the pituitary releases oxytocin, causing the uterus to clamp down (cramp) so that the uterus can return to its original size (Rock and Jones 2008). Plants that synergize with oxytocin, such as *Gossypium spp.* root bark, *Caulophyllum thalictroides*, and *Capsella bursa-pastoris* (Moore 1995) may help speed the process along, lessening the duration of cramps as well as preventing excessive blood loss.

To prevent post-abortion infection, immune stimulant or antibacterial herbs may be useful, particularly if a woman elects not to take the recommended 7-day round of antibiotics. Ellingwood (1919) recommends *Echinacea sp.* for sepsis after a badly conducted abortion, but it may be useful even after a *safely* conducted one. *Baptisia tinctoria* is recommended in King's American Dyspensitory (Felter and Lloyd 1898) for septicemia following retained fragments of placenta after abortion. Wagner et al (1985) found immune stimulating polysaccharides in *Echinacea purpurea, E. angustifolia, Calendula officinalis, Matricaria recutita,* and *Eleutherococcus senticosus,* all of which are useful post-abortion for their other properties. *Hydrastis canadense* is indicated both for its antibiotic and uterine tonic properties (Hoffmann 2003). A well-strained sitz bath may be a good use of *H. canadense* post-abortion. Both *Aristolochia paucinensis* (Gadhi et al. 1999) and *Capsaicin fructans* (Cichewicz and Thorpe 1996) are reported to have antibacterial properties specifically against *Clostridium spp*. If a woman chooses to take conventional antibiotics, pre and probiotics should be recommended (Haas 2006).

If bleeding is heavy post-abortion, consider supplementing with Iron rich herbs such as *Urtica dioica, Rumex Crispus*, *Rehmannia glutinosa,* and *Taraxacum officinalis.* Also, recommend dietary sources of Iron such as liver, leafy greens, molasses, beef, and apricots (Hoffmann 2003).

Plants indicated after any operation also apply post-surgical abortion. This includes *Arnica montanum* to lessen trauma after surgery (Karow 2008) and *Centealla asiatica* to aid in wound healing (Shukla 1999). Other supportive strategies post-abortion include counseling about birth control/fertility and encouraging self-care (sleep, good diet) for rebuilding. Planned Parenthood recommends deep uterine massage, heating pads, warm liquids, and rest for both cramping and heavy bleeding. They also recommend not doing the following for two weeks post-abortion: sex; strenuous exercise; placing anything in the vagina; douching; or using perfumes, bubbles, or oils in bath water (Planned Parenthood

2011).

Below are potential formulas for women of different constitutions (for simplicity's sake, they are formulated as tinctures, but some of the plants could be combined into lovely teas/decoctions.):

Post-abortion Formula for Vata Woman 10 ml (5ml BID)

Rosmarinus officinalis – antidepressant, antispasmodic, nervine, synergist (1 ml)
Glycyrrhiza glabra – adaptogen, antidepressant, hormone modulator (3 ml)
Rubus spp. (leaf) – uterotonic, iron rich (2 ml)
Asparagus racemosus – adaptogen, antibacterial, antidepressant, hormone modulator, immune stimulant (2 ml)
Matricaria recutita – antispasmodic, nervine, immune stimulant, uterotonic (2 ml)

Post-abortion Formula for Pitta Woman 10 ml (5 ml BID)

Lavandula angustifolia – antidepressant, antimicrobial, nervine, synergist (1 ml)
Caulophyllum thalictroides or *Viburnum spp.* – antispasmodic, nervine, uterotonic (2 ml)
Rehmannia glutinosa – adaptogen, anti-hemorrhage, hormone modulator, iron rich (3 ml)
Leonurus cardiaca – antispasmodic, nervine, menstrual cycle regulator (2 ml)
Matricaria recutita – antispasmodic, nervine, immune stimulant, uterotonic (2 ml)

Post-abortion Formula for Kapha Woman 15 ml (5 ml TID)

Artemisia vulgaris – antibacterial, antidepressant, antispasmodic, nervine (1 ml)
Calendula officinalis – antiseptic, antispasmodic, mild immune stimulant, uterotonic (1 ml)
Vitex agnus-castus – hormone modulator (4 ml)
Withania somnifera – adaptogen, antispasmodic, nervine, iron rich, immune stimulant (3 ml)
Alchemilla vulgaris – mild hormone modulator, uterotonic (2 ml)
Hypericum perforatum – antidepressant, antimicrobial, astringent, vulnerary (4 ml)

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Adverse Effects of Cross-sex Hormone Replacement Therapy

Linden de Voil

This paper explores potential side effects of long term cross sex hormone therapy in transsexual individuals, with focus on delineating which risks have been clinically observed and which are potential concerns that have not been seen in vivo. Although there is some potential to use herbs with feminizing or masculinizing effects as a substitute or adjunct to hormone therapy, my interest in this research is on supporting the organs and systems which may be negatively impacted by CSHRT. Hormone treatment is often accompanied by various surgical interventions, which involve a separate set of risk considerations not covered here. For the sake of brevity I have not outlined the positive/desired effects of hormones, which are covered extensively in many guides focused on gender transition; I have listed some excellent online resources with my references.

Worldwide incidence of transsexualism (defined as gender identity disorder) as estimated by the American Psychological Association DSM-IV at 1 in 100,000 female to male (FM) and 1 in 30,000 male to female (MF). However, as there have been no formal epidemiological studies, these numbers are based on indirect evidence of patients who have presented for referral to hormone therapy and surgeries, which they are presumably able to afford. There is likely a much higher incidence of transsexualism and, more broadly, gender dysphoria, with some estimates as high as
1:750 for MF and 1:1400 for FM (Horton 2008).

CSHRT is widely used and generally accepted as part of a standard protocol for individuals who want their external gender presentation to align more closely with their perceived gender. In MF transwomen, therapy usually consists of an anti-androgen (commonly spironolactone, sometimes flutamide or cyproterone acetate) alongside some form of estrogen, which may be taken orally, transdermally, or by injection. Estrogen is taken in high dose, three to five time the dose for a natal female undergoing hormone

replacement therapy, in order to produce feminization of secondary sex characteristics and to assist in suppression of testosterone. Progesterone may be included, although it is not a standard measure, and clinical evidence for efficacy is lacking. Transmen typically take only testosterone, in a dose sufficient to suppress menses and produce masculinization of physical characteristics, up to the standard
dose for hypogonadal natal men (Hembree et al 1994, Gorton 2005).

Various hormone preparations and administration routes have their own risks, which are outlined here in relation to the specific systems impacted. The greatest concern is use of ethinylestradiol in particular, and oral estrogens generally, which carry greater health risks than other estrogen preparations, including significantly increased risk of thromboembolism in the first year of treatment (Toorians 2003, van Kesteren
1997), greater prevalence of hepatotoxicity and increased markers of inflammation
(Goodman 2012, Wilson 2009).

Sex hormones have well documented slow acting gene-mediated effects through activation of nuclear receptors; more recently estrogen and testosterone have been shown to inducerapid effects through cell membrane receptors, which are found in the brain, reproductive organs, and cardiovascular tissues. In vitro, they appear to have some activity at neurotransmitter receptors, although this has not yet been shown to have clinical relevance (Falkenstein 2000).

Main sites affected or potentially affected by CSHRT include the breast tissue, ovaries and uterus, bone, liver, pituitary gland, adrenals, heart and vasculature. Research specific to trans populations is necessarily limited, with many studies comprising a very small population; in many cases, research remains inconclusive.

Cardiovascular health and inflammation:

While the effects of testosterone administration on transmen are not always the same as for natal males, some relevant research is worth noting. A 2013 review suggests that although evidence is conflicting, in hypogonadal natal males testosterone is primarily protective and anti-atherogenic, resulting in increased coronary artery flow, overall vasodilation and calcium-channel blocking effect; most negative effects result from its use in the frail elderly or in supraphysiological doses, which are not recommended for the

purposes of gender transition (Kelly 2013).

Sodium retention and mild edema are considered typical side effects of treatment with anabolic steroids, whether oral or injected; research on hypogonadal natal men also shows an increase in extracellular fluid volume under the influence of exogenous testosterone, without any corresponding changes in aldosterone, ANP, or blood pressure (Bassil 2009, Johansson 2005).

Overall, most FM trans-specific studies show changes in inflammatory markers, including increase of the vasoconstrictive protein endothelin-1, C-reactive protein, and homocysteine, with decreased HDL, increased triglycerides and lipid oxidation, and an increase in visceral fat deposition (Elamin 2009, Gooren 2008). There is an increased risk of polycythemia, which increases further with age; if severe, this includes increased risk of venous and arterial thrombosis (Gorton 2005).

However, multiple studies conclude there is generally *no change* in relative cardiovascular morbidity outcomes or increase in significant adverse events following CSHRT; a 2008 multi-study review (n=712) showed no increase in blood pressure, insulin sensitivity, clotting factors or arterial stiffness (Giltay 1998, Gooren 2008, Elamin 2009). The exception is administration of testosterone undecanoate, which was linked to increased blood pressure in 4 out of 35 users, with two cases of severe hypertension (Mueller 2007).

For MF transwomen, use of oral estrogens resulted in increased inflammatory markers associated with CV risk; however, transdermal estrogen does not produce the same result. (Transdermal applications are significantly more expensive than oral estrogens, and may not be covered by health insurance.) Risk increases dramatically when compounded by additional factors, particularly smoking. (Wilson 2009).

Compared to control groups, transwomen show increased rates of venous thromboembolism and cerebrovascular disease, with rates of heart attack comparable to those of natal men. In the same study, transmen showed no increase in any negative CV outcomes, but did have increased rates of type-2 diabetes (Wiercx 2013).

Changes in the Th1/Th2 ratio (as expressed by interferon gamma/interleukin 4) can be seen in both MF and FM, with a relative increase in Th1 resulting from androgen administration and decreased Th1 from estrogen (Giltay 2000). This shift toward Th2 phenotype could be an influential factor, considering that although some CV risk

markers appear to increase for transmen, actual adverse outcomes are increased only in transwomen.

Bone density

Because estrogen plays a significant role in maintenance of bone density, there is some concern about risk of osteopenia/osteoporosis following hormone replacement, especially in the case of oophorectomized transmen. The most relevant study shows that estrogen use in MF transwomen is linked to maintenance or increase in bone density. Although there should be some estrogen created from aromatization of testosterone in FM, this may not be adequate to prevent bone mineral loss after removal of the ovaries (van Kesteren 1996, 1998). It is suggested that post-oophorectomy transmen may want to supplement calcium, and possibly vitamin D, as a post-menopausal natal female would do (Gorton 2005).

Hepatic function

Multiple mortality and morbidity studies have shown no increase in hepatic pathologies in connection with CSHRT, other than higher rates of infectious hepatitis among transwomen (Asschman, van Kesteren, Gooren).

Alterations in hepatic function are considered a standard associated risk with use of testosterone replacement therapy. However, cholestasis, hepatotoxicity and hepatic tumors have only been documented in connection with oral forms of C-17 alkylated testosterone, generally in supraphysiological dose range; liver pathology has not been linked to use of transdermal or injectable unmodified testosterone within dose ranges used for HRT in either trans or natal men (Bassil 2009, Gorton 2005).

It is recommended that transmen be screened regularly (1-2 times per year) for changes in liver function. Unexplained elevated enzyme levels are found in 15% of transmen on hormone therapy (van Kesteren 1997). Although it is unclear if such changes are linked to overt pathology, reductions in dose may be recommended by the prescribing physician if this occurs (Gorten 2005, Hembree 2009).

In transwomen, use of anti-androgenic drugs other than spironalactone or finasteride, including leuprolide, flutamide, ketoconazole, and cyproterone acetate, has significant risk of hepatotoxicity, usually appearing as hepatitis with jaundice (Thole 2004). Cyproterone, leuprolide and flutamide are most commonly used in treatment of prostate cancer; the effective dose to induce desired feminization is much higher, and consequently carries greater risk of side effects.

Both testosterone and estrogen are primarily metabolized in the liver, through various CYP pathways; testosterone is a 3A4, 2C9, and 2C19 substrate (Choi 2005); estrogen is a 1A2 and 3A4 substrate (Tsuchiya 2005). Oral ethinylestradiol is often not recommended for CSHRT, due to the high dose required and increased risk for thromboembolism and changes in hepatic protein synthesis, but in some instances it is used. Ethinylestradiol persists for longer in the blood, with greater effect on the liver, than injectable or transdermal estradiol (Kapp 2009).

Evidence does not show increased hepatic risk for trans people using CSHRT, relative to same-sex HRT; however, it should be considered in the same light as long-term use of any hepatically-metabolized pharmaceutical drug.

Breast health and cancer

There is a theoretical concern for increased risk of breast cancer for transwomen; recent research on natal women shows an increase in breast cancer rates among recipients of estrogen and progestin combination therapy, which in some cases is also used for transwomen. (Chlebowski 2009).

However, a study of 816 MF transwomen found zero cases of breast cancer (van Kesteren 1997) and a recent study showed that both transmen and transwomen fall into the normal range of occurrence of breast cancer in natal men (Gooren 2013).

Many, though not all, transmen undergo a type of mastectomy that leaves some breast tissue in place. In a study of transmen following at least 6 months of hormone therapy, post-mastectomy histology showed atrophic reduction of glandular tissue, increase in fibrous connective tissue, and no atypical hyperplasia or carcinoma. (Grynberg 2010).

Another 2013 morbidity study showed equal rates of all cancers in both MF and FM transsexuals when compared with control groups of natal men and women (Wiercx 2013).

Based on the most recent research, there appears to be no increased risk for breast or other cancers.

Ovaries, uterus and prostate

Because of limited amounts of research and clinical evidence, there has been concern about the effects of long term use of androgens on the ovaries and uterus of transmen, and particularly for the potential risk of cancer. Bilateral oophorectomy-salpingectomy and hysterectomy are generally recommended within five years of starting hormone treatment. This is based on a risk-prevention strategy, the ability to subsequently decrease dose of testosterone, and on the idea that transmen are likely to have some degree of discomfort or trauma surrounding gynecological exam, and therefore are less likely to seek out the annual preventative screenings recommended if the organs are retained. (Gorton 2005, Hembree 2009).

No increase in cervical or uterine cancers has been reported. Three cases of ovarian cancer have been reported in transmen, although it is unclear if this represents an actual increase in rate of occurrence (Moore, Asscheman, Hembree). However, there is a demonstrated upregulation of androgen receptors in the ovaries following CSHRT, and some in vitro evidence links increased ovarian androgen receptor activity with growth of cancer cells. (Ahonen 2000, Sheach 2009).

Some research has pointed to potential connection between cross-sex androgen therapy and increased incidence of PCOS. However, this is complicated by the evidence that transsexuals have a higher incidence of pre-existing PCOS, compared to cisgender natal females, before beginning hormone therapy. (Bosinski 1997).

Research published in 1991 showed changes in the ovaries of FM transsexuals after androgen therapy, judged to be consistent with changes caused by PCOS (Pache 1991). Additionally, some associated markers for PCOS appear to increase in transmen after hormone therapy; in FMs, androgen therapy increases both ADMA and endothelin-1 levels, which have been shown to have positive correlation with incidence of PCOS; however, causation is unclear (Polderman 1993, Bunck 2009, Charitidou 2008).

A 2007 study in Japan found that although 58% of FM patients had PCOS ovarian characteristics, there were no corresponding increases in insulin resistance, which was linked only to obesity and not to PCOS morphology. (Baba 2007). Another study showed that although there was an increase in antral follicles consistent with PCOS in 79% of FM patients, 45% of patients also showed significant endometrial atrophy (Grynberg 2010). A third study showed universal atrophy similar to that found in post-menopausal women (Perrone 2009). This year a surprising new study out of Japan reported that although androgen therapy does induce some hyperplasia within the ovaries, there is no change to antral follicles, and the changes present are not consistent with PCOS (Ikeda and Baba 2013).

Within the last five years, research has begun to show that although there are morphological changes to the ovaries after androgen therapy, these changes do not necessarily mimic PCOS, nor is there evidence of increased rates of endometrial cancer or of metabolic changes similar to those linked with PCOS. Given that fertility is never reliably preserved after CSHRT, and that amenorrhea is a desired effect, the main relevant effect of PCOS in FMTs is metabolic disregulation and increased risk of cancer due to endometrial hyperplasia. Evidence appears to be mounting that neither of these is, in fact, increasing in occurrence following CSHRT.

While concerns regarding PCOS in transmen may perhaps be allayed, there is not enough evidence to come to a clear conclusion on risks for ovarian cancer. There is little research into the possible need for estrogen in transmen using CSHRT, other than reducing risks for osteoporosis. However, the lack of research does not assure us of benefit. The choice to undergo or refrain from oophorectomy and/or hysterectomy is individual and must involve weighing of risks of surgery, potential future health risks, and individual ability and desire to monitor gynecological health.

Effects on original gonads are generally, though not always, less relevant for transwomen, who are much more likely to undergo reassignment surgery that includes orchiectomy, as it dramatically increases feminization results while reducing the required dose of hormones. Although the prostate is not removed as part of GRS, it generally atrophies without the influence of testosterone; however, this is not always the case, and there have been reports of BPH significant enough to warrant surgical intervention, in one instance after 25 years on CSHRT (Casella 2005). Similarly, reported cases of prostate cancer in transwomen are extremely rare, and incidence is certainly not increased by CSHRT; risk results from lack of preventative screening or non-treatment. (Asscheman, van Kesteren 1997).

HPA / cortisol

Given that "close crosstalk exists between sex hormones and glucocorticoids at both neuroendocrine and peripheral levels, with different specificities according to sex," (Pasquali 2012), and that both estrogen and testosterone modulate HPA activity (Handa 1994, Rubinow 2005), we can reasonably expect that CSHRT may cause alterations to corticoids and other elements of the endocrine system. Trans-specific research in this area is extremely limited, and relevant primarily to transmen; to some extent we can extrapolate from other research, but more information is needed to draw any firm conclusions.

Introduction of exogenous testosterone into healthy natal men resulted in an increase of ACTH but an inhibition of cortisol; the ACTH:cortisol ratio was lower, suggesting decreased adrenal sensitivity, mediated by the adrenal gland itself, not the pituitary. (Rubinow 2005). In transmen, testosterone has been shown to result in increased adrenal androgen output by up to 70% (Polderman 1994) as well as overall higher levels of cortisol, DHEA, and androstenedione (Polderman 1995). Although inconclusive, this suggests that CSHRT in transmen could be linked with alterations in HPA function at the adrenal level.

In addition to physiological changes to adrenal function, cortisol production may be impacted by the well-documented increase in social stressors encountered by transgender and transsexual individuals (Israel 1997, Haas 2011.) A recent study explored transition-specific sets of stressors in FMTs, perceived levels of stress and correlated rise in cortisol, along with increased C-reactive protein and diminished decline in nocturnal blood pressure. The researcher linked these markers to acute stress caused by the transition process, which generally declined by the third year after beginning hormone therapy (du Bois, 2012). Interestingly, another study found that a.m. cortisol levels, which were already elevated in pre-CSHRT trans individuals, declined in the year following commencement of therapy, which was attributed to reductions in stress linked to transition (Colizzi 2013). What both of these studies have in common is the existence of elevated cortisol and perceived stress levels among trans individuals, regardless of causation.

There is also a reported correlation between use of testosterone and worsening or unmasking of obstructive sleep apnea; in addition to other health risks, sleep apnea can induce intermittent nocturnal cortisol spikes and correlative HPA disregulation. However, this connection is not solidly documented; although there have been a number of case reports, a recent meta-review suggests that there is not enough reliable evidence to posit a link between TRT and OSA (Hanafy 2007).

Conclusions

Endocrinologist Dr. Louis Gooren, who has co-authored many of the studies on transsexual health, summarized his perspective after two decades clinical experience and research: "It is clear now that sex reassignment of transsexuals benefits their well- being, although suicide rates remain high. Cross-sex hormone administration to transsexuals is acceptably safe in the short and medium term. However, potentially adverse effects in the longer term are presently unknown. The data, although limited, of surrogate markers of cardiovascular disease and the reports of cancer in transsexuals leave room for cautious optimism" (Gooren 2008).

When weighing risks of treatment, it is important to remember that transgender/ transsexual individuals are at an increased risk of suicide or self harm, with actual suicide attempts perhaps as high as 1:3 (Haas 2011). Risk of suicide is reduced from 20% when gender dysphoria is untreated to 1% after treatment; such a significant reduction may be considered to mitigate potential detrimental effects of hormone treatment (Levy 2009, Gorton 2005).

CSHRT is, for many individuals, a lifelong treatment plan that may begin as early as adolescence. In most areas, its risks are not greater than those associated with same- sex HRT, and are amenable to intervention, including preventative screening and supportive lifestyle choices.

Resources for more detailed info about CSHRT treatment, protocol and effects:

R. Nick Gorton, Medical Therapy and Health Maintenance for Transgender Men: A Guide For Health Care Providers. (Free online version available from nickgorton.org)

Transgender Health Care Info Archive: transgendercare.com. (Extensive info about MF hormone protocol and surgeries)

Hudson's guide to transition: ftmguide.org. (Excellent resource for FM hormone, surgical, and lifestyle transition)

The Center of Excellence for Transgender Health: transhealth.ucsf.edu.

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Fibromyalgia: Patterns, Pathology and Strategies for Herbal Support

Emily Peters

Introduction

Fibromyalgia (FM) is a syndrome characterized by widespread musculoskeletal pain, physical and mental fatigue, efort intolerance, non-restorative sleep and mood disturbance (Van Houdenhove 2006). Herbalist Chanchal Cabrera describes the clinical features of FM: “Symptoms generally begin as diffuse aching and stiffness which slowly worsen over a period of weeks or even months. Eventually the muscle stifness and pain become chronic and constant, and there are frequently 'trigger points' in the muscles. These are exquisitely tender nodules that are palpable. Symptoms also include local muscle spasm and low grade inflammation. … Frequently there will be poor sleep, overwhelming fatigue, irritability, anxiety, bladder and urethral irritation, irritable bowel symptoms, and general malaise.” (Cabrera).

There doesn't appear to be a clear cause of FM, but rather it is clinically associated with high levels of stress, depression, post-traumatic stress disorder (PTSD), childhood and adult abuse, and hypothalamus-pituitary-adrenal (HPA) dysfunction (Walker, Peres, Van Houdenhove 2006). Stress activates the sympathetic nervous system's fright, fight or freeze response, and long term stress can have great impacts on the HPA axis and overall neuroendocrine function (Van Houdenhove 2004). In this paper, I will describe the symptoms and patterns of FM, outline the relationship between FM and stress, and explain pathogenic mechanisms though which FM syndrome could arise. Ten I will briefy explore strategies for herbal support that take into account the person's constitution, energetics and symptom presentation.

Symptoms and Patterns

FM is far more common in women (Cabrera, Van Houdenhove 2005) and affects between 2 and 10% of people in industrialized countries. The criteria for diagnosis of FM is chronic

widespread pain in all four quadrants of the body and in the axial skeleton, tender points in 11 out of 18 designated areas, and is generally accompanied by an increased sensitivity to pain throughout the entire body (Clauw). FM is characterized by generalized allodynia, or pain elicited from stimuli that are not usually painful, and by hyperalgesia, or increased intensity and prolonged duration of pain caused by stimuli. Karl Henriksson states that “FM should be suspected in any chronic musculoskeletal pain condition when localised or regional pain spreads to several sites, when temporary or intermitted pain becomes more or less continuous, when pain on movement becomes pain at rest, or when segemental allodynia/hyperalgeia becomes generalised.” In one study comparing 191 patients with FM, 80% reported that localized pain gradually developed into widespread pain (Henriksson).

Overlap of symptoms as well as frequent comorbidity is found between FM and other unexplained symptoms, including: chronic fatigue syndrome, PTSD, low back pain, anorexia, insomnia, irritable bowel syndrome, headaches, migraines, multiple chemical sensitivity, joint disorders, major depression, anxiety, and panic attacks (Masé, Peres, Clauw, Mills & Bone pg 311). A general personality pattern common to people with FM includes low self-esteem, anxiety, depression, lack of emotional openness, excessive striving for achievement and recognition, aggression inhibition, and harm avoidance (Van Houdenhove 2004). Clinical observation suggests that FM often occurs in the aftermath of a long period of physical or emotional stress, often associated with depression. Additionally, FM seems to be precipitated by an additional triggering physical or emotional event such as an injury, infection, toxic exposure, or emotional trauma (Vertolli, Masé, Van Houdenhove 2006). Other possible triggering factors include Epstein-barr viral infection, hormone alterations, hypothyroidism, lyme disease, parvovirus infection, physical trauma and Q fever (Clauw). In the experience of Chanchal Cabrera, primary FM is the most likely to occur in healthy young women who tend to be stressed, tense, depressed, anxious, and striving, while men are more likely to develop FM from a particular physical strain (Cabrera).

A Close Relationship with Trauma and Stress

In a 1997 study comparing patients with rheumatoid arthritis (RA) and FM, patients with FM had significantly higher lifetime prevalence rates of all forms of abuse and greater severity of childhood trauma. In this study, Edward Walker states that “physical assault in adulthood [is] found to have a strong relationship with unexplained pain. Trauma severity was

correlated significantly with measures of physical disability, psychiatric distress, illness adjustment, personality, and quality of sleep in patients with fibromyalgia … ” Nearly all FM patients had experienced some form of trauma. In this study, associations were also found between trauma and pain intensity, number of pains, perceived stress, neuroticism and illness coping. Walker states that “... there is a high correlation between victimization history and the number of medically unexplained physical symptoms the patient has experienced ... Tis high correlation suggests that early experiences of victimization for which there were no timely interventions may be expressed over time through the appearance of medically unexplained physical symptoms. Te original trauma might actually gradually transform into physical symptom manifestations …” (Walker).

Walker speaks to research that suggests people can store trauma in our bodies, and this can manifest physically. Tis 'theory' that modern medicine is (finally) giving a voice to is a timeless lived experience that many, many people know in their bodies to be undoubtedly true. Nonetheless, this research is a signifcant step towards validating 'unexplained' physical illness, particularly for survivors of abuse and trauma.

A 2009 study explores the link between PTSD and FM. PTSD is characterized by the emergence of symptoms after a person experiences one or several/recurring traumatic event(s). Symptoms of PTSD include: re-experiencing the trauma via nightmares, memories or intrusive thoughts; emotional avoidance or numbness; and hyperstimulation characterized by insomnia, hyperarousal and irritability. Women with high disability pain have been found to be more likely to have experienced child abuse, adult sexual assault, more severe spousal abuse, lifetime abuse-related injuries and PTSD symptoms (Peres).

