The Healing Power of Rainforest Plants

Fibromyalgia

Hypothesis:
Fibromyalgia is caused by a mycoplasmal infection in the neuroendocrine system.

Justification:

The advances in research on Fibromyalgia Syndrome (FMS) over the last 5 years has targeted specific links and correlations which might indicate a dysregulation or imbalance of the neuroendocrine system, especially the HPA axis, which may well explain many of the seemingly unrelated symptoms presented by FMS patients. Research supports that various components of the central nervous system appear to be involved, including the hypothalamic pituitary axes, pain-processing pathways, and autonomic nervous system. The advances in gene research during this same time peroid has provided new evidence in the identification and pathogenesis of specific species of mycoplasmas which might have the ability to cause a dysregulation of the neuroendocrine system. To learn more about mycoplasmas,

how they deregulate cellular functions and cause a wide rang of diseases and syndromes in the body: Follow these links:

What is a Mycoplasma? (myco.htm)

What diseases can they cause? (myco.htm#disease)

Treatment Options (myco.htm#treatment)

Laboratory Testing for Mycoplasmas (myco.htm#testing)

Medical Research and Studies on Mycoplasmas (mycoresearch.htm)

Fibromyalgia is an extremely common chronic condition that can be challenging to manage. Although the etiology remains unclear, characteristic alterations in the pattern of sleep and changes in neuroendocrine transmitters such as serotonin, substance P, growth hormone, thyroid hormones, estrogen and cortisol suggest that dysregulation of the autonomic and neuroendocrine system now appears to be the basis of the syndrome.

Evidence of neuroendocrine system dysfunction in Fibromyalgia:

Thyroid

Virtually every feature of fibromyalgia corresponds to signs or symptoms associated with failed transcription regulation by thyroid hormone.(See Table) In the late 1980's to early 1990's some diagnoses of fibromyalgia and rheumatic diseases were subsequently changed to a hypothyroidism diagnosis after thyroid testing was performed.[4, 6, 7] In a 1992 german study thyroid function was tested in 13 female patients with primary fibromyalgia syndrome (FMS) 10 healthy age matched controls by intravenous injection of 400 micrograms thyrotropin-releasing hormone (TRH). Basal thyroid hormone levels of both groups were in the normal range. However, patients with primary FMS responded with a significantly lower secretion of thyrotropin and thyroid hormones to TRH, within an observation period of 2 h, and reacted with a significantly higher increase of prolactin. Total and free serum calcium and calcitonin levels were significantly lower in patients with primary FMS, while both groups exhibited

parathyroid hormone levels in the normal range. This phenomenon would not be discovered in a doctor's routine thyroid tests of measuring T3, T4, and TSH since it is indicative of secondary hypothyroidism (Euthyroid) rather than primary hypothyroidism. Recent research suggests the cause of this phenomenon to be a newly discovered gene mutation reporting: "in hypothyroid fibromyalgia, failed transcription regulation would result from thyroid-hormone deficiency. In euthyroid fibromyalgia, failed transcription regulation may result from low-affinity thyroid hormone receptors coded by a mutated c-erbA beta 1 gene, yielding partial peripheral resistance to thyroid hormone. The result would be tissue-specific hypothyroid-like symptoms despite normal circulating thyroid-hormone levels."[(1)]

In clinical practice is has been demonstrated that a significant percentage of FMS patients are tested with secondary hypothyroidism (euthyroid). This is missed by many health practitioners who routinely test thyroid function by measuring common thyroid hormones T3, T4, and TSH (Thyroid stimulating hormone). The majority of FMS patients will test in normal ranges. However, thyroid dysfunction is often revealed in FMS patients when a TRH stimulus test (Thyrotropin-releasing hormone) is administered. Medical practitioners have been able to manage symptoms of FMS patients with some degree of success by administering thryoid homone replacement therapy for several years now.[(2)] Other researchers suggest that FMS patients may have a form of thryoid autoimmunity, reporting the prevalence of thyroid microsomal antibodies were significantly higher in persons with than without chronic widespread musculoskeletal complaints. [(3)]

