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CHRONIC FATIGUE SYNDROME

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Chronic fatigue syndrome (CFS) is a mystery illness, at least according to medical authorities. Controversy continues regarding the cause and treatment of the condition. Though considered by many to be a "new" illness, syndromes with remarkably similar symptoms have been reported for more than 100 years.

During the 19th century, nervous exhaustion or ***neurasthenia*** (meaning lack of nerve force or nervous system weakness) was a popular diagnosis. It too was thought to be a new disease.

George Beard, M.D. (1839-1883), first coined the term *neurasthenia* to describe persons with unexplained chronic fatigue, lassitude, and weakness. Often accompanying these symptoms were complaints such as nervousness, irritability, anxiety, depression, headaches, insomnia, poor appetite, menstrual or sexual disorders, and more. Charles Darwin and Florence Nightingale may have suffered with this syndrome.

Today, one "has only to consult current medical journal articles on chronic fatigue syndrome to see that symptom puzzles that engaged our recent ancestors continue to intrigue us." CFS may also be known as myalgic encephalomyelitis, low natural-killer-cell syndrome, post-infective fatigue syndrome, postviral fatigue syndrome, chronic Epstein-Barr virus syndrome, viral exhaustion, atypical depression, and illness behavior. "Most of these labels have revealed more about the assumptions and attitudes of the doctors involved than about their patients."

For example, in 1984 two doctors in Nevada reported an 'epidemic' of severe fatigue among their patients. They proposed the cause was chronic infection with the Epstein-Barr virus. However, this 'virus' is ubiquitous -- it seems to be everywhere. Most people are found to have "antibodies" to it, theoretically indicating past exposure; but they do not all have CFS. And, not all CFS victims have the antibodies.

The disorder received the title "chronic fatigue syndrome" in 1988 by the Centers for Disease Control (CDC) along with a specific operational definition. In December 1994 a revision of the disease definition was made by a group of "international experts" convened by the CDC.

The primary complaint must be unexplained "**persistent or relapsing fatigue**" lasting six months or more, which cannot be relieved by rest, and results in substantial reduction in previous levels of work, school, social, or personal activities. This fatigue must be accompanied by four or more 'clinical' symptoms of the same or similar duration, including **unrefreshing sleep, headaches, lingering tiredness after sleep, significant impairment in short-term memory or concentration, muscle pain, joint pain, sore throat, and tender lymph nodes.** Many patients have

additional symptoms, but the above are the current "official" criteria.

A "careful medical history and examination" must rule out any other possible causes of fatigue such as untreated hypothyroidism, alcohol or drug abuse, severe obesity, or certain psychiatric conditions including major depression with psychotic or melancholy features, manic depression, schizophrenia, delusional disorders, dementias, and eating disorders.

Since the cause of CFS is "unknown" and there are no laboratory tests to prove its presence, doctors have been advised to diagnose it with caution. Nevertheless, when no other obvious cause for chronic fatigue is determined, CFS is frequently utilized as a readily accessible label. [i]

Estimating the prevalence of CFS -- even by the present diagnostic definition -- has been difficult. According to a large, four-city CDC study, there is a minimum of four to 10 cases per 100,000 persons. Yet, a Harvard Medical School professor, Anthony L. Komaroff, estimated that at least one in 1,000 Americans have the syndrome, or about 250,000 people in this country. [ii]

A concept that may underlie one of the "puzzling difficulties" with CFS is the distinction still generally drawn by doctors and patients between body and mind. "Real" illnesses have an organic "provable" source -- "a demonstrable abnormality of molecular or cellular structure, a lesion of some kind." On the other hand, depression and other psychiatric disorders are different -- "all in the mind" -- and are thus stigmatised. When physicians fail to find **clinical** indications of disease, they suggest that their distressed and bewildered patients see a psychiatrist. Usually the patient angrily refuses to do so, contending that something is actually and physically wrong. [iii]

CFS AND THE IMMUNE SYSTEM

Most CFS patients report that their symptoms began after some "infection" -- a cold, flu, bronchitis, skin disorder, hypersensitive state (often called an "allergy"), etc. -- but many other cases arise after other stressful events like major surgery, the death or serious illness of a family member, and similar traumas. Some cases develop gradually over time.

