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ATTENTION DEFICIT DISORDER – A NEW HYPOTHESIS

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Hyperactivity in children has been an enigma for several generations. The first account of hyperactivity in the medical literature can be traced back to 1902. In recent years with increased study, hyperactivity has been placed in a sub-category of Attention Deficit Disorders (ADD). Therefore, this discussion will concentrate on ADD and a relatively new theory for explaining and treating ADD. This new hypothesis is also suggested in order to account for ever increasing numbers of ADD children from one generation to the next.

Currently, the psychological/medical approach has been the major technique used to diagnose and treat these behavioral disorders. However, when there have been positive results, they have been empirical with little understanding of the underlying mechanisms contributing to the syndrome, or its successful treatment.

The interventions, which are used to treat ADD from a medical standpoint, are psychologically based. They tend to rely on psychological and behavioral data to assess the "need" for medication. The effects of medication are then assessed by the same type of psychological and behavioral data, which were used to determine the "need" for medication to begin with. For each child, this process is essentially experimental in nature. There are no biochemical markers used to make a diagnosis of ADD or to select a particular treatment. It is essentially a trial and error approach.

Two theories, which have been applied to medical treatment of ADD, are based on either a hypo- or hyper- arousal model. These models in clinical application are used for explanatory purposes rather than an *a priori determination* of what medication is likely to be effective for a particular child at a specific point in time. In other words, again there are no biochemical markers used to intelligently select a medication to use. As a result, there are interactions between medications and children with no adequate explanation of why medications seem to work with some children and do not work for others.

Another explanation applied to ADD has to do with neuro-transmitter function. In this theory, ADD is considered to be a problem of under-or over-production of neurotransmitters. With the neurotransmitter model, however, there is no satisfactory explanation of why a neurotransmitter is in excess in some children and why there is a deficiency in others. In addition, the manner in which the neurotransmitter model is applied, is limited by not being considered as part of the broader general neuro-endocrine metabolic system which plays a critically important role in generating and regulating neurotransmitters (metabolic pathway).

One of the major limitations of both the arousal and the neurotransmitter models is that they do not account for the apparent increase in ADD cases from one generation to the next.

When there appears to be no plausible clinical or scientific explanation to account for increasing numbers of ADD children, the alternative is to "explain away" the fact of increased numbers of ADD children as a artifact of better identification and diagnostic processes. If, in fact, there are increasing numbers of ADD children, then assuming that such a phenomenon is an artifact of better diagnostic processes has very serious implications because the magnitude of the actual problem of increasing incidence rates will be minimized and overlooked. Also, important steps for intervening to prevent increasing numbers of ADD children would not even be recognized or considered.

Nevertheless, dramatic changes have been observed in some children with the use of the Feingold diet, some medications, and single nutrients, which would strongly suggest that a biochemical factor does play a major role in many cases of hyperactivity. In addition, there is increasing evidence that heavy toxic metals such as lead, mercury, cadmium, and copper may contribute to behavioral disturbances in some children. An increasing number of studies have related data obtained from hair mineral analysis to ADD.

As stated earlier, the purpose of this discussion is to propose a new model for explaining and treating ADD, as well as accounting for increasing numbers of ADD children. The model proposed here focuses on the role of heavy metals, which accumulate in the ADD child's nervous system, particularly in the brain. This model also focuses on the role of nutrient minerals in the tissues and the psychophysiological functions of these minerals. Thus, this new model has two major components relating biochemistry to ADD - (1) excessive toxic metal accumulations, particularly in the brain, and (2) nutrient mineral imbalances which adversely affect the regulation of critical neuro-endocrine functions. Under this model, it is the combination and interaction of these two metabolic components, which is considered to be most strongly related to ADD with and without hyperactivity. However, this model does not discount the possibility that genetic and subclinical neurological conditions may also be contributing factors to ADD. But, most cases of ADD would have significant toxic metal tissue burdens and nutrient mineral imbalances as major contributing factors to the condition.

This model recognizes the inherent dynamic qualities of the body's metabolic processes, especially as they relate to the stress response. The constant fluctuations in these metabolic processes, especially as they relate to the stress response. The constant fluctuations in these metabolic processes help to account for the erratic patterns of behavior and attention span, which are so characteristic of ADD children. Dynamic changes can occur at two different levels of system functions. One level is within the individual child's own neuro-psychological-endocrine system. The other level is within the social-psychological-educations system.

