634 4/03
ANSR-APPLIED NUTRITIONAL SCIENCE REPORTS

Natural Therapies for Fibromyalgia Syndrome

By Kristi Hawkes, N.D.

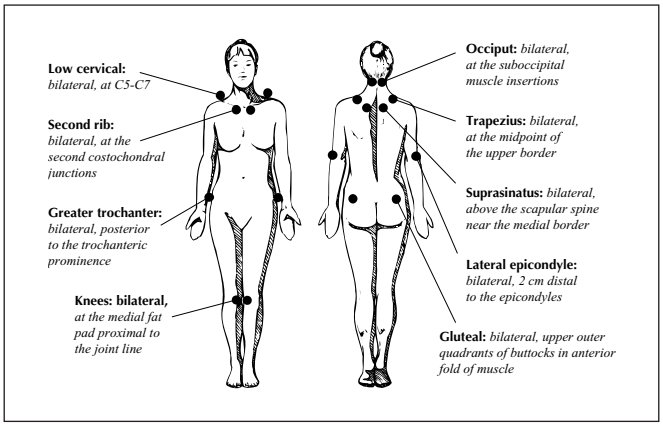
ABSTRACT: *Fibromyalgia is a syndrome characterized by generalized musculoskeletal pain and stiffness, chronic aching, fatigue, and multiple areas of local tenderness that can be identified during physical examination. Research studies suggest that fibromyalgia pain may be the result of a combination of factors affecting the activity of muscle cells and the central nervous system. Various conventional treatment modalities have been tested in fibromyalgia patients; unfortunately, these are often ineffective and may result in adverse side effects. A natural approach that incorporates nutritional support for the body processes implicated in the pathogenesis of fibromyalgia holds great promise for the millions suffering from this chronic syndrome.*

Fibromyalgia (FM) is a syndrome that presents with concurrent signs and symptoms characterized by fatigue; widespread musculoskeletal pain; and tenderness at specific sites in the neck, spine, shoulders, and hips referred to as "tender points" (Figure 1). Sleep disturbances, morning stiffness, headaches, irritable bowel syndrome (IBS), depression, and anxiety are also commonly associated with this syndrome.[1,2] Approximately 3-6 million Americans are affected by FM.[3,4] Women of childbearing age are primarily affected, but it can and does occur in children, the elderly, and men. Though reports have shown that nearly 30% of FM patients claim they cannot hold down a steady job, the condition has yet to gain recognition as a true "disease." Despite poor treatment outcomes, healthcare costs per patient have been documented at $2,274/year and the total annual drain on the U.S. economy is estimated to be over $20 billion.[5]

DIAGNOSIS

The tender point examination (i.e., tenderness in at least 11 of 18 defined points) has become the diagnostic gold standard for FM. Tender points are located over muscles and tendon insertions, and can range from mildly irritating to completely debilitating (Figure 1).[6,7]

Figure 1. Tender Points for Diagnostic Purposes.



Anatomic location of tender points according to the *American College of Rheumatology* 1990 classification criteria for FM.

Over the past decade, FM has been better defined in terms of physical and laboratory findings, which have suggested helpful guides for diagnosis.[2] Pain is the most prominent symptom, which generally begins in one region, such as the neck or shoulders, and seems to spread over time.[2] The most common histochemical and morphologic findings in affected muscle tissue are type II fiber atrophy (likely due to increased gluconeogenesis), glycogen deposition, mitochondrial abnormalities, and low levels of high-energy phosphates (e.g., ATP).[8]

Table 1. Common Physical and Laboratory Findings

• Tenderness of specific anatomical sites (at least 11 of 18 points)
• Chronic aching
• Stiffness
• Sleep disturbances
• Pain
• Headaches
• Anxiety
• Depression
• Chronic fatigue
• Intestinal disturbances
• Subjective soft tissue swelling
• Cardiovascular problems (dizziness, palpitations)
• Type II muscle fiber atropy
• Mitochondrial abnormalities
• Low Levels of ATP

It is important to note that FM resembles chronic fatigue syndrome (CFS) in several ways.[9] In both syndromes, patients experience symptoms of chronic musculoskeletal pain, aching, and stiffness, disturbed sleep, depression, and fatigue (Table 1).[6-12] While not all patients experience all symptoms, those with FM have a peculiar sensation of tenderness in specific areas of their body.[6,7,12-14] The presence and pattern of these "tender points" separate FM from CFS and other conditions.[2]

ETIOLOGY

A definitive cause of FM has yet to be elucidated. Various theories about disease trigger include stresses such as illness, injury, or trauma that affect the nervous system; hormone levels; muscle metabolism; and immune or endocrine function.[1,2] In addition, people with FM may often become inactive, depressed, and anxious about their health, all of which can aggravate the disorder.[2,15]

TREATMENT

The major classes of medications prescribed to FM patients include antidepressants, sleep-aids, anxiolytics, sedatives, non-steroidal anti-inflammatory drugs (NSAIDs), and muscle relaxants.[16] In controlled clinical trials, none of these agents have shown significant benefit. Additionally, they can also result in adverse side effects, which may include increased appetite, headache, nausea, anxiety, daytime drowsiness, constipation, dry mouth, and gastric bleeding.[2,16] On a positive note, exercise programs that involve muscle stretching and improve cardiovascular fitness have proven beneficial, most likely due to increased oxygenation of tissues and endorphin release.[2]

Understanding the etiologic theories of FM is an important step in the search for safe and effective therapies for the condition. A functional medicine approach would use this information to address the condition at the level of biochemical imbalances, rather than attempting to treat the end result of these biochemical imbalances—symptoms. Armed with knowledge and natural substances, the educated healthcare practitioner may be able to make a positive, long-term impact in the lives of his or her patients suffering from FM.

PATHOLOGY

Because FM is a multi-factorial syndrome that involves a wide range of bodily processes, treating biochemical pathology is helpful. A good treatment program addresses the four main areas that have established roles in the etiology or progression of the disease. These include mitochondrial dysfunction, hypothalamus-pituitary-adrenal dysregulation, toxicity, and intestine/nervous system abnormalities.

Mitochondrial Dysfunction

Malfunction of mitochondria is believed to be a primary factor in the etiology of FM, and abnormalities of the mitochondrial membranes in these patients have been reported. Mitochondria, which are concentrated in muscle tissue, are often referred to as the "powerhouses" of the cells due to their role in energy (ATP) production. Compromised mitochondrial activity can result in alterations in muscular function, as well as nervous, immune, and cardiac dysfunction.