The thyroid produces hormones that increase oxygen use in cells and stimulate vital processes in every part of the body. These thyroid hormones have a major impact on growth, use of energy, heat production, and infertility. They affect the use of vitamins, proteins, carbohydrates, fats, electrolytes, and water, and regulate the immune response in the intestine. They can also alter the actions of other hormones and drugs. The two key thyroid hormones are thyroxine (T4) and L-triiodothyronine (T3). Iodine is the raw material used in the manufacture of these hormones; it is extracted from the blood and trapped by the thyroid gland where 80% of the body's iodine is stored. The thyroid mostly produces thyroxine, which in turn, is converted into T3, the more biologically active thyroid hormone. Only about 20% of T3 is actually formed in the thyroid gland, however; the rest is manufactured from circulating thyroxine in tissues outside the thyroid. The whole process of iodine trapping and thyroid hormone production is directly influenced by another important hormone, thyroid-stimulating hormone (TSH or thyrotropin). This hormone is secreted by the pituitary gland and monitored by thyrotropin-releasing hormone (TRH), which is produced in the hypothalamus gland. Both glands are located in the brain. Any abnormality in this intricate system of glands and hormone synthesis and production can have far-reaching consequences.

In autoimmune diseases, the body's immune system attacks its own cells; in the case of autoimmune thyroiditis, the cells under attack are in the thyroid gland. Experts do not know why the immune system starts to injure the thyroid. One theory is that a virus or bacteria with a protein resembling a thyroid protein might trigger the response. This theory is backed up to some extent by the presence of recent infections in people with autoimmune disease.

Footnotes:

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Symptoms of Hypothyroidism	**Symptoms of Fibromyalgia**
Chronic fatigue	Chronic fatigue
Muscle and joint aches	Muscle and joint aches
Muscle cramps	Muscle cramps
Muscle weakness	Muscle weakness
Concentration and memory problems	Concentration and memory problems

Depression and mood swings	Depression and mood swings
Weight gain	Weight gain
Constipation	IBS including constipation
Obstructive sleep apnea	Sleep disorders and insomnia
Heavy menstruation	
Dry Skin & Hair	
Flaky/split fingernails	
Hair loss	
Sensitivity to cold	
Low blood pressure	

The hypothalamic pituitary adrenal axis

The brain and the immune system are the two major adaptive systems of the body. During an immune response the brain and the immune system "talk to each other" and this process is essential for maintaining homeostasis. Two major pathway systems are involved in this cross-talk: the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS).(6) Living organisms survive by maintaining an immensely complex dynamic equilibrium of the internal milieu or homeostasis. The systemic sympathetic and adrenomedullary (sympathetic) system (SNS) and the HPA axis are the peripheral limbs of the stress system, whose main function is to maintain both basal and stress-related homeostasis. At rest catecholamines (CA) maintain homeostasis as major regulators of fuel metabolism, heart rate, blood vessel tone, and thermogenesis. When homeostasis is disturbed or threatened by internal or external challenges, both the SNS and HPA axis become activated, resulting in increased peripheral levels of CAs and glucocorticoids that act in concert to keep the steady state of the internal milieu.

Researchers around the world have published articles and research which indicate "defects in the hypothalamus-pituitary-adrenal axis have been observed in autoimmune and rheumatic diseases, chronic inflammatory disease, chronic fatigue syndrome and fibromyalgia."(3) Research as early as 1989 indicated data suggesting alteration in the pituitary hypothalamic axis with respect to cortisol secretion in fibromyalgia syndrome.(4) A1998 German study discovered: "We found in FMS patients elevated basal values of ACTH and cortisol, lowered basal values of insulin-like growth factor I (IGF-I) and of triiodothyronine (T3), elevated basal values of follicle-stimulating hormone (FSH) and lowered basal values of estrogen. Following injection of the four releasing-hormones, we found in FMS patients an augmented response of ACTH, a blunted response of TSH, while the prolactin response was exaggerated. The effects of LHRH stimulation were investigated in six FMS patients and six controls and disclosed a significantly blunted response of LH in FMS. We explain the deviations of hormonal secretion in FMS patients as being caused by chronic stress, which, after being perceived and processed

