Trace Elements and Neuropsychological Problems as Reflected in Tissue Mineral Analysis (TMA) Patterns

David L. Watts, Ph.D., F.A.C.E.P.[1]

Introduction

Excerpts from the conference of "Research Strategies for Assessing the Behavioral Effects of Food and Nutrients" appeared in *Science* in 1982. The paper stated, "The effects are subtle, but a number of scientists are finding that people do react to what they eat." In 1983 Wurtman stated in *Lancet*, "Most drugs that modify normal or abnormal behaviors do so by changing the amounts of particular neurotransmitters present within the brain synapses or by influencing the interactions between transmitter molecules and their postsynaptic receptors. If a food constituent can be shown to cause similar changes in the release or the actions of one of these neurotransmitters, there is every reason to expect that the nutrient will also be able to influence behavior."[1] To further quote Dr. Wurtman, "There is no longer any real controversy over whether nutrients can affect behavior."

Dr. Wurtman and colleagues began studying the effects of food on brain biochemistry at M.I.T. over ten years ago. Their work appears to be focused on the investigations of amino acids and their role as precursors of neurotransmitters. However, vitamins and especially minerals are also known to affect brain function. This paper will focus on minerals and toxic metals.

It should be mentioned that the work of earlier clinical investigators has been largely ignored in this field. Approximately twenty years prior to Dr. Wurtman's reports, doctors Hoffer, Osmond and coworkers were pioneering the biochemical basis of mental illness. Their work has led to an effective nutritional treatment of serious mental disorders — a treatment that is continually being expanded in its application.

1. Trace Elements, Inc., P.O. Box 514, Addison, Texas 75001.

Toxic Metals and Brain Function

Heavy metals such as lead, cadmium and mercury are found ubiquitously in our environment: therefore, exposure is not uncommon. Excessive body burdens of heavy metals are known to contribute to adverse emotional changes and neurological impairment.

Lead

The adverse effects of lead have been known for centuries. The Romans used lead so extensively it has often been referred to as the "Roman metal".[2]

Symptoms of lead toxicity in adults include fatigue, anorexia, irritability, abdominal pain, muscle weakness and peripheral neuropathy.[3] Long term deficits in memory and psychomotor function, depression and hostility have been observed in individuals who are occupationally exposed to lead.[4] [5]

The signs of lead toxicity in children include encephalopathy, ataxia, speech abnormalities, seizures and coma. Chronic low levels of lead exposure have been associated with learning disabilities, spatial integration and motor coordination problems, hyperactivity, distractibility and aggressiveness.[6] [7] Even moderate exposure to lead affects I.Q. score.[8] [9]

Lead produces pathological changes in the anterior horn cells and peripheral nerves. Lesions develop throughout the brain but are most common in the cerebrum and cerebellum as a result of hemorrhages and thrombosis.[10] [n]

Mercury

Mercury is also a widespread heavy

metal whose toxic effects have been known for some time. Perhaps the most well known effect of mercury poisoning is tremors. These tremors have been referred to as "hatters shakes" due to the mercury exposure of workers in the hat industry. Methylmercury is a strong central nervous system (CNS) toxin that apparently produces cerebellar lesions. Other signs related to mercury toxicity include ataxia, weakness, irritability, headaches, rashes, slurred speech, pain and paranesthesia in the extremities. Manifestations of mercury toxicity in infants include cerebral palsy syndromes with psychomotor disturbances and mental retardation.[12]

Cadmium

Cadmium has only recently been recognized as a neurotoxin and is known to be damaging to the neonatal nervous system. Animal studies have revealed that cadmium exposure in the first thirty days of life produced increased locomotor activity, as well as increased MAO synthesis and turnover.[13] Behavioral abnormalities became more apparent as the animals matured. Other animal studies have shown that cadmium exposure produced a decrease in cortical acetylcholine and brainstem serotonin.[14]

Aluminum

Increased aluminum concentrations in the brain have been found to be associated with neurofibrillary tangles or degeneration in humans. Aluminum has been associated with Alzheimer's disease and dialysis dementia. Alzheimer's disease is characterized by memory and learning difficulties and personality changes. With further debilitation, cognitive abilities become markedly decreased. Animal studies have revealed some neurochemical effects of aluminum primarily affecting cholinergic function[15] and inhibition of Na-K-ATPase.