Scientists in Sweden have conducted several studies on patients with FM. Muscle morphology, chemistry, and physiology were carefully examined, as were the most prominent symptoms, including muscle pain, muscle fatigue, and muscle stiffness. The authors of a comprehensive review of these studies found that FM patients appear to have microcirculation disturbances, along with mitochondrial damage and abnormally low phosphate counts—strongly suggesting an energy deficient state in the muscle tissues.[17]

HPA Dysregulation

Many patients with FM have had exposure to significant life stress and/or have inordinate responses to daily life stressors.[18] Altered reactivity of the hypothalmic-pituitary-adrenal (HPA) axis, resulting in hyposecretion of adrenal androgens (e.g., cortisol) has been observed in these patients.[19] Unfortunately, little information has thus far been obtained on how HPA disturbances can be related to the major symptomatic manifestations of pain, fatigue, sleep disturbances, and psychological distress.[20] One study has postulated that HPA dysfunction may involve serotonergic neurotransmission and alterations in the activity of arginine-vasopressin (AVP) and corticotropin-releasing hormone (CRH).[21]

HPA dysregulation is far-reaching and can interfere with proper functioning of the hypothalamus-pituitary-thyroid (HPT) axis. Neuroendocrine abnormalities along the HPT axis are also common in FM patients.[7,10,13] Furthermore, due to the association between hypothyroidism and fibrositis and myalgia, it is recommended that FM patients be given a comprehensive thyroid hormone test.[9,11,12] And with fatigue as one of the major complaints associated with both FM and CFS patients, hypometabolism due to secondary hypothyroidism fits very nicely into this hypothesis. (For more information on HPA and HPT function in the stress response, please refer to the *Applied Nutritional Science Report* titled "Nutritional Management of Stress Induced Dysfunction," by Richard L. Shames, M.D.)

Toxicity

Every day we are exposed to numerous toxins that, when ingested, inhaled, or absorbed, can damage or disturb various physiological functions.[22,23] Toxins may include pesticides, food additives, and other non-natural chemicals. Whatever the source, both natural and man-made toxins affect many people in ways that science does not yet fully realize.

Excessive toxin exposure can result in prolonged firing of peripheral pain receptors, resulting in central nervous system sensitization and exaggerated stimuli response.[24-26] According to researchers, increased sensitization and stimuli response are thought to contribute to the chronic pain of FM.[27] In fact, toxin exposure has been suggested to play a significant role in the development and progression of both FM and CFS, as approximately 47% to 67% of patients with FM and 53% to 67% of patients with CFS have reported at least one episode of symptom exacerbation after specific chemical exposure.[27,28]

Yet another area of concern for those with FM surrounds the role of excitotoxins—substances found in a variety of food additives, including monosodium glutamate (MSG), aspartame, hydrolyzed vegetable protein, and sodium caseinate.[29] It is becoming widely accepted that excitotoxins have neurotoxic effects on the human central nervous system, thereby disrupting various hormone levels.[29] (For more information on the influence of toxins on health, please refer to the *Applied Nutritional Science Report* titled "The Role of Detoxification in the Prevention of Chronic Degenerative Diseases," by DeAnn Liska, Ph.D. and Robert Roundtree, M.D.)

Intestine/Nervous System Abnormalities

Another area of interest is the link between intestinal dysfunction and FM. Although the statistics vary, research suggests that up to 70% of patients with FM complain of symptoms associated with irritable bowel syndrome (IBS). IBS is a functional disorder characterized by chronic abdominal pain with alternating diarrhea and constipation.[30] In comparison with healthy subjects, patients with IBS also tend to experience extraintestinal symptoms that overlap with FM complaints, including increased nerve sensitivity, morning stiffness, headaches, sleep disturbances, and fatigue.[31]

Since IBS and FM have shared clinical features, a common etiology has been suggested.[31] The autonomic nervous system regulates responses from smooth muscle, cardiac muscle, glands, and intestinal sensory neurons, and is interrelated with the enteric (intestinal) nervous system both anatomically and functionally.[32] An imbalance in autonomic function may result in the overexpression of sensory information, influencing factors such as intestinal motility, pain sensitization, and the response to psychological stress.[31,33] Research suggests an autonomic imbalance may be the pathway by which both intestinal and extraintestinal symptoms are seen in FM and IBS patients.[34] It is further postulated that chronic overstimulation of the autonomic nervous system while the patient is at rest contributes to a weakened autonomic response to physical challenge, contributing to the increased fatigue, decreased tissue oxygenation, and reduced threshold for pain seen in FM patients.[35]

Amino Acid Transport

Dietary protein is broken down into peptide fractions in the stomach and further digested into free amino acids and short chains of two or three amino acids in the intestines. Coupled with sodium, the amino acids are transported across the small intestine into circulation. Once absorbed, they influence multiple biochemical processes including the synthesis of neuropeptides and neurotransmitters. Several studies suggest that patients with FM have a defect in amino acid homeostasis, in particular, deficiencies in L-tryptophan, L-leucine, L-isoleucine, and L-valine. Researchers hypothesize that the amino acid deficiencies seen in patients with FM may be the result of defective intestinal amino acid transport mechanisms.[36-38]

L-tryptophan deficiency is a good example of how reduced amino acid transport may be related to FM symptoms. L-tryptophan is a precursor to 5-hydroxytryptamine, or serotonin. Serotonin is contained in the brainstem and is active throughout the central nervous system. Known as a "neuromodulator," serotonin is involved in the configuration of emotional, cognitive, and motor functions, as well as circadian and neuroendocrine rhythms.[32,39] Research suggests that decreased L-tryptophan and the resulting low serotonin levels play a pathophysiologic role in fibromyalgia.[37-39]

While the serotonergic system has been repeatedly discussed, elevated levels of substance P—a neuropeptide that mediates pain perception—has also been described in FM.[40] In a study performed on 51 patients with FM, substance P concentrations were negatively correlated with both tryptophan and its metabolite 5-hydroxyindoleacetic acid (5-HIAA). While high substance P levels were associated with strong sleep disturbances, high levels of 5-HIAA were associated with good quality of sleep. Furthermore, a relationship between high substance P concentrations and high pain scores trended toward significance, whereas high levels of tryptophan and 5-HIAA were associated with reduced pain in FM patients. These findings support the hypothesis that decreased serotonergic activity and increased substance P activity influence circadian rhythm and sleep, as well as neurogenic pain perception in FM.[40]

The amino acids L-leucine and L-isoleucine are involved in the synthesis of oxytocin, a neuropeptide thought to have antidepressant, antianxiety, and analgesic properties. In a study of FM patients, low levels of oxytocin were significantly correlated with higher scores of depression, stress, and pain. Researchers postulated that together with other neuropeptides and neurotransmitters, oxytocin may play a role in the mechanisms responsible for FM symptoms.[41]

Collectively, leucine, isoleucine, and valine are referred to as branched-chain amino acids (BCAAs)—essential amino acids that are highly concentrated in muscle tissue. BCAAs are metabolized into biochemical compounds that mediate energy production and protein synthesis. Thus, low levels of BCAAs may also partially explain the depleted muscular energy seen in FM patients.[42]

NATURAL TREATMENT APPROACHES

An important nutritional foundation for optimal health and healing in both healthy individuals and patients suffering from chronic conditions (e.g., FM) includes a diet rich in fruits and vegetables, an adequate intake of omega-3 fatty acids, and a balanced multivitamin/mineral supplement.