by the central nervous system (CNS), activates hypothalamic CRH neurons. CRH, on the one hand, activates the pituitary-adrenal axis, but also stimulates at the hypothalamic level somatostatin secretion which, in turn, causes inhibition of GH and TSH at the pituitary level."(1) In several studies, histologic muscle abnormalities of membranes, mitochondria, and fiber type have been well described at both the light microscopic and ultrastructural levels. These abnormalties found in muscle tissues of FM patients have been reported to be "consistent with neurologic findings and disturbances in the hypothalamic-pituitary-adrenal axis" and suggests, "muscle abnormalities may be elicited by intrinsic changes within the muscle tissue itself and/or extrinsic neurologic and endocrine factors.(5) Another research group found "The influence of maximum exercise has been studied in 10 patients with primary fibromyalgia syndrome (PFS) and 10 healthy sedentary control persons. The exercise consisted of a bicycle ergometertest and a steptest, both till exhaustion. In both tests, the mean maximum workload of the PFS patients was lower than that of the controls. Significantly lower values of serum creatinekinase, myoglobin, cortisol, epinephrine and norepinephrine were found in PFS patients. A striking finding was a lower heart rate in PFS patients compared to the controls under the same workload. The lower (nor)epinephrine concentration together with the lower heart rate suggests a disturbance of the sympathetic activity in PFS patients. The preliminary conclusion is that there is a disturbed reactivity of the sympathetic system as well as of the HPA axis in PFS."(7)

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Hypothalmus

Recent research this year has demonstrated FMS patients have a decrease in GH (growth hormone) over healthy subjects suggesting "Severe GH deficiency is not a significant pathogenic factor in most patients with FMS. We observed an impaired reactivity of the somatotropic axis in one-third of patients with FM, in keeping with a functional alteration of the hypothalamus."(1) "Patients with fibromyalgia were found to have an impaired ability to activate the hypothalamic pituitary portion of the hypothalamic pituitary adrenal axis as well as the sympathoadrenal system, leading to reduced corticotropin and epinephrine response to hypoglycemia." (2) In fact, FMS patients complaining of "fibro-fog" have later been diagnosed with intermitent hypoglycemia which is causing sort term memory loss, periods of confusion or "fog" and even stupor-like states as blood sugar levels drop unexpectedly. This intermittent hypoglycemia may result from this impaired hypothalamic pituitary function.

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The Correlation between Fibromyalgia and Mycoplasmas

Mycoplasmas require a large amount of cholesterol and other sterols for growth and reproduction. If mycoplasmas are present and are competing for these sterols intracellularly, less is available to the body and especially the neuroendocrine system for the sythesis and manufacture of steroids like estrogens, growth hormones and cortisols which many FMS patients have been found to be deficent in.

Mycoplasmas also need and utilize proteins derived from amino acids. Amino acids are the core building blocks in the neuroendocrine system for the synthesis of most chemicals produced and used in the complex intricate pathways of the neuroendocrine system including thyroid hormone production and other chemicals taken up and used in the HPA. This may cause the deregulation of the neuroendocrine system thru the loss of vital nutrients required to maintain regulation.

Mycoplasma maintains a defense mechanism which encodes tryptophan to hide from normal immune responses. If enough tryptophan was utilized from host cells in the neuroendocrine system for this

purpose, less would be available to the body for the normal synthesis of tryptophan to seratonin, thus possibly deregulating or lowering seratonin levels and causing depression and sleep disorders most FMS patients present.

Mycoplasmas have the ability to attached to any cell in the body and cause that cell to malfunction, acting differently, thereby causing different interactions with other cells. If mycoplasmas invaded and attached to various cells in endocrine organs, it could cause the widespread deregulation of the entire endocrine system described in the previous research shown above, because of the complex interactions between these organs and the chemicals they produce and utilize.

For more information on how mycoplasmas cause disease and deregulation of systems, organs and cells, see the Simple File on Mycoplasmas (myco.htm) which also includes treatment protocols or the more Technical file on Mycoplasmas (mycoresearch.htm) which provides more links to clinical research published about their actions and pathogenisis.

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"Never doubt that a small group of thoughtful committed citizens can change the world; indeed, it's the only thing that ever has."

Margret Mead

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