Studies have attempted to link CFS with various viruses or groups of viruses such as Epstein-Barr Virus, Cytomegalovirus, Herpes Simplex Virus 1 and 2, Human Herpes Virus 6, Human T-Lymphotropic Virus Type II, spumaviruses, retroviruses, enteroviruses, Coxsackie ("polio") or Echo viruses, and other entities. However, "to date none of these have been exclusively associated with the illness."

Overall, the "complexities" associated with CFS "relate to diagnosis and reflect the inability of investigators to identify pathognomonic [causative] findings" for the syndrome. The CDC hypothesizes that an interaction and combination of immunological, infectious, psychological, and environmental factors cause CFS symptoms. Put succinctly, they don't know what causes it. [iv]

Generally, investigators have noted some "immunological abnormalities." While a study at the University of Miami found that many CFS sufferers had a "dysfunction" in their natural killer cells, another study did not find this abnormality. "Other research has found similarly contradictory results." It is not known whether the observed immunologic abnormalities or the inconsistent frequency or pattern of seropositivity or "other entities" are causal or are "merely epiphenomena" -- just there by chance. The data are obviously vague. [v]

"Virtually everybody in the [medical] field agrees there is some immunologic disturbance in these patients," evidenced

by a multitude of scientific articles based on this opinion. "The big problem is that the presumed immune dysfunction "is different in each article." [vi]

CFS AND MYALGIC ENCEPHALOMYELITIS

It has been noted that there are "striking similarities" among neurasthenia, CFS, and ***benign myalgic encephalomyelitis*** (ME) -- inflammation of the brain and spinal cord with resultant muscle pain. Symptoms include **unexplained chronic fatigue** and **profound muscular weakness** made worse by exercise.

Many symptoms of ME (and of CFS) "can only be understood as disturbances of cerebral functioning." **Depression, anxiety, nervousness, irritability, insomnia, impaired concentrtion and memory**, etc., are not attributable to localized muscle disease or muscle abnormalities. Obviously, the nervous system is involved -- physiologically, biochemically. Indeed, "the symptoms of most patients with chronic fatigue states are real, pervasive, and often incapacitating," NOT all in the mind. [vii]

In 1956 the term *benign myaligic encephalomyelitis* was introduced to describe cases of a "mysterious infective illness with symptoms and signs that strongly suggested active inflammation of the brain and spinal cord." As such, could ME or CFS be a result of vaccination, specifically polio vaccination -- an insult to the central nervous system? William Campbell Douglass, M.D., thinks so. He says that "chronic fatigue syndrome is not a new disease, but simply an '**aborted form**' of the more serious paralytic polio." He explains that, after the introduction of the polio vaccines in the 1950s, "the trend toward a **new** polio rapidly increased and it has been recognized by the neurologists for **40 years**. The terms 'atypical' and 'abortive' polio have been quietly dropped because they would point to the awful realization that polio is more common than ever **and caused by polio vaccination**." (Emphasis added)

Dr Elizabeth Dowsett, a microbiologist from Britain, unequivocally states that true CFS (as differentiated from other fatigue states) "strikes one clinically as being **polio-like** and it has often been diagnosed as a '**nonparalytic polio**.'" [viii]

CFS AND THE NERVOUS SYSTEM

Whether or not the polio vaccine is a cause, CFS is largely a neurological illness which affects the musculoskeletal system. Profound fatigue is only part of the symptom picture. As exhausted and immobilized as they may be, many victims say their lives are even more deeply affected by changes in their brains. "They forget things. They stumble over simple words or figures. They can't concentrate enough to read a few paragraphs." A 42-year old woman with CFS laments: "Of all the things I've lost, I miss my mind the most." [ix]

A high prevalence of "psychiatric disorders" -- up to 70% -- has been found in most studies of CFS patients. About half suffer with **major depression**. They are nearly six times more likely than others to be depressed or anxious or to have a history of these disorders. Other emotional factors are also commonly observed as already mentioned. [x]

While the medical establishment continues to seek a clinically diagnosable 'disease,' perhaps it is time to consider **subclinical nutritional deficiency**. One candidate is a ***vitamin B complex deficiency syndrome*** -- a deficit of the vitamin B complex (all the B vitamins are interrelated so deficits of one accompanies deficits of others) and related nutrients such as specific minerals, trace minerals, amino acids, other vitamin complexes, and enzyme synergists required for nerve health and function. Symptoms of **subclinical**

deficiencies of the vitamin B complex (and cooperative nutrients) may include:

Unusual fatigue, exhaustion, weakness, muscle soreness or achiness, numbness, tingling or even pain, craving for sweets, nervousness, restlessness, anxiety, apprehension, vague fears, irritability to rage, uneasiness to panic, indigestion, constipation or diarrhea, stomach pains, decreased or increased appetite, mood swings, morbid thoughts, severe depression, loss of ability to concentrate, loss of memory, mental confusion, menstrual complaints (in women), cold hands and feet, headaches, soreness of the mouth, dermatitis (dry, greasy skin), facial oiliness, acne, slow pulse or fast pulse, burning feet, insomnia or sleep disturbances, flights of ideas, lightheadedness to dizziness, palpitations, pain in the chest, noise sensitivity, acoustic hallucinations (hearing voices, etc.), inability to handle stress, suspicions, hypochondria, heightened sensitivity (to pain, touch), burning or itching of the eyes, 'electric shock' sensations, a constant feeling that "something dreadful is about to happen," difficulty swallowing, sore throat.

The above complaints mirror those of persons diagnosed with CFS. [xi]

CFS AND THE ADRENAL GLANDS

The appearance of an "immune system engaged in some kind of chronic war" and the consistent findings indicating that CFS patients have altered levels of certain brain hormones brings up another aspect. Scientists at the National Institute of Mental Health, the University of Michigan, and the National Institute of Allergy and Infectious Diseases found that the hypothalamic-pituitary-adrenal (HPA) axis in CFS was often altered. Patients consistently showed decreased levels of the steroid hormone, **cortisol** (from the adrenal glands), and increased levels of the pituitary hormone, **ACTH** (adrenocorticotropic hormone). ACTH stimulates the adrenal cortex to produce hormones such as cortisol, the most potent of the naturally-occurring glucocorticoids. Cortisol is important for (1) its regulatory action in the metabolism of fats, carbohydrates, sodium, potassium, and proteins, (2) its influence on the nutrition and growth of connective (collagenous) tissues such as bone, ligament, cartilage, tendon, muscle, skin, etc., and (3) its regulation of the immune system in many ways.

When the adrenal cortex cannot adequately produce its steroid hormones, the pituitary gland -- via ACTH -- attempts to "nudge" or signal the adrenals to pick up production and secretion. IF the adrenals cannot keep up with the body's demands, elevated ACTH levels and decreased cortisol levels can be expected.

This finding by researchers "provides an alternative explanation for some of the immune findings in the syndrome." Instead of some illusive "infection," adrenal gland fatigue would account for the various immune system deficits and compensations -- depending upon individual biochemistry -- usually seen in CFS. It would also account for the common history of some inflammatory incident that precedes CFS. Such an incident (or incidents, if chronic) would stress the adrenals, no doubt already somewhat depleted, into further debilitation. This stress would be especially disruptive if medications or high-potency, crystalline-pure vitamin fractions were used to interfere with any stages of inflammation and repair. [xii]

The laboratory findings, according to the above researchers, are "incompatible with a primary adrenal insufficiency," that is, with a **clinical** or serious disease. A pituitary "disease" is also unlikely. Why

not simply consider **subclinical adrenal fatigue** which would clearly explain the findings? [xiii]

Subclinical adrenal fatigue could -- especially in conjunction with a subclinical vitamin B complex deficiency syndrome -- account for the "neurally mediated hypotension" (NMH) found in some CFS patients. This disorder is triggered when the body's nervous system incorrectly adjusts blood pressure and heart rate. **Faintness** (dizziness, loss of postural tone, lightheadedness) caused by diminished cerebral blood flow, is common with this type of hypotension. "Susceptible individuals develop an increased **catecholamine** [epinephrine and/or norepinephrine] response, resulting in symptoms such as **vasodilation** (dilation of the blood vessels), **bradycardia** (slow pulse), **hypotension, stomach discomfort, nausea, pallor, blurred vision, sweating, headaches** after exercising, etc. -- all parasympathetic responses.

Epinephrine and norepinephrine are secreted by the adrenal medulla in response to stimulation (stress) of the sympathetic nervous system. Epinephrine causes some of the physiological expressions of **fear and anxiety**. A disturbance in the metabolism of norepinephrine at important brain sites has been implicated in affective disorders -- **disturbance of mood accompanied by full or partial manic or depressive syndrome**. [xiv]

These adrenal-produced catecholamines are important neurotransmitters, linking the nervous system with adrenal gland function. If one or both areas are nutritionally depleted, symptoms of CFS can readily ensue.