Diet

Several studies suggest that a mostly raw (uncooked) food, primarily vegetarian diet helps to improve symptoms in patients with FM.[43-45] In one study, 30 patients participating in a dietary intervention were told to consume a diet consisting of 24% fat, 65% carbohydrate, and 11% protein from fresh fruit, green salad, carrot juice, dehydrated barley grass juice, omega-3 fatty acid dietary supplements, and minimal amounts of animal products. The mean intake of beta-carotene from carrot juice was 52 mg/day and regular consumption of fruits and vegetables resulted in high intakes of vitamin C, folate, potassium, and magnesium. After 7 months, 19 of the 30 patients responded favorably with significant improvements in quality of life, including pain, range of motion, and flexibility. The researchers concluded that this form of dietary intervention may play a significant role in helping patients with FM.[43]

Multivitamin/Mineral Formula

While the body requires available supplies of vitamins and minerals for maintaining optimal health and vitality, attaining adequate dietary intake is not easily assured. A good multivitamin/mineral supplement containing essential micronutrients helps to ensure optimal nutrient intake and reduce the risk of chronic disease development.[46]

EPA/DHA 30:20 Formula

EPA (eicosapentaenoic acid) and DHA (docosahexaenoic acid) are omega-3 fatty acids that are known to play a key role in maintaining cell membrane structure and modulating inflammatory pathways. The phospholipid-rich cell membrane contains high concentrations of omega-3 fatty acids, which influence tissues throughout the body by improving cell membrane fluidity, receptor function, enzyme transport, and gene expression.[47] Omega-3 fatty acids are also essential to healthy eicosanoid synthesis, protecting tissues from damage by excessive cytokine production that induces painful inflammation.[48] Research suggests that dietary intake of approximately 6 grams per day of omega-3 fatty acids, as seen in a vegetarian diet, may significantly improve the quality of life of FM patients.[43]

While a healthy diet and adequate nutrient and essential fatty acid intake are important in maintaining health, patients with FM may also need more intensive support. A basic program that addresses the physiological factors previously discussed may be beneficial for patients with FM (Table 2).

FOCUS: MITOCHONDRIAL SUPPORT

Certain nutritional factors play a role in maintaining the integrity and proper functioning of the mitochondria. The following formulas have been used in FM patients in clinical settings:

Mitochondrial Antioxidant Formula

A combination of nutrients known to positively influence mitochondrial energy production, along with antioxidants, may support a higher rate of ATP synthesis, as well as protect mitochondria from free radicals. Nutrients such as lipoic acid and B vitamins serve as cofactors for cellular energy production and metabolism; thiamin supports the activity of enzymes (e.g., transketolase, pyruvate and alpha-ketoglutarate dehydrogenase complexes) that influence citric acid cycle activity and activities of the respiratory chain; amino acids like creatine allow muscle to regenerate ATP, and N-acetyl-L-carnitine helps transport nutrients (i.e., fatty acids) into the mitochondria for energy production. Furthermore, antioxidants such as coenzyme Q_{10}, L-glutathione, N-acetylcysteine, and vitamins C and E help prevent oxidative damage that can affect cellular and/or mitochondrial function negatively.[49-52]

Malic Acid/Magnesium Complex

Malic acid is a natural compound found in apples and various other fruits that is necessary for ATP production.[53] Combined supplementation with 1,200 mg of malic acid and 300 mg magnesium administered in a dose-escalated fashion produced significant reductions in the severity of 3 primary pain/tenderness measures in FM patients. In another study, supplementation with malic acid (1,200-2,400 mg) and magnesium (300-600 mg) resulted in subjective improvements in myalgia within 48 hours and reduced tender point index scores were recorded at 8 weeks.[54] Malic acid is thought to increase the rate of ATP substrate transport into mitochondria, thus generating mitochondrial ATP production.[54,55] In addition, malic acid has an oxygen-sparing effect that may counter the relative hypoxia demonstrated in FM patients.

Magnesium/Potassium Aspartate Complex

Magnesium, potassium, and aspartate have roles in intermediary metabolism (enzyme-catalyzed processes that extract energy from nutrients to build new cells) that may mitigate physical fatigue.[56,57] Aspartate is a non-essential amino acid that can be depleted during times of stress. Among their many functions, magnesium is important in maintaining the integrity of the mitochondrial membrane, whereas potassium is important in maintaining cell electrical stability and growth.

In a study examining the effects of potassium, magnesium, and aspartate (1,000 mg potassium aspartate and 1,000 mg magnesium aspartate per day for 1 to 2 weeks) on 4 subjects undergoing extremely fatiguing physical exercise, non-athletes demonstrated improved physical endurance.[57] These findings were supported by another study wherein 6 men, who were above average in physical fitness, exercised until severe exhaustion and muscle pain ensued. Potassium-magnesium-aspartate supplementation of 1.75 g every 6 hours for 4 days was shown to prolong exercise capacity. Researchers postulated that the mechanism behind this anti-fatigue effect was the resynthesis of ATP and phosphocreatine.[58]

FOCUS: MANAGING STRESS/NORMALIZING HPA FUNCTION

The use of adaptogens—herbs that help normalize bodily processes and increase the ability to "adapt" to stress—and B vitamins provide a general approach to managing stress.

Traditional Holy Basil Combination

Adaptogens such as holy basil (*Ocimum sanctum*), ashwagandha (*Withania somnifera*), and brahmi (*Bacopa monnieri*) have a history of use in Ayurvedic medicine and are scientifically well supported to improve stress tolerance.[59,60] In an animal study, holy basil was observed to reduce the incidence of gastric injury induced by cold stress and restraint stress.[61] Holy basil has also been found to inhibit the lipoxygenase and cyclooxygenase pathways, antagonize histamine, enhance gastric mucosal strength, and help prevent adrenal cortisol depletion.[62,63]

Ashwagandha was shown to enhance adaptability to both physical and chemical stress in animals, showing the ability to suppress adrenal enlargement, as well as adrenal ascorbic acid and corticosterone depletion.[64] It appears to have a corticosteroid sparing effect, which may be mediated via the HPA axis. Ashwagandha also produces positive changes in stress-related prostaglandin and catecholamine production. In other animal testing, brahmi was shown to improve adaptations in sensory, motor, and motivational systems.[65] In humans, it exhibits beneficial effects on anxiety, as well as mental functions such as mental fatigue.[66]

B_6/Pantothenic Acid Complex

The B vitamins pantothenic acid and B_6 are important in energy production and the response to stress by supporting adrenal hormone production and regulation.[67,68] In conjunction with ATP and cysteine, pantothenic acid plays an integral role in the synthesis of coenzyme A, which initiates various metabolic processes including the production of glucocorticoids.[67] In an animal study, vitamin B_6 was found to stimulate the secretion of adrenal catecholamines; these results were confirmed when it demonstrated virtually no effect on adrenalectomized rats.[68]

Thyroid Support

While normalizing stress-induced changes in HPA function will have a positive influence on the HPT axes, some patients may need additional thyroid support. Several nutrients are known to support healthy thyroid hormone synthesis, to promote the conversion of thyroxine (T4) to the more bioactive triiodothyronine (T3), and to address receptor dynamics and the expression of thyroid hormone sensitive genes. Such nutrients include iodine, selenium, zinc, and vitamins E, A, and D.[69-74]

FOCUS: DETOXIFICATION

Detoxification is the process of inactivating toxins that involves two phases of reactions, Phase I bioactivation and Phase II conjugation. Phase I activation is catalyzed by enzymes that transform toxins into reactive intermediates, preparing them for conjugation by the Phase II system, which then renders the substances non-toxic and facilitates their excretion. Phase I generates reactive intermediates, which can act as mutagens or carcinogens if they do not immediately undergo conjugation by the Phase II system. That is, these reactive intermediates can bind to and damage DNA. Patients with excessive toxin exposure may require additional hepatic support to restore balanced Phase I and Phase II activity, or bifunctional detoxification.[43,75,76] When balanced bifunctional detoxification is restored, FM patients may experience increased energy and vitality.