Many FM patients' symptoms resemble PTSD; individuals with PTSD often present with chronic pain and vice versa; both conditions involve similar pathways through with the symptoms can escalate. FM patients with PTSD reported significantly higher levels of avoidance, hyperarousal, re-experiencing, anxiety and depression than FM patients without high levels of PTSD. The prevalence of PTSD amongst FM patients was found to be much higher than the general population and women with FM reported more traumatic events than their male counterparts. Both disorders seem to be induced by relatively short term stress followed by chronic pathology, suggesting that the stress may induce a self-perpetuating vicious cycle. Clinical experience suggests that clients with PTSD and FM, especially with high levels of dissociation, will present with worse symptoms in response to exposure to traumatic triggers (Peres). It is also possible that early life stress and trauma can alter the set point of the stress response system, contributing to an increased

susceptibility towards stress-related disorders and stress in general later in life. (Van Houdenhove 2004)

The clinical relationship between pain and depression is well recognized; FM and depression are both stress-related conditions and lifetime depression is a risk factor for FM. Te relationship between stress, depression and FM exists in a recursive pattern that traps the patient in a vicious cycle (Van Houdenhove 2006). Pain in FM should not be considered merely an expression of depression, as the stress of the illness may lead to depression, contributing to further symptoms such as sleep problems, less adaptive coping, physical and mental dysfunction and decreased quality of life. Anxiety also plays a perpetuating role in FM by increasing arousal, irritability, muscle tension, hyperventilation and avoidance behavior, resulting in higher pain intensity, more tender points, increased functional limitations and increased fatigue (Van Houdenhove 2004). Constant pain is both a physical and emotional stressor, often leading to psychological distress including depression (Henriksson). A high correlation has been found between the number of tender points reported by a patient and measures of anxiety, distress and depression (Clauw). FM patients tend to had a difficult time dealing with emotional stressors, which could partially explain the role of stress in the onset of the condition; their symptoms, particularly chronic pain, lead to increased stress, contributing to a vicious cycle that is self-perpetuating. (Van Houdenhove 2005).

Pathogenic Mechanisms

FM is thought to arise from acute stress, medical illness and various pain conditions in conjunction with a variety of neurotransmitter and neuroendocrine imbalances, including HPA dysregulation, excessive pronociceptive input, and defcient modulatory signaling via noradrenergeic and seratonergic pathways (Peres). A study found that those who had chronic widespread pain were three times more likely to have signifcantly lower levels of saliva cortisol and higher levels of post-stressor serum cortisol than the control group, demonstrating an HPA axis dysregulation (McBeth). FM is characterized by HPA axis hyporeactivity to physical and mental stressors. Tis altered neuroendocrine responsiveness could be described as a 'loss of resilience' of the stress response system. “Te available evidence seems to confrm … that the stress response system of patients with chronic pain and fatigue may be impaired after a period of overburdening by physical and/or emotional

stressors, Tis may imply a biologic 'switch' from hyperactivity to hypoactivity of the system, associated with function or even structural receptor changes, and followed by a cascade of disturbances in immunologic, neurotransmitter, and central pain processing mechanisms" (Van Houdenhove 2005).

It is important to understand how the stress response can lead one to hypoactivity of the stress response system, which is a presentation of HPA dysregulation, hypocortisolism and adrenal exhaustion. Te hypothalamus and/or amygdala perceive and judge danger, which is impacted by past experiences and emotional memory. When a threat or stressor is identifed, the hypothalamus stimulates three pathways: the alarm reaction via sympathetic nervous system, stimulating the locus ceruleus; intermediate reaction via sympathetic nervous system, stimulating the adrenal medulla; and the adaptation or resistance reaction, via hypothalamic corticotropin releasing factor (CRF) to pituitary, then ACTH to stimulate the adrenal cortex to secrete cortisol (Bunce July 2013).

Cortisol impacts virtually every tissue in the body. It stimulates muscle protein breakdown, liberating arachidonic acid to be used by the liver in gluconeogenesis leading to elevated blood sugar. It increases resistance to trauma, infection, hemorrhage and exertion in the short term, while decreasing immune competence in the long term. It down-regulates macrophages, stimulates T lymphocytes, and suppresses serotonin, histamine, and eicosanoids (prostaglandins and leukotrienes). It decreases levels of GnRH, LH/FSH, progesterone and testosterone, decreases delta wave (deep) sleep patterns, increases prolactin, and stimulates morning wakefulness (Bunce July 2013).

Te amygdala can bypass the frontal cortex when a stressor is perceived as an emergency, even when there is no real danger. Te amygdala becomes hyper-aroused, stimulating the hypothalamus and amygdala to both release CRF. Trauma can be reactivated, anchoring us in old learned emotional patterns via the amygdala; this is where emotions become physiology. Chronic exposure of HPA to corticoids can down-regulate sensitivity, interrupting negative feedback loops. Arousal can be maintained because of this positive feedback from repeated amygdala stimulation. Tis shifts one's perception of 'reality' to become hypervigilant. Te cycle continues, as continued perception of stress maintains the stress response. Over time, this cycle will eventually lead to adrenal exhaustion and hypocortisolism (also called the allostatic load). Tis chronic long term exposure to stress response causes symptoms that overlap signifcantly with FM including: reduced immune response, anxiety/depression, fatigue and sleep disruption, cognitive defcit, and muscle weakness. Insulin-resistance, hypothyroid function, sex hormone dysregulation, PCOS,

osteoporosis and hypertension can also be a part of a HPA dysregulation picture (Bunce July 2013).

In FM, the HPA axis and the sympathetic nervous system are both dysregulated. Tis causes changes in neurotransmitter function, immune function and central pain mechanisms. Dysregulated immune activity includes the release of pro-infammatory cytokines that can promote lethargy, fu-like symptoms, social withdrawal, concentration difculties, mood lowering and pain-threshold lowering, which all can ft the symptom picture of FM. Changes in central pain processing has been confrmed by brain imaging studies in FM patients, linked to the inadequate descending pain inhibition and a dysfunction of endogenous opioid and low CRF (Van Houdenhove 2006). A study found that 80% of FM had elevated levels of Substance P in the CSF, which has been linked to the release by neurons in the CNS. Te constant pain hypersensitivity could be due to changes in the function of sensory neurons and how they send information to the CNS in response to noxious stimuli (Henriksson). Stress-induced pro-infammatory cytokines such as IL-1, IL-6, and TNF-alpha also may contribute to the pathogenesis of pain. Research suggests that prostaglandins, leukotrienes, pro-infammatory cytokines, and nitric oxide could also play a role in the pathophysiology of FM (Van Houdenhove 2004).

Traumatic experiences may increase vulnerability to FM by psychosocial mechanisms, such as hypertension of muscles, sleep problems, HPA axis dysfunction, and inadequate stress coping from low self-esteem, depression and abuse. Perpetuating stressors can decrease quality of life and likelihood of recovery, such as: not accepting the condition and thus not adjusting lifestyle accordingly, a lack of social support, and not being accepted as sufering from a legitimate illness. (Van Houdenhove 2006).

An Herbalist Approach to FM

In caring for a person with FM, it is very important to understand people's stories. Listening and giving emotional support can help to understand how their condition is afecting them. If appropriate, we could help people shape their views diferently (Bunce Dec. 2013). Conceptualizing FM as a stress disorder may help with client-practitioner rapport and encourage patients to address psychosocial issues (Van Houdenhove 2004). It is important to evaluate a client's diet, nutrition and lifestyle factors and help them to envision healthy changes and assess whether there is gut infammation that is complicating symptoms (Bunce Dec 2013, Cabrera). Toxic exposure has also been found to be a precursor to FM; in

assessing a client's history it is important to consider this as a possible causative factor and formulate accordingly (Bunce Dec 2013, Vertolli).

Looking through the lens of the Ayurvedic doshas, we can see how FM can arise in diferent constitutional picture. In the cold and dry Vata, we are likely to fnd a client with little to no resources, gone through adrenal fatigue to exhaustion with anxiety. A Kapha will likely be stagnant, depressed and prone to worry about others. A Pitta will likely exhibit the infammatory picture of autoimmunity and hypersensitivity with FM as a secondary complaint (Bunce Dec 2013). Matthew Wood considers FM to be an expression of the damp stagnation, torpid tissue state. He explains that a build-up of unneeded fuids, precipitating into thick phlegm, prevents tissues from getting enough nutrition so they become fabby and weak and this causes pain in the muscles, with accompanied symptoms of skin lesions, backed up digestive and liver function, lymphatic stagnation, and in some cases hypothyroidism. He indicates that blood purifers or alteratives are the most appropriate herbal remedy (Wood 2004). Tis seems to best ft the Kapha picture that Bunce describes. Michael Vertolli views the typical constitutional pattern as autoimmune and infammatory, following emotional or toxic stress (Vertolli), ftting the Pitta pattern the most clearly. In any person, of course we fnd many overlapping energetic patterns and there usually is not a perfectly clear constitutional picture – people are complex and we should tailer their specifc herbal protocols based on their unique presentation.

Herbal goals for support include: pain management, decrease substance P, improve detoxifcation, bufer stress perception and anxiety, improve circulation, improve sleep, normalize immune function, regulate HPA axis and modulate infammation (Bunce Dec 2013, Cabrera, Masé, Vertolli). Tere are many dozens of herbs that can meet these goals; I am going to give examples of herbs that seem to be the most specifc for FM based on my research. An herbalist can choose other plants based on the person's set of symptoms, for issues such as GI infammation, insomnia, acute pain, anxiety, hypothyroidism, etc.

Anti-infammatory and nervine herbs help to relieve symptoms of pain and improve mood. Hypericum perforatum, St John's wort, is useful for depression, as an anodyne and a nerve tonic, if the medication list allows (Masé, Cabrera, Mills & Bone). Actaea racemosa, black cohosh, is used as an anti-infammatory for the muscles and for someone with a dark and brooding mind, signs of congestion, and with shifting, sharp, spasmodic pain especially in the back and neck (Cabrera; Wood 1997); it is also indicated for a person with a history of sexual abuse, alcoholism or drug abuse; with heavy, aching pain, muscular tension and rheumatic pain made worse with activity (Alfs pg 35). Uncaria tomentosa, cat's claw root

bark, is immunomodulant and anti-infammatory for joint pain, rheumatism and FM (Bunce Dec 2013, Alfs pg 43). Curcuma longa, turmeric, is a useful warming anti-infammatory (Cabrera, Mills & Bone). A cognitive stimulant and brain tonic would be useful, such as Rosmarinus of., rosemary; Centella asiatica, gotu kola; Avena sativa, milky oats; or Ginkgo biloba, ginkgo (Cabrera, Alfs pg 62, Mills & Bone).

Alterative, circulatory stimulant herbs can help support elimination and detoxifcation. Arctium lappa, burdock, and Apium graveolens, celery seed, are useful alteratives with a specifc afnity for the musculoskeletal system (Cabrera, Mills & Bone, Wood 2004). Te bitter nervine Verbena spp., vervain, has a tonic efect on the glands of the body (Cabrera). Urtica dioica, nettle, can be indicated for FM as a nutritive tonic. Juglans nigra, black walnut hulls, and Fucus vesiculosus, kelp, are both useful when hypothyroidism accompanies FM (Cabrera, Wood 2009). Zanthoxylum, prickly ash bark, is a useful circulatory stimulant specifcally for encouraging blood fow to the muscles (Bunce Dec 2013, Cabrera). For stagnation of fuids and to help eliminate toxins, a lymphatic such as Phytolacca decandra, poke; or Galium aparine, cleavers, would be useful (Cabrera, Wood 2004). For supporting the immune system, Echinacea spp., has been found useful (Cabrera, Mills & Bone).

Adaptogenic herbs have an important role to play in supporting the adrenals, normalizing the HPA axis, and generally improving one's stress response. Adaptogens that have been used for FM include: Eleutherococcus senticosis, eleuthero, as an adrenal tonic and energy enhancer; Glycyrrhiza glabra, licorice, as an adrenal-restorative and anti-infammatory; and Ganoderma lucidum, reishi, for disturbed Shen, to modulate immunity, for pain and joint weakness, insomnia, and especially for a pro-infammatory, hypersensitive person (Bunce Dec 2013, Mills & Bone, Cabrera, Alfs pg 92). Any adaptogens would be useful here as well, chosen based on the person's constitution and specifc presentation, and are most useful for long-term support (Bunce Dec 2013).

Complementary Strategies

Strategies to reduce pain and stress, improve sleep, reduce anxiety and depression, detoxify, modulate immunity and regulate the HPA axis should be complemented by body work and therapy. When FM overlaps with a history of trauma, it is very important that treatment focus on both physical and emotional dimensions. Te role of evaluating someone's traumatic history and helping them process it should be taken on carefully by someone trained to do so – a therapist or psychologist who understands the process of

uncovering and healing from trauma; for many people it can be very painful and difcult to look at and reconstruct these memories (Peres). Careful low-intensity aerobic exercise, tailored to the person's abilities can also be useful (Van Houdenhove 2004). Herbalists have found it helpful to refer clients to massage therapy, craniosacral therapy, counseling, and spiritual/awareness type practices (Masé, Vertolli).

Cognitive behavioral therapy is particularly helpful to optimize coping, implement adaptive lifestyle changes and encourage long-term self-care (Van Houdenhove 2004). Cognitive therapy is a structured, directive form of psychotherapy where the goal is to help the patient identify maladaptive cognitions and change them. Te approach of mindfulness meditation has been found to be efective in the treatment of patients with chronic pain. It is a system of self-inquiry that stems from Buddhist philosophy and leads to increased awareness of one's thoughts, sensations, feelings, consciousness and the nature of one's mental processes. A study found that a mindfulness meditation based stress reduction program signifcantly improved the symptoms of patients with FM (Kaplan).

Conclusion

Conventional medicine has not established strategies to cure FM. Tey can use medications for sleep, IBS, pain management, depression and anxiety, as well as recommending cognitive behavioral therapy and aerobic exercise (Clauw). HPA axis dysregulation is not recognized as being curable . With a holistic mindset, we are able to step back and evaluate the root causes of imbalances that can cause debilitating syndromes such as FM. Te research suggests that HPA axis dysregulation, usually stemming from long-term trauma, toxicity, or another physical/mental stressor, seems to be a likely cause of FM. In our herbal strategy we can assess the person's energetic constitution and explore what stressors may have led to this imbalance for them. Along with referrals to therapy and body work, we can formulate herbal remedies with alteratives, anti-infammatories, nervines, and adaptogens to help relieve symptoms and support the body, particularly the stress response, to come into balance.

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Herbal Therapeutics for physiological hyperprolactinemia concurrent with PMS symptoms

Angie Barger

Hyperprolactinemia is characterized by higher than normal blood levels of PRL. Elevated PRL levels can be physiological or pathological. 10 Pregnancy, lactation, nipple stimulation, and stress can physiologically induce hyperprolactinemia. 10 The most common pathological causes of hyperprolactinemia are prolactinomas (PRL producing tumor), hypothyroidism, drugs, renal failure and cirrhosis of the liver. 10 This paper will address the physiology, pathophysiology and herbal therapeutics for lactation-induced hyperprolactinemia concurrent with PMS.

In lactation, PRL secretion inhibits dopamine. 9 PRL is a peptide hormone from the anterior pituitary that controls milk production in the breast, and appears to play a role in regulation of the immune system. 9 Dopamine is an amine Central Nervous System neurotransmitter associated with sensing pleasure. 9 PRL is primarily controlled by an inhibiting hormone from the hypothalamus, Prolactin inhibiting hormonr (PIH). 9 There is good evidence to suggest that PIH is actually dopamine.10

Pre-menstrual syndrome (PMS) is defined by psychological, behavioral and physical symptoms which occur in the luteal phase of the menstrual cycle.10 A syndrome is a collection of possible symptoms. In PMS, there are estimated to be 150 collective symptoms. This paper focuses on those included in PMS-A: tension, irritability, insomnia, depression, anxiety, etc. and PMS-D: low estrogen levels with overlapping PMS-A symptoms and PMS-H. These specific PMS groups have been linked to excess PRL in the bloodstream. 10 PMS theory currently supports an abnormal tissue response to the normal changes of the menstrual cycle. 4 Fluctuating estrogen and progesterone levels may be implicated, but serotonin, endorphins, androgens and/or other neurotransmitters and hormones can be part of the physiological picture. 8 The menstrual cycle is a transformative cycle, generated by the interplay of secretory sites, the hypothalamus, the anterior pituitary and the ovaries, and of the hormones they produce. 10 Relevant anatomy in a woman's body actually changes throughout the month, characterizing the female reproductive system's mobility

and changeability. 10

PMS-A, PMS-D and PMS-H are linked to elevated levels of PRL in the bloodstream. 11 With improved liver function, hormones will be processed more efficiently, leading to less PMS symptoms – and specifically less PMS-A, D and H resulting from excess PRL. 11 Excess PRL, in addition to stress, inversely affects dopamine levels, leading directly to depression or anxiety. Elevated PRL levels are also indicated in the following PMS categories and symptoms:

- PMS-A : nervousness, tension, irritability, mood changes and anxiety. 10
- PMS-D : depression, forgetfulness, crying, confusion, and insomnia. 10
- PMS-H : fluid retention, weight gain, swelling of extremities, breast tenderness and abdominal bloating. 10

The combination of these female sex hormones secreted into the bloodstream simultaneously demands a healthy metabolic center: the liver. The liver metabolizes female sex hormones that control the menstrual cycle: Follicle stimulating hormone (FSH), Lutenizing hormone (LH), Gonadotropin releasing hormone (GnRH), Estrogen, Progesterone and Inhibin. In PMS, the liver's processing speed can be improved with herbal therapeutics. 9 While menstrual female sex hormones are spiking in the bloodstream PRL is steadily secreted to produce breastmilk.11 When the liver has a low level of function, sex hormones recirculate in the bloodstream instead of being metabolized efficiently. 10 If the tissue's capacity to regenerate new cells is compromised by disease, the body's entire metabolism will be affected. This results in elevated blood serum levels of PRL, inducing hyperprolactinemia. Regardless of our lifestyle, the liver is heavily taxed simply living a daily life in the 21st century which includes 16,000 chemicals which did not even exist one hundred years ago. The liver should be herbally supported to increase optimal performance, especially in the concurrent case of hyperprolactinemia and PMS. 15

Pathophysiology

In PMS dysfunction, treatment should support the return to underlying functional rhythms. Conventional medicine treats menstrual cycle rhythm dysregulation with the birth control pill - . After a two to three month cycle, the pill is withdrawn, upon which time the body seems to “reboot” itself and the medication can be withdrawn after regulation.10 The most widely used birth control pill contains either two hormones: estrogen and progestin, or sometimes just progestin.1 When released in a timely manner throughout the menstrual cycle, progestin will prevent ovulation.1

Just prior to menstruation, levels of progesterone and estrogen, as well as Calcium (Ca), and Magnesium (Mg) – which allows dopamine in the brain – drop. This may influence pain, insomnia, muscle cramping and stress reactions. 9, 6 Dopamine is a Central Nervous System (CNS) Neurotransmitter connected with sensing pleasure. 9 A dopamine deficiency is indicated in PMS, anger and irritability alike.

As hormones drop, so do endorphins, which can result in insomnia, anxiety and irritability. Immune response also drops, leading to flare-ups of chronic issues like herpes. These physiologies are interconnected and linked to an abnormal tissue response to the normal changes of the menstrual cycle 11,10

Further study is needed to determine which factors effect milk output in breastfeeding mothers. 6 Further study is also warranted to determine if breastfeeding mothers with a large output tend more towards the pathologies associated with elevated levels of prolactin in the blood. An elevated state of hyperprolactinemia in the situation of a prolactinoma appears different in the body than elevated levels of prolactin in a breastfeeding woman.7 During lactation, the pituitary down regulates the mRNA gene receptor expression of prolactin receptors, but not in the case of a prolactinoma .7 Therefore, elevated levels of prolactin will continue to circulate in the bloodstream for longer in a breastfeeding mother but not necessarily in a person with a prolactinoma. 7 PRL inhibits the activity of aromatase which plays an important role in biosynthesizing steroid hormones and is present throughout the body, including the liver and fatty tissue, further leading to decreased liver function during high PRL production. 10

Some strategies for decreasing PRL levels can result in decreased milk production, and so should be monitored if a mother intends to continue breastfeeding while using herbs like *Vitex agnus-castus* to deal with pathologies attributed to low levels of dopamine.6 Focus should instead be on supporting the liver to most efficiently process the sex hormones in her bloodstream. Further research is warranted to determine if breastfeeding mothers with high output of breastmilk can utilize strategies for decreasing prolactin levels and still successfully breastfeed with a lower output and possibly reduced hyperprolactenemia. Research does not currently show whether women with high output of breastmilk have high levels of PRL as opposed to women with low breastmilk output.

Therapeutics

PMS-A (anxiety), is characterized by a HOT and dry tissue state, nervous tension, anxiety, irritability, mood changes and insomnia. PMS-A is associated with high estrogen and low progesterone, either from corpus luteum deficiency or the liver's inability to break down excess estrogen/sex hormones. PMS-A also may result from increased CNS stimulation, stimulating effects of estrogen, high environmental exogenous estrogens: animal products/plastics, etc. 10 **PMS-D** (depression), is characterized by a COLD and moist tissue state, depression, forgetfulness, crying, confusion, insomnia, and/or withdrawal. PMS-D is associated with excess progesterone, which causes CNS depression, lowered serotonin. 10 **PMS-H** (hyper hydration) is characterized by a COLD, stagnant tissue state and reveals itself as bloating, breast tenderness, abdominal bloating and weight gain. 10 These three conditions are linked to excess prolactin in the bloodstream. 10

Herbal Therapeutics Goals, Actions and Herbs

Herbally, we aim to address the following goals:

- Correct any hormonal imbalance through the HPA axis.10 **Hormonal normalizers** are used when the endocrinology (not the liver) is indicated in excess hormone levels. *Paeonia lactiflora* contains paeoniflorin, a monoterpene glycoside which contributes to its effectiveness in the treatment of PMS and hyperprolactenemia among other gynecological conditions. 20 A low dose of *Vitex agnus-castus* lowers PRL levels wile improving the secretion of breastmilk as a galactogogue.23 Dosage for *Vitex agnus-castus* is 1:2 or 1:3, 60-75% EtOH, 1 ml TID of fluid extract for three to nine months. 9

- Correct Essential Fatty Acid (EFA) status, responsible for the normal function and development of most tissues including the liver and blood vessels. 8 Mills and Bone suggest a supplement of Evening primrose Oil at 3000 – 4000 mg per day.10 EFA status can also be remedied with Flaxseed Oil, as suggested by Ruth Trickey in Women, Hormones and the Menstrual Cycle, at 1-2 tablespoons/day (T/d), oily fish 12-20 oz/week, or Cod Liver Oil supplements at 1-2 T/d. 8

-Treat the main physical symptoms as they occur. **Circulatory stimulants** like *Zingiber officinale* and *Ginko biloba* aid the body in detoxifying by moving the blood to muscles and joints. *Zingiber officinale* is dosed at 0.7-2 mL daily of 1:2, 1.5-5 ml per day of 1:5.10 In China, Zingiber is mainly used to promote sweating and as an expectorant for colds and chills. Western herbalists also regard it as a good circulatory stimulant. 27 **Diuretics** are used for fluid retention in PMS-H: *Taraxacum officinale* leaf decoction can be drunk to "purify the blood", and for nervousness. 31 *Galium aparine* is traditionally used for

cystitis, indicating it as a diuretic by Michael Howard in. *Traditional Folk Remedies.* 26 Dosage for *Galium aparine* is 3.5-7 ml at 40% EtOH of 1:2 liquid extract daily. *Urtica dioica* is appropriate as tincture or tea. *Urtica dioica* is suggested at 2-6 ml at 40% EtOH of 1:2 liquid extract or 2-3 T/quart infused per day. 28 *Urtica* is energetically dry and may not be appropriate for vatas with PMS-A. **Analgesics** address aches & pains in PMS-H: *Zingiber officinale* contains pungent qualities which produce analgesic effects. 22 *Corydalis cava* is fast and the strongest pain remedy when pain comes from stasis in the abdomen. It is usually paired 4:1 with Eschscholtzia californica, inducing a powerful anti-inflammatory action as well. *Viburnum opulus, or* Crampbark, attends to spasms within 10 minutes. 29 Crampbark, also an antispasmodic nervine, has a liquid extract dosage of 1:2.5, 40% alcohol, 10% glycerin (to get tannins), 3-4 mL TID/QID;favor frequency over larger dose. 29

-Treat emotional disturbances : tonics for depression, sedatives for anxiety.10 **Nervine relaxants** are indicated for anxiety in PMS-A, *Valeriana officinalis,* or Valerian root, referred to as "Nature's Tranquilizer in Penelope Ody's *Complete Guide to Medicinal Herbs,*calms the nerves with chemicals called valepotriates that seem to depress the nervous system, especially potent in a fresh plant extract.27 Valerian is dosed at 2-6 ml of 1:2 liquid extract daily at 60% EtOH to capture the volatile oils.10 *Piper methisticum,* or Kava kava, is known for its kava lactones which have potencies similar to cocaine and procaine as local anasthetics.30 Kava also reputes anti-anxiety effect without sedation or hypnotic effects. 31 Dosage for *Piper methisticum* is 3-6 ml daily of 1:2 liquid extract at 60% with coconut milk fat to extract kava lactones. *Leonorus cardiaca,* or Motherwort, was once described by Mrs. Maude Grieve as "especially valuable in female weakness and disorders, allaying nervous irritability and inducing quiet and passivity of the whole nervous system".32 It is a safe nervine relaxant at doses of 2-4 mL TID 1:2, 50% alcohol, or 1mL as needed for immediate anxiety relief.33

Nervine tonics are indicated for anxiety in PMS-A, depression in PMS-D. A particularly noted formula is *Schisandra chinensis* in combination with *Hypericum perforatum* and *Withania somnifera*. 8 Vitamin B complex may help with PMS-A symptoms relating to stress. 24

-Compensate for adverse effects of stress on the body with adaptogens. 10 **Adaptogens** are indicated to transform the perception of stress and balance the Hypothalamic-Pituitary Axis. *Eleutherococcus senticosus,* Siberian Ginseng, is noted to have similar properties to *Panax Ginseng, both worthy of mention as "the king of tonics".34* Eleuthero will help a person get through the stress, but Ginseng will help them recover

from having been stressed out – on your nerves, vasculature, entire physiology - starting with endocrine system.35 Dosing of Eleuthero is 1:4 at 50% EtOH, 2-8 ml daily.30 *Withania somnifera,* or Ashwagandha, is indicated as a substitution for psychotropic drugs in Premenstrual Dysphoric Disorder (PMDD), a PMS serious mood disorder.8 Ruth Trickey suggests the choice herb *Hypericum perforatum* formulated with *Withania somnifera*, an anxiolytic such as *Lavandula officinalis*, and a hormone-modulating herb such as *Vitex agnus-castus*.8 This combination would also be appropriate especially for PMS-A or PMS-D. *Withania somnifera* is taken in large doses, and is a food like herb. Suggested dose of the fluid extract is 35-90 ml per week of a 1:2 fluid extract at 40% EtOH to preserve the steroidal withanolides, believed to be responsible for many of the adaptogenic qualities.8, 10 *Schisandra chinensis* is the key player in our team of herbs: adaptogen, hepatoprotectant, hepatic anti-inflammatory, anti-oxidant, cholagogue and nervine – it is indicated in every tissue state of hyperprolactinemia concurrent with PMS where the liver is to blame. Dosage is 1-1.5 ml, 1:3 at 60-70% (to preserve lignans) EtOH TID.10

-Balance hepatic metabolism (Stage 1 and Stage 2). **Hepatoprotectant** herbs are indicated to protect the liver from further damage and secondarily efficientize hormone metabolism. The main indications for *Schisandra chinensis* is the primary hepatoprotectant due to its anti-oxidant activity and production of hepatic glutathione levels.18, 19 *Silybum marianum*, Milk Thistle, or isolated Silymarin, also a galactagogue and a cholagogue, protect the liver by producing an anti-oxidant effect on liver tissues and enhancing the metabolism of some drugs (phytochemicals) via enzyme pathways in the liver.12,13 The standard dosage of Milk Thistle is 200 mg 2 to 3 times a day of an extract standardized to contain 70% silymarin.37 *Taraxacum officinale* root can be used generally as a liver tonic by increasing bile production and secretion.14 Tincture the fresh root 1:2, 35%, 5-10 mL TID. 25 Mills and Bone suggest the following formula as hepatoprotectant when a taking the contraceptive pill: *Taraxacum officinale* 1:2, 35 ml, *Silybum marianum* 1:1, 35 ml and *Schisandra chinensis* 1:2, 35ml for a 100 ml formula, dose 5-10 mL TID.10 *Cynara scolymus* works as a cholagogue, choleretic antioxidant and promotes regeneration of the liver cells. 16, 17 Cynara is dosed at 3-8 ml of 1:2 liquid exract per day. 10

-Treat the liver if signs of sluggishness are apparent with bitters, cholagogues, choleretics ; reduce oxidative damage, remove offending substances. **Anti-inflammatory** herbs can decrease the initial site of inflammation in the liver; **Anti-oxidants** improve liver function and avoid further damage. *Schisandra chinensis* and Circumin extract*, from Curcuma longa,* are excellent liver anti-inflammatory herbs with anti-oxidant properties.21 Circumin extract is dosed at 500 mg, 1.755 g/day.36,37 **Cholagogues** release more bile

from the liver into the bloodstream as fat metabolizes enhancing liver metabolism. Bitter qualities of *Schisandra chinensis*, *Taraxacum officinale* Root and *Cynara scolymus*, mentioned also as hepatoprotectants, serve as cholagogues at the same dosage mentioned.8

Because of the individual nature of PMS, conventional treatment is primarily symptomatic and non-pharmacologic. Stress management, counseling, conflict resolution, biofeedback and guided imagery are all recommended. Exercise, supplements and dietary changes are also suggested. If improvement is still needed, medications are prescribed and can include birth control pills, Selective Serotonin Reuptake Inhibitors (SSRIs), Non-steroidal anti-inflammatory drugs (NSAIDs) or progesterone which is clinically shown ineffective for mood and behavior symptoms of PMS/Premenstrual Dysphoria (PMDD), and sertonergic antidepressants. 11, 2

Non-herbal supportive strategies are of utmost important when balancing hyperprolactinemia concurrent with PMS. Exercise increases dehydroepiandrosterone (DHEA), a precursor to female sex hormones, and balances the impacts of cortisol. Cortisol, an anti-inflammatory agent of stress reactions, exits the bloodstream system when one exercises. 10 Otherwise it is involved in a negative feedback loop when in regulation. If cortisol exits the body, the HPA axis will become more sensitive to it, thus the body will not have to produce it in continuous high amounts if it perceives a stressor. As with any sensitization in the body, less of the neurotransmitter or hormone is needed if more receptors are available to receive the signal.9 A woman's awareness of the timing of her cycle could establish compassion for oneself. Beer should be avoided, which further increases prolactin levels.8

In conclusion, an abnormal tissue state is currently the scientific explanation for PMS. From an herbal perspective, tissue state will aid the selection of herbal treatment. When hyperprolactinemia accompanies PMS, elevated prolactin levels can be attributed to physiological conditions if a woman is lactating. High levels of hormones in the bloodstream in addition to the myriad of chemicals in the daily environment are taxing to even a slightly damaged liver. Extreme care is given first to her liver and then to balance hormones if the liver therapeutics are ineffective. Prolactin-inhibiting/ Dopaminergic herbs may be indicated if the woman has high output of breastmilk, without threatening her breastfeeding

relationship with her baby. This theory is currently unsupported by research and deserves a bit of attention. Herbs are the best supportive strategy to improve liver health and subsequently decrease symptoms of physiological hyperprolactinemia concurrent with PMS through efficient hormone metabolism.