Adrenal fatigue or **subclinical hypoadrenia** simply refers to adrenal glands that are not functioning up to their full or normal potential. These are the "stress glands." Any stress -- physical, mental, chemical, thermal -- can drain or weaken these glands **if they are not adequately nourished**.

One ironic aspect of some current CFS therapies is the recommendation of a "healthy" diet which includes no or low salt intake. As Dr. Hugh Calkins, a cardiologist and one of the researchers in the NMH study, puts it: "That may not be a good idea." Salt (specifically sodium) is imperative to adrenal gland health and function. Other trace minerals (potassium, zinc, iron, etc.), minerals (magnesium, calcium, etc.), vitamin complexes (especially the vitamin C complex with its organic copper enzyme, tyrosinase) and amino acids are, of course, also important. But adrenal "defects" frequently create sodium deficiencies, symptoms of which include **muscle weakness, poor memory and concentration, anorexia, acidosis, dehydration, and tissue atrophy**. [xv]

A chronic vitamin B complex deficiency syndrome can also place great stress on the adrenals. When the adrenals can no longer compensate for the demands thus placed on them, nervous exhaustion follows.

Scientists have concrete evidence of adrenal hormone receptors in the brain which "appear to play a key function in the integrative response to stress and participate in regulation of ongoing as well as long-term influences on the adaptive process."

Adrenal hormone actions in many regions of the brain -- including the hippocampus, important both in cognition (reasoning, judgment, perception, and memory) and in affective (emotional and/or mental) response. -- "appear to participate in the response of the brain to repeated stress." The brain's ability to adapt to stressful situations, then, depends, not only on the vitality and well-being of the nervous system itself, but also of the adrenal glands.

And the reverse is true: the nerves have a profound effect on the adrenals. The distribution and close proximity of several nerve plexuses in the outer zone of the adrenal cortex establishes that there is a direct neural effect on adrenal cortical cell functions. Therefore, the brain and rest of the nervous system affects the adrenals, and the adrenals affect the nerve tissues. Subclinical depletions of either area will affect the other. Another "chicken and egg" dilemma, perhaps, but the answer would be to nutritionally support both. [xvi]

Symptoms of sublinical adrenal fatigue include: **the insidious onset of slowly progressive fatigability, weakness, malaise, and asthenia (loss or lack of strength, debility, profound muscular weakness**). Early on such complaints may be sporadic, most evident during or following times of stress. As adrenal function becomes more impaired, the weakness progresses until the patient is continually fatigued, necessitating bed rest. **Gastrointestinal complaints** are common, such as **vague abdominal pain, nausea, indigestion, even vomiting, diarrhea, and mild anorexia.** Findings may include **orthostatic hypotension** (systolic usually below 110, often below 100), **decreased sodium levels** (if severe, low serum sodium) and/or **increased potassium levels** (if severe, elevated serum potassium), **craving for salty foods, mild metabolic acidosis, unexplained eosinophilia** (unusual number of eosinophils in the blood), **relative lymphocytosis** (elevated lymphocytes in the blood), and **fever**. The latter three items indicate acute inflammation. If the medulla is affected, **hypertension, paroxysmal symptoms (such as profuse sweating, episodic palpitations, and headache), tremulousness, and pain in the chest or abdomen** can be present. Whether affecting the cortex or medulla or both, **personality or emotional changes** frequently occur: **excessive irritability and restlessness, agitation, confusion, apprehension, anxiety, depression**, and others. [xvii]

CFS AND NUTRITION

Neurologist George Beard, when describing neurasthenia in 1867, attributed the condition to exhaustion of nerve cells "through depletion of their stored nutriment" or nutritious substance(s). Julius Althaus, a physician who published a neurology textbook in 1893, wrote of neurasthenia and its cerebral origins, most of which he felt were caused by "severe influenzal infection" or inflammation. His favorite treatment for neurasthenia was "cerebrine extract" taken from the brains of young animals. This was the basic equivalent of a modern glandular supplement of brain or nerve extract. It would supply the protein configuration -- the exact same amino acids in the exact same arrangement -- as the human brain and other nerve tissues, as well as some vitamin B complex, minerals, and trace minerals supportive to nerve health and function. [xviii]

Some clinical and experimental findings have revealed deficits of magnesium (including low red cell levels), vitamin B_{12}, and essential fatty acids among CFS patients. These nutrients are all essential to both nerve and adrenal function. At the neuromuscular junction, for example, magnesium ions antagonize the release of acetylcholine -- an ester of choline (of the B complex) which plays a role in nerve impulse transmission. Unless acetylcholine is properly modulated (with magnesium playing a role), excessive muscular stimulation and consequential muscular tension, spasm, or pain will result. [xix]