Bifunctional Detox Support Formula

While eliminating toxins such as MSG, aspartame, and other excitotoxins from the diet can play a crucial role in reducing toxin exposure, targeted nutritional support may be necessary.[43,75,76] In particular, detoxification therapies designed to lessen exposure to toxins while facilitating the elimination of stored toxins from the body.[75] A formula containing silymarin, ellagic acid, catechins, and N-acetylcysteine (NAC) combined with other detoxifying and

antioxidant factors promotes bifunctional detoxification, while simultaneously protecting cells from reactive metabolites generated by Phase I enzymes that cause oxidative stress. The mechanisms by which these four nutritional factors function include:

Silymarin

Silymarin, the active constituent in milk thistle (*Silybum marianum*), has a long history of traditional use as a hepatoprotectant that is supported by recent scientific research.[77] In patients with hepatic disorders of various etiologies, including exposure to industrial phenolics (e.g., toluene, xylene), 400 mg/day of silymarin significantly improved liver function.[78] Further research suggests that silymarin increases serum glutathione and related enzyme activities such as glutathione peroxidase, thereby inducing Phase II activity and reducing hepatic oxidative stress.

Ellagic acid

Ellagic acid from pomegranate induces Phase II activities such as the production of glutathione-S-transferases at the gene level, while modulating the activity of Phase I enzymes so that they are not over-induced. Furthermore, ellagic acid was shown to bind to DNA and protect it from mutating carcinogens by promoting their methylation.[79] Ellagic acid has also been shown to ameliorate nickel toxicity via chelation and excretion of metal ions from cells or tissue. Biochemical markers suggest that ellagic acid protects the integrity of cell membranes during this sequestration.[80]

Catechins

Catechins are a class of flavonoids that are highly concentrated in green tea extract and have been shown to possess multiple health-promoting qualities. Considered to be bifunctional modulators, data suggest that catechins induce Phase II glucuronidation and glutathione conjugation enzymes, and are postulated to selectively inhibit or induce Phase I activity.[81] As potent antioxidants, catechins have also been shown to possess anticarcinogenic and antimutagenic potential in animals and have demonstrated the ability to directly bind to many toxins.[82] Furthermore, catechins may also support the detoxification process by promoting healthy microflora and pH in the gastrointestinal (GI) tract, which is important to healthy bowel function and the elimination of toxins.

N-Acetylcysteine (NAC)

NAC has been shown to increase glutathione production, an important cofactor in glutathione conjugation. Known to be a sulfur donor, NAC may facilitate the detoxification of heavy metals via binding of metals to the sulfur in glutathione.[83] Cysteine, an important support factor in heavy metal detoxification, is depleted in the presence of elevated toxic metal load. Supplementation with 200 to 500 mg/day of cysteine in the form of NAC helps to maintain healthy cysteine levels and support sulfation cofactor and glutathione levels.[84]

FOCUS: MAINTAINING INTESTINAL/NERVOUS SYSTEM HEALTH

Maintaining intestinal health is essential to communications along the brain-gut axis. Improved GI health can be achieved with a nutritional regimen known as the 4R® GI Restoration Program, which addresses four primary stages of healing: *Remove, Replace, Reinoculate, and Regenerate*. This regimen focuses on *removing* pathogenic microbes and toxins, *replacing* digestive enzymes and other digestive factors that may be lacking, *reinoculating* the GI with healthy bacteria, and *regenerating* the GI lining. A basic protocol consisting of probiotics, prebiotics, and other GI-supportive factors can address each of the four areas.

L. Acidophilus NCFM and Bifidobacteria

A large body of scientific evidence suggests that supplementing with "friendly" probiotic organisms, such as *L. acidophilus* NCFM and bifidobacteria, helps to restore a healthy microbial balance in the GI tract.[85,86] A healthy microbial balance helps to prevent pathogenic organisms from proliferating, and strongly influences multiple bodily functions including intestinal and immune function. Preliminary research suggests that oral bacteriotherapy improves the composition of intestinal microflora and may play a role in the prevention and treatment of symptoms associated with IBS.[87]

Prebiotic Blend

A prebiotic blend of nutritional factors, such as plantain fruit, phosphatidylcholine, and arabinogalactins, helps to promote GI health by removing pathogens and protecting the GI lining.

Plantain

The gastric mucosal lining helps to protect the body from invading microbes and toxins, as well as from damaging acid and medications that can contribute to the development of gastric lesions. Extracts of plantain fruit that are rich in flavonoids have been used therapeutically for their anti-ulcerogenic properties, and have been shown to stimulate mucus secretion and protect the gastric lining from potentially harmful exogenous agents.[88,89]

Phosphatidylcholine

Toxins and pathogens can compromise the amount of phosphatidylcholine in the GI mucosa lining.[90] Recent research suggests that supplementing with soy lecithin, a rich source of phosphatidylcholine, helps to protect the GI mucosa from injury by ulcerogenic substances, such as nonsteroidal anti-inflammatory drugs (NSAIDs) or bile salts, and accelerates healing.[90]

Arabinogalactins

Arabinogalactins are a type of fiber found in many vegetables and grains that function as a prebiotic; that is, arabinogalactins have been shown to promote a healthy intestinal microbial balance by preferentially increasing the beneficial intestinal bacteria *L. acidophilus* and bifidobacteria over potentially harmful bacteria such as *Clostridia*.[91,92]

Table 2. What a Basic Nutritional Supplement Program for FM Patients May Include.

Nutritional Supplement	Area of Support
Multivitamin/Mineral Formula	Overall health and energy levels
EPA/DHA 30:20 Formula (Omega-3 fatty acids)	Overall health of cells that house mitochondria
Mitochondrial Antioxidant Formula	Muscular system: comprehensive mitochondrial support
Malic Acid/Magnesium Complex **OR** Magnesium/Potassium Aspartate Complex	Muscular system: mitochondrial energy production
Bifunctional Detoxification Support Formula	Nervous system: elimination of toxins
L. Acidophilus NCFM and Bifidobacteria	Digestive system: intestinal health and function
If stress is an issue, then add:	
Traditional Ayurvedic Holy Basil Combination	Endocrine system: hormone balance and stress response
B_6/Pantothenic Acid Complex	Endocrine system: adrenal hormone production and energy

EXERCISE

Cardiovascular and muscular deconditioning are common in FM. Difficulties remaining active may lead to extreme deconditioning and eventually perhaps impaired ability to complete activities of daily living.[15] Therefore, exercise that combats deconditioning without triggering pain is a key component of FM treatment.[18] Exercise is reported to alleviate some symptoms of FM, perhaps via augmentation of neurotransmitter levels or stimulation of muscle metabolism.[93]