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Diabetes Mellitus: Herbal, Nutritional and Life Style Therapeutics

Aisling Badger

Pathophysiology:

Diabetes is a life long illness where there are continual high levels of glucose in the blood. An endocrine system failure can also result when the beta cells produce insulin but the cells are unable to use it properly- leading to insulin resistance(Kumar, et al 1189). Through my research I have discovered that herbs can play a supportive role in decreasing blood sugar levels while increasing insulin production, specifically in type II. A healthy well educated diet is also crucial in treating diabetes, and seems to be one of the leading problems in the development of this disease. Most commonly we see Type I, II and Gestational Diabetes. Type I is an auto immune destruction of the beta cells where over time the pancreas loses the ability to produce insulin, making us dependent on daily injections. The cause is unknown but may be linked to a genetic component, and is often developed at a young age. Type 2 or
" adult onset" is a non insulin dependent diabetes, which is the most common among adults. In type II, the cells are not able to respond to the insulin that is present, causing insulin resistance. As a result blood glucose cannot get into cells to be stored for energy, leading to high levels of sugar in the blood, creating what we know as Hyperglycemia. Insulin resistance can also often become apparent in people 10- 20 years before the onset of diabetes type II (Kumar, et al,1194+).
Gestational Diabetes is high blood sugar levels that develop during pregnancy. It usually disappears after delivery, although the mother becomes more at risk for type 2 diabetes later in life. (University of Maryland Medical Center, alternative medicine index) According to the American Diabetes Association, all women should be screened for diabetes in their third trimester.

The endocrine system is composed of glands that are responsible for secreting hormones directly into the bloodstream. All of the glands can have an inter-relational connection to each other and the diseases that arise from endocrine system disfunction, such as Diabetes(Kumar,1156). Insulin; a hormone secreted by the pancreas, is produced by beta cells to control blood sugar in our body. After eating, our body starts the process of

breaking down sugars and starches into glucose which enters the blood stream and our cells take up as energy. Resistance to insulin accompanied with high levels of sugar in the blood leaves our liver, body fat and muscle cells to inappropriately respond to the insulin that is present(Kumar). If left untreated complications that arise from insulin deficiency and high levels of sugar in the blood, can cause an array of vascular and oxidative diseases especially in the retina, kidney, and blood vessels, thus effecting other organs(University of Maryland Medical Center), Some research has linked an increase of Diabetes to obesity levels, the growing rate of poverty, sedentary life styles and poor diet(Adam Drewnowski,Journal of Clinical Nutrition). Development of diabetes may also stem from genetic heritage, certain infections, and other chronic illnesses(University of Maryland Medical Center). Inflammation is also a key role for those with type II Diabetes. Inflammatory markers such as cortisol induce insulin resistance in the liver. Certain inflammatory markers and stress hormones have been found to be increased in those with type II diabetes(University of Maryland Medical Center). Diabetes effects 16 million people in the US, half of whom have never been diagnosed, Each year 800,000 people in the US develop diabetes and 54,000 die from the complications related to diabetes. It is currently the leading cause of end stage kidney failure, onset blindness, and lower limb amputations(American Association of Diabetes, Statistics).

THERAPEUTICS:

While alongside conventional therapy, there are many herbs that have been well researched as ways to improve insulin resistance, and its production from the pancreas, as well as support the body to lower blood sugar levels.

Bitter melon (*Momordica charantia*) an ayurvedic herb, is widely used for diabetes. It seems to stimulate insulin sensitivity, by increasing the rate that cells bring in sugar and decreasing the rate of sugar production by the liver. Studies have shown that compounds extracted from bitter melon were similar to that of the enzyme AMPK (activated protein kinase)which controls the movement of glucose transporters to the cell surface- important for the uptake of glucose from the blood stream. (Garven Institute of Medical Research, 2008). One study looked at the effect of bitter melon on non insulin dependent, and non diabetic rats and found that the fruit extract significantly reduced blood glucose during the 50 g oral glucose tolerance test. (Leatherdale BA, Et al. 1981)

Gymnema *(Gymnema sylvestre*) is known as “the sugar destroyer”. It interferes with the taste receptors on the tongue so that that taste of sweetness cannot be perceived at the moment. It has been shown in several clinical trials to help reduce blood glucose, blood lipids , body weight and suppress appetite. It showed ability in vitro to stimulate insulin release from the pancreas. This may be a good herb for people who are slightly over-weight with metabolic syndrome, or for those who really need to get past the sugar cravings. (Shanmugasundaram ER, et al, 1990)

Fenugreek (*Trigonella foenum-graecum***)** has been highly researched and shown in several studies to have consistent ability to reduce blood glucose, cholesterol and triglycerides in people with elevated glucose levels. In a 2-month study of 25 people with type 2 diabetes, fenugreek (1 g daily of a standardized extract) significantly improved levels of blood sugar and insulin response while their triglyceride levels decreased and HDL cholesterol levels increased compared to those on the placebo(Gupta A,et al 2001).

Cinnamon *(Cinnamomum zeylanicum)* is used as an effective treatment for those with type II diabetes accompanied with high cholesterol. A study in Pakistan conducted a 40 day study with 60 people who all had type 2 diabetes. Daily they were given doses of either 1, 3 or 6 Gm of powdered cinnamon. The results showed cinnamon to improved blood glucose levels by 18-29 %, and cholesterol levels by 12-26%. They indicated that cinnamon used by folks with diabetes could help reduce the risk factors associated with diabetes(Khan A, et al. 2003). Another trail using 3 g daily also found that cinnamon improved blood sugar levels(Mang B et al. 2006). While another showed that daily HbA1c (a measurement of blood sugar levels over a period of time) levels were reduced when cinnamon was taken daily (Akilen R, et al. 2010).

While certain herbs are used for their effectiveness of controlling blood sugar and insulin, its important to mention the role that **Supportive Herbs** play in a disease like diabetes.

Adaptogens – herbs which help us adapt to stressful situations by normalizing our stress response and the inflammation it caused. They are particularly helpful for blood sugar imbalances because of their effects on cortisol and glucose metabolism. They directly effect our adrenals which are responsible for mediating stress in the body. If our adrenals are deficient then we cannot properly respond to stress. Our blood sugar levels can fall on the low side and leave us hypoglycemic and fatigued, which temporarily can be remedied by sugar to boost energy levels. The adrenals also secrete the hormone nor-epinephrine when

our blood sugar levels fall. By supporting our adrenals, adaptogens can help to normalize balanced blood sugar, maintain our stress response and reduce the symptoms of hypoglycemia (Hoffman 483).

American Ginseng (*Panax quinquefolius*) is supportive to digestion, the absorption of nutrients and good for those who are truly exhausted. In a study performed on American Ginseng, it appeared to improve blood sugar control (Vuksan V, Sievenpiper JL, 2000) while another study found benefits with ginseng and its ability to improve the mood and energy of patients(Sotaniemi EA,et al 1995).

Eleuthero(*Elutherococus senticoscus*) is supportive in the assimilation of nutrients, helpful for improving energy levels, and normalizing immune function. Often given to those who are truly depleted. Eleuthero would be helpful for those with immune impairment and severe fatigue. (Tierra)

Medicinal mushrooms all seem to help with blood sugar balance especially Reishi *(Ganoderma Lucidum)* and Agaricus blazei, or subrufescens. Both offer rejuvenation effects by effecting the liver and making it more sensitive to insulin. They mediate inflammation while providing a rich antioxidant and free radical scavenging effect to the body. Reishi is known for its anti inflammatory calming effect on our Shen, while supporting the body to build its strength. It is a wonderful adaptogen, and useful for long term use (Tierra, 115).

A study on agaricus blazei murill extract improved insulin resistance in type 2 diabetes. The placebo trial used 536 type II patients all of whom had been taking Glcazide and metformin for more than 6 months. Patients were given 1500 mg Algaricus blazei Murill (AMB) extract or the placebo daily for 12 weeks. The Homeostasis model assessment for insulin resistance (HOMA-IR) was used to measure the effects and outcome of the study. After, it was reported that the patients that received the supplement of ABM had a significantly lower HOMA-IR index than those in the placebo group. It was concluded that extract of ABM improves insulin resistance among people with type 2 diabetes. (Hsu CH, et al 2007)

Milk thistle (*Silybum marianum***)** has been used extensively for protecting the liver against various strains of hepatitis, poisoning, cirrhosis, or from liver toxic medications. A small but well conducted study found that milk thistle extract silymarin, could be useful in improving blood sugar levels. One group received a silymarin (200 mg) tablet 3 times along side conventional therapy. The second group received the placebo. The patients were monitored monthly and at the end of the trial the results showed a significant decrease in cholesterol, and LDL levels in group who was given the silymarin. It was reported that

silymarin treatment in type II diabetic patients for has potentail to improve the glycemic index (Huseini HF, Larijani B et al 2006).

Bitters are stimulating to the appetite and support the absorption and assimilation of nutrients, as well as enhancing liver function. Most tend to also have a balancing effect on blood sugar by helping to regulate the secretions of pancreatic hormones (Hoffman, 498).

Dandelion root (Taraxacum officinale)helps to slows transit time which increases insulin. It is a hepatic antioxidant, and is helpful in hypoglycemia, and a wonderful herb especially if the diet it low in soluble fiber (Hoffman, 587).

Schisandra (Schisandra chinensis) has a strong liver effect which also effects sex hormone imbalance. It has been used to regulate blood sugar and improve the digestion of fats. Its sour taste gives it a cholegogue effect that stimulates bile and excretion, which stabilizes imbalances within the body(Tierra, 118).

Nervines are supportive to the nervous system, and are beneficial because of the calming, relaxing effect which can be helpful to reduce anxiety. They are grounding, nourishing and strengthening, and are often used to reduce irritability and mood swings that accompany intense cravings (Hoffman, 517).

Oats (*Avena sativ*a) are rebuilding and deeply regenerative to the nervous system,useful for people who feel depleted and weak. They have been specifically used for people who have anxiety and and exhaustion that is also associated with depression(Hoffman, 532).

Hawthorn (*Crataegus laevigata)* is rich in flavonoids and is a herb for the heart, both emotionally and physically. Its tonic, anti-inflammatory and circulatory effect helps to reduce anxiety and gives a sense of protection and connection to the heart while physically reducing the risk for cardiovascular disease and other heart related problems (Hoffman, 542).

Linden (*Tilia europea)* a relaxing herb that also has an affinity to the heart, has been used specifically for the prevention of atherosclerosis, and high blood pressure; common side effects of diabetes. It has a tonic like action to the nervous system, and combined with hawthorne makes an fantastic circulatory tonic (Hoffman, 589).

Stevia (*Stevia rebaudiana)* could also be helpful for people in diabetes because of its slower absorption time of sugar into the body. It has also been shown to be useful in weight management(Anton SD, et al, 2010).

NUTRITIONAL APPROACHES:

Diet playa a key role in the prevention and management of diabetes. It is important to learn how to **Nourish** ourselves**.** Refined sugar and carbohydrates do not sustain or nourish us in the way our body truly wants. They provide a quick rise and fall of blood sugar, while temporarily relieving the cravings for sugar, or in the case of taste: sweetness. In most traditional systems of healing, sweet tasting foods are considered to be those that are building and nourishing to our bodies. Full sweets would include foods such as whole grains, legumes, nuts and seeds, dairy, fruits, and colorful root vegetables(Pitchford, 189-190).

Paul Pitchford, Author of Healing with Whole Foods says that " the obvious remedy is to consume less of the foods that are stressful to the liver and weaken the spleen and pancreas". This would include foods that greasy and fatty foods; such as red meats, eggs, seeds, excess oils, and foods that have been denatured or refined; sugar, white flours, synthetic fats etc, and excessively sweet, salty or spicy foods.

These foods increase inflammation, hypersensitivity, and an increase of sugar into the diet. Small frequent meals rich in protein and good fat are helpful to stimulate insulin production, while stabilizing and balancing our blood sugar(371-372). Common deficiencies within a diabetics diet should be looked at closely.

Protein is often lacking a person's diet who regularly consumes food rich in sugar and carbohydrates, especially true to those who eat a vegan or vegetarian diet that is not well prepared or balanced. It provides the overall energy that stabilizes our blood sugar levels longer. Providing a protein source at every meal could be especially helpful for those with diabetes(Hass,60).

Fat is crucial because it lubricates our bodies, feeds the nervous system and allows the break down process to happen over a longer period of time thus delaying the absorption of sugar into the blood. Consuming a good oil source such as olive, flax or pasture raised butter will help to stabilize the breakdown and prevent rapid blood sugar spikes. Fat is also a source of energy and can supply the body with a fuel that can be used when needed(Hass, 73).

Fiber which is a naturally occurring source of sugar, provides a steady drip of glucose into the blood stream rather than all at once. Rich fiber containing foods leave less of an impact on blood sugar levels, which can be determined by what we know as the glycemic index of food(Hass, 37-38).

Supportive Strategies:

Exercise is crucial for the management and prevention of diabetes. It improves the bodies use of insulin by lowering blood glucose levels and properly using sugar within our muscles to produce energy. Beneficial in cardiovascular health exercise improves the flow of blood throughout our body and increasing the heart's pumping ability. It helps promote weight loss by supporting the decrease of excess body fat, improving insulin sensitivity and lowering blood pressure. Studies have also shown that people with type 1 diabetes who get regular exercise reduce the need for insulin injections(University of Maryland Medical Center).

Learn to **Relax.** It is important to be aware about the relaxing effects that sugar and the typical american diet has on the brain. The physiological action beyond this is that sugar creates a temporary increase of dopamine and opioid levels, as well as the effect of our seratonin levels. Under stress the adrenals secrete adrenaline, which raises blood sugar levels and sends the body into a "fight or flight" mode. Continual stress results in a steady release of adrenaline which leads to an overstimulation of glucagon to keep blood sugar high. The body tries to counter act this is by pushing the pancreas to secretes more insulin in order to bring the high levels of blood sugar down. Relaxations strategies such as deep breathing, yoga, walks, baths or a hot cup of tea can help reduce anxiety, and tension and increase circulation of the blood(Thomas Cowan, Weston A Price Foundation).

It is important to recognize that over the years within our society, cases of diabetes have been rapidly increasing. This is partially due to the fact that as we have evolved, sugar has become more readily available to us, and partially due to the lack of education on how to nourish and care for our bodies. Perhaps there is a lack of purpose or desire to care for ourselves, or nourishment missing on a deeper level that makes us live the way we do. While the search for a cure to diabetes continues along all medical fronts, research proves that by educating ourselves on ways to live healthier lives, we may be able to support and manage a disease that can be so life threatening.

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Understanding the Diagnosis and Treatment for Generalized Anxiety Disorder from an Integrative Perspective

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Anxiety is a normal and even beneficial emotion to experience at times. It is part of our hard wiring for safety and survival, giving us the internal cue to gear up for "fight or flight" when sensing potential harm or dis-ease. Anxiety from health issues, financial concerns or troubled relationships are ordinary responses to stressful situations. When these worrisome emotions become excessive, unrealistic, persistent, and interfere with normal daily activities, this may be a form of mental illness (ADAA). Anxiety disorders are pathological presentations of arousal, tenseness, and increased autonomic activity, such as heart rate, blood pressure and respiration (Bunce, APA). For people with anxiety disorders, the constant and overwhelming worry can be crippling. This paper will engage in a discussion about the causes of anxiety and the current modalities of therapy, with a specific focus on generalized anxiety disorder.

Understanding Anxiety Disorders:

Anxiety disorders are the most common psychiatric illness in the United States, with each year an estimated 18% (40 million) adults in America affected and 8% of teenagers (NIMH "Anxiety disorder"). There are several categories of anxiety disorders, including panic disorder (PD), obsessive-compulsive disorder (OCD), post-traumatic stress disorder (PTSD), social anxieties, specific phobias, and general anxiety disorder (GAD).

Although the varieties of anxiety disorders each specify unique characteristics, all

have common feelings of excessive and irrational fears and dread (NIMH “Anxiety disorder”).

The overwhelming percentage of Americans with anxiety disorders is all together heartbreaking and concerning. If the evolutionary purpose of anxiety is to foreshadow danger ahead, what does this say about the population of our country? Anxiety is not only a serious psychological disorder; it is also a ‘canary in the coal-mine’ for underlying health concerns. Compared to the general population, clients with anxiety disorders are more likely to develop a medical illness (Rogers), as well as prolong the duration of a medical illness (Shader). Conversely, patients with a chronic medical illness are more likely to be diagnosed with an anxiety disorder (Wells). Certain medical conditions have higher comorbidity with anxiety, including Grave’s disease, anorexia nervosa, Alzheimer’s disease, mitral valve prolapse, heart arrhythmias, hypertension, chronic obstructive pulmonary disease, irritable bowel syndrome, and diabetes (NIMH “Generalized anxiety disorder (GAD),” Winston, Masé). Overall, patients with anxiety disorders have higher rates of mortality from all causes (Gliatto). Although there have been incredible advances in western medicine’s understanding of the body and brain chemistry, it appears the deeper researchers look, the more difficult it is to separate what is an emotional or physical ailment (Winston).

Tendency towards chronic anxiety may be a behavior that is learned, inherited, triggered or chemically imbalanced (Faustino, “Natural medicines database”). From an integrative perspective, it is important to understand the root cause of the disorder in order to effectively treat the person. Much like depression, growing up in a household with an anxious parent increases the chances of an anxious child (Baldwin, Sarris and Panossian, Winston). There is also evidence that a genetic component is at hand (Kendler), making the idea of coming from “a long line of worriers” much more genetically plausible! Emotional triggers may induce anxiety for any number of reasons, such as with depression, trauma, illness, financial concerns, family issues, abuse, divorce, and other major life changes. Chemical triggers that may exacerbate anxiety are long-term alcohol (Carguilo), nicotine (Morisette), and caffeine use (Bruce, Wise); as well as medications like benzodiazepines (Ashton),

steroids, over the counter sympathomimetics, selective serotonin reuptake inhibitors (SSRIs), digoxin, thyroxine, theophylline, and antihistamines ("Natural medicines database", Wise). Environmental triggers may also be an overlooked cause for increasing anxiety in the US and around the world (Bunce, Winston). Life today may not have the same primitive fears of survival it once had, but it may be argued that it is more stressful, complicated and confusing than ever before. The social expectation for the standard of a "happy" life has become nearly unachievable. As a culture, we have shifted away from being a reflection of nature and rocketed into a technological craze of consumerism and perpetual dissatisfaction. When expectations of security are unrealistic and excessive, it is no wonder that anxiety will follow this same pattern.

The pathophysiology of anxiety disorders is still being unraveled, although current evidence hypothesizes some degree of imbalance of serotonin, noradrenaline, glutamine, and GABA neurotransmitter levels and transmissions (Sarris and Panossian). Neurotransmitters are an infinitely complicated orchestra of chemical messengers that help move information from nerve cell to nerve cell, and without the proper levels, messages cannot be communicated properly. This break in communication will then alter the brain's reaction and initiating stress responses (NIMH "Anxiety disorder"). The understanding of the "neurotransmitter imbalance" theory is based on the observable mood improvements that occur when taking selective serotonin reuptake inhibitors (SSRIs), selective serotonin and noradrenalin reuptake inhibitors (SNRIs), and benzodiazepines (Mukherjee, Sarris and Panossian).

Whether the stressors stems from physical or emotional root, the body is equipped to respond in the same defense through the Hypothalamus-Pituitary-Adrenal axis (HPA axis) (Bunce). The HPA axis is a major part of the neuroendocrine system that controls reactions to stress and regulates many body processes, including mood and emotions, digestion, immune system, sexuality and energy storage / expenditure (Bunce, Pariante). HPA exhaustions involves the suppression of dehydroepiandrosterone (DHEA), testosterone and estrogen synthesis, all hormones which work to improve mood (Bunce). Low estrogen is associated with decreased serotonin production, while progesterone acts on GABA receptors, showing correlation between both sex hormones deficiencies and increased anxiety (Antonijevic, Bunce). This is also evident pre-menstrually and peri- menopause,

when estrogen levels decline and there is a distinct change in mood, specifically anxiety (Antonijevic, Bunce).

Generalized Anxiety Disorder:

Many anxieties can remain on-going, debilitating and "beyond the control" of the client, ultimately adversely affect their daily life (APA). The diagnosis can be difficult since many anxiety disorders share common symptoms, but for now I will highlight the most common diagnosed anxiety, Generalized Anxiety Disorder (GAD). GAD affects 3.1% of US adult population (6.8 million US adults) and twice as many women as men (NIMH "Anxiety disorder"). There has been some refining of the definition of GAD over time, as originally there was little distinction between panic disorder and GAD. Panic disorders are now better understood to be intense, brief, acute anxiety, with variable periods of remission and relapses ("Natural medicine database"). GAD on the other hand, is not associated with these intense physical attacks, but as defined in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) it is a "chronic state of apprehensive expectation and uncontrollable worry concerning multiple daily life events or activities and accompanied common manifestations of psychic or motor tension for more than half the time of at least 6 months" (APA, Boulenger).

Symptoms and behaviors associated with GAD fall under 3 categories

(APA): (1) Excessive physiological arousal

Muscle tension, irritability, fatigue, restlessness, insomnia

(2) Distorted cognitive processes:

Poor concentration, unrealistic assessment of problems, worries

(3) Poor coping strategies:

Avoidance, procrastination, poor problem-solving skills

Understanding the client's medical and mental history is the first step to an initial assessment, as well as gathering an understanding of family relationships, career, spiritual connection, acute and chronic stressors, and somatic ailments (Bunce,

Winston). Client's symptoms can present in a wide range and degree of severity. Many complaints may be the result of anxiety without the client aware of their connection, just as other seemingly unrelated factors may be triggering anxiety (Schweizer). It is important to differentiate the anxiety between "Acute" (brief or intermittent episodes lasting hours to weeks, often preceded by stressors) and "Chronic" (persistent or unremitting lasting months to years, can even be seen as a personality trait) in order to better understand if the anxiety tends more towards panic attacks or depression (Schweizer). This will help differentiate the most effective therapeutic path to recommend.

There is also a well established trifecta between anxiety, depression and sleep disorders, with nearly 50% of adults with depression also diagnosed with an anxiety disorder (Gliatto), 65-90% of adults with depression experience a sleep disorder, and 50% of adults with sleep disorders experience generalized anxiety ("Sleep and mental health").

Although this connection and intertwining of ailments is not the focus of this paper, I feel it is critical to point out how closely symptoms of imbalance will perpetuate each other, and therefore how important it is to address sleep, depression, anxiety, and underlying health concerns together.

Therapeutics for Generalized Anxiety Disorder:

Psychological Therapies -

Non-pharmacologic modalities should be the first line intervention for clients with GAD symptoms and behaviors. There are a variety of therapy options, each focusing on different ways to discover what anxiety triggers and how to lessen and even reset them. Psychotherapy involves talking with a trained mental health professional to discover different ways of thinking, behaving and reacting to situations (NIMH "Generalized anxiety disorder (GAD)"). Some of its methods include relaxing and breathing techniques, and finding new ways to support a balance in the patient's life. Psychotherapy practitioners listen and offer objective feedback, while helping clients examine stressors in life and find better ways of coping or eliminating them (Grohol).

Cognitive-behavioral therapy (CBT) works with people to change thinking and behavioral patterns when reacting to anxiety-provoking situations (NIHM “Anxiety disorder”). Through psych education, relaxation training, cognitive restructuring and behavioral aspects, fears can be confronted and desensitized (Sarris and Moylan). Mindfulness-based cognitive therapy is clinically effective at relieving anxiety and depressive symptoms in clients with generalized anxiety disorder (Kim), although only when the client is ready to confront their fears (Sarris and Moylan).