A deficit of serum acyl-carnitine has been found in some patients with CFS. Carnitine, found in skeletal muscle and the liver, is required for mitochondrial beta oxidation of fatty acids, and is thus

important in the production of energy within skeletal muscle. Deficiencies may result in **myalgia, muscle weakness, general fatigue, and postexercise fatigue**. Though serum acyl-carnitine levels return to normal when fatigue improves, "replacement treatment" of acyl-carnitine does not help. This indicates that the deficit is a **result of**, rather than a cause of, CFS. [xx]

The amino acids **lysine** and **methionine** are needed for carnitine synthesis. Both are essential amino acids (not synthesized by the body) and heat-labile (easily denatured by heat, then no longer bioavailable to form proteins). Cooked and pasteurized foods would not carry these amino acids in usable form. Heat damaged amino acids render the blood proteins unacceptable to the tissues for either maintenance or repair. The consequence is atrophy and degenerative changes in areas including the adrenal glands, thymus gland (essential to immune response), pancreas, and so on. The process for making carnitine also requires **iron** and the **vitamin C complex** (imperative to adrenal gland health and function). [xxi]

A "basic characteristic" of many individuals with chronic fatigue is "inadequate availability of appropriate amino acids." Inadequate intake (in bioavailable form), poor digestion or absorption, or increased needs (due to emotional or physical stress, consumption of refined and processed foods, periods of inflammation or "infection," pollution, ingestion of certain drugs) can -- any or all -- be contributing factors. Deficits of amino acids have been discovered in patients who meet the established definition of CFS. Of course, **all** the amino acids must be available to the cells and be provided simultaneously for proper protein synthesis. If even one of the essential amino acids is ingested at a different time than the other essential and nonessential amino acids, "assimilation of all into protein is curtailed." Total protein is imperative. [xxii]

Back in 1921 Sir Robert McCarrison gathered data showing that the endocrine glands were the first structures to atrophy or degenerate after nutritional deficiencies. The adrenal glands would slacken in their function and show evidence of atrophy with inadequate vitamin A complex, B complex, C complex, E complex, alkaline-ash minerals, and other nutritives. The pituitary, which stimulates the adrenals by ACTH, suffers in function with similar deficits. In 1950, Dr. Royal Lee (D.D.S.) was able to state that adrenal (glandular) extract could be used therapeutically for "atrophic diseases of the muscle and nerve systems."[xxiii]

A patient with CFS can therefore only benefit from a healthful diet which includes plenty of raw foods, as well as supplementation with natural food concentrates to support the nervous system, adrenal glands, and total protein supply. A supplement schedule would include **total vitamin B complex, brain glandular extract, ionizable (free) calcium, the alkaline-ash minerals (which include magnesium, potassium, sodium, zinc, iron, etc.), adrenal glandular extract, vitamin C complex (including tyrosinase), vitamin A complex, essential fatty acids, and total protein such as raw veal bone meal**. The mystery in this "mystery illness" may in this way be solved.

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[i] Robert L. Martensen, M.D., Ph.D., *Journal of the American Medical Association*, Vol.271, No.16, 27 April 1994, p.1243; University of California at Berkeley *Wellness Letter*, Vol.7, Issue 9, June 1991, pp.1-2; Christine Russell, *The Washington Post*, Tuesday, 28 May 1996.

[ii] Don Colburn, *The Washington Post*, Tuesday, 16 January 1996; Christine Russell, *The Washington Post*, 28 May 1996; Dedra Buchwald, M.D., et al., *Annals of Internal Medicine*, Vol.123, No.2, 15 July 1995, pp.81-88.

[iii] R.E. Kendell, *The Lancet*, Vol.341, 1 May 1993, p.1137.

[iv] *Nutri News*, Vol.1, No.5, October 1992, p.1; *Science News*, Vol.139, No.15, 13 April 1991, p. 236; Ron Winslow, *The Wall Street Journal*, Monday, 16 September 1991, p.B1; and 16 January 1992, p.B6; *Journal of the American Medical Association*, Vol.269, No.14, 14 April 1993, pp.1779-1780.