Numerous exercise intervention trials have been conducted—the majority of which have been combined aerobic, flexibility, and strength training. Aerobic interventions included such activities as cycling, walking, jogging, and pool exercises. These clinical trials have offered evidence that many FM patients can improve their aerobic capacity and muscle strength, and decrease their symptoms.[4,15,18] However, earlier studies that used exercise programs designed for healthy individuals suffered a high rate of attrition and worsening of symptoms. More recently, researchers have utilized lower intensity activities with a variety of frequencies and durations, which have been met with better compliance and results.[3,4,15]

In a recent study, the effect of graded resistance exercise on 10 patients diagnosed with FM was measured. Patients had an 8-year mean duration of symptoms. Subjects participated in an exercise program 2 days per week for 8 consecutive weeks that consisted of resistive exercises such as the leg press, shoulder press, bicep curl, and military press. The twice-a-week exercise frequency was adopted to avoid the risk of exacerbating symptoms. Training load was based on the maximum weight lifted in one repetition through the complete range of motion. At the end of the 8-week period, it was demonstrated the program attenuated the major clinical symptoms characteristic of FM. Assessment methods included a pressure algometer, the Fibromyalgia Impact Questionnaire (FIQ), and the Symptom Checklist-90-Revised (SCL-90-R). Dynamic strength, as assessed by shoulder and leg press one repetition maximum, increased by 43% and 51% respectively; the pain threshold, presented as a combined score, increased by 36%; the pain and disordered sleep rating declined by 49% and 43% respectively; and the score for psychological distress declined by 52%. In particular, patients emphasized diminished fatigue and improved mood and sleep.[93] These data supports similar findings in another study of muscle strengthening exercise.[4]

To further investigate the safety, feasibility, and effects of a progressive strength training and cardiovascular exercise program, 24 women with confirmed FM were recruited for a 20-week intervention. For the 4 four weeks, participants performed pool exercises that focused on major joint range of motion. The following 16 weeks consisted of land-based exercises for improving cardiovascular endurance, muscle strength, and joint range of motion; these consisted of a broad range of exercises such as walking, hip flexion/extension, knee extension/flexion, and stretching. All subjects who completed the intervention (n=15) experienced improved muscle and cardiovascular fitness; improved 6-minute walk time without a change in heart rate; and a significant improvement in FIQ scores.[3]

Deconditioned muscles are more likely to experience muscle microtrauma, causing more pain after exercising.[15] Whether exercise is good or bad for patients with FM depends on many variables including age, current level of conditioning, rate of increase of exercise intensity, frequency of exercise, ratio of concentric to eccentric muscle use, and more.[15] Attention to these variables must be considered when giving patients an exercise protocol.[18]

COMPLEMENTARY THERAPY

Frequency-Specific Microcurrent

The application of small amounts of electricity, measured in microamperage, is believed to stimulate healing on a cellular level.[94] For years, such low-dosage electricity has been used to increase the rate of healing in injured athletes, control pain, increase the rate of fracture repair, and treat myofascial pain and dysfunction.[95] Injured tissue has altered electrical dynamics as compared to healthy, surrounding tissue. Altered electrical dynamics cause changes in cellular and mitochondrial function, resulting in impaired healing and inflammation.[95]

Frequency-specific microcurrent therapy works by sending electric currents to injured cells. This "bio-electric therapy" supports the natural current flow in the tissue, which is necessary for transporting nutrients to the cells, as well as facilitating the removal of wastes away from the cells; it is also critical to protein and ATP synthesis.[95] The current is administered to various parts of the body for periods up to 90 minutes via the fingertips of vinyl graphite gloves or via small, cotton-tipped probes. The actual electrical current administered to the patient is so diminutive that it cannot be felt. Response is frequency specific.

The most commonly used frequencies are 0.3 Hz for increasing healing, 3 Hz for stimulation of acupuncture points, 30 Hz for pain control, and 300 Hz for reducing edema and stimulating lymphatic flow. The sequence of frequencies used in each patient is somewhat dependent on the condition of the muscle and the operator's perception.[95] Treatments can last from several weeks to 2 years with less frequent administration as the patient progresses. Contraindications for use of microcurrent therapy would include treatment through the chest of a patient wearing a pacemaker, of the abdomen of a pregnant woman, or in the area of malignancy.

A study performed on rat skin by Cheng et al. found that microcurrent therapy using 50-1,000 microamps of electricity can increase ATP production three- to five-fold; augment membrane transport, which helps to increase nutrients to the area; and boost protein synthesis in animal skin. It is important to note that ATP production actually decreased at 5,000 microamps, highlighting the importance of dose-response.[96]

CONCLUSION

Because FM is a multifactorial condition of questionable etiology, treatment that addresses the various bodily systems or processes known to be defective may be of benefit. While medications prescribed for FM complaints may provide temporary relief, they may not address the underlying factors involved and can cause undesirable side effects such as headache, nausea, drowsiness, or constipation. On the other hand, nutrition and dietary supplements that support the nervous, endocrine, and digestive systems and facilitate the removal of toxins may assist the body in the healing process.

REFERENCES

1. National Institute of Arthritis and Musculoskeletal Skin Diseases. National Institutes of Health. *Fibromyalgia*. Retreived July 2, 2002 from, http://www.niams.nih.gov/hi/topics/fibromyalgia/fibrofs.htm
2. Arthritis Foundation Disease Center. *Fibromyalgia*. Retrieved July 22, 2002 from, http://www.arthritis.org/conditions/DiseaseCenter/fibromyalgia.asp
3. Rooks DS, Silverman CB, Kantrowitz FG. The effects of progressive strength training and aerobic exercise on muscle strength and cardiovascular fitness in women with fibromyalgia: a pilot study. *Arthritis Rheum* 2002;47(1):22-28.
4. Jones KD, Burckhardt CS, Clark SR, et al. A randomized controlled trial of muscle strengthening versus flexibility training in fibromyalgia. *J Rheumatol* 2002;29:1041-48.
5. Thorson K. *Fibromyalgia syndrome (FMS) political case statement*. Retrieved March 6, 2002 from, http://www.fmnetnews.com/pages/case.html
6. Smythe HA. "Fibrositis" and Other Diffuse Musculoskeletal Syndromes. In: Kelley WN, et al., eds. *Textbook of Rheumatology*. 1st ed. Philadelphia: WB Saunders; 1985:481-89.
7. Wolfe F. The clinical syndrome of fibrositis. *Am J Med* 1986;81:7-13.