Pharmacological medication:

For clients whose anxiety is significantly impairing their daily function and quality of life, pharmaceutical medications are very often prescribed. However, it is important to note these medications do not cure or address the root of the anxiety, they simply control the symptoms (Andreatini and Lacerda). Starting with the very first dose, pharmacological drugs work by altering brain chemistry, although full effect requires a series of changes to occur which sometimes takes several weeks (NIMH “Anxiety disorder”). A variety of drugs have proven effective in generalized anxiety disorder management, although each drug has its benefits and drawbacks that need to be carefully considered for each individual (Andreatini and Lacerda, Faustino). There are many cases that prescription medication is specific and warranted, such as anxiety that is unresponsive to therapy, herbal, dietary and lifestyle modifications or for severe disorders while other therapeutic support is in progress. However, prescription drugs are most often the primary action for addressing anxiety, with 11% of middle-aged women and 5.7% of middle-aged men using anti-anxiety medications, and 11% of the entire US on anti- depressants (Pratt).

Antidepressants

Antidepressants were developed to treat depression but are also effective for anxiety disorders, although generally take 4 to 6 weeks before taking full effect (NIHM “Anxiety disorder”). Antidepressents such as venlafaxine (Effexor®), paroxetine (Paxil®) and imipramine (Tofranil®), have a high incidence of non-adherence to treatment (NIMH “Anxiety disorder”), and side effects of cholinergic symptoms, sexual dysfunction, insomnia, and withdrawal issues (Adreatini and Lacerda, Sarris

and Panossian)

SSRIs

Some of the newest antidepressents are called selective serotonin reuptake inhibitors, or SSRIs (Faustino). These work to alter levels of serotonin in the brain, which, like other neurotransmitters, help brain cells communicate with one eachother (Faustino). SSRIs must be started at low doses and gradually increase until they reach the beneficial effect over several weeks (Sarris and Moylan). Popular SSRIs to anxiety disorders are fluoxetine (Prozac®), sertraline (Zoloft®), escitalopram (Lexapro®), paroxetine (Paxil®), and citalopram (Celexa®). Venlafaxine (Effexor®) is closely related to SSRIs and is often used to treat GAD (NIMH "Anxiety disorder"). SSRIs have fewer side effects than older antidepressants, but still can cause an initial increase in anxiety in early stages (problematic for patient compliance), nausea, headaches, sleep difficulties or sexual dysfunction in over 50% of users in the long term (Sarris and Moylan).

Anti-Anxiety Drugs

High potent benzodiazepines are the most commonly prescribed anxiolytic and act on gamma-amino-butyrc-acid (GABA) / Benzodiazepine receptors (NIMH "Anxiety disorder"). They have established efficacy for quick relief of many anxiety disorders but do not actually decrease worrying (Sarris and Moylan). They act to lower anxiety by decreasing vigilance and by eliminating somatic symptoms (ex. Muscle tension). Some popular benzodiazepines are diazepam (Valium®) for anxiety, clonazepam (Klonopin®) for social phobia and GAD, lorazepam (Activan®) for panic disorder, and alprazolam (Xanax®) for panic disorder and GAD (NIMH "Anxiety disorder"). Benzodiazepines are quick acting, but not without limitations and side effects. They are not suitable for long term because of concerns of dependency and tolerance development. Benzodiazepines risk sedation, amnesia, potential abuse and/or dependency, withdrawal syndrome, and possible long-term cognitive effects from interactions with depressants of the central nervous system (Andreatini and Lacerdo, Faustino, Sarris and Moylan, Shader).

Buspirone (Buspar®)

Buspirone is a newer anti-anxiety medication for GAD (NIMH “Anxiety disorder”). It is similar in the mechanism of action to a benzodiazepine, but take at least 2 week for effectiveness and without the concern for tolerance and dependency. Possible side effects include dizziness, headaches, and nausea (NIMH “Anxiety disorder”). Also, despite the potential interest of many new pharmacological treatments of GAD, recent years have shown that the development of new anxiolytic drugs often appear limited by high-rates of placebo response in numerous clinical trials (Boulenger).

Herbal Medicine:

In the human body, there are infinite molecular processes involved in the stress response mediated by the central nervous system (CNS). Many of these compounds are active against a wide range of targets, and may cause numerous effects and changes (Sarris and Panossian). Considering the complexity of mental disorders, the modulation of a single neurotransmitter target may not necessarily treat the patient as successfully as approaching multiple targets of the neuro/endocrine systems (Sarris and Panossian,). Supporting this theory is the ever-increasing validity of traditional herbal medicine to treat anxiety (Sarris and Panossian, Ernst 2007, Faustino). Unlike synthetic drugs made in a laboratory, plants are influenced by a phytochemical profile that is as different as the soil it was grown in, resulting in overall biological effects that rely on synergistic interactions between plant constituents (Faustino, Kennedy and Wightman, Sarris and Panossian). Furthermore, anxiety disorders are more both under-treated and over prescribed, motivating patients of all kinds to seek non-conventional treatment (Sarris and Moylan). In a recent US cross sectional and longitudinal survey (2012), 43% of individuals diagnosed with an anxiety disorder use a variety of complementary therapies (Sarris and Moylan). With the rising cost of prescription medications and their unwanted side effects, patients are exploring herbal and other natural remedies (Lakhan).

The main goal for supporting a client with GAD is to help reduce their perception of stress. This may be regulated through supporting the HPA axis, the CNS function via neurotransmitters, and sometimes sedating or supporting cognition function (Bunce, Faustino). Secondary goals may be to improve digestion and nourishment

since the mind-gut connection is so tightly connected, and address inflammation exacerbated by chronic stressors (Bunce). When supporting someone with GAD, the herbal actions may include antidepressant, anxiolytic (relaxing nervine), adaptogen, bitters digestives, nootropic (cognitive enhancing), sedative, hypnotic, anti-inflammatory, and analgesic effects (Bunce).

Herbal medicines work in similar mechanisms as pharmacological drugs, which makes sense since it is estimated that 25% of all drugs on the market today contain compounds that are directly or indirectly derived from plants (Faustino, Koehn). Some plants modulate anxiety disorders through the modulation of neuronal communication and through the alteration of neurotransmitter synthesis (Sarris and Panossian). Anxiolytic herbs may have effects on the GABA system, either via inducing ionic channel transmission by voltage-gated blockage, through alterations of membrane structures, GABA transaminase or glutamic acid decarboxylase inhibition, or less commonly via biding with benzodiazepine receptor sites (e.g. GABA-A) (Sarris and Panossian). Other actions may involve stimulating or sedating CNS activity, and regulating or supporting the healthy function of the endocrine system and HPA-axis (Gliatto).

A comprehensive review of plant-based medicines that have clinical evidence of anxiolytic activity (as of 2012) revealed 21 human clinical trials (Faustino). Efficacy was found for several herbs for treating a range of anxiety disorders (Sarris and McIntyre). Specifically for reducing generalized anxiety with herbal preparations, the most promising evidence supports the use of Kava (*Piper methysticum*) (Ernst and Pittler, Sarris and Laporte). Additional research points towards a beneficial effect from Ginkgo (*Ginkgo biloba*) (Woelk), Passion flower (*Passiflora incarnata*) (Akhondzadeh, Aslanargun, Movafegh), Chamomile (*Matricara recutita*) (Amsterdam, Wong), Scullcap (*Scutellaria lateriflora*) (Wolfson), Lemon balm (*Melissa officinalis*) (Kennedy and Scholey, Kennedy and Little), Bacopa (*Bacopa monniera*) (Pase), Rhodiola (*Rhodiola rosea*) (Bystritsky), Hawthorne (*Crataegus oxyacantha*) (Hanus), California poppy (*Eschscholtzia californica*) (Hanus), and Ashwagandha (*Withania somnifera*) (Cooley) (Sarris and Panossian). There is currently little evidence supporting the use of St. John's Wort (*Hypericum perforatum*) for anxiety disorders, while there is strong evidence for its use in depression (Sarris and Panossian, Schüle, Singer). Many of these anxiolytic herbs have the potential for additional applications to support

secondary goals often paired with anxiety, such as improving mood (Chamomile, Kava, Lemonbalm, St. John's Wort), support for insomnia (Passion flower, Scullcap), enhancing cognition via nootropic activities (Bacopa, Ginkgo), and adaptogenic tonics to combat chronic stress (Ashwagandha) (Sarris and Panossian, Mills).

Diet and Lifestyle:

The connection of diet and physical activity to mood regulation is clearly linked (Bunce). There is much research in this area of study, but for brevity I will just skim the surface.

To start, anxiety levels are greatly decreased by walking for 60 minutes, or running 20-30 minutes, for at least four days per week (Gliatto). Other modalities of exercise that show beneficial results in modulating stress and anxiety are mindfulness, yoga and tai chi (Sarris and Moylan). Diet and nutrition are gaining evidence everyday about their close relationship with anxiety and mental disorders. With strong evidence for the prevention and treatment of psych disorders with Omega-3 fatty acids, which have shown specific support in mood disorders and depression (Freeman).

Conclusion:

It is clear that anxiety disorders are a destructive pandemic that is affecting nearly 1 in 5 adults in the US. Anxiety is too easily becoming a way of life and accepted state of mind. Without fully understanding where these mood disorders are stemming from, they will continue to perpetuate a blurring memory of how it feels to be truly content.

Prescription drugs may be effective at masking the symptoms, but not without the cost to health and our right for pure, non-medicated happiness. The topic is not whether synthetic drugs or natural methods are better, as this is as complicated as the individuals and compounds in question, but simply that complimentary therapies can support each other while the root of the anxiety disorders are being addressed. Through herbs, nutrition, lifestyle, therapy, and pharmacological medications, there is great potential to increase the efficacy of not simply repressing the symptoms of anxiety, but serving to better understand and overcome them.

"Worry is a thin stream of fear trickling through the mind. If encouraged, it cuts a channel into which all other thoughts are drained."

— Arthur Somers Roche, American journalist, writer, 1883-1935.

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Kudzu in alcohol abuse

Beracah Sullivan

I grew up in a home where drugs and alcohol weren't consumed but were discussed a great deal. My mother is a recovering alcoholic and my stepfather is a recovering drug addict. My stepfather ran a drug treatment center for many years before he went on to work for NY State to put together policies, procedures and protocol for drug treatment facilities. There are as many factors that go into the development of ones dependency and addiction as there are in treating it. Often addicts are given prescription drugs in order to help them through withdrawal symptoms and detox from alcohol and narcotics. Some of which can be just as addictive as the substances they are looking to stop using. Though we haven't formally touched on the subject herbal medicine to help break the pattern of addiction yet in our studies, there have been brief mentions of it here and there during class discussion. What plants have been traditional used in the treatment of alcohol and drug addiction and in what ways can they assist in breaking the cycle of dependency.

I was able to find more abstract then full text studies on Kudzu (Pueraria lobata), a Chinese Medicinal herb that has a long history of being used to treat alcohol addiction. A study published last year involving 17 men ages 21-33 years old, looking to reduce their alcohol consumption in a non-treatment environment. For a period of 8 weeks, (2 weeks to set a base line, 4 weeks of treatment and then 2 weeks follow) the participants were given a standardized Kudzu extract of 250 mg isoflavones t.i.d or a matched placebo on a daily basis. They reported their alcohol consumption and desire to use alcohol via a wrist actigraphy device and had laboratory visits twice a week so that medication adherence could be monitored as well as any adverse effects. The study found that patient compliance was very good and that there were no adverse changes to vital signs, blood chemistry and renal or liver function. While the patients reported no change in their desire and craving for alcohol, they did see a reduction in the amount of alcohol drinks consumed within the week by about 34-57%, there was a reduction in the amount of heavy drinking days and there

were longer periods of abstaining from alcohol consumption (1). While this particular study mentioned the fact that they used isflavones of the Kudzu plant they didn't specify which ones specifically or if it was a combination of a few that were given.

The next abstract I found identified the isoflavones present in Kudzu that are believed to be responsible for the reduction of alcohol consumption in animals and humans, daidzin, daidzein and puerarin, though it admitted that it was unsure as to how exactly it suppressed alcohol consumption. It speculated that extracts of Kudzu may work by antagonizing the opioid receptors. This is similar to the action that naltrexone has on those same opioid receptors (2).

The one full text study I was able to find focused on the kudzu isoflavone puerarin as the constituent that reduces alcohol consumption. The authors weren't completely clear as to how or why puerarin worked with in the body, but the suggested that it may be due to it's ability to alter ALDH2 or that it might be working on the monoamine oxidise-acetaldehyde pathways (4). 10 adults participant who had reported having an average of 9-17 drinks per week took 2 capsules of 300mg of purified puerarin two times a day. There was a placebo week that was administered in order to create a base line and then participants were a placed in a relatively realistic setting in order to track and observe their drinking. The study found that the participants had a reduction in the amount of beer they consumed, the time it took and the number of sips that it took to finish those beers where increased. Daily diaries showed that participants drank slightly less during the treatment week vs the placebo week. While it is a pilot study, the results seem to suggest that puerarin and kudzu root could help in the reduction of alcohol consumption as well as alter their drinking patterns (3).

While I was a little disappointed that I couldn't find any studies about other herbs/herb combinations that I have seen or heard mentioned in class discussions, it is good to know that there are some studies on the topic. I think that realistically a single isoflavonoid, or a single herb for that matter, isn't going to be the end to alcohol abuse, but I think that it should be seriously considered as part of the creation of a support system that is built in around people who are looking to start making healthier choices for themselves and their lives. Because addiction is such a layered issues I believe plants, with their multiple constituents and secondary metabolites and layers of ways in which they can reach us and heal us, could be really key in helping to support and nourish people through the process of getting and staying clean.

1. A standardized **kudzu** extract (NPI-031) reduces alcohol consumption in nontreatment-seeking male heavy drinkers.
Lukas SE, Penetar D, Su Z, Geaghan T, Maywalt M, Tracy M, Rodolico J, Palmer C, Ma Z, Lee DY.
Psychopharmacology (Berl). 2013 Mar;226(1):65-73. doi: 10.1007/s00213-012-2884-9. Epub 2012 Oct 16.

2. [Medicinal plants in the phytotherapy of alcohol or nicotine addiction. Implication for plants in vitro cultures].
Ozarowski M, Mikołajczak PŁ, Thiem B.
Przegl Lek. 2013;70(10):869-74. Polish.

3) The isoflavone puerarin reduces alcohol intake in heavy drinkers: a pilot study.
Penetar DM, Toto LH, Farmer SL, Lee DY, Ma Z, Liu Y, Lukas SE.
Drug Alcohol Depend. 2012 Nov 1;126(1-2):251-6. doi: 10.1016/j.drugalcdep.2012.04.012. Epub 2012 May 10.

4) Keung WM. Preclinical Studies of Kudzu (Pueraria lobata) as a Treatment for Alcohol Abuse. In: Keung WM, editor. Pueraria: The genus Pueraria. Taylor & Francis; New York: 2002. pp. 144–158

This is the info regarding the study that I had mentioned in the class discussion about Declinol that I passed on using because I found follow up articles regarding a federal lawsuit against them.

Declinol, a Complex Containing **Kudzu**, Bitter Herbs (Gentian, Tangerine Peel) and Bupleurum, Significantly Reduced Alcohol Use Disorders Identification Test (AUDIT) Scores in Moderate to Heavy Drinkers: A Pilot Study.
Kushner S, Han D, Oscar-Berman M, William Downs B, Madigan MA, Giordano J, Beley T, Jones S, Barh D, Simpatico T, Dushaj K, Lohmann R, Braverman ER, Schoenthaler S, Ellison D, Blum K.
J Addict Res Ther. 2013 Jul 2;4(3). pii: 153.

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3835486/

Interestingly enough, If I had taken the time to read the whole study I would have seen that it incorporated some of my original questions about bitter herbs and alcohol consumption.
Lesson learned here- read the study all the way through!

In addition, when I went back and read more into the link about Declinol, the lawsuit was more of a trademark and copyright infringement.

http://www.prweb.com/releases/declinol/10/prweb11269894.htm

Here is an editorial article that addresses some of the concerns I've heard within the drug treatment community about the fine line that treatment facilities must walk in transitioning patients from one addiction to possibly another with a pharmaceutical.

http://www.psychologytoday.com/blog/ending-addiction-good/201304/facing-the-root-causes-addiction

I think that this article out lines some key points that play into what I was going for in my research project. It is important to keep in mind that 1) herbalism should be considered as a partner in the transition of Detox and that in many cases medical supervision is a must. 2) Taking herbs without some other kind of support system to help a patient address the root issues of alcohol and drug abuse is probably going to lead to a false sense of security, which may eventually lead to relapse.

Pathophysiology and Therapeutics of Post-Myocardial Infarction Ventricular Remodeling

Steve Byers

INTRODUCTION:

Cardiac remodeling refers to changes that take place in the architectural shape, size, and physiological function of the heart, after an injury to the myocardium. There are many complex mechanical and biochemical processes that result in cardiac remodeling and many different diseases or dysfunctions (such as in the heart valves), which may lead to remodeling of the myocardium. Generally, most of the remodeling takes place in the left ventricle in post-myocardial infarction (MI) patients. Yet globally, the heart is vulnerable to remodeling in all areas, especially where there has been damage. The focus of this paper will be primarily on post-MI ventricular remodeling.

Mild cardiac remodeling is a “normal process” that occurs throughout our lifetime as a result of normal aging.[i] Various researchers apart of Framingham study have found that the most common form of remodeling is of the left ventricle (LV) which occurs concentrically where the LV thickness slowly increases and the LV cavity decreases in size when observing preserved or increasing ejection fractions.[ii] LV thickness occurs more predominantly in women, especially older women, than men across all ages, which is perhaps due to reduced cardiomyocyte dropout and slightly higher cardiomyocyte turnover displayed in aging women.[iii] Despite this evidence, there is still no clear correlation between age-related concentric thickening and heart disease. [iv] That said, considering the effects of obesity, hypertension, and diabetes, LV wall thickness does increases yet LV cavity size does not decrease proportionately with age which is perhaps indicative of an inability to compensate for increased wall stress from increased blood pressure. [v] Other factors such as family history with heart disease has become a predictor of increased risk of developing eccentric LV geometry in offspring who do not have heart failure.[vi]

MYOCARDIAL INFARCTIONS

According to recent Center for Disease Control statistics[vii], every 1 in 4 deaths in America are caused by heart disease. Of those 600,000 deaths, 385,000 deaths are from coronary heart disease. On average, 715,000 Americans have a heart attack every year and of those, 525,000 are a first heart attack (about 75% of those people). It is estimated that coronary heart disease expenses (health care services, medications, and lost productivity) collectively cost $108.9 billion each year. Identifying and supporting areas of prevention is critical to reducing the prevalence of this epidemic in America. Risk factors for having a MI include diabetes mellitus, hyperlipidemia, smoking, obesity, hypertension, male gender (higher incidence in men of all ages but difference between men and women narrows with age), and family history.[viii]

A myocardial infarction occurs when a coronary artery is occluded beyond the critical point that its normal cellular function and homeostasis are maintained. When a thrombus forms on an ulcerated or unstable atherosclerotic plaque, coronary arteries can be occluded beyond their critical threshold (>75%) to move blood.[ix] A thrombus large enough will occlude the artery and cause a MI resulting from ischemia of the myocardial tissue and eventually necrosis of myocardial tissue down stream from the thrombus. This occlusion causes a decrease in oxygen, glucose, and nutrients to the myocardium through the coronary arteries and leads to ischemia of those cells.

A MI can also be triggered by increased myocardial metabolic demand where the heart is unable to compensate for the demand (i.e. the out of shape man with hypertension shoveling the driveway after a big snow storm and has a heart attack). Other forms of increased myocardial metabolic demand include severe hypertension, severe aortic stenosis, cardiac valvular pathologies, and low cardiac output states associated with a decreased mean aortic pressure necessary for coronary perfusion.[x]

The more the artery is occluded and the longer amount of time that it is occluded, will contribute to increased amounts of ischemic damage. Two types of ischemia have been defined for MIs. Ischemia that spreads fully through the heart muscle from the endocardium through the myocardium to the epicardium is called a transmural MI. If only partial muscle death occurs through the muscle, it is referred to as a sub-endocardial or non-transmural MI. Emergency medical services often use a protocol of thromolytic medications referred to as “clot busters” and now even more commonly use catheterization with a metal stent in order to clear the blockage as soon as possible. Stents did not exist until 1978, when the death rate of myocardial infarction patients was 25% and now after 35 years of improved medical protocols, that number has dropped to 5% in hospitals with catheterization labs.[xi]

The sooner the clot is removed, the less ischemic damage will occur and the better the outcome of survival and quality of life post-MI.

Another consequence of MI damage is the repair process (discussed more below) involving fibrosis, which is initially integral in providing structural integrity with scar tissue in the areas of necrosis of myocardial tissue. Yet in the long run, post-MI fibrosis becomes one of the causes of continuous LV remodeling leading to progressive ventricular dilation, diminished cardiac performance, and ultimately chronic heart failure.[xii] Research has shown that it is possible to predict morbidity and mortality based on the extent of remodeling.[xiii]

PHASES OF LEFT VENTRICULAR REMODELING

Three phases of adverse left ventricular (LV) post-infarction remodeling have been defined to outline the physiological effects of the infarction and the proceeding myocardial remodeling. Vanhouette et al. (2006)[xiv] describes the phases in detail which will be the main reference for the following phases described below unless otherwise noted (see Figure 1 for a overall schematic of the pathophysiology of ventricular remodeling):

Phase 1- Early Wound Healing Phase (0-7 days post-MI):

The death of myocytes induces a cascade of mechanical and biochemical responses that help balance cardiac output as well as initiate necrotic myocyte clean up and compensation for fewer functioning myocytes. Reduced cardiac output leads to activation of the Renin Angiotensin Aldosterone System (RAAS) as well as signaling of the adrenal medulla and sympathetic nerve terminals to release Norepinephrine (NE) and to block its reuptake. Both responses to reduced cardiac output cause vasodilation and signal the release of Angiotensin II (ANG2). NE and ANG2 stimulate endothelin (ET-1), which is a stimulus for myocyete hypertrophy and signals the secretion of Ventricullar Natriuretic Peptide (VNP) and Atrial Natriuretic Peptide (ANP), an inhibitor of catecholamine, ANG2, ET-1, and aldosterone. Both ANP and VNP are powerful vasodilators, and are released in the atrium and ventricles, respectively, in response to increased ventricular stretch. They both increase cardiac output by reducing systemic blood volume through naturesis and therefore reduce blood pressure and also reduce vascular resistance.

During this phase, depending on the severity of the ischemia, the infarction zone expands throughout the heart tissue as a result of serine protease degradation of intermyocete collagen struts and neutrophil release of metalloproteinases (MMPs).[xv] An imbalance between MMPs and Tissue Inhibitor of metalloproeinases (TIMPs), allows MMPs to degrade the extra-cellular matrix (ECM) surrounding the myocardial tissue by signalling

inflammatory cytokines, neutrophils, macrophages, growth factors, and angiogenic factors to clean up and remove necrotic myocytes as well as initiate tissue regrowth. This is the beginning of a process that leads to the laying down of collagen and hypertrophy.

Within hours of infarction, myocardium wall thinning and ventricular dilation results and leads to an insidious cycle of higher blood pressure which places more stress on the walls of the LV. In response to an increase in macrophages, fibroblasts, and wall stretch, ANG2 and ET-1 are released and initiate hypertrophy of the cardiomyocytes.[xvi] Non-infarcted myocardial tissue also takes on the burden of extra work to keep the heart functioning normally and becomes stretched, consequentially adding to further hypertrophy of myocytes.

Phase 2 - Granulation and Early Remodeling Phase (7-21 days post-MI):

This phase is largely distinguished by a process of removing the necrotic tissue and initial rebuilding of tissue. Macrophages continue to phagocytose necrotic myocetes while myofibroblasts increase activity as they proliferate into the infracted area. A mixture of collagens, proteoglycans, and matricellular proteins (such as osteopontin, thrombospondin-1 and -2, and fibronectin) is deposited by fibroblasts to make granulation tissue. Dead myocardial tissue, which normally looks like thin, strait columns under a microscope when it is healthy, is replaced by scar tissue that is much wider and disorganized in comparison. Later, granulation cells apoptose and are replaced by new collagen laid down by myofibroblasts who leave a thin hypocellular scar. Also during this phase, the non-infarcted myocetes experience hypertrophy from stretch and an expanding infarction zone. Several animals studies measuring MMP and TIMP levels during this phase show a rising and falling of levels of various MMPs as well as a rise in TIMP levels but not until day 14. This evidence demonstrates a heavy signaling of MMPs during this phase, which is responsible for increased LV remodeling.

Phase 3 - Late Remodeling Phase (21 days post-MI):

LV regional remodeling takes place for weeks, months, and years after a MI. Animal studies suggest a continually fluctuating level of MMPs and TIMPs, which contribute to the process of chronic inflammation and collagen formation leading to chronic LV remodeling.

BIOMARKERS OF HEART HEALTH

Several biomarkers have been identified in association with LV hypertrophy. C-reactive proteins, biomarkers of inflammation as well as fibrinogen and plasminogen

activator inhibitor-1 (PAI-1) have been found to be indicators of altered LV geometrical changes[xvii] and collagen biomarkers such as collagen types I and III[xviii]. Homocysteine has also been used as a marker for predicting risk of heart failure, though when levels are reduced to "normal levels" when they were once high, the risk of heart failure is still prevalent.[xix] The most reliable biomarker for predicting remodeling has been aldosterone to renin ratio (ARR) which has been clearly associated with eccentric and concentric ventricular hypertrophy. [xx] This is significant since it is clear that activation of the RAAS and ANG 2 levels have already clearly been associated with LV remodeling as mentioned above.

THERAPEUTICS

Despite the polypharmacy of drugs used to prevent post-MI remodeling, heart failure can still occur. There are several approaches, which can be considered to inhibit and prevent the processes involved in cardiac remodeling which may incorporate both pharmaceuticals and herbal medicine where appropriate:

- Preventing ischemic damage
- Lowering high blood pressure
  - Regulating RAAS and ANG2 expression
- Reducing fibrosis and collagen formation
  - Down regulating TGF-beta1
- Inhibiting MMP activity
- Inhibiting Endothelin formation
- Reducing excessive aldosterone levels
- Addressing secondary factors such as insulin resistance, which contributes to myocardial hypertrophy[xxi]. Improve insulin sensitivity.
- Reducing overall inflammation
- Address compounding factors contributing to further heart damage such as
  - Reduce stress, anger, tension
  - Improve circulation
  - Prevent atherosclerosis progression

- Make necessary lifestyle/diet changes to reduce risk of another MI and improve overall health perhaps with the guidance of Cardiac Rehabilitation Services.[1]

In the process of considering potential medicinal plants for use in post-MI scenarios, a combination of traditional uses will be mixed with modern scientific studies that have created interest in several plants and their actions on reducing remodeling and hypertrophy. While many studies focus on isolated constituents used in animal studies, they will still be included for the sake of potential extrapolation for indicated actions in congruence with traditional use.

The general energetic picture of the post-MI patient is somewhat dependent on their overall constitution and the phase of remodeling they are in. Generally, the overall energetic picture of someone in a post-MI condition will be cool and deficient lacking vitality and Qi. In general, the herbal strategies will be to warm and rebuild the deficient person.