The changes, which occur within the neuro-psychological-endocrine system, may be related to stages of stress response. In particular, this hypothesis suggests that ADD children will tend to show hair HTMA patterns reflecting strong trends towards adrenal insufficiency. These HTMA patterns would be one of two primary types (1) Fast Metabolism with an "inversion: of the ratio of sodium-to-potassium (Na/K), i.e. a HTMA ratio significantly below the ideal ratio of 2.4, or (2) Slow Metabolism with a very low HTMA sodium level. In terms of Dr. Hans Selye's three stages of stress (alarm, resistance, and exhaustion), Fast Metabolism with a HTMA sodium/potassium ratio significantly below 2.4 is an earlier stage of stress than is Slow Metabolism with a very low HTMA sodium level. Fast Metabolism with an Na/K inversion is indicative of a transition from the resistance stage to the exhaustion stage, whereas, Slow Metabolism with a low tissue level of sodium is reflective of being chronically in the exhaustion stage of stress.

A stimulant medication such as ritalin is most likely to be effective with a Fast

Metabolizer who has a HTMA sodium/potassium inversion, whereas, an anti-depressant drug such as norpomin is most likely to be effective with a Slow Metabolizer who has a very low HTMA sodium level. These relationships are suggested because the artificial stimulation effect of ritalin will tend to deplete the adrenal glands over an extended period of time. This will result in a more advanced stage of adrenal insufficiency (Slow Metabolism with a very low HTMA sodium level) resulting in a depressed neuro-psychological-endocrine system.

At the social-psychological-educational level, the proposed cumulative toxic metal hypothesis suggests that, from one generation to another, toxic metals from the environment accumulate in greater quantity in the body's cells and tissues. During the course of a pregnancy for example, when a severe stress occurs, a woman's body will tend to release toxic metals from her own cells and tissues into the blood stream for elimination from the body. However, some of these released toxic metals can cross the placenta into the fetus. Depending on how toxic and how stressed the mother is, the fetus may be born with a very heavy load of toxic metals affecting neuro-muscular, and neuro-endocrine functions.

It is strongly suggested by this hypothesis that, over several generations, the population of children in each succeeding generation will have increasing numbers of ADD children as the toxic metals accumulate and are transmitted in utero. This trans-generational change is accelerated by several other environmental events, which have occurred since the end of World War II. These particular events have had a particularly strong effect on copper accumulations to toxic levels in many people. One major environmental development involved the introduction of copper water pipes into homes, offices, apartments and schools. The expanded use of water softeners with these copper plumbing systems increased the leeching of copper into the drinking and cooking water consumed by people.

Another adverse environmental factor contributing to increased copper toxicity from one generation to another was the introduction of the birth control pill with its estrogen content added to a woman's own natural estrogen production. Estrogen tends to increase copper accumulation in the body's cells and tissues. The introduction of the copper-7 IUD also tended to produce significant copper elevations in the tissues of some women.

Copper is an essential nutrient mineral, but in excess accumulation, it can become very toxic to the neuro-psychological-endocrine system. Substantial elevations of tissue copper are commonly found in many ADD children along with significant deviations in ratios between essential nutrient minerals, such as, Ca/Mg, Na/K, and Zn/Cu. The addition of tissue mineral analysis (HTMA) to the diagnostic assessment and treatment of children and adolescents with ADD will help to provide a more comprehensive data base for understanding the metabolic problems of these children. The role of nutritional factors and toxic metal accumulation also will become much clearer. HTMA may provide the biological markers, which, so far, have been absent with medication treatment.

This writer's experience with these concepts and with hair mineral analysis profiles of several hundred children and adults seen in an outpatient child development clinic suggests that there may be distinct biochemical/nutritional characteristics of the profiles of children diagnosed as ADD and hyperactive.

When the underlying biochemical/nutritional profile is known and can be nutritionally treated, then there tends to be a better biological foundation for psychological and educational services rendered to the child. Changes in a child's hair mineral profile can be monitored over time along with changes in behavior observations and ratings. This should result in much more effective help for the ADD child and family.

Further research should investigate the relationship between the factors discussed here and the role of toxic and heavy metals, which contribute to ADD.