8. Abraham GE, Flechas JD. Management of fibromyalgia: rationale for the use of magnesium and malic acid. *J Nutr Med* 1992;3:49-59.
9. Goldenberg DL, Simms RW, Geiger A, et al. High frequency of fibromyalgia in patients with chronic fatigue seen in a primary care practice. *Arthritis Rheum* 1990;33:381-87.
10. Masi AT, Yunus MB. Concepts of illness in populations as applied to fibromyalgia syndromes. *Am J Med* 1986;81:19-25.
11. Campbell SM, Clarks, Tindall EA, et al. Clinical characteristics of fibrositis. A blinded controlled study of symptoms and tender points. *Arthritis Rheum* 1983;26:817-24.
12. Henrikson KG, Bengtson A. Fibromyalgia—a clinical entity? *Can J Physiol Pharmacol* 1991;69:672-77.
13. Smythe HA. Nonarticular Rheumatism and Psychogenic Musculo-skeletal Syndromes. In: McCarty DJ, ed. *Arthritis and Allied Conditions*. 9th ed. Philadelphia: Lea & Febiger; 1989:1241-54.
14. Wolfe F, Smythe HA, Yunus AB et al. The American College of Rheumatology 1990 criteria for the classification of fibromyalgia. Report of the Multicenter Criteria Committee. *Arthritis Rheum* 1990;33:160-72.
15. Jones KD, Clark SR, Bennett RM. Prescribing exercise for people with fibromyalgia. *AACN Clin Issues* 2002;13(2):277-93.
16. Arthritis Foundation. *Fibromyalgia medications*. Retrieved July 22, 2002 from, www.arthritis.org/conditions/DrugGuide/fibromyalgia.asp
17. Bengtson A, Henrikson KG. The muscle in fibromyalgia-a review of Swedish studies. *J Rheumatol* 1989;16 (Suppl 19):144-49.
18. Littlejohn GO, Walker J. A realistic approach to managing patients with fibromyalgia. *Curr Rheumatol Rep* 2002;4(4):286-92.
19. Dessein PH, Shipton EA, Joffe BI, et al. Hyposecretion of adrenal androgens and the relation of serum adrenal steroids, serotonin and insulin-like growth factor-1 to clinical features in women with fibromyalgia. *Pain* 1999;83(2):313-19.
20. Crofford LJ. The hypothalmic-pituitary-adrenal stress axis in fibromyalgia and chronic fatigue syndrome. *Z Rheumatol* 1998;57 (Suppl 2):67-71.
21. Demitrack MA, Crofford LJ. Evidence for and pathophysiologic implications of hypothalmic-pituitary-adrenal axis dysregulation in fibromyalgia and chronic fatigue syndrome. *Ann N Y Acad Sci* 1998;840:684-97.
22. *EPA: Environmental Protection Agency position paper: Terms of Environment.* (n.d.). Retreived September 9, 2002 from, http://www.epa.gov/OCEPAterms
23. Rainville P, Bushnell MC, Duncan GH. Representation of acute and persistent pain in the human CNS: potential implications for chemical intolerance. *Ann N Y Acad Sci* 2001;933:130-41.
24. Bennett GJ. Update on the neurophysiology of pain transmission and modulation: focus on the NMDA-receptor. *J Pain Symptom Manage* 2000;19(Suppl 1):S2-S6.
25. Pall ML. Common etiology of posttraumatic stress disorder, fibromyalgia, chronic fatigue syndrome and multiple chemical sensitivity via elevated nitric oxide/peroxynitrite. *Med Hypotheses* 2001;57(2):139-45.
26. Ursin H, Eriksen HR. Sensitization, subjective health complaints, and sustained arousal. *Ann N Y Acad Sci* 2001;933:119-29.
27. Bell IR, Baldwin CM, Schwartz GE. Illness from low levels of environmental chemicals: relevance to chronic fatigue syndrome and fibromyalgia. *Am J Med* 1998;105(Suppl 3A):74S-82S.
28. Buchwald D, Garrity D. Comparison of patients with chronic fatigue syndrome, fibromyalgia, and multiple chemical sensitivities. *Arch Intern Med* 1994;154(18):2049-53.
29. Smith JD, Terpening CM, Schmidt SO, et al. Relief of fibromyalgia symptoms following discontinuation of dietary excitotoxins. *Ann Pharmacother* 2001;35(6):702-06.
30. Chang L. The association of functional gastrointestinal disorders and fibromyalgia. *Eur J Surg Suppl* 1998;583:32-36.
31. Canataroğlu A, Gümürdülü Y, Erdem A, et al. Prevalence of fibromyalgia in patients with irritable bowel syndrome. *Turk J Gastroenterol* 2001;12(2)141-44.
32. Vander A, Sherman J, Luciano D. *Human Physiology*. 5th Ed. McGraw-Hill Publishing Company: New York; 1990.
33. Tougas G. The autonomic nervous system in functional bowel disorders. *Can J Gastroenterol* 1999;13(Suppl A):S15A-S17A.
34. Backman E, Bengtsson A, Bengtsson M, et al. Skeletal muscle function in primary fibromyalgia. Effect of regional sympathetic blockade with guanethidine. *Acta Neurol Scand* 1988;77(3):187-91.
35. Cohen H, Neumann L, Kotler M, et al. Autonomic nervous system derangement in fibromyalgia syndrome and related disorders. *IMAJ* 2001;3:755-60.
36. Russell IJ. Neurohormonal aspects of fibromyalgia syndrome. *Rheum Dis Clin North Am* 1989;15(1):149-68.
37. Yunus MB, Dailey JW, Aldag JC, et al. Plasma tryptophan and other amino acids in primary fibromyalgia: a controlled study. *J Rheumatol* 1992;19(1):90-94.
38. Neeck G, Riedel W. Neuromediator and hormonal perturbations in fibromyalgia syndrome: results of chronic stress? *Baillieres Clin Rheumatol* 1994;8(4):763-75.
39. Juhl JH. Fibromyalgia and the serotonin pathway. *Altern Med Rev* 1998;3(5):367-75.
40. Schwarz MJ, Spath M, Muller-Bardorff H, et al. Relationship of substance P, 5-hydroxyindole acetic acid and tryptophan in serum of fibromyalgia patients. *Neurosci Lett* 1999;259(3);196-98.
41. Anderberg UM, Uvnas-Moberg K. Plasma oxytocin levels in female fibromyalgia syndrome patients. *Z Rheumatol* 2000;59(6):373-79.
42. Maes M, Verkerk R, Delmeire L, et al. Serotonergic markers and lowered plasma branched-chain-amino acid concentrations in fibromyalgia. *Psychiatry Res* 2000;97(1):11-20.
43. Donaldson M, Speight N, Loomis S. Fibromyalgia syndrome improved using a mostly raw vegetarian diet: an observational study. *BMC Complement Altern Med* 2001;1(1):7.
44. Kaartinen K, Lammi K, Hypen M, et al. Vegan diet alleviates fibromyalgia symptoms. *Scand J Rheumatol* 2000;29(5):308-13.
45. Hostmark AT, Lystad E, Vellar OD, et al. Reduced plasma fibrinogen, serum peroxides, lipids, and apolipoproteins after a 3-week vegetarian diet. *Plant Foods Hum Nutr* 1993;43(1):55-61.
46. Fletcher R, Fairfield KM. Vitamins for chronic disease prevention in adults. *JAMA* 2002;287:3127-29.
47. Alexander JW. Immunonutrition: the role of omega-3 fatty acids. *Nutrition* 1998;14:627-33.
48. Horrocks LA, Yeo YK. Health benefits of docosahexaenoic acid (DHA). *Pharmacol Res* 1999;40(3):211-25.
49. Kwong LK, Kamzalov S, Rebrin I, et al. Effects of coenzyme Q(10) administration on its tissue concentrations, mitochondrial oxidant generation, and oxidative stress in the rat. *Free Radic Biol Med* 2002;33(5):627-38.
50. Dlugosz A, Piotrowska D. Lipid peroxidation stimulated by Solvesso, Bawanol and methanol, and its counteraction by antioxidants in human placental mitochondria. *Toxicol In Vitro* 2002;16(6):649-56.
51. Custodio JB, Cardoso CM, Almeida LM. Thiol protecting agents and antioxidants inhibit the mitochondrial permeability transition promoted by etoposide: implications in the prevention of etoposide-induced apoptosis. *Chem Biol Interact* 2002;140(2):169-84.
52. Nakai A, Shibazaki Y, Taniuchi Y, et al. Vitamins ameliorate secondary mitochondrial failure in neonatal rat brain. *Pediatr Neurol* 2002;27(1):30-35.