ACTIONS:

Primary:	Cardioprotective	**Secondary:**	Diaphoretic
	Anti-inflammatory		Nervine
	Anti-fibrotic		Anti-cholesteremic
	Circulatory Enhancement		Lymphatic
	Vasodilator		Adaptogen/Endocrine Modulator

HERBS:

Hawthorn- *Cratageus oxycanthoides:*

Anti-inflammatory, Anti-oxidant, Nervine, Vasodilator, Cardiac Trophorestorative

Hawthorn is a well known and highly researched cardio trophorestorative that improves conditions such as mild congestive heart failure, mitral valve prolapse, myocarditis, arrhythmias, atrial fibrillation, palpitations, angina, improved recovery from MIs, preventing atherosclerosis, mildly lowering high blood pressure, and preventing or improving ischemic heart disease conditions.[xxii]

Tea (Infusion): 1-2 tsp. dried berries, 8 oz. hot water, steep for 1 hour, take 3

[1] This component of healing from a MI is critical to determining the quality of life and reduction of post-MI remodeling. While it is essential to discuss diet and exercise, the limits to the length of this paper have left me with no other option than to put the thought into a footnote and encourage further reading for those interested. Also, it would be a great loss not to mention the emotion influences, such as anger, grief, heart break, and anxiety as well as the benefits of spiritual practices such as prayer, meditation, and positive thinking.

cups/day

Tincture (1:5), 30-35% ETOH, 10% Vegetable Glycerin Dose: 3-5 ml (60-100 gtt.) QID

Capsules: 2 (00) capsules TID

Solid extract - 1/4 - 1/2 tsp. 2x/day

Rosemary *Rosmarinus officinalis:*

Anti-inflammatory, Anti-oxidant, Diaphoretic, Nervine

In an animal study using a diterpene phenol extract of Rosemary, pulmonary fibrosis was inhibited through the mechanism of inhibiting up-regulation of TGF-beta1 and inhibiting excessive collagen deposition, especially collagen-I formation.[xxiii]

Tea (Infusion): 1/2 tsp. powdered herb, 8 oz. hot water, steep covered for 15-20 minutes, take 2-3 cups/day

Tincture (1:5) 65% ETOH Dose: 1-2 ml (20-40 gtt.) TID/QID

Night Blooming Cereus - *Selenicereus grandiflorus:*

Cardiotonic, nervine, and diuretic

This plant was used by the Eclectics for “Athlete’s heart” and more recently has been used by David Winston for lowering blood pressure in people, especially those with insulin resistance and blood pressure and cholesterol levels that have been creeping up every year starting at age 30[xxiv]. He has also used it for heart disease with an irregular, feeble heartbeat and pulse accompanied with anxiety, dyspnea, or depression. Winston also finds it effective for mild to moderate congestive heart failure with angina, decompensation of the heart due to nicotine use, and for mitral valve prolapse.

Tincture (1:2), 40% ETOH Dose: .5-1.5 ml (10-30 gtt.) TID

Boswellia *Boswellia serrata:*

Anti-Inflammatory, Analgesic, Circulatory Stimulant.

In an animal study, a primary constituent of its resin, incensole acetate, was shown to inhibit TNF-α, IL-1β and TGF-β1 expression, as well as NF-κB activation following head trauma and had a therapeutic window of treatment up to 6 hours[xxv] following the ischemic injury. It is a very effective anti-inflammatory and analgesic especially in cases of congealed or stagnant blood where there is pain and trauma.

Tea (Decoction): 1/2-1 tsp. dried resin, 12 oz. water, decoct 15-20 minutes, steep covered 45 minutes take 4 oz. 3x/day

Tincture (1:5), 70-80% ETOH Dose: 1.5-2.5 ml (30-50 gtt.) TID

Capsules-Standardized to 25-37% Boswellic acids: 300-400 mg TID

Gotu Kola – *Centella asiatica*

Adaptogen (mild), anti-inflammatory, anti-oxidant, circulatory stimulant, anti-ulcerogenic, nervine, vulnerary.

Gotu Kola is used for any degenerative disorder pertaining to the muscles or fascia. It is a useful adjunct in a protocol for mitral valve prolapse, endocarditis, cardiomyopathy, and ischemic heart disease and for inhibiting scar tissue formation.[xxvi] In an animal study with rats subjected to a myocardial infarction and were given an extracted constituent from Gotu Kola called madecassoside, which was shown to reduce c-reactive protein and super-oxide dismutase levels as well as reducing ischemia reperfusion injury.[xxvii]

Tea (Infusion): 1-2 tsp. dried herb, 8 oz. water, steep covered 45 minutes, take 4 oz. 3x/day Tincture (1:2), 30% ETOH Dose: 1.5-2 ml (30-40 gtt.) TID

Capsule: 200 mg standardized extract (40% asiaticosides): BID/TID

Red Sage – *Salvia miltiorrhiza- Dan Shen (TCM)*

Analgesic, anti-coagulant, anti-inflammatory, anti-oxidant, cardioprotective, peripheral vasodilator

Dan shen is useful for angina pain, reducing blood pressure in hypertension, reducing cholesterol and trigylceride levels, reducing palpitations, improving cardiac circulation, and for preventing the development of atherosclerosis.[xxviii] In TCM it is used for conditions of blood stasis in the chest (including the heart) and abdomen as well as for symptoms of "disturbed Shen." There are several animal studies validating the use the constituent salvianolic acid B for reducing cardiomyopathy and remodeling by inhibiting TGF-beta1, MMP-2 and MMP-9 expression.[xxix] [xxx] [xxxi]

Tea (Decoction): 1-2 tsp. dried root, 8 oz. water, decoct 10-15 minutes, steep 1 hour, take 2-3 cups/day.

Tincture (1:4 or 1:5), 45-50% ETOH Dose: 1.5-3 ml (30-60 gtt.) TID/QID

Chinese Figwort - *Scrophularia ningpoensis*

Anti-inflammatory, cardiotonic, hypotensive, vasodilator, lymphatic

An animal study conducted on rats subject to a myocardial infarction were given an extract of Scrophularia ningpoensis for 4 weeks. Rats given the figwort showed a decrease in the left ventricular weight index, heart weight index, a decrease in ANG2, hydroxyproline, and

reduce collagen deposition as well as down regulate TNF-alpha andTGF-beta1 in the myocardium when compared to the control group.[xxxii] It has a mild positive inotropic effect on strengthening the heart and can be used along with stronger remedies for mild cardiac edema.[xxxiii]

Tea (Decoction): 1 tsp. dried, powdered, root, 8 oz. water, decoct 10 minutes, steep 1 hour, take 2 cups/day

Tincture (1:4-1:5), 50% ETOH Dose: 1-2 ml (20-40 gtt.) TID

Corydalis - *Corydalis yanhusuo*

Anti-inflammatory, analgesis, anti-arrythmic, anti-spasmodic, anti-ulcerogenic

An animal study found it to attenuate myocardial hypertrophy and reduce levels of collagen formation in rats that were fed 200mg/day of corydalis extract starting 2 weeks after being induced with myocardial pressure overload.[xxxiv] In one animal study with cats, it was shown to decrease resistance of blood flow and increase blood perfusion to coronary arteries by improving the contractile function of the heart and reducing the consumption of oxygen by the heart muscle.[xxxv] In TCM, pain is associated with stagnation. Corydalis has been used to move stagnant Qi and stagnant blood especially where there is chest pain, angina, arrythmia, and coronary artery disease.[xxxvi] It can also be used for preventing myocardial ischemia.

Tea (Decoction): 1 tsp. dried rhizome, 10 oz. water, decoct 15 minutes, steep 1 hour, take 2-4 oz. 3-4x/day.

Tincture (1:4 or 1:5), 50-60% ETOH Dose: 1-2 ml (20-40 gtt.) QID

Asian Ginseng – *Panax ginseng*

Adaptogen, anti-inflammatory, anti-oxidant, cardioprotective, nervine, immune amphoteric

It is used in TCM for total depletion of upright qi with yang collapse where the person is experiencing SOB, shock, a cold sweat, shallow respiration, and had a feeble pulse.[xxxvii] Several animal studies on ginsenosides from Panax ginseng have shown them to have an ability to reduce adverse post-MI remodeling[xxxviii] and reduce myocardial ischemia and reperfusion injury. [xxxix]

Tea (Decoction): 1-2 tsp. dried root, 12 oz. water, decoct 30 minutes, steep 1 hour, take 2 cups/day

Tincture (1:5), 50% ETOH Dose: 1-2 ml. (20-40 gtt.) TID

Reishi - *Ganoderma lucidum*:

Adaptogen, anti-cholesteremic, Anti-inflammatoy, Anti-oxidant, Cardiotonic, Nervine

Reishi helps prevent atherosclerosis, lowers blood pressure, inhibits platlet aggregation, reduces triglyceride levels, and cholesterol. [xl] It is also used for symptoms of disturbed Shen.

Tea (Decoction): 1-2 oz. dried mushroom, 32 oz. water, decoct 2-4 hours, take 3-4 cups/day

Tincture (1:5), 25% ETOH Dose: 4-5 ml (80-100 gtt.) 4-5x/day

Capsules (mycelia extract): three 500-1000 mg capsules, 3x/day.

Turmeric - *Curcuma longa*:

Anti-inflammatory, anti-oxidant, anti-cholesteremic

In a study with rats induced with a MI, curcumin was found to reduce the size of infarction by protecting against myocardial ischemia and reperfusion injury after 30 minutes of occlusion of the left anterior descending coronary artery.[xli] Curcumin may thin the blood and is used for preventing abnormal cell signaling. Turmeric can reduce C-Reactive protein levels and prevents atherosclerosis and lipid oxidation.[xlii]

Tincture (1:2 or 1:4), 60% ETOH Dose: 2-4 ml (40-80 gtt.) TID/QID

Curcumin Capsules - Standardized 80-90% Curcumin: 500 mg TID

Barberry - *Berberis vulgaris*:

It helps rebalance heart rhythm, is an endothelial fibrosis inhibitor, and stabalizes plaque.[xliii] It may also inhibit MMP activity in patients with congestive heart failure.[xliv] The berries have been found to be hypotensive and vasodilating in rats with hypertension.[xlv]

Tea (Decoction): 1 tsp. dried root bark, 8 oz. water, decoct for 10 minutes, steep 45 minutes to 1 hour, take 4 oz. 3 -4x/day

Tincture (1:5), 60% ETOH Dose: 1.5-3 ml (30-50 gtt.) TID/QID

Capsule: 1000-2000mg QD- 15-20+ g of barberry root a day at a 5-6% berberine content.

Rhodiola – *Rhodiola rosea*

Adaptogen, antioxidant, cardio-protective

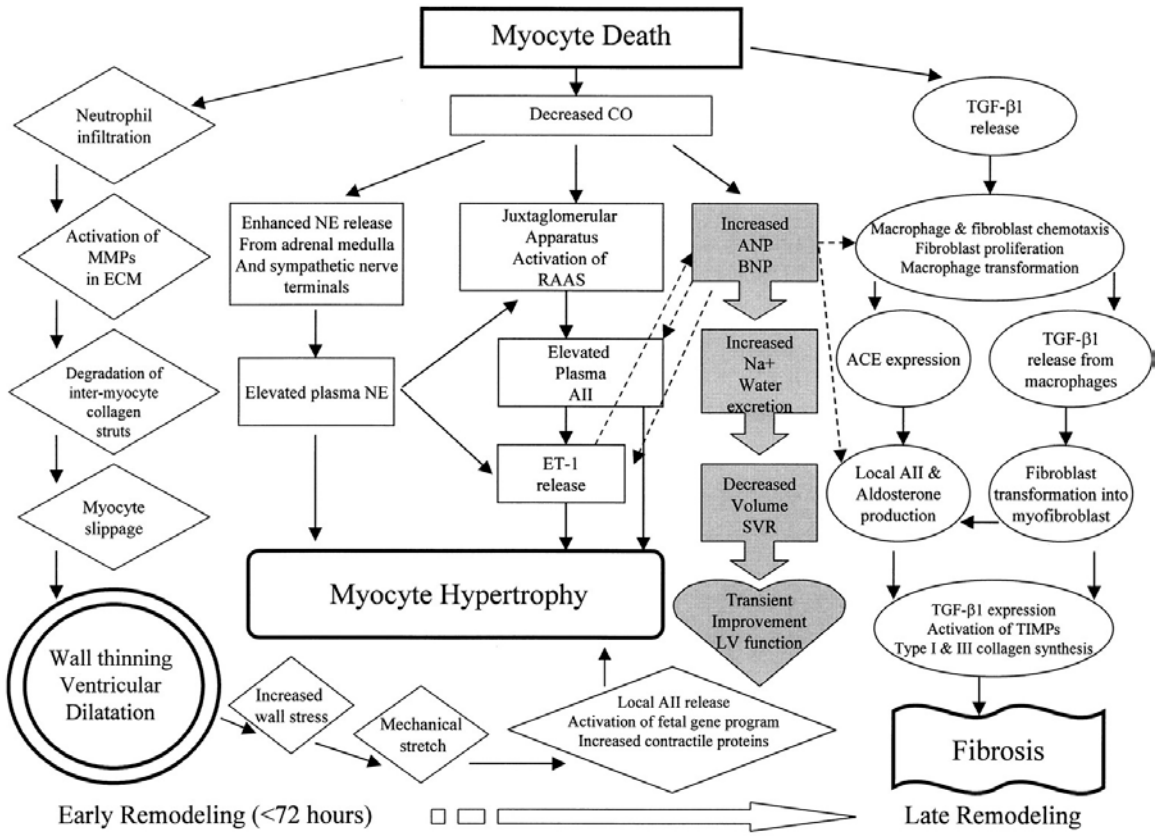
Rhodiola has been used for lowering c-reactive protein levels, preventing atherosclerosis, preventing myocardial infarctions, and reducing symptoms of angina and mild congestive heart failure.[xlvi] It is also used in TCM for relieving heart blood stagnation and heart yang deficiency.

Tea (Decoction): 1-2 tsp. dried root, 10 oz. water, decoct 15 minutes, steep covered for 45 minutes take 1-2 cups/day

Tincture (1:5), 30% ETOH Dose: 4-6 ml (80-120 gtt.) TID

APPENDIX

Figure 1: Pathophysiology of Ventricular Remodeling



Diagrammatic representation of the many factors involved in the pathophysiology of ventricular remodeling. ECM indicates extracellular matrix; RAAS, renin-angiotensin-aldosterone system; CO, cardiac output; SVR, systemic vascular resistance; LV, left ventricular; and AII, angiotensin II.

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Transforming Trauma Stored in the Body: A Holistic Approach to Post Traumatic Stress Disorder

Danielle Rissin- Rosenfeld

Post Traumatic Stress Disorder (PTSD) affects people's lives profoundly but because it is not something that is visibly debilitating it is often over looked. The purpose of this paper is to provide a more comprehensive look at the reasons for PTSD and the ways that it manifests itself in the body. It is also a platform to briefly overview supportive strategies, healing practices and herbal therapeutics.

Post Traumatic Stress Disorder (PTSD) is a reaction to one or more traumatic events which presents anywhere between one month and a year after the occurrence. People experience trauma in situations that are emotionally/physically painful and distressing and overwhelm their ability to cope. Trauma may begin as acute stress from a perceived life-threat or as the end product of cumulative stress. (33) Trauma can stem from childhood abuse and neglect, medical/ surgical interventions, war and violence, physical emotional and sexual abuse, accidents and natural disasters, grief or witnessing acts of violence. (30) To be diagnosed with PTSD one must exhibit the symptoms of mood functional disturbance, substance abuse and suicidal thoughts. (35)

Symptoms of PTSD may include catatonia (speechless), black-outs (memory loss), robotic actions (mind control), flashbacks (remembering when you don't expect to), sleeplessness, irritability, quickness to anger, listlessness, anger, hopelessness, purposelessness, defeatism, depression, anxiety, panic attacks, nightmares, bad dreams, and disassociation. (20) Risk factors for PTSD may include experiencing dangerous events or traumas, having a history of mental illness, getting hurt, seeing people hurt or killed, feeling horror, experiencing helplessness or extreme fear, having little or no social support after the event or dealing with extra stress after an event such as loss of a loved one, pain and injury, or loss of a job/ home. (20)

Hypthalamic-Pituitary-Adrenal Axis

During a traumatic event the individual usually mounts a “fight, flight, or freeze” response. If the person’s body is unable to fully process the stressor and release the shock of the event it gets stored in the cerebral cortex and muscle memory. (29) The stress during the traumatic event, as well as ensuing stress, activates the Hypthalamic-Pituitary-Adrenal axis, which leads to the suppression of DHEA, Testosterone and Estrogen synthesis, all of which affect mood. Low estrogen is associated with lower serotonin production, which can impact anxiety. While the Acute Stress Response is necessary for survival, prolonged stress is ultimately damaging. Allostatic load is a term for wear and tear on the body that accumulates when an individual is exposed to repeated or chronic stress. A high allostatic load can result in long-term physiological changes such as atherosclerosis and stroke. A high allostatic load can cause insomnia, depression, and diabetes due to disruption of endocrine function. (4, 29)

Chronic stress can cause prolonged elevated levels of Corticoliberin CRF, a peptide hormone and neurotransmitter. This may down-regulate CRF receptors in the pituitary and brain. Decreased Adrenocorticotropic hormone (ACTH) response and decreased CRF receptor concentrations are found in the frontal cortex of depressed individuals who commit suicide, as well as in people suffering from PTSD. (7)

Traumatic events can be replayed and re-experienced months to years after the event. Fear related memories are stored in the cerebral cortex, and when triggered, may also activate neural circuits in the temporal lobe and brain stem, potentiating feelings of panic. (4) Panic, stress and emotional distress are perceived by the hypothalamus which signals your body regulating hunger, thirst, sleep and wakefulness, as well as most involuntary mechanisms, including body temperature. When these signals are disturbed, the body is confused about how to regulate itself (36). This can contribute to the symptoms of PTSD and exacerbate self-destructive patterns.

Historical Trauma

Beyond the acute presentations of trauma, there are social, political and spiritual contexts which engender ongoing trauma. Dr. Maria Yellow Horse Braveheart defines historical trauma as:

> “Cumulative emotional and psychological wounding over the lifespan and across generations, emanating from massive group trauma. Native Americans have, for

> over 500 years, endured physical, emotional, social, and spiritual genocide from European and American colonialist policy. The effects of historical trauma include: unsettled emotional trauma, depression, high mortality rates, high rates of alcohol abuse, significant problems of child abuse and domestic violence." (21)

It's hard to heal from trauma when the conditions which created it haven't changed. Repeated on-going traumatic events such as genocide, residential schools, slavery, war, displacement, repeated sexual, emotional and physical abuse, environmental destruction are not only held in an individual's body but can be carried over to one's descendants (14). Although healing from trauma is important for the individual, it is also often a community healing process which is required. When a person lives in isolation and is displaced from their community or landbase and home it is a harder and longer journey towards healing. (21)

In Judith Lewis's book "Trauma and Recovery", she says that, "*recovery can take place only within the context of relationships; it cannot occur in isolation. The first principle of recovery is the empowerment of the survivor."(*18) There is a lot to be said about the incredible strength and resilience of people who have endured so much. One of the most major factors that encourages is community as well as positive feelings towards oneself and healthy coping mechanisms like exercise and ritual. (25)

Therapeutic Practices for PTSD

Therapeutic protocols depend on a person's symptoms, experiences and constitution. People have found success with alternative therapies such as cranial sacral therapy, acupuncture and somatic experiencing.

Somatic experiencing is a psychobiological method for resolving trauma symptoms and relieving chronic stress. Other therapies include Eye Movement Desensitization and Reprocessing (EMDR), Hypnosis and Emotional Freedom Technique (EFT). (35)

Somatic experiencing offers a framework to assess where a person is "stuck" in the fight, flight, freeze, or collapse responses and provides clinical tools to resolve these fixated physiological states.

Acupuncture is also a huge support in enabling victims to cope with trauma and to heal from PTSD. When giving acupuncture treatments for PTSD acupuncturist Janette Cormier tends to do the, “NADA (north American Detox Acupuncture) as a basic protocol combined with more specific points for them as an individual” She also does, “'grounding' treatments and treats many kidney issues (fear). It's often Kidney Yang deficiency.” (22)

Psychotherapy is also used as a healing modality for PTSD. However the pure mental health focus, “often individualizes the experience, leaving people isolated with the impact and the concrete circumstances of their specific situation.” (14)

Eye Movement Desensitization and Reprocessing (EMDR) is a therapy in which a person moves their eyes rapidly from side to side while recalling a traumatic event.

Biofeedback is a therapy to help understand how the body reacts to stress. It involves using a machine, at first, to see bodily functions that are normally unconscious and occur involuntarily like heart rate and temperature. The purpose is so that one will learn to control the reactions, and eventually be able control their reactions to stress without using a machine.

Hypnosis induces a deep state of relaxation, which may help people with PTSD feel safer and less anxious, decrease intrusive thoughts, and become involved in daily activities again.

Emotional Freedom Technique (EFT) helps a person revisit traumatic events while tapping on acupuncture points in order to release the experience. (36)

Ceremony encourages spiritual nourishment and release of a traumatic event especially ceremony that incorporates shaking and crying. In Peter Levine’s Book Waking the Tiger he talks about how when responding to an inescapable or overwhelming threat humans and animals both use an immobility response. Levine believes that for humans to heal from trauma they should mirror the fluid adaptation of wild animals, by shaking and passing through the immobility response. This will enable them to move through the trauma and becoming fully functional again. (26)

To begin the journey to healing a person must be able to look at their trauma and be ready to transform it. When talking with clinical herbalist and acupuncturist Janette Cormier she said,

> "One of the most difficult things about healing from PTSD is that you need to gently, but firmly push people forward out of victimization. This can be really challenging for the practitioner and the patient and must be done with care and integrity. There is also a timing issue regarding a sort of 'grieving period'. Obviously it is not enough to say/totally inappropriate to tell people they must "get over it", but within the context of treatment and support this is still the ultimate goal - that they leave victimhood beyond for a life of freedom from their past 'demons'" - whether they be people or experiences. In order for people to move forward in this way they need to be strong, and I often find myself working on strengthening their vitality and spirit, working on their constitution, nourishing them and encouraging them always to move forward and not to stagnant. Ceremony can be huge for this. I feel like acupuncture treatments are like a personal ceremony when done appropriately... and can be massively profound at times." (22)

Herbal Therapeutics for PTSD

Herbal medicine is only one of the many supportive strategies needed when approaching PTSD. When addressing PTSD it is important support the person's endocrine function, nervous system and help them adapt to the stress. When approaching it from Chinese Medicine perspective, PTSD can be manifested as Shen disturbance or insufficient/ blocked qi. In that case it may be useful to move or build the blood. (18) Addressing acute anxiety and depression can also be useful in improving the quality of one's daily life. The lasting effects of PTSD can result in permanent physiological changes such as chronic pain and illness (4,13,36). In this case one will want to support a person's body by modulating their immune system and supporting their neuroendocrine function. With acute symptoms of PTSD such as insomnia, anxiety and panic it may be prudent to address physical pain and emotional turmoil. However, suppressing these symptoms in the long term may prolong the problem. Sedating someone is not addressing the underlying issue of why the trauma is reoccurring. (27) Herbal actions may include nervines, adaptogens, blood builders and movers, anxiolytics, immunomodulants and yin tonics etc. The herbs need to be different

for each person depending on their constitution, symptoms, barriers to healing and lifestyle. In my research the herbs that were prominent:

Milky Oats (Avena Sativa) is a restorative nervine for self-induced adrenal exhaustion. The milky tops are used internally as tea or fresh tincture. The fresh tincture is used for acute nerve injury. The sooner taken the better. Milky oats are specifically indicated for concussion, compressed nerve, cut nerves and traumatic brain injury. It is helpful in recovery from neurasthenia/chronic fatigue syndrome, insomnia, depression, anxiety, and opiate withdrawal. They help restore, rebuild and regenerate nerves.

Dose: 3-5 ml 3xday (traumatic acute nerve (esp. brain) injury recovery) less for other maladies. Tea: 6tbs/quart infusion preferably overnight. (9)

Hawthorn (Crataegus monogyna) Hawthorn nourishes and calms the heart, helping to settle the Shen. At the same time, her thorns offer protection from those who would harm you. Hawthorn can potentiate digitalis drugs, caution w/ bata blockers.

Dose: solid extract jam- Itsp TID (10)

Wood Betony (Stachys officinalis) is used for harmonizing the gut brain action, parasympathetic tone, headaches and for aiding digestion in cases of all the consequences of eating while experiencing stress. It is specifically indicated for grounding the solar plexis for people who are fixated in mental patterns, providing protection from the belief in evil spirits, nightmares and visions.

Dose: 1 teaspoon TID (17)

Anemone pulsatilla is for underlying anxiety, people who are sad, pale withdrawn and need sleep. It is for people who are fearful and weep easily when everything that can go wrong has. It is specifically used for panic attacks, insomnia, nervousness, and a generally agitated emotional state with gloom and distress.

Dose: 3-10 drops, to 4X a day. USE WITH CARE. (30,31)

Dan Shen (Angelica sinensis) infuses courage where there is disturbance and fright. It is specific in cases of insomnia, dementia, nourishes the blood while moving and has calming effects. It is a blood tonic, mild analgesic for pain from stagnation and moves energy in the body.

Dose: 3-15 g or 4-3 ml- 1-4 times daily. (9)

Ashwagandha (Withania somnifera) is indicated for the wired and tired person. It helps in re-regulating the HPA axis and helps shift a person's perception of stress and cognitive decline. It is a yin tonic and is building, helping to store energy rather than use it.
Dose: 2-4 ml TID (2)

Skullcap (Scutellaria lateriflora) is a calming nervine for depression or anxiety, restless leg syndrome, muscle spasms and obsessive compulsive behaviour. It is for those people who are tired and wired. The eclectics used it for irritability of the nervous system and restlessness.
Dose: 10g per day, tincture 1:2 30-150 drops TID. (9)

Frankincense (Boswellia sacra) is warm and pungent, and enters the heart and lung meridians. It promotes blood circulation and movement of qi, and is used following acute physical trauma. It can also relax tendons. Frankincense is especially suitable for conditions where the joints and muscles are very stiff, swollen, and painful. (10)

Mimosa (Albizia julibrissin) is also used for disturbed shen, the bark and blossoms (more calming). In Chinese tradition it is used for disturbed shen symptoms, including bad dreams, irritability, anger, depression, and poor memory. Combined with Hawthorn berries and Rose petals it is used for "broken hearts", grief and deep sadness.

Mimosa blossoms (He Huan Hua) also can be used to calm the shen and elevate the mood, but they are weaker and less effective than the bark. (32,17)

Dose: Tincture (1:5): 40-80 drops TID. (32)

Passionflower (Passiflora incarnata) is a mild anodyne, anxiolytic, hypnotic and nervine, antispasmodic and hypotensive. It's specifically used for circular thinking and people who can't relax or sleep because the mind is going around and around. Passionflower is an antispasmodic relaxant and is a warmer alternative to blue vervain. It's good for anxiety, irritability, restlessness, stress induced spasm, backache, tension headache, bruxism (grinding teeth) disturbed shen, convulsion, stress induced heart symptoms, nerve pain, exhaustion with spasm and twitch, insomnia when you can't turn off your mind, restless sleep, sleeplessness from anxious irritability and worry. It is great combined with Jamaican dogwood for someone who is sleepless with pain.

Dose: Fresh tincture (1:2 40%) 3-6 mls TID. For sleep 5ml at bedtime and for tea: 1-2 tsp/8 oz steep (30 min ideal) (6)

Rosa rugosa pedals are used for grief. They are used to astringe and protect the boundaries of the heart. It can help people who feel disempowered feel more empowered. The combination of mimosa, hawthorn and rose can help people who feel more deeply and help deal with unresolved issue. (27,9)

Dose: petals 1:1 50%ETOH, 40%h2o,10%glycerin.

