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MULTIPLE SCLEROSIS

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For many years we have observed patterns of minerals found in the hair to be associated with different diseases. Since Multiple Sclerosis (MS) is not such a common condition, relatively speaking, in the past I would pay particularly close attention to the laboratory results of patients who were diagnosed as having MS. These patients, suffering from MS, have shown a strikingly similar and well-recognized pattern. The hair tissue mineral analyses (HTMA) almost always revealed a Fast Metabolic pattern (sympathetic dominance). The next most common finding was a very low tissue copper level, usually found to be below 1 mg% (milligram percent), {T.E.I. ideal 2.5 mg%}. This finding would be expected since copper deficiency has long been known to be associated with neurological defects.

The mineral copper is involved in the myelination of nerve tissues. The myelin sheath is largely made up of a lipid/protein substance that surrounds the nerve in a spiral wrapped fashion, insulating the nerve much like the insulation used to surround electrical conducting wires. In the absence of this insulation or myelin, sclerosing develops around the nerve, disrupting normal nerve conduction.

There has been independent verification of our findings by other researchers. Ryan, et al, reported their findings in *Clinical Chemistry,* Volume 24, number 11, in 1978. Their paper, which was titled, "Trace Elements in Scalp-Hair of Persons with Multiple Sclerosis and of Normal Individuals", told of a study consisting of forty MS patients and forty-two controls. Both groups included males and females. In this study, they found a significant difference in the hair copper levels of the two groups, with a 99% confidence range. Their findings revealed the following results: the median copper values in the control group of men were, 2.2 mg%; the female control group had a median copper of 5.9 mg%. Of the MS group, median hair copper levels in males was 0.7 mg%; females, 1.0 mg%. The average between the control and study group were 2.9 and 0.9 respectively. The mean zinc\copper ratio in the MS group was markedly elevated at 18:1, {T.E.I. ideal Zn\Cu ratio = 8}, additionally indicating decreased copper bioavailability, due to zinc's well known antagonism of copper function.

Smith, et al, reported their findings of plasma ceruloplasmin, selenium, zinc, and copper, as well as anti-oxidant status in a group of MS patients. The paper entitled "Trace Element Status in Multiple Sclerosis", was reported in *The American Journal Of Clinical Nutrition,* 50; 1989. The study revealed a significant increase in the red blood cell Zn\Cu ratio in the study group, as compared to the control group without MS.

One special case I recall began in 1989, when our laboratory received a sample from a client whose patient had been diagnosed as having MS. The patient had been diagnosed and treated at the Massachusetts General Hospital beginning in 1979. At that time the patient was in her early twenties. She was experiencing the typical signs and symptoms of MS, and CAT scans showed involvement in the left cortex and spinal cord. Treatment involved physiotherapy and steroids. Over the next ten years, the patient experienced exacerbations and remissions. The 1982 medical report stated that although she experienced multiple regressions, there was a steady course of increased exacerbations. Steroid therapy had to be increased to help control the debilitating symptoms. At the time the patients' hair sample was submitted to our laboratory, the patient was experiencing ataxia, severe headaches, extensive numbness in the extremities and face, and visual difficulties. These symptoms were so severe, they contributed to almost complete incapacitation.

Remarkably, the HTMA result did not show a low tissue copper level as we would suspect. The pattern also showed a Slow Metabolic type, or Para-sympathetic Dominance instead of the Sympathetic pattern usually seen in patients diagnosed with MS. In other words, the laboratory results showed a totally opposite pattern as seen in other MS patients. This made me question whether or not the patient actually had MS, even though her symptoms paralleled those of the disease.

The patient was placed on a metabolic rebalancing regime based upon the HTMA laboratory results, and not on her symptoms or diagnosis. Remarkably, the patient responded dramatically within a short period of time. In past experiences with MS, the response was slow, usually taking several months to more than a year before a significant response could be seen. However, within six weeks, this patient noticed an improvement in all of her symptoms; and within a year, she reported that the severe headaches had completely disappeared, the numbness, double vision, and extreme fatigue had subsided. The patient’s activity level greatly increased and she also reported a necessary weight gain of over sixty pounds. I should mention that the patient did experience ups and downs during this period, but the ups occurred more often than the downs. I would also like to emphasize the courage of this lady. She was diligent in following her regime of, diet, exercise and supplements. She received constant encouragement from those close to her, but ultimately the decision was hers. She has even gone before a number of groups to relate her experience to others, something that she would not have been able to do previously. The patient has been free of any major exacerbations since 1989, and has shown a steady improvement.