53. Werbach MR. Nutritional strategies for treating chronic fatigue syndrome. *Altern Med Rev* 2000;5(2):93-108.
54. Abraham GE, Flechas JD. Management of fibromyalgia: rationale for the use of magnesium and malic acid. *J Nutr Med* 1992;3:49-59.
55. Bobyleva-Guarriero V, Lardy HA. The role of malate in exercise-induced enhancement of mitochondrial respiration. *Arch Biochem Biophys* 1986;245(2):470-76.
56. Kruse CA. Treatment of fatigue with aspartic acid salts. *Northwest Medicine* 1961;597-603.
57. Nagle FJ, Balke B, Ganslen R, et al. The mitigation of physical fatigue with "spartase." *FAA Office of Aerospace Medicine* 1963;63-12:1-10.
58. Ahlborg B, Ekelund LG, Nilsson CG. Effect of potassium-magnesium-aspartate on the capacity for prolonged exercise in man. *Acta Physiol Scand* 1968;74:238-45.
59. Sen P, Maiti PC, Puri S, et al. Mechanism of anti-stress activity of *Ocimum sanctum* Linn, eugenol and *Tinospora malabarica* in experimental animals. *Indian J Exp Biol* 1992;30(7):592-96.
60. Mishra LC, Singh BB, Dagenais S. Scientific basis for the therapeutic use of *Withania somnifera* (ashwagandha): a review. *Altern Med Rev* 2000;5(4):334-46.
61. Bhargava KP, Singh N. Anti-stress activity of *Ocimum sanctum* Linn. *Indian J Med Res* 1981;73:443-51.
62. Singh S, Majumdar DK, Rehan HM. Evaluation of anti-inflammatory potential of fixed oil of *Ocimum sanctum* (Holy basil) and its possible mechanism of action. *J Ethnopharmacol* 1996;54(1):19-26.
63. Sembulingam K, Sembulingam P, Namasivayam A. Effect of *Ocimum sanctum* Linn on noise induced changes in plasma corticosterone level. *Indian J Physiol Pharmacol* 1997;41(2):139-43.
64. Singh A, Saxena E, Bhutani KK. Adrenocorticosterone alterations in male, albino mice treated with *Trichopus zeylanicus*, *Withania somnifera* and *Panax ginseng* preparations. *Phytother Res* 2000;14(2):122-25.
65. Singh HK, Rastogi RP, Srimal RC, et al. Effect of bacosides A and B on avoidance responses in rats. *Phytother Res* 1998;2(2):70-75.
66. Stough C, Lloyd J, Clarke J, et al. The chronic effects of an extract of *Bacopa monniera* (Brahmi) on cognitive function in healthy human subjects. *Psychopharmacology* 2001;156:481-84.
67. Fidanaza A, Floridi S, Lenti L. Panthenol and glucocorticoids. *Boll Soc Ital Biol Sper* 1981;57(18):1869-72.
68. Lau-Cam CA, Thadikonda KP, Kendall BF. Stimulation of rat liver glycogenolysis by vitamin B_6: a role for adrenal catecholamines. *Res Commun Chem Pathol Pharmacol* 1991;73(2):197-207.
69. Witchl M. Herbal drugs and phytopharmaceuticals. London: CRC Press; 1989.
70. Brown KM, Arthur JR. Selenium, selenoproteins and human health: a review. *Public Health Nutr* 2001;4 (2B):593-99.
71. Nishiyama S, Futagoishi-Suginohara Y, Matsukara M, et al. Zinc supplementation alters thyroid hormone metabolism in disabled patients with zinc deficiency. *J Am Coll Nutr* 1994;13(1):62-67.
72. Gupta P, Kar A. Cadmium induced thyroid dysfunction in chicken: hepatic type 1 iodothyronine 5'-monodeiodinase activity and role of lipid peroxidation. *Comp Biochem Physiol C Pharmacol Toxicol Endocrinol* 1999;123(1):39-44.
73. Pallet V, Audouin-Chevallier I, verret C, et al. Retinoic acid differentially modulates triiodothyronine and retinoic acid receptors in rat liver according to thyroid status. *Eur J Endocrinol* 1994;131(4):377-84.
74. Shrader M, Muller KM, Nayeri S, et al. Vitamin D3 - thyroid hormone receptors heterodimer polarity directs ligand sensitivity of transactivation. *Nature* 1994;370(6488):382-86.
75. Bland JS, Barrager E, Reedy RG, et al. A medical food-supplemented detoxification program in the management of chronic health problems. *Altern Ther Health Med* 1995;1(5):62-71.
76. Rigden S. Entero-hepatic resuscitation program for CFIDS. *The CFIDS Chronicle* 1995;Spring:46-49.
77. Saller R, Meier R, Brignoli R. The use of silymarin in the treatment of liver diseases. *Drugs* 2001;61(14):2035-63.
78. Wellington K, Jarvis B. Silymarin: a review of its clinical properties in the management of hepatic disorders. *BioDrugs* 2001;15(7):465-89.
79. Barch DH, Rundhaugen LM, Stoner GD, et al. Structure-function relationships of the dietary anticarcinogen ellagic acid. *Carcinogenesis* 1996;17(2):265-69.
80. Ahmed S, Rahman A, Saleem M, et al. Ellagic acid ameliorates nickel induced biochemical alterations: diminution of oxidative stress. *Human Exp Toxicol* 1999;18:691-98.
81. Bu-Abbas A, Clifford MN, Walker R, et al. Selective induction of rat hepatic CYP1 and CYP4 proteins and of peroxisomal proliferation by green tea. *Carcinogenesis* 1994;15(11):2575-79.
82. Ahmad N, Muktar H. Green tea polyphenols and cancer: biological mechanisms and practical implications. *Nutr Rev* 1999;57(3):78-83.
83. Olmstead MJ. Heavy metal sources, effects, and detoxification. *Altern Ther Complement Med* 2000;Dec:347-54.
84. Quig D. Cysteine metabolism and metal toxicity. *Altern Med Rev* 1998;3(4):262-70.
85. Clements ML, Levine MM, Ristaino PA, et al. Exogenous lactobacilli fed to man - their fate and ability to prevent diarrheal disease. *Prog Ed Nutr Sci* 1983;7:29-37.
86. Alm L. The effect of *Lactobacillus acidophilus* administration upon the survival of *Salmonella* in randomly selected human carriers. *Prog Food Nutr Sci* 1983;7(3-4):13-17.
87. Bazzocchi G, Gionchetti P, Almerigi PF, et al. Intestinal microflora and oral bacteriotherapy in irritable bowel syndrome. *Dig Liver Dis* 2002;34 (Suppl 2):S48-S53.
88. Wakahayshi H, Orihara T, Nakaya A, et al. Effect of *Helicobacter pylori* infection on gastric mucosal phospholipid contents and their fatty acid composition. *J Gastrenterol Hepatol* 1998;13:566-71.
89. Lewis DA, Shaw GP. A natural flavonoid and synthetic analogues protect the gastric mucosa from aspirin-induced erosions. *J Nutr Biochem* 2001;12:85-100.
90. Wakahayshi H, Orihara T, Nakaya A, et al. Effect of *Helicobacter pylori* infection on gastric mucosal phospholipid contents and their fatty acid composition. *J Gastrenterol Hepatol* 1998;13:566-71.
91. Robinson RR, Feirtag J, Slavin JL. Effects of dietary arabinogalactan on gastrointenstinal and blood parameters in healthy human subjects. *J Am Coll Nutr* 2001;20:279-85.
92. Kelly GS. Larch arabinogalactan: Clinical relevance of a novel immune-enhancing polysaccharide. *Alt Med Rev* 1999;4:96-103.
93. Geel SE, Robergs RA. The effect of graded resistance exercise on fibromyalgia symptoms and muscle bioenergetics: a pilot study. *Arthritis Rheum* 2002;47(1):82-86.
94. Lambert M, Marcus P, Burgess T, et al. Electro-membrane microcurrent therapy reduces signs and symptoms of muscle damage. *Med Sci Sports Exerc* 2002;34(4):602-07.
95. McMakin C. *Treatment of Resistant Myofascial Pain with Microcurrent Using Specific Microcurrent Frequencies Applied with Graphite/Vinyl Gloves*. Retrieved December 11, 1997 from, http://www.drcarol.org/page.asp?PID=1064
96. Cheng N, Van Hoof H, Bockx E, et al. The effects of electric currents on ATP generation, protein synthesis, and membrane transport of rat skin. *Clin Orthop* 1982;(171):264-72.

