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## Dehydroepiandrosterone, Melatonin, and Chronic Fatigue Syndrome

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Chronic fatigue syndrome (CFS) has symptoms similar to neurally mediated low blood pressure, and treatment for this kind of low blood pressure helps some with CFS (*J. Am. Med Assoc.* 1995; 274: 961). If you are in the treatable group, good for you. For those with CFS who read this, I would like to propose an explanation of CFS that may lead to an alternative treatment.

My explanation depends on my work with cycling of the hormones melatonin and dehydroepiandrosterone (DHEA). My idea is that melatonin and DHEA levels determine blood pressure. If DHEA is too high, blood pressure is too high; the opposite occurs with melatonin. For example, melatonin is very high in children, but melatonin begins a steady decline following childhood (puberty) to very low levels in old age. Blood pressure is lowest in children and increases thereafter. Problems with neurally mediated hypotension (low blood pressure) are more common in the young than old. Melatonin is highest at night, and blood pressure decreases slightly at night. I suggest melatonin reduces blood pressure by reducing DHEA production, Exercise increases DHEA and blood pressure. Excitement, according to my work, also increases DHEA, excitement increases blood pressure. In this case, I think DHEA stimulates the smooth muscle of blood vessels and makes them constrict, this increases blood pressure. In people with normal DHEA, exercise also uses DHEA. The increased blood pressure of exercise, therefore, is temporary. People with CFS exhibit lingering tiredness **after** exercise. They burn their small supply of DHEA during exercise, but do not replenish-it in sufficient quantities to produce the sense of well-being, I attribute to DHEA

I suggest chronic fatigue syndrome results from a disruption of this cycle that increases melatonin, while DHEA declines. My work suggests this cycle is increased by viral infection. The majority of patients in the JAMA study reported an abrupt onset of symptoms in association with an infectious illness that resembled influenza or mononucleosis. I think the first response of this cycle to viral infection is an increase in melatonin, which then increases DHEA. (These cycle to produce the chills and fevers of viral infections.) It is known that DHEA increases dramatically in response to HIV infection, then declines thereafter. I suggest the same mechanism is at work in AIDS and chronic fatigue syndrome. That is, a virus attacks these people who cannot maintain their DHEA response. One group dies, the others live miserable lives. DHEA is necessary for normal mental and physical health.

My work suggests melatonin is our natural narcotic. In the JAMA tilt tests, people with CFS, experienced symptoms quite similar to those of people who have taken narcotics, i.e., nausea and vomiting, sweating, light-headedness, etc. These are also symptoms of syncope caused by neurally mediated hypotension. They are also symptoms of viral infection. I suggest they **all** are produced by too much melatonin. Narcotics mimic the effects of melatonin. (Melatonin is our way of maintaining day/night rhythms, and narcotics (derived from plants) are used by plants to maintain this rhythm.) People with CFS are not producing the DHEA response of the cycle, the virus has probably harmed their adrenal glands, which make DHEA, A symptom of CFS is unrefreshing sleep. In normal people, melatonin (sleep) leads to increased DHEA in the morning.

I suggest chronic fatigue syndrome results from DHEA insufficiency. DHEA taken orally in the morning may alleviate these symptoms. DHEA is currently available in a specific form that produces physiological levels of DHEA in the blood, and is available upon prescription. Surely this should be better than the complicated drug regimen currently being used for CFS.

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DHEA (dehydroepiandrosterone): DHEA is a steroid hormone secreted by the adrenal glands. Structurally similar to other steroid hormones (including estrogen and progesterone), DHEA is converted into whatever hormone the body needs at that time. In addition, DHEA has some specific functions of its own.

For example, DHEA blocks an enzyme (G6PD) which promoted fat production. In laboratory studies, DHEA appears to be the first known substance that, without changing eating **habits**, causes the loss of fat (as opposed to mere **weight** loss due to the breakdown of primary lean muscle tissue or fluid loss). Calories consumed are simply converted to heat rather than stored as fat. At the same time, DHEA helps the body to retain nitrogen, important for lean muscle tissue.

In humans, DHEA first appears in the bloodstream by the age of 7 and peaks in production at about 25 years. Beyond that, there is a gradual decrease in DHEA production until the point in one's 80s production is as little as 5% of that at age 25. As the DHEA levels drop, so does the ratio of fast to slow motor units drop. This gradual conversion to slow muscle is due to both inactivity and loss of DHEA. This creates a vicious cycle of decreased muscle power leading to inactivity, which in turn leads to a decrease in muscle power. By raising DHEA levels in people over 40 to those of a 25 year old, one can increase the desire and ability to exercise, which in turn burns more calories.

Even keeping exercise levels constant throughout one's life, the decrease in DHEA with age decreases the ratio of lean muscle to body fat. DHEA affects body fat by influencing metabolism. Ingesting DHEA improved thyroid function, which in turn increases mitochondrial respiration. Since the mitochondria are centers of energy (ATP) production in all cells, DHEA increases the consumption of oxygen and calories throughout the entire body.

There are several animal studies which point to DHEA as being effective in treating obesity. In a type of mice that have a tendency to become obese, administering DHEA at a dose of 500mg per kg of body weight, three times per week, prevented the mice from becoming overweight. DHEA did not cause any toxin effects, and since it did not suppress appetite, it suggests that its effect was to increase metabolic rats. (1)

A second study involved the administration of DHEA (0.6% of the diet) in both lean and obese rats. The result was a decrease in body weight and body fat in both lean and obese rats. In the lean rats, the decrease in body fat was primarily due to a decrease in the number of fat cells. In the obese rats, the decrease in body fat was due to decreases in both the number and size of fat cells. (2)

DHEA has a milder effect than weight loss formulations based on ephedrine and other stimulants. DHEA affects the endocrine system in a very natural way, while stimulants cause the cardiovascular system to speed up in an unnatural way.

In making our DHEA, we start with selected strains of wild yam that are rich in diosgenin and other important plant sterols and then use a unique process for extracting the diosgenin and other sterols. Our wild yam extract has more of the active DHEA precursors than do other types of wild yam. Adrenal and **liver** enzymes convert these precursors into DHEA.

Diosgenin cannot be utilized effectively in the human body. Normally, only about 1 percent of diosgenin is converted into DHEA in the intestines. In our process, we use fermentation, which provides a much more complete conversion of the diosgenin into DHEA before it enters the intestines.