For astringency and anti-inflammatory effects- 3-5 ml

For emotional effects- 1-2 ml/day

Hips in syrup/jam- 1-2 TBS/day

Hip tea- 2-5 grams (9)

Ghost pipe (Monotropa uniflora) is used as a nervine to relieve symptoms of neurological chemistry disruption and pain, to stop seizures, convulsions, insomnia, mental disorders, and chronic muscle spasms. Ghost pipe is specific for: overwhelming physical pain when combined with anodyne herbs, migraines associated with traumatic brain injury, anxiety and panic associated with emotional or sensory overload, the triggering of emotional memories that make the subject feel beside herself with unpleasantly intense mind altering experiences.

Caution: consumption of 15 ml or more of Monotropa tincture can bring deep sleep and ultra -vivid dreams, often bizarre, frequently erotic. (1,12)

Dose: start with three drops of the tincture, but go up to 1ml if the person doesn't respond to a 3 drop dose. In cases of severe panic/agitation give 1ml drop doses at 5 minute intervals. (1)

American Ginseng (Panax quinquefolius) is an endocrine amphoteric and adaptogen that is useful for mild to moderate depletion of the HPA axis and adrenal glands. Because of its effects on the HPA axis, it can help correct dysfunction of the immune system, including depletion that leads to a susceptibility to catching colds. It is also a mood regulator, good for regulating blood sugar in recovering junkies, chronic fatigue syndrome helping regenerate the body in people with long term amphetamine use.

Dose: Do not use longer than 3 months, 50-80 drops TID (9)

Holy Basil (Ocimum tenuiflorum) is an adaptogen and helps re regulate a person's perception of stress in HPA dysregulation. It is also used specifically in eating disorders, which are a common occurrence in PTSD because of the disassociation and body dysmorphia that may occur.

Dose: Whole plant juice 1-3 ml, Tincture 3-5 ml in a formula, Tea 2-5 g (5)

Reishi (Ganoderma lucidum)
For women who have hyperimmunity (immune system hyper vigilant see everything outside of itself as a threat) the caregiving type who are more prone to fibromyalgia. It is used for disturbed Shen, anxiety, insomnia, bad dreams, and listlessness.
Dose: 1:2 ml 2 stage extract, 5ml BID, 2 tsp QD- BID powders, 7:10g in decoction (28).

Flower essences help deal with emotional and spiritual trauma. Some flower essences that clinical herbalist Jasmyn Clift recommended for PTSD are:

Star of Bethlehem for shock and trauma. It comes with work. People will need a coping mechanism because they will revisit the trauma to move through it. They need to be up for the work. If they're going through "crazy" times they shouldn't take it. It is ideal to help work with and transform triggers. (8, 16)

Mimulus helps a person get over fear. It is for the person who is shy and retiring and prone to hide their anxiety. For the person who is deterred by chronic fears such as fear of the dark, injury, poverty etc. (8, 16)

Aspen is an inability to explain the fear, sympathetic state and worry. For the person with unexplainable fear, who has fears by day or night for no known reason, and fear accompanied by trembling and sweating. (8, 16)

Rock Rose is for complete and utter terror and extreme fear caused by facing an unexpected or unfamiliar experience. It is for the person who experiences fear from terrifying sights or nightmares. When their fear and panic is so severe it is projected in the atmosphere. Rock Rose enables a person to be calm, courageous and be able to look out for the well-being of others. (8, 16)

Gentian is for people who are starting to lose faith in themselves. It is for people who attract negativity because of their own negative state of mind and are experiencing a deep depression from a known cause. Gentian aids a person in finding faith in their own resources. (8, 16)

Gorse is for people with no self- sufficiency who are living with chronic illness and/or in poverty. For people with diminished vision, lack of ambition and interest due to hopelessness. Gorse helps confidence and enables one to overcome obstacles and not be overly influenced by others. (8, 16)

Clematis is for people who cope by disassociating. They are day dreamers that indulge themselves in drugs and alcohol, TV and other distractions. Clematis helps a person live in the present and complete tasks it is especially for people with A.D.D and PTSD. (8)

Honeysuckle is for people living in the past who can't be present it is specifically for cases of PTSD. Honeysuckle is for the people who dwell on thoughts of the past and hold a pessimistic outlook both for the present and future. For the people who are chronically nostalgic and regretful. Honeysuckle helps people overcome past experiences and be able to creatively transform trauma and grow from it. (8, 16)

Olive is for people who are totally exhausted to the point of tears, drained of energy and unable to cope with daily thing. It helps people find motivation, depend on their inner-self and attract happiness and pleasure despite their exhaustion. (8, 16)

Pine is for people who have immense guilt and self-reproach. This is a common occurrence in PTSD because people internalize their trauma and blame themselves for it. Pine is for people who over work themselves and stress out because they are never good enough. Pine helps resolve remorse and enables people to live without useless guilt. (8, 16)

Bach Rescue Remedy is for acute trauma situations and helps calm and ground a person in times of panic and anxiety. (8)

Other flower essences that may be useful in PTSD are: Indian pink for remaining centered and focused even under stress. Bleeding heart in a spirit dose of the whole is for mass hysteria.

Eating the flower of borage will enhance courage. (25)

Red clover is used to create a calm and steady presence. (25)

Yarrow is useful for boundaries of psychic protection. (25)

Sunflower is for people who have relationship issues with their fathers. It also helps a person express themselves fully, truly and vibrantly. (25)

Additional nutritional and supplementation support may include: Omega 3 fatty acids for supporting mood. Eating kale and other leafy greens high in magnesium reduces spasms. Co-Q10 and magnesium support immune and muscular support. It is good to avoid coffee and other stimulants and it is recommended to look into other food sensitivities. (34)

Herbs are just one of the supportive strategies needed in the journey towards healing from PTSD. The process of healing is different in each person. Healing is most effective when done within community; the more support a person experiences the quicker and more likely it is for transformation to occur. Displacement and isolation hinder the healing process. Ideally, a person receives multiple forms of care. Trauma is usually not a singular occurrence and traumatic events may be unavoidable. When addressing PTSD, it is important not just to look at it as an individual issue, but to look at the larger issues and context of why the trauma has occurred.

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Case Study – Anxiety, Stress management

Marguerite Gregory

RM is a 48 year old female, H 5' 3 ½ ", W 133 lb., who came to the City Market clinic, accompanied by her daughter, MM, in June, 2013. Her primary goals were to boost her immune system, prevent Lyme Disease, and get help with insomnia related to extreme stress and PMS. During this period of time her stress was tremendous – her daughter had Lyme disease, she was going through a major break in an important family relationship, and the proposed gas pipeline through Monkton was planned to pass close to her home well. The initial visit and subsequent follow-up visits were conducted back-to-back with both mother, daughter and two student clinicians present each time.

RM presented with frequent twitchy movements and seemed nervous, on the verge of tears. She had cold hands and her face was pale, but with heat signs. At the initial visit her pulses were thin, tense, and hard to occlude. They were somewhat weak in the Metal (LI/Lu), Fire (TB/P) and Fire (SI/H) positions, and moderate in all other positions. Heart rate variability was present, but slight. Her tongue was pink with a red tip and a slight white coat. It appeared moist with slight quivering, lots of scallops and a crack near the tip. She had slight blue sublingual veins.

Two years previously, in 2011, she was diagnosed with environmental illness from mold in the house that was subsequently removed. She had also been diagnosed with candida on the phone by a medical intuitive. She was leary of physicians and did not see a doctor regularly.

During the client interview, most complaints centered around stress, although she reported that during the last 3 months, her stress and related symptoms had begun to improve. She was taking proactive steps to resolve the external factors causing her stress and was also receiving Reiki that was helping. Symptoms of stress included insomnia, low energy, PMS, global and specific anxiety, reduced libido and tinnitus. She also reported a loose, mushy stool, PMS with breast tenderness (she has fibroids), increased appetite and craving for sweet, insomnia and mild, dull cramps. Both mother and daughter were continuing to

experience cyclical symptoms that may have been related to the environmental illness. For 3 days every other week they experienced fatigue with brain fog, nausea and depression.

The constitutional summary was yin deficiency (false heat) with both Vata derangement (tendency to cold, variable energy, nervous system disorders (insomnia and anxiety) and shallow breathing and Pitta derangement (red tip of the tongue, hypersensitivity to mold).

The summary of relevant physiology included nervous tension affecting mood, sleep, GI tract and libido, and immune system hypersensitivity.

Initial Goals:

1. Support adrenals and HPA-axis and increase energy.
2. Reduce perception of stress.
3. Support digestion.
4. Support sleep.
5. Support the immune system and reduce hypersensitivity.

Herbal actions centered around adaptogens, nerviness (trophorestorative and relaxing), digestive tonic, immune modulators and immune tonics. She was given powders to make into bliss balls: ashwagandha, shatavari, astragalus, and codonopsis (15 g/day), and a tincture of blue vervain, skullcap, reishi, and yarrow (5 ml TID). It was recommended that she and her daughter use air purifiers in their bedrooms, and that she continue with Reiki and the community support group that she was involved in.

First follow-up, 6 weeks later, July 2013

All of RM's pulses had strengthened, the weakest now being Fire (SI/H). It was further noted that her tongue was swollen and had a jing saddle. She was fidgety and her eyes blinked rapidly and frequently.

She reported that both her and her daughter's health and emotional well-being were improving. RM reported much less anxiety (2/10), though she felt that a baseline of anxiety was part of her make-up. Sleep related to stress and PMS had improved (insomnia now only 1-2 days instead of 4), her energy was as good as it had been 5 years ago and she had not noticed the tinnitus lately. PMS had generally improved except that she still had bothersome

fibroids and mild, dull cramps. There was no change in her loose stool, but the mystery cycle of fatigue and depression had disappeared and she attributed this improvement to her work with Barbara Clearbridge, a medical intuitive who practices Reiki and other healing modalities.

Dampness (swollen tongue and mushy stool) was added to her constitutional summary, a goal of reducing dampness also added, and astringent action introduced. The bitter taste, which had been considered part of the "digestive tonic" action was revisited, because RM could not stand the taste of the tincture, although she liked the bliss balls.

Herbal support formulas:

1. Bitters tincture: dandelion, blue vervain, yarrow (1 tsp TID).
2. Bliss balls: add reishi to ashwagandha, shatavari, astragalus, and codonopsis.
3. Nervine, astringing, and adaptogenic tea: tulsi, skullcap, gotukola, nettles, licorice.

It was also suggested that she could try eliminating some foods that tend to be allergenic such as dairy, wheat, nuts and seeds.

Second follow-up, 8 weeks later, Sept. 2013

Her pulses continued to strengthen and to resemble one another in both strength and quality, that is slightly deep, tense and thin. On her tongue, a coat that was white with some yellow was noted only at the back now, and red papillae were noticed at the rear and sides. Quivering was not seen this time.

RM reported much less anxiety. She had begun to enjoy social events with large groups of people and felt she was managing better with events that formerly had caused her more distress. Sleep and energy continued to improve, but she still felt unable to return to the vigorous yoga she used to enjoy because she felt too depleted. She asked for a remedy that she could take at times of acute stress. Her digestion had improved; she was more comfortable, experiencing less gas and bloating and her stool was firmer. She had eliminated wheat and was doing only very little dairy. Lately, stress was causing more PMS breast tenderness. She had been diagnosed with fibroids in her 20's (2 in one and 1 in the other breast) and they were always tender during her periods, 6/10 on a pain scale.

She reported that she was not taking enough of her powders and wanted the daily amount

to be measure into separate baggies. She decided that she did not want to add any new herbs yet, either for endurance or for PMS and fibroids, but wished to continue with the herbs that were helping a lot.

Third follow-up, 8 weeks later, November, 2013

It was observed that RM looked very much better. Her face was shining and she seemed very relaxed, even though her hands still twitched and her eyes still blinked fairly frequently. Pulses and tongue observation were not done.

RM reported that she was managing stress much better, sleeping better and had less anxiety and anger. PMS symptoms except for tender fibroids were fewer and less intense. Endurance and stamina were improved but she wanted a boost. Her only digestive upset was now with kale, so ways to cook it to lessen the indigestion were suggested to her. She'd had the stomach flu and wanted an herbal remedy to help for another time. She'd had a cold and was told how to make an elderberry, lemon and ginger remedy and a boneset oxymel. She felt that this would now be a good time to begin working on the breast fibroids.

To the summary of relevant physiology was added:
Hormone imbalance (PMS, fibroids)
Stagnation (PMS, fibroids, blue sub-lingual veins)

Herbal actions: lymphatics and hormone modulators were added.

Herbal support formulas:
1-3 remain the same, with the option to substitute passionflower and oats when skullcap was unavailable
4. Add eleuthero as a simple, ½ tsp TID.
5. Lavender bee balm as needed for the stomach flu
6. Vitex, 2 ml once a day in the AM
7. Tincture formula of viola, calendula and red root to add to castor oil as a liniment for a breast massage every other day.

Other recommendations: It was suggested that she add omega-3's, and legumes and avoid methylxanthines (things with caffeine). It was also recommended she take vitamin E,

flaxseed oil and evening primrose oil, include seaweed in her diet 3 times a week, and increase her exercise.

Email follow-up, February 2014

RM had stopped the vitex because she said it was giving her bad gas. She had been doing the breast massage for about a month, but there was as yet no noticeable change to report.

Case Study – Mood support, sleep latency, menstrual difficulties

Emer McKenna

MC is age 30, 5'8.5", and weighs about 140lbs. I first saw her in clinic on January 22, 2013, she was 29 then. Her primary concerns were anxiety and depression, skin dryness and eruptions, and poor sleep. She described alternating between mild anxiety and depression with one or the other being worse at different times in her life but in the past three years there had been a shift that felt worse, less manageable, and more hopeless. Her skin was sensitive to heat, cold, sun, and different foods particularly wheat and sugar. She viewed her skin as the canary in the coalmine of her body. She expressed having poor circulation, perennially cold feet, purple-looking legs in the winter, and blotchy skin on her face and neck when she was stressed. The blotchy skin thing was new to the past two or three years. Her sensitivity extended to her sleep concerns. When she ate wheat or sugar, or even drank juice or ate a piece of fruit, too close to her bedtime, she would not be able to get to sleep. If she drank alcohol she would not be able to get to sleep. If she stayed up too late, past approximately 12:30 or 1 am, she would not be able to get to sleep. When she was able to get to sleep she would sleep well, but lightly, and if she were awoken in the night she would not be able to fall back asleep. Not being able to sleep and anxiety compounded one another. She also described waking up in the morning with her heart racing and feelings of panic.

M's digestive system was sensitive, obviously. She described needing to eat simple foods to avoid digestive upset and bloating. She noted sensitivities to wheat, gluten, sugar, and dairy. She moved her bowels about once a day. Her stool was generally a light to medium brown and formed. When her digestive system was not doing well she tended to loose stools.

M said she drank at least a gallon of water a day and described herself as a very thirsty person. Her urine tended to be clear and she felt as though she drank and drank but did not feel hydrated.

During our intake, when the topic of menstruation came up, M informed me that recently her periods had become very light and much further apart. Her cycle was 28-34 days long and she bled for only 3 days. She used to have more "normal" periods and this change had come on gradually in the past couple of years.

M has a history of migraines that began when she was a child, possibly related to a head injury she sustained at age 5. They peaked in intensity about 3 or 4 years ago and that lasted for 2 years. Eventually she eliminated several different foods from her diet and she found that gluten and sugar were major triggers for her migraines. She also went through a huge breakup and Hurricane Irene, moved houses, and became more physically active. Her migraines were no longer a primary concern although she did describe getting more minor headaches behind her eyes recently, perhaps related to her job working in front of a computer.

Her pulse was thin, slack with some flooding in the first two positions, on the right hand side, at the deep level. Her tongue was pale, very quivery, with a white coat only on the back third of her tongue. It was moist on the edges and dry in the center.

My energetic assessment of M was Blood Deficiency and Liver Stagnation leading to Qi Depression. Signs of Blood Deficiency included her thin and slack pulse, pale tongue, and light menstruation. Signs of Liver Stagnation were her light stool, very sensitive skin, headaches behind her eyes, and her frustrated depression. Physiologically she seemed to be suffering from adrenal insufficiency and HPA dysregulation. Clues of this were the recent traumas of breaking up, Hurricane Irene, and having to move. Her issues with sleep and waking up in the morning in a panic with her heart racing were also indicators.

My therapeutic goals were to support her adrenals and HPA function, build the Blood, and move and support the Liver. The actions required to achieve these goals were: nervine, adaptogen, Blood builder/mover, hepatic.

M had told me that she was extremely sensitive to the bitter taste so we had a conversation about all the benefits bitters could offer her and she agreed to be brave and try them for a while. Since she was so sensitive, I wanted to give her a bitter formula that was mild, but would still do the job.

I gave her a tincture formula as follows:
3mL dong quai
2mL ashwagandha
1mL burdock
1mL blue vervain
.5mL cardamom

The dosing instructions were to take 1/2 tsp, three times per day, before meals. I chose dong quai for it's blood building and moving qualities and mildly bitter and sweet taste; ashwagandha as the primary adaptogen, to build the blood, and to promote sleep; burdock as a mild bitter and hepatic and for her skin concerns; blue vervain for the liver and as a nervine; and cardamom for flavor and as a catalyst.

I also gave her the following tea formula:
2g skullcap
2g wood betony
1g tulsi
1g anise hyssop

M did not flowery tasting teas at all so I was restricted considerably in the plants i could choose for her. I wanted to give her a tea to encourage her to drink something other than plain water in the hopes that that would prove more hydrating. I chose skullcap as a leafy tasting anxiolytic; wood betony as a nervine and digestive; tulsi as an adaptogen and for her skin, and anise hyssop as a nervine for her Heart and Liver and to make the tea somewhat less drying. I also recommended that she drink switchel as an electrolyte better to improve hydration.

I heard from M about one week later. She did not like the tea, it gave her an itchy feeling in the back of her throat and she wondered if I could change it. She still was not open to anything flowery-tasting. I wasn't sure exactly what was going on with the throat itchiness so I consulted with Betzy who advised me that it was likely the essential oils in tulsi and advised me to add oats to make it more moist. I did this and dispensed the formula again with an additional 2g of oats. Unfortunately, that did not help, M reported only drinking her tea sporadically, and as far as I know she never refilled it. Looking back now, I think that M was sensitive to dryness, likely related to her lack of Blood, and that the tea was too drying

for her. I think linden could have helped make the tea more palatable, but unfortunately has a distinctly flowery taste and she likely would not have tolerated it anyway.

M is a friend of mine so we had a lot of informal communication regarding her progress. At first she found the bitter taste very difficult to swallow and had to force herself to do it. A few weeks later I received an excited report that her period was heavier and more red. This trend of improvement continued and she got to the point where she loved the taste of her bitters and would carry her bottle around and take swigs off it. She had reported that she seemed to be increasing her dose slightly with the swigging and I gave her my blessing. She also told me that bitters was making her stool darker and her bowel movements more regular.

I next saw her in clinic on September 16. She was a little late to her appointment and had some emotional stuff going on that she needed to talk about and that comprised most of our follow up. She told me that improvements in her periods had continued, that she was having much less difficulty falling asleep at night, and that her mood was feeling somewhat better, even in the face of stress. I found her pulse to be thin, tight, and still slack. Her tongue was still pale and quivery though more moist. She reported feeling muscle pain while sitting. I felt that M needed even a little bit more blood building than she was getting and i decided to add 3mL of white peony to her tincture, to synergize with the dong quai to build blood and as an antispasmodic. She took the new formula to California this fall to work but her backpack was stolen and she lost it. Being off her formula for a couple months made her even more aware of it's benefits in her life. In correspondence with me she told me that her sleep is much more solid now, her skin has mostly cleared up, her "poops are AMAZING," and that her periods have become more regular. She even found that taking bitters before having alcohol prevents sleep problems and hangovers. Since she has been off the bitters, for the past two months or so, her periods have gotten lighter again. It seems as though her Liver and Blood need some more tonifying but that she is on the right path and that these plants have made a huge difference in her health and will likely continue to do so.

Case Study – Dermatographism, recurring vaginosis

Elise Walsh

Female 36 y/o

Background / History:

The client's primary concern was *dermatographism* (also called *dermatographic urticaria* or *skin writing*). When any part of her body is scratched or rubbed, her skin becomes raised and inflamed, forming wheals or welts. She experiences it on her back, and on seams of clothing in particular. The condition is windy in nature, arising at different places on her body. It becomes worst when she is lying in bed trying to sleep. She first experienced this condition when she stopped lactating two years ago. She finds relief from anti-histamine creams about once a week, but does not want to rely on them long-term. She has a sense that it may be stress-related.

The client presented with high stress levels due to her work in the field of data assessment and analysis. She did not report a sense of meaning in her work and her working environment was one where she did not feel valued, which caused a sense of anxiety. Working over 50 hours each week, she had a desire to spend more time with her daughter. Her health was also a source of stress. She found alone time rare, mood "ok", and was waking unrested regularly. Upon rising she also felt stiff and sore in her back, which resolved soon after rising.

Her second concern was reoccurring vaginal infection, which involved a greenish discharge and occasional itching. A test done by her doctor after our first meeting confirmed it was bacterial vaginosis. Her cycle lasts 30 days, with 4-5 days of bleeding. Over the past three month period she had one month of "normal" flow and two months of nearly absent flow. PMS involves some lower back pain, and mild emotional sensitivity. She has been anemic in the past, although current hemoglobin levels are at the low end of normal at 12.7. The client also reported pain with intercourse.

Her vegetarian diet is rich in vegetables, fruits, beans, soy, nuts, and seeds. The onset of the dermatographism encouraged her to get tested for food sensitivities – she found she is sensitive to eggs and brewer's yeast. While she is not outright allergic to dairy, she eats it sparingly. Having been vegan in the past, her oils sources include canola and olive oils, avocado and Earth Balance for cooking.

Digestive symptoms involved some bloating, a mild alternating diarrhea and constipation pattern which tends towards loose stool, passed 1-2x/day. She experiences hemorrhoids every six weeks, which are somewhat painful and began with pregnancy. She applies a topical salve to support wound healing.

She has a tendency to work through her illnesses, which generally reach the lower respiratory system and persist up to two weeks. She had one sinus infection in the past year, in previous years she did not become sick as readily.

Her blood pressure is low to normal 90/60, she experiences cold hands and feet, and bruises easily. Skin and hair are dry.

Assessment:

The precipitation of her skin condition at the same time as she finished lactating was suggestive of possible endocrine imbalance. The dermatographism, recurrent vaginal infection, painful intercourse and dry skin were suggestive of poor tissue integrity.

The client presented as a vata-pitta constitution. She is highly driven, action oriented, experiences poor circulation to the periphery and work-related anxiety, suggestive of a pitta imbalance. Her pulses were thin suggesting deficiency of fluids (possibly blood and yin). They had generally good force though the fire pulses were the weakest. Tongue was scalloped, with a sulcus, which along with her digestive symptoms and hemorrhoids pointed towards a need for digestive support. The variability in her digestion, windy skin rash, absent sense of social safety, and tendency to forget to breathe were suggestive of a vata constitution and/or imbalance.

Plan:

Our strategy has been to support tissue integrity, promote endocrine system balance, tone down her inflammatory response through tonification of the liver, support digestive function, build blood, reduce perception of stress and shift stressors. The following tinctures and tea along with dietary suggestions were utilized:

Tincture:

5ml Fresh Nettle leaf

3ml White Peony root

3ml Shatavari root

2ml Mugwort leaf and flower

1ml Burdock seed

1ml Stoneroot

= 15ml daily dose: 1tsp TID

Tea infusion:

5g Goldenrod flower

4g Tulsi leaf and flower

3g Gotu Kola leaf

3g Lemon Balm leaf

=15g/day

Other recommendations:

Algae-based source of Omega-3 EFAs

Blueberries as vascular tonic

Focus on colorful fruits and veggies

Follow-up:

Client returned two months later (Follow-up #1, June) having utilized the tincture, tea and omega-3 supplement consistently. As well as starting a new, more meaningful job, she found improvement in her skin condition. In the past years, May has been the worst month for her skin condition, but this year she has seen improvement.

She found an asthmatic pattern arising, however, involving a sense of tightness in her lungs and an inability to get enough air into her lungs. The triggers seem to be anxiety-provoking experiences (speaking in front of large groups) and when out of breath (pulling her daughter up a hill). We added 1ml of Reishi to her tincture (replacing the Stoneroot), to give her formula more of a focus on reducing the inflammatory response in her skin and lungs.

Bacterial vaginosis has continued to persist for her, we discussed using a garlic suppository and boric acid protocol, along with tissue tonification and endocrine balancing strategies through herbs and diet.

After using her formula for four months (Follow-up #2, September) and shifting job stressors she found complete relief from pre-sleep onset dermatographism. When she ceased using her herbal formulas, the itching returned, along with asthma and hemorrhoids (stoneroot was added to her formula again).

Her emotional health has been good, though her menses are still scant. Energy is all right, she still feels tired upon waking after seven hours of sleep. We discussed using a whole food fat source such as butter, coconut oil, and olive oil to replace Earth Balance.

Discussion:

The herbal formulation has been effective for management of the inflammatory skin condition through minimizing the hyper-reactive mast cell response in her skin and supporting tissue integrity. The herbs also support her endocrine system balance by estrogen enhancement, yin and blood building, and liver tonification. The client plans to continue to work with these formulas in the coming months. While this client-practitioner relationship will conclude at the end of the clinical internship, future goals should be to build blood in order to support a menstrual flow. The client plans to continue her own tea blends and to work with a Burlington herbalist as the need arises.

Case Study – Lower leg edema, sugar cravings

Susan Nova Staley

I first met this client on July 16, 2013 at the VCIH Student Clinic in Montpelier, VT. Client is a 37yr old female, roughly 5'4'' and 140lbs. Tongue appeared pink in color with a slight white coat that yellowed and darkened towards the back. The tongue was dry in quality with a slight quiver. It was slightly swollen with a red tip that was a bit pointy. Pulses were tight in quality and more deep than superficial; some weakness in the Kidney and Pericardium positions, otherwise present.

Primary concerns were to reduce swelling and pain in her lower legs, improve the skin condition on her face and reduce her sweet tooth. Client works as a full time pastry chef where she is constantly on her feet (which contributes greatly to the lower leg edema). I observed varicosities and spider veins on her legs. Her complaint of edema was visually represented and presented as non-pitting on both sides. She had experienced this swelling for about three weeks. Current pain was around 1 on a scale from 1-10 though rated at a 5 during running (which she had stopped due to discomfort). Also found relief from compression stockings. 3-second capillary refill time on exam. Also acutely presenting was a swelling that her acupuncturist identified as a Baker's Cyst behind her right knee; this was causing her pain and difficulty reaching full range of motion. There was also an acute pain in her right ankle.

Client has a history of asthma that was significant as a child but now presents only in the very cold weather, as well as allergies (mold and pollen). Diagnosis of Plantar Fasciitis 15 years ago and has since changed to barefoot running shoes that have offered great relief. She wears these shoes at work now. Family history of Cardiovascular system weakness (stroke, heart disease, high blood pressure and diabetes) as well as some thyroid disorder.