NATURAL THERAPIES FOR FIBROMYALGIA SYNDROME: A SUMMARY

By Kristi Hawkes, N.D.

Fibromyalgia (FM) is a condition characterized by symptoms such as fatigue, muscle and bone pain, and specific areas of tenderness referred to as "tender points." Other common symptoms include sleep disturbances, morning stiffness, headaches, bowel irregularities, depression, and anxiety.[1,2] Approximately 3-6 million Americans suffer from FM, most of whom are women.[3,4] However, FM can also occur in men, children, and the elderly.

How is Fibromyalgia Diagnosed?

Fibromyalgia can be diagnosed by a tender point examination. Mildly irritating to completely debilitating tenderness in at least 11 of 18 defined points, such as in the neck, spine, shoulders, and hips, signifies FM.[6,7]

What Causes it?

The cause of FM has yet to be determined. But research suggests that abnormalities in the muscular, endocrine, nervous, or digestive systems should be addressed, and exposure to toxic substances should be reduced.[1,2]

Muscular System: Mitochondrial Dysfunction

Mitochondrial dysfunction is thought to be a primary cause of FM. Mitochondria are found in all cells of the body, but are highly concentrated in muscle cells. They are responsible for cellular energy production. Decreased cellular energy production in muscle cells can result in increased muscle stiffness, pain, and fatigue in FM patients.[17]

Nervous System: Toxin Exposure

Every day we are exposed to numerous toxins such as pesticides, food additives (e.g., MSG, aspartame), and chemicals that can adversely affect various bodily functions, including nervous system function.[22,23] Excessive toxin exposure can result in increased and prolonged pain sensation by the nervous system, contributing to the chronic pain associated with FM.[24-27]

Digestive System: Intestinal Tract Abnormalities

Data suggest that up to 70% of patients with FM complain of symptoms associated with irritable bowel syndrome (IBS), such as chronic abdominal pain, alternating diarrhea and constipation, morning stiffness, and fatigue.[30,31] Since IBS and FM have overlapping symptoms, it has been suggested they may have a common cause.[31,34]

It is further theorized that dysfunction in nerve pathways between the intestinal tract and the brain help to explain the increased pain sensitivity and fatigue in FM.[35]

Endocrine System: Hormone Imbalance

Many patients with FM have experienced significant amounts of stress, which can contribute to abnormalities in the endocrine system.[18] The endocrine system regulates hormone (e.g., serotonin, cortisol, thyroxine) production by portions of the brain, as well as by the adrenal and thyroid glands. Imbalanced hormone production caused by stress can contribute to the increased fatigue, sleep disturbances, and psychological distress experienced by FM patients.[19,20]

How is it Treated?

Conventional Medical Approaches

Doctors may prescribe antidepressants, sleeping pills, muscle relaxants, and/or pain-relief medications.[16] Unfortunately, none of these medications have been shown to be of significant benefit and they can cause adverse side effects (e.g., headache, nausea, stomach bleeding).[2,16]

A Natural Treatment Approach

In addition to a healthy diet and regular exercise program as prescribed by your healthcare provider, incorporating a basic nutritional regimen that addresses the abnormalities previously discussed may be of benefit (Table 1).

Table 1. What a Basic Nutritional Supplement Program for FM Patients May Include.

Nutritional Supplement	Area of Support
Multivitamin/Mineral Formula	Overall health and energy levels
EPA/DHA 30:20 Formula (Omega-3 fatty acids)	Overall health of cells that house mitochondria
Mitochondrial Antioxidant Formula	Muscular system: comprehensive mitochondrial support
Malic Acid/Magnesium Complex **OR** Magnesium/Potassium Aspartate Complex	Muscular system: mitochondrial energy production
Bifunctional Detoxification Support Formula	Nervous system: elimination of toxins
L. Acidophilus NCFM and Bifidobacteria	Digestive system: intestinal health and function
If stress is an issue, then add:	
Traditional Ayurvedic Holy Basil Combination	Endocrine system: hormone balance and stress response
B_6/Pantothenic Acid Complex	Endocrine system: adrenal hormone production and energy

Additional Therapy

Frequency-Specific Microcurrent

Injured tissue has altered electrical dynamics as compared to healthy tissue. These altered electrical dynamics can cause changes in cellular function, resulting in impaired healing and painful inflammation.[95] Frequency-specific microcurrent therapy is the application of an extremely low frequency of electrical current (approximately one millionth of an amp) to control pain and stimulate healing of injured tissue, such as muscle, on a cellular level.[94,95] This "bio-electric therapy" supports the natural healthy electrical current flow in tissue, which is important to mitochondrial energy production, the transport of nutrients to cells, and the removal of wastes from cells.[95] The actual electrical current administered is so small that it cannot be felt.

Conclusion

Because FM has been associated with abnormalities in multiple body systems, treatment that addresses each of these systems may be of benefit. While medications commonly prescribed for FM complaints may provide temporary relief, they may not address the underlying factors involved and can cause undesirable side effects such as headache, nausea, or stomach bleeding. On the other hand, good nutrition, exercise, and dietary supplements that support the muscular, nervous, digestive, and endocrine systems and facilitate the elimination of toxins may assist in the healing process.