As a result of this patients improvements, other samples of patients diagnosed with MS were received from our clients. Again, we saw similar HTMA patterns and response. These cases led me to investigate exactly why these HTMA patterns were so different from our previous studies, and the studies of other researchers on MS patients. Knowing that copper is usually elevated following viral conditions, we began checking the history of these patients. In almost every case, the patient recalled having a viral condition prior to developing MS symptoms. One patient recalled having a flu shot prior to developing the symptoms. I have speculated that although the symptoms of these patients are closely related to MS, we may be seeing a "false" MS condition, and that actually the symptoms may be due to a virus that has crossed the blood-brain barrier, into the nervous system. The nervous system may be affected to the point that neurological symptoms develop almost identical to those of "true" MS. Thus, I coined the term "True and False " MS patterns. The true MS pattern being associated with Sympathetic dominance, and low tissue copper level, and the false MS pattern being associated with Para-Sympathetic dominance and elevated tissue copper.

In summary, the true MS pattern is seen in Fast Metabolic types who show a very low HTMA copper level. This would be expected since increased adrenal activity is known to enhance copper loss from the body. The Fast Type I individual is considered to have increased adrenal activity. Lewis, et al, published findings of animal studies which confirmed the effects of adrenal hormones on copper homeostasis in the report, "Severity of Copper Deficiency In Rats Fed Fructose Is Not Solely Dependent On Hepatic Copper Concentration: Effects Of Adrenalectomy"***, Journal of Nutritional Biochemistry,*** Volume 2, 1991. They found that increased adrenal hormones reduced hepatic copper storage. Animals that had their adrenal glands removed had a higher concentration of liver copper; while elimination of excess copper from the liver was reduced in adrenalectomized animals. This study helps to explain why we often see increased copper retention in Slow Metabolic Type 1's who have a corresponding adrenal insufficiency.

The myelin defects found in True MS is speculated to be caused in part, by lipid peroxide formation. Free radicals are destructive to the lipid membranes of all cells, but particularly to nerve tissue, which are composed mostly of phospholipids. Copper deficiency can lead to free radical formation in a number of ways. First, due to an imbalance in the iron-to-copper ratio; secondly, due to the decrease in superoxide scavenging; and third, due to excessive adrenal cortical production of glucocorticoids.

Excess tissue copper retention can also result in increased free radical production. For example, the zinc and manganese activated superoxide activity may be reduced in the presence of excessive copper. Since copper is antagonistic to, and increases the oxidation of vitamin C, extracellular radical scavenging is also reduced. Recently, Maeda and Akaike reported their findings of oxygen free radicals being generated during influenza viral infections. "Oxygen Free Radicals As Pathogenic Molecules In Viral Disease", *Proceedings of The Society For Experimental Biology And Medicine****,*** Volume 198, Number 2, 1991. This may explain the MS symptoms found in the False MS patterns, as well as the close association of the condition manifesting after a viral infection. In past issues of the *Newsletter****,*** we discussed the other minerals associated with viral manifestations. A deficiency, or increased requirement of these nutrients seems to fit the pattern of the nutritional requirements of patients with the "false" MS pattern. As previously mentioned, excessive tissue copper has been associated with viruses. Calcium is also known to produce an increase in the proliferation of dormant viruses, whereas, phosphorus decreases viral proliferation. Zinc deficiency is known to increase the susceptibility to viruses as well. Maeda also states that the immune reaction may account for the increase in free radical production associated with viral infections. We have long associated an increase in immune response with the Slow Metabolic Type 1. Their immune system becomes overactive, resulting in adrenal suppression. Supporting the immune response, specifically thymus function, would be contraindicated in the Slow Type 1 patterns. These are just a few more examples of the importance of treating the patient, not the condition. Symptoms of MS and other conditions can develop in different individuals. And although their symptoms are almost identical, the cause may be completely different in each person. HTMA can be extremely beneficial in recognizing biochemical individuality, thus providing a more specific and appropriate therapeutic approach.