Client's experience of edema started 15 years ago. In the past she experienced some "arm bloating" that was relieved by lymph massage. Client exercises daily preferring

running, yoga, and weight training. Edema had stopped her present running practice. Client reports experience of heat and sweating after exercise well after stopping exercise. She rated her daily energy levels around 7.5, though she says that her husband would rate her at a 12. ☺ Without daily exercise she feels “cranky” and as though “something is missing”. Client also reports having experienced low libido and poor concentration over the past year.

Sleep is variable; it takes her a while to fall asleep and she wakes 2x/night on average to pee. (Stops fluid intake around 8pm each night). She can be distracted by making lists in her head regarding past, present and future as well as minor obsession with “doing the right thing” for self and others. Part of her difficulty sleeping is that her schedule doesn’t permit her to sleep the hours she prefers (2am-9pm) so she uses an alarm clock and feels unrested upon waking (6am).

Client sips water throughout the day, drinking an average of 64oz . She reports that she urinates all the time and that the urine is clear or close to clear in color. And although she reports that she “feels pretty quenched” she will frequently pee before leaving her house in the morning and arrive to work feeling the need to urinate again, which may only be 10 minutes later. She also says that she doesn’t feel fully voided. No history of UTIs or painful/burning upon urination.

Client reports sensitivity to changes in temperature; that her hands and feet will be freezing cold in the winter-time and that her veins will tend to pop out and swell during warm weather. Hands swell in heat. Skin becomes “worse” in heat and humidity with increase in “bumps” and be oiler. Client generally prefers cold over hot. Muscles store tension easily (esp bisepts and neck) Tends to pull muscles in her back.

28 day menses cycle. Scant, 2 days of bleeding for the past 10 years, down from 3 days. Starts red with no clotting. She experiences some swelling in her breasts and a little moodiness premenstrual with little to no experience of cramping (1x/year mild). Menses is not a disturbing experience.

Diet is well rounded and has variety. It includes ample fruits (berries) and vegetables. Sweet tooth causes client to indulge in daily desserts (usually in the form

of a baked good). She also may avoid “rich” foods because they “make her feel terrible”. My impression was that she feels heavy and uncomfortable after eating greasy foods and that she avoids them. My impression was that she thinks greasy foods make everyone feel bad and everyone should avoid them, although this may point to some insufficiency of liver function. She practices intermittent fasting 1x/week. Whole grains in diet in addition to refined carbs. Some use of cooking with olive oil (her favorite fat).

Initial constitutional assessment was imbalance between water and fire, and some interaction between the two. Excess heat (potentially coming from dryness) seen in her preference for cold over heat, tendency to become irritated in hot weather and the redness at the tip of her tongue. Some suggestion of Pita from relief felt through exercise. Water element is suggested from the fluid imbalance seen in edema, history of lymphatic insufficiency, frequent urination and dry tongue. Other notable phases are wood related to liver function, issues with tendons and ligaments and muscular tension. Potential tension/stagnation in Liver. She also gave the impression of “brewing beneath the surface” so to speak, or perhaps the feeling that she works to present everything as “ok”. Also worth noting is the Earth element seen in sweet tooth and tendency to worry/want the best for others.

Initial physiological assessment would show some venous insufficiency/poor circulation/Lymphatic weakness (decreased liver function, family history of cardiovascular challenge, sugar consumption, being on feet all the time and tension in the musculature). This insufficiency looks like edema, varicosities, swollen hands and feel in the heat and frozen hands and feet in the cold. Lymphatics may be a life-long area of weakness. Liver function is diminished/burdened and seen in skin rashes, tension, poor circulation of blood and trouble digesting fats.

Initial goals included: support liver function, decrease sweet tooth, increase circulation, support lymphatic function, support cardiovascular system, decrease tension (improve sleep), decrease edema and decrease stress.

Herbal Actions: Hepatic/bitter, circulatory stimulant, lymphatic, astringent, cardiovascular tonic, adaptogen, nervine (mild/relaxant), anti-inflammatory.

There were 3 initial formulas. The first was a tonic 15ml (1.5tsp BID) and included 5 parts Hawthorne, 4 Dan Shen, 3 Schisandra and 2 Prickly Ash. Second was to be taken during acute edema and was comprised of 2 parts cleavers, 2 parts Red Root and 1 part Horse Chestnut, 5ml BID. Also included was a tea totally 5g/day this included 2 parts Dandelion Leaf, 2 parts Linden, 1 part Peppermint and 1 part Tulsi. Last piece of the protocol was a bitter formula to be taken in the evening before dinner or afterwards in an effort to begin the wind down process as well as reduce the sweet craving some through the bitter taste. This formulas was 2 parts Dandelion root, 1 part Mugwort, 1 part Chamomile and 1 part Lavender to be taken 5ml prn.

Hydrotherapy was suggested for supportive therapy and dietary suggested included 1/2c of blueberries daily, increasing garlic in her diet and eating more greens to decrease sugar cravings. Also to switch to saturated fats for cooking and to reserve olive oil for dressings.

Follow-up concerns were to ask into blood pressure, cholesterol levels and if she is experiencing any heart palpitations. Also questions regarding blood sugar levels (tingly feet? Vision?) And if she has had her thyroid levels tested in recent history or to suggest such tests in the future because of family history.

Client returned for a follow-up visit on September 3rd of the same year. She disclosed that she had been very compliant and followed the protocol completely since receiving the formulas and had refilled them twice. (yay!) She reported that during a gym class she found herself in a kneeling position she had otherwise been unable to achieve and she accounted her success to the herbs. She also said that her eyes are a little blurry in the morning and increasingly so. She spoke of worry about a mole on her husband and I encouraged her to make an appointment at the dermatologist for him. Her tongue was slightly less swollen looking though otherwise the same and pulses the same.

I want to keep an eye on the client with Dan Shen because of her scanty periods. 2 ml of Dan Shen was added to the tonic formula to help build blood, Yin and provide a bit more antispasmodic action. New formula as of September 13th 2013 is: 5 parts Hawthorne, 4 parts Dan Shen, 2 parts Schisandra, 2 parts Prickly Ash, 2 parts White

Peony. Other formulas remained the same. No further contact with client at this time as she lost access to her car and was having trouble getting to the clinic. Will be in touch through email shortly.

Health Access Issues

Emer McKenna

Migrant workers in Vermont face a variety of challenges when it comes to accessing health care. Firstly, many agricultural migrant workers in Vermont have not arrived through official channels and therefore lack the proper documentation to work in the United States.[1] There are programs that allow for migrant workers to enter the United States legally, but few permits are given out (400 for the entire state of Vermont)[2], and they have stipulations which allow them to be given only to laborers who are both migrant AND seasonal.[3] In Vermont, the largest industry employing migrant workers is dairy[4]. These jobs are year-round, so these H2-A visas are not available to workers in this field.[5] Lack of documentation prevents workers from accessing state programs such as VHAP or Catamount, which are available to residents and foreign workers with visas.[6]

A lack of documentation also puts workers at risk of deportation by US Immigrations and Customs Enforcement (ICE), a department of Homeland Security.[7] To avoid potential exposure to law enforcement, some workers avoid leaving their farm at all for any reason.[8] Of course, some workers do leave their farms to buy groceries, visit nearby friends or family members, and to visit doctors.[9] Quite recently, outside of a dental clinic in Richmond, a migrant worker was stopped by local authorities for “suspicious activities” and subsequently turned over to ICE.[10] It can be extrapolated, then, that state and local police or immigration officials may target health clinics and other organizations that serve migrant workers and engage in racial profiling to apprehend undocumented foreign nationals. Though some districts in Vermont have passed Bias-free Policing Policies[11] racial profiling is still considered a problem in much of Vermont.[12] Vermont has been named by the Mexican Consulate in Boston as the state with the most severe immigration enforcement in the Northeast[13] and fear of deportation was ranked highest among barriers to receiving health care in one study which surveyed 70 workers in northern and central Vermont.[14]

The other most frequently reported barriers to health care were lack of insurance (discussed above) and high cost of care[15] [16]. Most migrant workers who leave their countries of birth to work in the US plan on coming for a short time, working as much as possible, saving money, supporting family members abroad, and then returning to their countries.[17] Therefore, many workers are reticent to spend money on health care unless facing a serious medical condition, in which case workers often return to their home country to receive care.[18] When workers were asked if they would be more likely to seek health

care if they had access to a free or sliding-scale community clinic, one hundred percent of those surveyed said yes.[19]

Other frequently reported issues for migrant workers in Vermont are transportation and language barriers.[20] [21]

Population Demographics

An accurate estimate of the number of migrant farm workers in Vermont is difficult to attain because of issues such as obtaining access to marginalized and vulnerable populations, the desire of these populations to avoid interaction with government officials (or those perceived as such), and the inherent fluctuation of migrant populations due to political or economic factors.[22] That said, the most frequently cited figures come from the Vermont Agency of Agriculture, who commonly cite between 2000 and 3000 workers[23] and from the Vermont Migrant Education Project who estimated 1500 workers in Vermont in 2009.[24] A study conducted by the Vermont Department of Health in 2007 cited a total migrant worker population of 2500, 1200 of whom resided in Franklin, Grand Isle, and Addison Counties.[25]

Compatibility with the Clinic

A number of historic influences have combined to create the modern cultural make-up of Mexico and Central America. The ancient Maya[26], Aztecs[27], *conquistadores* from Europe[28], and Africans brought to the region as slaves,[29] have all contributed to the pool of knowledge which informs modern day traditional medicine of Mexico and Central America. This is significant since most migrant farm workers in Vermont come from Mexico or Guatemala, with an increasing number migrating from indigenous communities in southern Mexico.[30] The use of traditional herbalists (*yerber@s*[31]*),* healers (*curander@s)*, midwives (*comadron@s),* bone setters (*gueser@s)*, and massage practitioners (*sobadores)* is common, in both indigenous and non-indigenous communities in Mexico and immigrant and native-born populations in the United States.[32] [33] [34]. Even without the services of an herbalist, people of Latina/o or Chicano/a heritage in the United States and Mexico frequently use herbs such as peppermint, cinnamon, chamomile, eucalyptus, aloe vera, yarrow, et al for various complaints.[35] The most commonly reported health issues of migrant workers in Vermont have been skin problems such as dermatitis and fungal infections of the feet, soreness and/or injuries caused by repetitive stress and hard physical labor, respiratory conditions, gastrointestinal issues, dental concerns, and vision problems.[36] Also mentioned were high blood pressure, parasites, anxiety, and depression.[37] [38] Migrant

farm workers are an underserved population, in terms of health care, in the United States in general, and Vermont, in particular.[39] They are also a population under a significant amount of stress, due to long hours, hard labor, social/cultural isolation, fear of law-enforcement authorities, and exposure to chemicals and other occupational hazards.[40] Our clinic could be an incredible resource to these populations, and be an excellent health care adjunct, capable of addressing many commonly reported health complaints.

Issues for the Clinic at VCIH

In order for the clinic to become more of an open resource to migrant farm workers in the area, there are certain issues that will need to be addressed. Matters involving interpretation will require significant attention. Intake documents will need to be translated into Spanish and once these documents are filled out by the client, a practitioner may need assistance in comprehending them. Setting appointments may also be an issue for our clinic if there are not bilingual staff members answering the phones. Since interpretation will take away from the actual time spent in conversation with a client, the length of appointments may also warrant consideration.

For the purpose of this research paper, extensive enumeration of potential clients within our service area was not conducted. Due to the transience and immigration status of migrant farm workers, these populations are not particularly visible or easily accessible to outsiders. If the clinic decides to go forward this project, outreach will be conducted to both workers and their employers. I believe this process will give a much more concrete estimate of the number of people our clinic could look forward to serving.

There are still many questions in my mind, regarding transportation, length of travel, timing of appointments, safety from immigration, and the efficacy of serving clients in Montpelier, Burlington, or elsewhere. I believe the connections made with nearby communities through outreach would greatly help illuminate the best ways to provide herbal resources to migrant workers in Vermont.

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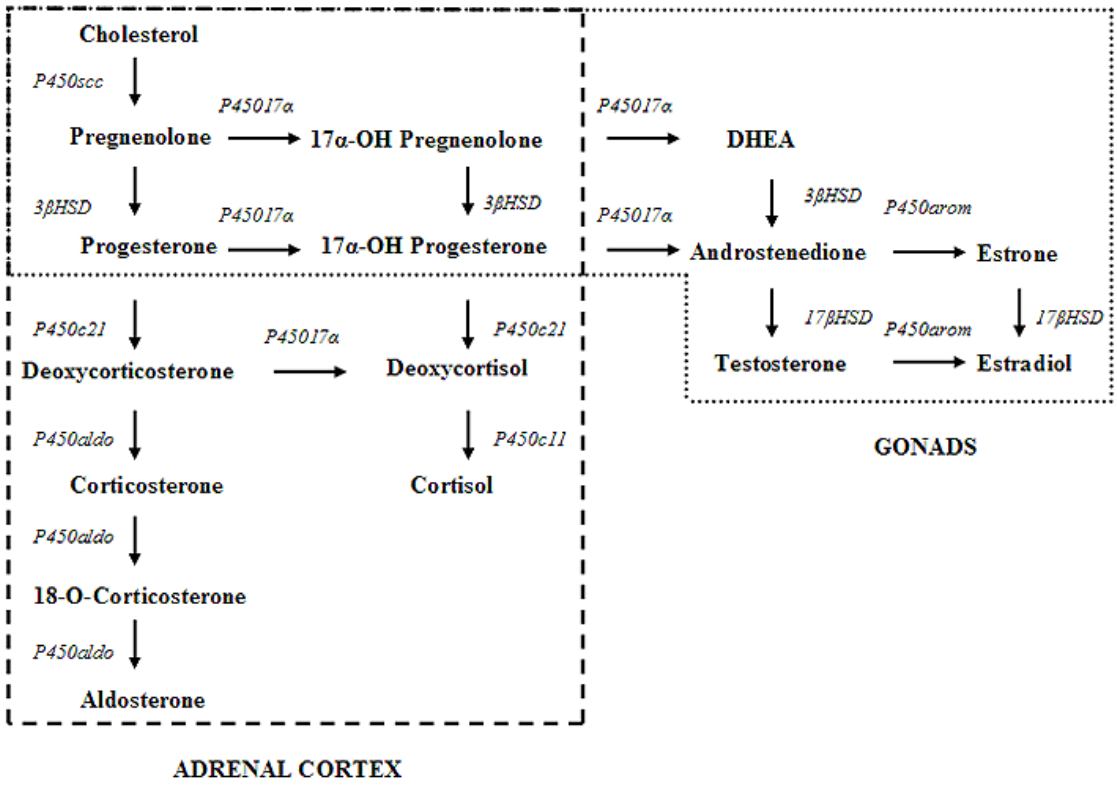
Why You Should Chill Out: Stress Hormones Affecting Endogenous and Exogenous Sex Hormones

Kelly McCarthy

I want to explore the interconnection between stress hormones and our endogenous sex hormones. Humans have evolved to cope with life-threatening stress quickly and efficiently, but these mechanisms can get out of balance when the stress is chronic or prolonged, without a chance to recover and come back to a state of relaxation. I want to focus on what the effects are when our bodies prioritizes making cortisol, a stress hormone, instead of making our endogenous sex steroid hormones. I hope to work with trans* clients (including those who choose to take exogenous sex steroid hormones) so first understanding the body's process regulating its endogenous sex steroids under stress is a key part of my understanding.

The Hormone Cascade

The hypothalamic-pituitary-adrenal (HPA) axis controls our stress response via hormones.



The hypothalamus is considered the master gland, directing many life sustaining functions. It secretes Corticotropin Releasing hormone (CRH) in response to stress, which stimulates the anterior pituitary to release Adrenocorticotropic hormone (ACTH), which increases the production and secretion of corticosteroids, most notably cortisol and aldosterone. When cortisol is released from the adrenal cortex, it provides a negative feedback to CRH and ACTH, telling both of them to decrease activity so less cortisol is produced. The smooth function of these feedback loops are essential to regulated HPA activity.

As seen in the hormone cascade chart, all steroid hormones are derived from cholesterol. Cholesterol converts to pregnenolone which can take one of two paths, to either be converted to progesterone or DHEA. From progesterone, we primarily get cortisol, although progesterone can convert to androstenedione (instead of cortisol) which can convert to testosterone or estrogens . From DHEA, we first get androstenedione, which then converts to testosterone and then estrogens.

Pregnenolone and progesterone are the places along the hormone cascade at which the production of cortisol can be favored over that of the sex hormones. Evolutionarily, surviving the stress of a given moment gets prioritized over the potential to make a baby in the future. When people experience high levels of stress, be it physical or mental, the body will use the pregnenolone or progesterone to make cortisol rather than making DHEA or androstenedione. This prioritization of cortisol is one of the ways hypothalamic-pituitary-adrenal (HPA) axis dysregulation can manifest.

Cortisol and DHEA are measurable ways to assess level of stress in the body and many studies are conducted assessing the ratio of cortisol and DHEA. Because cortisol and DHEA act in the body to balance each other, when blood levels of one are high, the other is low. DHEA and cortisol have an inverse relationship. I will look at their physiological effects and some of their pathophysiological effects when they are out of balance.

Cortisol is a glucocorticoid steroid hormone, released from the adrenal cortex in response to stress and low blood glucose levels. Its primary functions in the body are to suppress the immune system, metabolise macronutrients, and increase blood glucose levels through gluconeogenesis. Cortisol uses two types of receptors, glucocorticoid and mineralocorticoid. The glucocorticoid receptors have less binding affinity, and the mineralocorticoid have more. Because plasma levels of cortisol have not only circadian fluctuations throughout the day, but can also spike in response to stress, both receptor systems ensure that cortisol can be fast acting (glucocorticoid receptors) or slow acting (mineralocorticoid receptors), depending on need. “Under pathological conditions, for example, severe major depression or Cushing's syndrome, continuing corticosteroid

hypersecretion leads to overexposure of GRs (glucocorticoid receptors) and MRs (mineralocorticoid receptors) which, in turn, are down-regulated." (Holsboer). This offers one explanation of how having chronically high plasma levels of cortisol can disrupt the negative feedback loops that regulate our stress response.

Cortisol is kept in balance by DHEA-S's antiglucocorticoid activity. DHEA-S is the most abundant circulating steroid in the body, produced by the adrenals. Testing serum levels of DHEA-S is one way to test for adrenal dysfunction, as a progression towards adrenal fatigue is characterised by the inability of the adrenals to produce adequate DHEA-S. The sulfate group (-S) can get removed to create DHEA, and DHEA can have the sulfate group added back on the create DHEA-S. DHEA functions primarily as a metabolic precursor to testosterone and estrogens; there is limited information about its function in its own right but it seems to act as a neurosteroid and to have mild androgenic properties (Friess). Healthy levels of DHEA means healthy LDL levels, strong bone density, normal sleep patterns, as well as positively affecting emotional mental health, memory, and cognition. DHEA helps us recover from emotional and physical stress. It is sold as a supplement with claims that it is the source of the fountain of youth. If only.

As the primary precursor to our endogenous sex hormones, DHEA is responsible for converting into testosterone and estrogen. Testosterone acts as an anabolic and androgenic hormone in people of all genders, although it is circulating unbound to sex hormone binding globulin (and therefore more readily available) at about a 7 times greater concentration in people with testes. Testosterone increases muscle mass and strength, increases bone density, acts as the precursor to DHT, which is responsible for secondary sex characteristics like deepening voice, pubic, facial, and armpit hair, and adult body odor. It is also responsible for the maturation of genitalia and plays a role in sexual arousal. Testosterone inhibits alpha-2 receptor formation, which are adrenergic cell receptors that sense the circulating epinephrine and tell the hypothalamus there is enough epinephrine circulating, which stimulates lipolysis. So testosterone acts as a direct feedback inhibition of the hypothalamic sympathetic discharge and inhibits fat storage. (Mitrovic)

Testosterone can be aromatized to three forms of estrogen - estrone, estradiol, and estriol. Estrogens are responsible for female secondary sex characteristic, such as breast development and are involved in the build up of the endometrial lining. They also have an important role in healthy bone density, encourage fat stores, influence a healthy lipid profile, protect vascular endothelium, affect blood coagulation. This is all to say these sex steroids do have profound effects on our bodies other than activities related to reproductive sex.

In order for there to be adequate plasma levels of DHEA from which to make these sex hormones, there needs to be a healthy ratio of DHEA and cortisol. I will look at two possible ways for cortisol and DHEA to get out of balance, first high cortisol/low DHEA, then high DHEA/low cortisol.

Having high levels of cortisol circulating in the blood raises blood sugar, and high levels of blood sugar over prolonged periods of time will lead to insulin resistance by downregulating the GLUT-4 insulin receptor. The detrimental effects of insulin resistance are beyond the scope of this paper, but it is directly implicated in some of the most prevalent health concerns of modern American's: Type II Diabetes, Metabolic Syndrome, atherosclerosis, heart disease, and obesity. Cortisol in the bloodstream also downregulates the inflammatory process by inhibiting IL-2 receptors on helper T-cells, which means the interleukin 2 cannot produce as much Th2 response, leading to a Th1 dominant immune response.. Th1 dominance is often expressed in auto-immune and high inflammatory conditions. So prolonged high cortisol will have a negative effect on immune function.

The correlating low levels of DHEA have been implicated in a variety of medical conditions, including rheumatic disease, cardiovascular disease, and immune system disorders. (Imrich, 2002; Thijs, 2003; Chen, 2004; Dharia, 2004). Low DHEA is often seen in people with clinical depression. (Zaluska). A 2013 study showed women with major depression have lower levels of androgens (which are metabolites of DHEA), as compared to the control group, and within the women with major depression , there were even lower androgens if they had severe anxiety. (Oulis) Paradoxically, another 2013 study looked at DHEA-S levels in women with PCOS as compared to women with PCOS *and* diagnosed with either Major Depressive Disorder or Generalized Anxiety Disorder; the women with either MDD or GAD had significantly higher levels of DHEA-S than those without these psychological conditions. (Zaluska). PCOS is a condition characterised by sex hormone dysregulation in people with ovaries, and comparing these two recent studies illustrates the interconnectedness (and complicatedness) of stress, DHEA, and sex hormones.

If cortisol levels stay high over a prolonged period of time, eventually the cortisol producers - adrenals, ovaries, and testes, will not be able to keep producing adequate amounts. Low levels of cortisol are often described as a flatline, where there is no cortisol spike upon waking in the morning, people experience high levels of inflammation, low immune function, and generally feel exhausted. Low cortisol levels will be seen with corollary high DHEA. Elevated levels of DHEA have been observed in connection with obesity and Type II diabetes, female hirsutism, and in people subjected to prolonged physical stress (Krobath. Bergfield 2000.) There is a strong connection between PCOS and elevated DHEA

(Yildiz). These are also conditions associated with elevated levels of androgens, which makes sense considering DHEA has mild androgenic properties and is the precursor to testosterone and DHT. Elevated levels of DHEA will inhibit cortisol binding, meaning cortisol cannot aid in mounting an appropriate stress response.

All people should have testosterone and estrogens acting in their body. In people with ovaries, about 60% of testosterone comes from the conversion of adrenal DHEA, the remaining 40% is produced by the ovaries. DHEA is also estimated to contribute 70% of estrogens before menopause and nearly all after menopause. Compare that to people with testes, where about 95% of the testosterone is produced in the testes; the remaining five percent is supplied by conversion of adrenal DHEA to testosterone. (McCormick) People without testes, therefore, are more at risk for overworked adrenals, because the body needs to use the adrenals for producing cortisol *and* sex steroids, and cannot rely on the testes to produce testosterone. The adrenals will prioritize making cortisol over making sex hormones, and an imbalance of these hormones has numerous, noted negative effects.

Considerations With Trans* Clients

This paper will look briefly at potential effects on the HPA axis in transpeople who are taking exogenous hormones. Identifying as trans* does necessitate cross-sex hormone replacement therapy, nor does it imply a gender binary, and many trans* people choose not to take exogenous hormones. Because of the immense variables, this paper can only hope to generalize some possible interactions between exogenous hormones and our bodies stress hormones.

Mental Health and Stress

Much of the literature looking at trans* people focuses on people who choose to take exogenous sex steroid hormones, although there is a significant amount of research looking at LGB and queer people, which can include trans* people who are and are not taking hormones. There is much research documenting that trans* people experience disproportionately high levels of stress, depression, anxiety, and suicide attempts. (Mustanski 2008. Nuttbrock 2002.) A 2011 study of nearly 200 trans patients reported 61% of them to experience depression and anxiety before beginning cross-sex hormone treatment. (Gomez-Gil) In 2013, 70 transsexual patients had their cortisol awakening response measured before beginning cross-sex hormone replacement -- overall, their cortisol levels were higher than non-trans patients. (Colizzi). According to a 2001 British study, “socio-economic and psychosocial handicaps are probably central inducers of

hyperactivity of the hypothalamic-pituitary adrenal (HPA) axis" (Björntorp). Knowing this, and not because trans* identity is one to be pathologized, a practitioner would be wise to inquire if a trans* patient experienced or perceived a high level of stress (whether currently or in the past), by virtue of being trans* in a gender-binary world.

Exogenous Hormones

Female-to-Male Transmen

Transmen who choose to take testosterone can opt for a range of doses, and commonly patients are aiming for similar circulating blood levels as cismen. I have looked at studies looking at the effects of testosterone in cismen to know how testosterone and cortisol possibly interact under "normal" circumstances.

In trials involving cismen, high cortisol inhibits the effects of T on target tissues and also downregulates androgen receptors. Chen et al. found "evidence that the opposing physiological effects of the androgen and glucocorticoid hormones are due to the direct physical interaction between their receptors at the transcriptional level," From this, one could extrapolate that even if someone is taking exogenous testosterone, if the cortisol levels are high, the testosterone may not be able to bind to receptors and act on target tissues.

There is also a concern with exogenous testosterone being aromatized to estrogen. In general, "the conversion of androgens into estrogens is favored by a pro-inflammatory environment." (Edwards). So if someone is in a state of adrenal exhaustion with lots of inflammation *and* they are trying to transition or maintain masculinity with exogenous testosterone, it could be possible that the exogenous testosterone is getting aromatized and not having the desired effect.

OB-GYN Nurse Practitioner Marcelle Pick focuses her practice on ciswomen with adrenal fatigue. According to her literature, she sees a strong connection where post-menopausal (or post-hysterectomy) ciswomen have pathologically high DHEA and this high DHEA makes imbalanced conversions either strongly towards testosterone overproduction or creating a situation of estrogen dominance (Pick) Both overproduction of testosterone or estrogen would have negative implication when someone is taken exogenous testosterone.

Male-to-Female Transwomen

Transwomen often will use an anti-androgen (such as spironolactone or finasteride) to suppress testosterone *and* a form of estrogen (oral, injectable, or transdermal) to encourage feminization. Spironolactone competitively binds with aldosterone, increasing

testosterone clearance and estradiol production. It also blocks the conversion of potent androgens to weaker ones at target tissues. It has been shown in multiple studies that the administration of estrodiol in ciswomen leads to elevated cortisol. Studies have examined the effect of exogenous estradiol in post-menopausal ciswomen on cortisol levels. In 2008, 2mg estradiol administered daily (which is a common dose for transwomen) to post-menopausal women was found to increase plasma levels of cortisol. (Edwards) A Mexican study showed administering estrogen to post-menopausal women increased DHEA and ACTH, both of which were low because of hypoestrogenism. The authors hypothesized this was because the estradiol was having a stimulating effect on the pituitary gland. (Fonseca).