[v] University of California at Berkeley *Wellness Letter*, June 1991, p.2; David P. Dooley, M.D., *Journal of the American Medical Association*, Vol.267, No.7, 19 August 1992, pp.873-874.

[vi] Paul Cotton, *Journal of the American Medical Association*, Vol.266, No.19, 20 November 1991, pp.2667-2668.

[vii] R.E. Kendell, *The Lancet*, Vol.337, No.8734, 19 January 1991, pp.160-161.

[viii] William Campbell Douglass, M.D., *Second Opinion*, Vol. VI, No.8, August 1996, pp.1-6; Charles Shepherd, *The Lancet*, Vol.343, No.8891, 22 January 1994, pp.242-243.

[ix] *The Healthkeepers Journal*, Vol.16, No.2, February 1995, p.19.

[x] *The Lancet*, Vol.339, No.8805, 30 May 1992, p.1349; Helen Cope, et al., *The Lancet*, Vol.345, No.8942, 14 January 1995, p.131, and Vol.344, No.8926, 24 September 1994, pp.864-868; *American Health*, Vol.15, No.9, November 1996, p.21.

[xi] *Cecil Textbook of Medicine*, 20th Ed., Philadelphia: W.B. Saunders Company, 1996, pp.1148-1150; *Harrison's Principles of Internal Medicine*, 13th Ed., New York: McGraw-Hill, Inc., 1994, pp.472-476.

[xii] Joseph Palca, *Science*, Vol.254, 20 December 1991, pp.1726-1728.

[xiii] M. Demitrack, et al., *Journal of Clinical Endocrinology and Metabolism*, Vol.73, No.6, December 1991, pp.1224-1234.

[xiv] Issam Bou-Holaigan, M.D., et al., *Journal of the American Medical Association*, Vol.271, No.12, 27 September 1995, pp.961-967; Peter C. Rowe, et al., *The Lancet*, Vol.345, No.8949, 11 March 1995, pp.623-624.

[xv] Rick Weiss, *Health*, Vol.10, No.1, January/February 1996, pp.42-45; Robert H. Garrison, Jr., R.Ph., and Elizabeth Somer, M.A., R.D., *The Nutrition Desk Reference*, New Canaan, : Keats Publishing, 1995, pp.172-173.

[xvi] E.R. DeKloet, et al.; Bruce McEwen, et al.; M.A. Holzwarth, et al., *The Hypothalamic-Pituitary-Adrenal Axis Revisited*, New York: Annal of The New York Academy of Sciences, 1987, pp.351-361, 394-401, 449-464.

[xvii] *Cecil Textbook of Medicine*, pp.1249-1253; *Harrison's Principles of Internal Medicine*, pp.1970-1971.

[xviii] Kendell, *The Lancet*, 19 January 1991, p.160; B.M. Greenwood, *The Lancet*, Vol.342, No.8878, 23 October 1993, pp.1039-1040.

[xix] John McLaren Howard, et al., *The Lancet*, Vol.340, No.8816, 15 August 1992, p.426; I.M. Cox, et al., *The Lancet*, Vol.337, No.8744, 30 March 1991, pp.757-760; Charles Shepherd, *The Lancet*, Vol.337, No.8749, 4 May 1991, p.1095.

[xx] Hirohiko Kuratsure, et al., *Clinical Infectious Diseases*, Vol.18, 1994, Supplement 1, pp.S62-S67; P.H. Levine, "Chronic Fatigue Syndrome: Current Concepts," Lecture delivered at the Symposium on Advances in Clinical Nutrition, Meeting of The American College of Nutrition, San Francisco, California, 11-13 October 1996.

[xxi] *Nutritional Biochemistry & Metabolism*, ed., Maria C. Linder, Ph.D., New York: Elsevier, 1991, p.180; Richard Murray, D.C., *Biomedical Critique*, Vol.5, No.5, August 1984, pp.1-4.

[xxii] Paul A. Goldberg, M.P.H., D.C., *Today's Chiropractic*, Vol.25, No.2, March/April 1996, pp.32-38; E.A. Newsholme, E. Blomstrand, *Experientia*, Vol.52, 1996, pp.413-415.

[xxiii] Harold Burrowes, *Biological Action of the Sex Hormones*, London: Cambridge University Press, 1949, p.90; Royal Lee, D.D.S., "Recent Advances in Clinical Nutrition," Lecture to the 52nd Annual Convention of the Michigan Association of Osteopathic Physicians and Surgeons, Grand Rapids, Michigan, 5 October 1950.