Our body's propensity to prioritize cortisol combined with a high stress culture means HPA dysregulation is all too common. It is clear that endogenous sex hormones interact with the HPA axis, although we still have a limited understanding here. People taking exogenous hormones, whether to present in the world with a certain gender identity or to regulate their menopause, especially want to modulate their stress so at the very least, their bodies endogenous sex hormones and stress hormones are functioning as smooth as possible. It is in optimal health that our body can best take in foreign substances (be it food, drugs, or hormones) and process those substances to use or pass through.

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Some reflections on action-based classification of medicinal plants

Linden de Voil

For many contemporary herbal practitioners, the categorization of medicinal plants according to action on the body can seem as common as the air we breathe; the classification of a plant as astringent, expectorant, or vulnerary may be one of the first ways we understand that plant and its role as medicine. This system is so familiar that we may forget that classifying and applying herbs according to these action categories is just one approach to their use - certainly one with many useful applications, but not without its limitations.

We can gain some insight into the historical application of such classification by looking at a partial history of terms applied to the common medicinal plant mullein (Verbascum thapsus) within the Western European herbal tradition.

Although Dioscorides uses the classification astringent for other plants, he does not apply it to mullein, which is listed as a specific remedy "for old coughs" and said to help with the spitting of pus.[i] Hildegard of Bingen also suggests specific indications, recommending Mullein for "one who is hoarse or has pain in his chest."[ii] By the time of the electic physicians we see a clear transition into use of categories of actions; mullein is classified as a demulcent and anti-spasmodic, and a specific for cough with catarrh,[iii] as well as an alterative.[iv] Maude Grieve's 1931 herbal highlights its "markedly demulcent, emollient and astringent properties, which render it useful in pectoral complaints."[v]

Modern research on the uses of verbascum is limited. Contemporary herbalists commonly continue to apply the same primary categorizations seen since the eclectic era with varying emphasis on its demulcent, astringent and expectorant qualities.[vi] We often see it listed as having a virtual parade of actions; a quick glance into just one desktop reference text lists twenty-one actions for mullein, ranging from alterative to yin tonic.[vii] Some of these (antihistamine) may be specific enough to assist in applied use, though many (anti-inflammatory) are extremely broad. This points to one of the potential shortcomings of the action-based classification system: the application of an extremely long list of actions

for a single herb, which can quickly defeat its own usefulness, overwhelming any sense of how the herb is most appropriately applied. Jim McDonald offers one potential clarification by dividing classes of action into foundational actions and secondary properties. McDonald is not the only herbalist to offer an explanation of a subcategorical system. Culpeper famously interpreted Galen's system of degrees in his 17th century herbal;[viii] Hoffman offers another approach to primary and secondary uses,[ix] while Wood delineates a system very similar to McDonald's, based on pathological tissue conditions and affinities to specific organ systems.[x]

McDonald's foundational classes include astringent, demulcent, bitter, and nervine, among others, while secondary actions are "those properties attributed to it that owe their effect to one or more of the plant's primary actions," including categories such as anti-inflammatory, expectorant, or lymphatic. By this system, we understand mullein to have foundational astringent, demulcent, and relaxant action, with secondary actions including expectorant, anti-inflammatory, and lymphatic qualities.[xi]

In addition to the sheer number of actions applied to any given plant, there may be confusion about or overlap in meaning of some terms; the breadth of definition applied to some terms of classification may be another limitation within an action-based organizational system.

Expectorant herbs provide a relevant example here. Hoffman points out that the term 'expectorant' itself is often "used somewhat loosely to refer in a general way to remedies that 'do something' for the respiratory system,"[xii] as do the terms 'pulmonary' and 'respiratory tonic,' although pharmacologically expectorants are "agents that promote removal of mucus secretions from the lungs, bronchi and trachea."[xiii] Translations of historical texts present their own challenges in this arena. To return to a previous example: we read Dioscorides in translation to state that mullein helps with the spitting of pus. Does this simply mean that it has expectorant qualities, and if so by what action? Might there be alternative translations that suggest additional qualities, or more specific action?

As terminology continues to evolve with our understanding and interpretation of herbal actions, there is at least one instance where we we encounter the problem of significantly differing definitions for a single term, arising through shifting historical use. Medical phytotherapist Kerry Bone argues against the use of the categories of 'relaxing' and 'stimulating' expectorants, equating them with antiquated divisions in which expectorants "were formerly classified into stimulating and depressing groups, the former being supposed

to increase blood pressure and diminish secretion, the latter to lower blood pressure, increase secretion and promote expulsion. These terms are now generally discarded."[xiv]

However, other contemporary herbalists continue to use these categorizations to differentiate between those expectorants that facilitate increased mucosal secretion via irritation of bronchioles or thinning of sputum, and those that act, via reflex, as demulcents within the lower respiratory tract, simultaneously encouraging expectoration and loosening mucous by production of less sticky sputum that effectively 'pushes' out stickier mucous.[xv]

Not surprisingly, increased understanding of physiology and emphasis on pharmacology at the molecular level has shifted and complicated our understanding and usage of the action-based classification system; again expectorants provide an interesting lens to watch this development.

In 1927 Dr JA Gunn offered a system of categories of expectorants based on their site of action: local, via central nervous stimulation, directly on vagal nerves, or directly on secretory glands, and specified the drugs which have most action for each category, and for certain clinical presentations. He explained, "This classification is not to be regarded as a mere academic curiosity. It is upon differences in site of action that the superior suitability of certain expectorants for certain actions largely depends."[xvi]

Subsequent research clarified the expectorant action of substances which trigger the gastropulmonary reflex, inducing the preliminary stage of emesis, salivation, and thin watery secretion from goblet cells in bronchial glands, and linked the action with saponin-containing plant extracts.[xvii] Research also shows that expectorant herbs containing saponins function to reduce the surface tension of the secretions, facilitating their separation from the mucous membranes.[xviii] The demonstrated demulcent action of mucilaginous polysaccharides, which form a protective coating lining the respiratory mucosa, has been hypothesized to work through a similar reflexive action.[xix]

After tracing its historical legacy in traditional medicine, it comes as no surprise that mullein contain both saponins[xx] and polysaccharides.[xxi] By comparing the biomedical, evidence-based arc of classification with the action-based system of traditional herbalist McDonald, we arrive at some similar conclusions. This leads us finally to a major strength of the action-based classification system, which makes it so useful at this particular moment in time: the ability to help bridge traditional uses of herbs with the language and paradigm of orthodox western medicine.

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[vii] Mars, 208.
[viii] Culpeper, 376.
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[x] Wood.
[xi] McDonald, "Mullein."
[xii] Hoffman, MH, 320.
[xiii] Moini, 248
[xiv] Bone, 100.
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Permeability Matters:
A Primer on Leaky Gut and Related Auto-immune Conditions

Kelly McCarthy

Clinical herbal practitioner, Betzy Bancroft, relates that often people who come to the VCIH clinic are people with auto-immune conditions for which western medicine does not offer promising treatments. (Bancroft 2013) According to herbalist Paul Bergner, primitive people did not suffer from auto-immune conditions with the same frequency that modern people do. (Bergner 2001) Current research is showing the connection between lack of gut health and many auto-immune conditions. If looking at the human body as a microcosm of the macrocosm, one can see that as our external ecosystem is seriously struggling because of resource extraction, overuse, and factors too various to list for the scope of this paper, the internal ecosystem of our gut is in a state of disharmony for many people and auto-immune conditions are often seen in clinic. Herbal medicine has an important place in the intervention and prevention of leaky gut and the related auto-immune conditions.

The epithelial cells in the gut serve multiple functions: they create a protective barrier and they regulate the exchange of contents of the lumen (the external environment) with the inside of the body. (Silverthorn 2001). As a protective barrier, the cells are responsible for keeping out bacteria, pathogens, parasites, and other antigenic substances. But simultaneously the cells must allow nutrients from food and drink to pass from the lumen to the bloodstream. The inherently “leaky” junctions between these cells allow solutes across the barrier according to their size and charge. (Shen 2006) The lumen of the gut is also home to the largest concentration of mucosal cells in the body, known as the Mucosa Associated Lymphoid Tissue (MALT), which plays a major role in immune function. It is estimated about 80% of lymphocytes are located here. (Silverthorn 2001) MALT constitutes sites of great importance for both innate and acquired immune functions. (Turner 2009) If the mucosa is damaged, so is the body's ability to differentiate self from non-self and when the body can’t differentiate self from non-self, immune cells can attack healthy self. (Fassano 2005)

Ideally, the intercellular junctions are tight enough to keep antigenic material from passing through into the bloodstream. Many things in the modern world have been shown to irritate the lining of the gut, causing small perforations and therefore creating a larger junction for antigenic material to pass through. Some known irritants are, according to medical herbalist and Ayurvedic practioner Todd Caldecott, “antibiotics, alcohol, caffeine, parasites, pathogenic bacteria, peroxidized fats, some food preservatives and food additives, enzyme deficiencies (e.g. celiac disease, lactose intolerance), NSAIDs, corticosteriods, refined carbohydrates, oral contraceptives and mycotoxins (fungal toxins found in stored grains and dried fruit)” (Caldecott 2000) Damage to the lining of the gut creates wider junctions and damages mucosal immune tissue.(Lichtenberger 2012) When antigens are allowed by an overly leaky gut to pass from the lumen into the bloodstream, an immune response is initiated. The immune system creates antibodies to these antigens. (Caldecott 2000) Unfortunately, many tissues possess antigens that are similar enough to these exogenous antigens that the antibodies that have been created attack the self. Hence, the pathogenesis of an autoimmune condition. (Bergner 2001)

It is more obvious to see how a damaged gut epithelium could lead to digestive system related auto-immune conditions such as Crohn’s disease and Ulcerative Colitis. (Ma 1997; Zanjanian 1976) Research has shown a link between leaky gut and many non-gastrointestinal related auto-immune conditions including multiple sclerosis, ankylosing spondylitis, Behcet's syndrome, diabetes, and rheumatoid arthritis (Orlando 2002; De Keyser 2002; Fresco 2001; Vaarala 2002; Malosse 1992). To see the mechanism of action, take for example Diabetes mellitus. According to researchers de Kort, Keszthelyi, and Masclee, the increased immune reaction caused by the leaky gut leads to the destruction of beta cells in the pancreas which can lead to increased cytokine production which can lead to insulin resistance. (de Kort et al 2011).

It could be said that auto-immune conditions are a product of the modern world and its associated ills. It would behoove people suffering from auto-immune conditions to look to more primitive times for potential solutions: diets high in fiber and exercise, eating bitter foods, avoiding toxic substances like pharmaceutical drugs. Herbal medicine can be useful because so many plants can lower inflammation, soothe the gut lining, balance the immune system, and tone epithelial tissue. Because there is limited historical herbal treatment protocols for these specifically modern conditions, auto-immune conditions exemplify the need to combine an energetic approach with current medical research.

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Selecting native plants for a beneficial insects garden

Denise Quick

The Community Teaching Garden (CTG) is a project of the Friends of Burlington Gardens (FBG) whose goal is teaching organic gardening to adults. It is located at the Ethan Allen Homestead in Burlington, Vermont, a site where there have been community gardens since the1976 (Flint, 2007). In addition to the CTG, the Ethan Allen Homestead is currently the location of 50 community garden plots and the Visiting Nurses Association's Family Room Garden (FBG, n.d.).

As gardens have been established at this site for over 35 years, there are also established populations of garden pests. For example, during the summers of 2009 and 2010, there was a noticeable population of Colorado potato beetles (*Leptinotarsa decemlineata*) on solanaceous crops, especially potatoes, and relatively high populations of Mexican bean beetles (*Epilachna varivestis*) on pole beans. Damage from striped cucumber beetles (*Acalymma vittatumwas*) was evident on cucurbits during the summers of 2009, 2010 and 2011, while significant impact from flea beetles (family Chrysomelidae) was seen on leafy brassicas during the early summer in 2011. As the CTG is an organic garden, cultural methods are emphasized, and students picked pest insects from crops regularly during the growing season. Use of ORMI-listed (Organic Materials Review Institute) pesticides has also occurred at the CTG (personal observation). Additional pest control methods that support the local ecosystem are desired at the garden. In late summer 2011, the board of the Winooski Valley Park District (WVPD), who manages the land where the CTG in located, approved development of a beneficial insect garden. This garden is to be located on land contiguous to the CTG garden (Yumi Jakobcic, personal communication, 28 Oct 2011).

Healthy populations of predatory and parasitoid insects are desirable in agricultural settings because they feed on herbivorous pest insects during at least one life-stage. A review of the literature by Fielder and Landis (2007a) reveals that plant foods are also important to these insects. Adult parasitoid insects with access to nectar have shown increased fecundity or length of life, and pollen resources are needed by some predatory insects to produce

mature eggs. Therefore, the availability of appropriate plant resources for beneficial insects may reduce populations of pest insects in fields and gardens.

A number of non-native, annual plants are commonly recommended to support populations of beneficial insects, but Fielder and Landis (2007a, 2007b) suggest there are advantages to using perennial, native plants rather than non-native annuals. Native plants are adapted to local environments, so may have a reduced likelihood of invasiveness. It is likely that perennial plants provide better overwintering habitats for beneficial insects, thus assisting in establishing a more permanent community of beneficial insects. Additionally, the use of native plants can increase native biodiversity in a given area (2007a, 2007b).

Some plants that appear attractive to beneficial insects also attract pests. For example, in Michigan Fielder and Landis (2007a) observed a high number of herbivorous Japanese beetles (*Popillia japonica*) on common evening primrose (*Oenothera biennis*), a native Michigan perennial. They also found a number of *Phyllotreta spp*. flea beetles, common pests of brassica, on sweet allysum (*Lobularia maritima*). So, although sweet allysum is often recommended as a beneficial insect attractor, planting it near brassicas may increase the pest load on those crops (2007a).

Research on an organic farm in California's Central Valley compared insect occurrences in hedgerows of native shrubs, native perennial grasses and weedy patches (dominated by mustard and knotweed species) located between farm fields. Over two growing seasons, the levels of beneficial insects (both predators and parasitoids of pest insects) and pest insects (herbivores on crop plants) were tracked. Researchers found a ratio of beneficials to pest was approximately 4:1 on the native shrubs, approximately 1.5:1 in the perennial grasses, and approximately 1:1.5 in the weedy patches. The shrubs held the highest numbers of beneficial insects while they were in bloom, suggesting that floral resources were being used. (Morandin et al, 2011).

Another research study, performed at a research farm in Ingham County, Michigan, compared the presence of beneficial and herbivorous insects on native perennial plant with insects found on commonly recommended non-native annuals. Forty-three native plant species were tested, and 24 of those species were found to contain beneficial insect populations at least as large as the populations found on the five non-native annuals. Some

of those 24 species also held a high population of pest insects, so were considered unlikely to be useful as host plants for beneficial insects (Fielder and Landis, 2007a).

In the work of both Morandin etal (2011) and Fielder and Landis (2007a), the number of beneficial insects observed on the native plants increased over the two years of the study, suggesting that an established population of perennial plants can help to support a healthy population of beneficial insects. As the research of Fielder and Landis (2007a) was performed in Michigan, it has more applicability to Vermont (the current USDA Plant Hardiness Zone Map shows Ingham County, Michigan as zone 5b, while Burlington, Vermont is categorized as zone 5a (USDA ARS, 2012)). Table 1 lists plant evaluated by Fielder and Landis (2007a) whose home range includes Vermont. These plants should grow well in Burlington, Vermont, and should provide floral resources thought out the growing season.

Table 1: Native plants found to be used by beneficial insects in Michigan (Fielder and Landis, 2007a), whose range includes Vermont (USDA NRCS, 2012).

Scientific Name	**Common name (s)**
Zizia aurea	Golden zizia or golden Alexanders
Angelica atropurpurea	Purplestem angelica
Coreopsis lanceolata	Lanceleaf tickseed
Ratibida pinnata *	Barnhart pinnate prairie coneflower
Spiraea alba	White meadowsweet
Monarda punctata *	Spotted beebalm
Silphium perfoliatum	Cup plant
Eupatorium perfoliatum	Common boneset
Lobelia siphilitica	Great blue lobelia
Symphyotrichum novae-angliae	New England aster

* The USDA Plants Database shows Vermont to be the far eastern edge of the ranges for *Ratibida pinnata* and *Monarda punctate*.

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Heating or Cooling?
Amphotericity of Zingiber officinale and Capsicum annuum in Pyrogenesis

Robin Shapero

The famous nineteenth century herbalist Samuel Thomson refers to cayenne(*Capsicum annuum)* as "being powerful only to raise and maintain that heat on which life depends," and ginger(*Zingiber officinale)* as "being the next best thing to raise the inward heat and promote perspiration."[i] These two plants do indeed have an undeniably "hot" taste to them. That being said, many an herbal refers to them each as diaphoretic,[ii] and diaphoresis, or sweating, while being a well-known part of the fever process, is actually cooling to our body.[iii] There are several ways in which plant constituents can insert themselves into the complex process of the pyrogenic cascade to show marked effect, and I will explore just a few of the ways these two plants can do so.

Fever, or pyrexia, is not simply a symptom of the disease process but a deliberate and necessary action of our body's internal defense system.[1] Indeed, "fever is not failure of the body to regulate temperature, rather, body temperature is being regulated at higher level than normal."[iv] The main regulator of temperature in the body is our hypothalamus. A simplified view of the pyrogenic cascade is that "a trigger of the fever, called a pyrogen, causes a release of prostaglandin E2 (PGE2). PGE2 then acts on the hypothalamus, which generates a systemic response back to the rest of the body, causing heat-creating effects to match a new temperature level."[v]

Arachidonic acid is the first rate-limiting factor of the pyrogenic cascade. It is necessary for the synthesis of prostaglandin E2(PGE2)[vi], and constituents of ginger inhibit the metabolism of arachidonic acid.[vii] This could cause more AA to be available for conversion to PGE2, and therefore be helpful in raising a higher fever response. However, in pharmacological trials on rats, ginger was actually shown to *lower* PGE2 levels.[viii] Cyclooxygenase(COX), an enzyme that is responsible for the conversion of arachidonic acid into prostaglandin precursors, has been shown to be inhibited by a breakdown-constituent of ginger, shogaol, in certain isolated tissues. COX inhibition is also the mechanism of action by which common NSAIDs such as aspirin[ix] and ibuprofen[x] effect their anti-pyrexial action. These seemingly contradictory effects of ginger constituents on the body's

biochemical activities are characteristic of the often amphoteric effects engendered by whole-plant use of medicinal herbs.

Capsaicin is the principle active constituent of cayenne and is what gives its characteristic hot taste.[xi] Capsaicin, like certain ginger constituents, has also been shown to have an amphoteric effect on fever and inflammation, although unlike ginger, this has been shown to be dose-dependent(this effect may well be dose-dependent for ginger as well, but this has not been studied).[xii] A 1996 study stated that "…acute treatment at low concentrations will produce an increase of IL-l-a."[xii] An increase of interleukin-1-alpha(IL-1a) causes increased PGE2 production, which, as stated earlier, causes the hypothalamus to raise its set point, inducing increased thermogenesis. [vi] Capsaicin has also, in low doses, "been reported to increase thermogenesis by enhancing catecholamine secretion from the adrenal medulla in rats, mainly through activation of the central nervous system."[xiii] This same study, however, also shows that high doses of capsaicin over a longer time period would increase nitric oxide production. NO is a potent vasodilator, causing increased blood circulation to peripheral tissue, which effects lowered core body temperature and possible increased diaphoresis. [xiv] Once again, a herb's amphoteric qualities are proved and ratified by scientific data.

The contradictory indications of ginger and cayenne that are evidenced by the scientific literature can be quite confusing. We as herbalists, though, have other resources than just pure scientific data for our clinical applications of these two plants. Historic texts of herbal practice give us many recommendations on how to use a phytomedicine appropriately. Samuel Hahnemann, the originator of homeopathy, states that "if experience should show that by medicines that possess similar symptoms to the disease the latter would be most certainly and permanently cured, we must select for the cure medicines with similar symptoms."[xv] Anyone who has ever eaten a hot pepper can attest to the fever-like symptoms that occur immediately thereafter. The Materia Medica of the Hindus, a collation of thousands of years of traditional knowledge compiled from original Sanskrit works, declares ginger to be "heating."[xvi] In the Galenic system both ginger and cayenne are considered to be "heating in the third degree." That is, capable of causing and maintaining fever.[xvii]

The only conclusion that can be drawn from such a wealth of contrary information is that ginger and cayenne can each, given the right dose, the right client, the right timing and the right state of disease can assist in either abatement or enhancement of fever response. The many factors involved and the importance of outcome necessitates further research and experiential learning in this matter.

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The Use of Animal Fats in Topical Applications

Anna Powell

By the ninth edition of the US pharmacopeia (1916) lard had been replaced by petrolatum in many of the official ointments and pastes. By the 1940's it was common thought that hydrogenated oils were better than lard as an ointment base due to the fact that they are less susceptible to rancidity and more stable (Fiero, 1940). Before the introduction of petroleum, most ointments were prepared from animal fats. The animal oils used were numerous including, bear's grease, dog fat, beaver fat, bat oil, eel oil, porpoise oil, raccoon oil, rattlesnake oil and skunk oil. Lard, tallow, wool fat and goose grease were more commonly used up until the early 1900's (American Druggist and Pharmaceutical Record, 1912). The use of animal fats in medicine dates back to early human history. Ancient Egyptian papyrus scrolls, from 1700 BC contain information on their medical remedies of the time. Animal products appear in 42% of the prescriptions found on the Ebers papyrus, and approximately 1/3 of these contain fat or grease, both of which were likely used as vehicles. Animals that were mentioned included cows, geese, donkeys, humans, cats, pigs, mice, goat, sheep, bats, hippopotami, antelope, dogs, fish , ostrich, pigeons, pelicans, ravens, frogs, lizards, snakes, tortoises and crocodiles (Parkins, 2001). In a review of medieval cosmetics, butter, bear, deer, goat, hen, and pig fat are referred to as being used topically and in ointments (Cavallo, 2008). Wilhelm Fabry, a "surgeon-in-ordinary", published a book in 1614 on burns, *De Combusionibus*, which utilizes bear and hen grease as a part of a formula to smooth and soften the scars (Naylor, 1996). In studies done of the compositions of residues of apothecary jars from eighteenth century Italy and Spain, traces of fatty material possibly belonging to a pig and also to a ruminant animal were identified in samples (Saliu, 2011). In another study on the organic materials found in a series of glass jars recovered from a Roman villa in the ancient town of Oplontis traces of lipid based materials were detected, including beeswax, animal fat and vegetable oils. These are thought to have been used for the macerations and/or enfleurage of plant based materials (Ribechini, 2008). Records of Native American medicine cite the use of eagle fat, raccoon grease, fish oil, hog grease, seal oil, pond frog fat, bear grease, fox oil, rattlesnake fat and oil, skunk oil, goose fat, beaver fat, mountain goat fat, tallow and buffalo fat being used as liniments and for the bases of ointments (Duke, 1986) (Josselyn, 1886) (Lloyd,

1951) (Moerman, 1998) (Youngken, 1925). The Noatak Eskimos used seal oil and whale blubber in their topical ointments (Lucier, 1971). Sauer's eighteenth-century colonial American herbal mentions goose fat, butter, and lard as emollients. Goose fat is said to be used in place of olive oil when it was too expensive or unavailable (Weaver, 2000).The Eclectics used lard in ointments, cerates, liniments, laxative enemas, and as simple dressing for ulcer and blisters *(Cook, 1869)* (Sayer, 1917) (Scudder, 1898) (Thomson, 1905) In the Foxfire collections of traditions of the Appalachian peoples mutton tallow, ground hog oil, goose fat, wild cat oil, and beef tallow are included in a list of home remedies for various ailments (Fund, 1972). Today in Poland, badger fat is sought after via the Internet and press ads and is widely used as a salve for rheumatism by folk medicine practitioners (Zalewski, 2007).

The penetration of materials from the outer world into and through the skin has long been an object of discussion and investigation (MacKee, 1945). Wild (1926) experimented with the absorption of various mercurials with different ointment bases, including lard, hydrous wool fat and paraffin. Lard showed the greatest absorption, increasing with prolonged rubbing. Hydrous wool-fat was absorbed to a greater extent than lard, but the absorption of mercury was actually less than from lard ointment. The paraffin base absorptions were always less than the lard bases (Wild, R.B., 1926). Soft- paraffin remains on the skin as a protective layer for a considerable period and appears to be hardly absorbed at all. From some hundreds of cases of impetigo, it was found that patients treated with the paraffin base required on an average three weeks of treatment compared with two weeks for those who had the same ointment, but with a lard base (Wild, 1911). Petrolatum poorly penetrants the skin unless applied with friction (Sutton, 1908) (Sayer, 1917). The poorest vehicles are the ones which have been most frequently incorporated in ointments, namely, petrolatum, mineral oil and lanolin (Laug, 1947).

When choosing a base for an ointment it is important to consider if the ointment is to be absorbed by the skin, as seen with lard and hydrous wool fat, or if a protective coating is desired where only the epidermal layer is affected, as seen with paraffin and petroleum bases (Wild, 1911).

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Late Autumn & Early Winter Grain Bowl For Optimal Immunity

Al Scotton

Bowl:

Wild Rice

Quinoa

Shiitake mushrooms

Carrots

Kale

Broccoli

Nori

Almonds

Walnuts

Pepitas

Sunflower Seeds

Sauerkraut

Sprouts (any sort!)

Sauce:

Raw Garlic

Olive Oil

Tamari

Ginger

To make sauce, chop 1 head of garlic and 1 thumb of ginger and blend with 1 part tamari or soy sauce. With blender running, slowly pour in 2 to 3 parts olive oil, until sauce thickens. To assemble bowl, soak, rinse, and cook grains. Sautee shiitake mushrooms and mix with shredded nori in a bowl; add the warm grains. Steam chopped carrots, kale, and broccoli, and add to bowl. Top with a handful of nuts and seeds, sauerkraut, and sauce. Also great with avocado and sprouts!

Carrots, broccoli, and kale are rich in many vitamins and minerals including carotenoids, which protect the mucous membranes of the lung and intestine during the onslaught of viruses in cold and flu season. Kale helps to dispel mucous, and shiitake helps to balance the immune response. Garlic is aromatic, pungent, and dispersive, warming, and highly antimicrobial, with an affinity for the upper respiratory system. Nori helps to moisten dryness during late autumn and winter, nourish the kidney yin (governed by the water element in TCM), support the nervous system and endocrine systems with key minerals, and also helps to bring body warmth inward. Almonds and seeds nourish the jing and moisten the body with healthy fats, while walnuts, quinoa, and wild rice nurture the kidney yang. Ginger is pungent and warming, and benefits the metal element in late fall.

Most of these foods are fibrous, helping to maintain a healthy colon, healthy gut flora, and stable blood sugar levels, all of which contribute to healthy immune function. Most are also sweet, nourishing, and moistening, which is important during the dry and cold Vata season. The grain bowl is filled with sources of omega-3s, protein, complex carbohydrates, vitamins, minerals, and phytonutrients. The meal is a good source of magnesium, phosphorus, copper, iron, vitamin A, vitamin C, vitamin K, and manganese. All five tastes are present: pungent (garlic), bitter (kale), sour (sauerkraut), salty (nori, tamari), sweet (grains, nuts/seeds, carrots). Each of these tastes works on different body systems, bringing nourishment and balance to the entire organism for optimal health and optimal immunity.