It is interesting that parathyroid hormone has been found to increase aluminum absorption and brain concentrations in animals.[16] [17] When parathyroid hormone is withdrawn, brain aluminum concentrations decrease regardless of intake.

Nutritional Minerals

Many nutritional trace elements are known to cause abnormal mental function when either a deficiency or excess exists. When a nutritional mineral accumulates excessively in the body, it can become toxic.

Copper

A deficiency of copper is known to affect the CNS. Animal studies have revealed the association between copper deficiency and myelination defects. TMA studies have shown low tissue copper levels in patients suffering from multiple sclerosis[18] and Parkinson's disease.[19] Low dopamine levels have been found in animals and patients with Parkinson's.[20] Menke's disease, an inherited inborn error of copper metabolism in infants, is characterized by abnormal CNS development. Progression of the disease leads to psychomotor disturbances, mental retardation, seizures and death.[21]

The late Dr. Carl Pfeiffer, et al postulated the relationship between copper toxicity and some types of schizophrenia, i.e., histapenic. Excess copper has also been related to postpartum psychosis, autism, depression and premenstrual syndrome.[22]

Wilson's disease is a condition associated with increased copper accumulation and toxicity. Neurological manifestations may include tremors, disturbances in coordination, dysphagia or severe psychiatric disorders.[23]

Iron

Research has concluded that iron deficiency can lead to deficits in attention and cognitive functions[24] [25] and has indicated a correlation between hemoglobin levels and intellectual performance in teenagers[26] and confirmed in adults and children.[27] Iron deficiency is known to lead to impaired neuronal development in infants. MAO, an iron dependent enzyme, which is reduced in the presence of iron deficiency, can lead to an increase in other neurotransmitters such as serotonin, dopamine and norepinephrine.

Excess iron accumulation also affects the CNS. Increased iron deposition in regions of the brain has been implicated in Parkinson's disease.[28]

Zinc

Fetal zinc deficiency in animals is known to

result in CNS defects. Psychological disturbances are frequently noted in individuals suffering from acrodermatitis en-teropathica.[29] Zinc-induced deficiencies in humans have been associated with neurological symptoms such as depression, poor concentration, nervousness and moodiness.[30] The hippocampus is particularly rich in zinc. Zinc deficiency is associated with learning and memory defects similar to behaviour syndromes resulting from destruction of the hippocampus.[31]

Acute oral zinc toxicity has been reported to produce drowsiness and somnolence.

Manganese

A deficiency of manganese in animals produces CNS disorders. The most recognized neurobehavioural effects of manganese, however, are due to its toxicity. Manganese toxicity in humans is associated with Parkinsonian-like symptoms, which include hypokinesia, rigidity, tremor and masklike facies.[32] The symptoms of manganese toxicity have been divided into three stages. The first includes apathy, asthenia, anorexia, euphoria, insomnia, muscle pains and compulsive behaviour. During the next stage, speech disturbances, incoherence, ataxia and altered balance may develop. Muscular rigidity, staggering and tremors develop in the later stages of manganese toxicity.[33]

Calcium

Depression is universally associated with hypercalcemia due to hyperparathyroidism[34] and/or hypervitaminosis D. Many patients suffering from hyperparathyroidism receive diagnosis of psychoneurosis, schizophrenia or schizoid personality.[35] Irritability, mood swings and paranoid psychosis are associated with hypocalcemia.[36] Emotional symptoms resulting from disturbances of calcium homeostasis are quickly alleviated with normalization of calcium metabolism.

Reports of animal studies show that by manipulating the calcium content of the cerebral spinal fluid behavioral changes could be induced. Reduction of calcium produced agitation, irritability and insomnia while increased calcium levels resulted in sedation.[37]

Flach demonstrated changes in calcium metabolism associated with improvements in psychiatric patients receiving various types of therapies.[38]

Magnesium

The effects of magnesium are similar to those of calcium. Excess magnesium produces sedation, and deficiency produces irritability. Psychological changes associated with abnormal magnesium metabolism include personality changes, hyper-irritability, psychosis, depression and schizophrenia.[39] [40]

Sodium and Potassium

Disturbances in sodium and potassium metabolism are commonly seen in psychiatric patients.[41] [42] [43] Manifestations of sodium and potassium deficiency include severe depression, apathy and schizophrenic syndromes. Clinical studies have indicated that post operative psychosis is produced by potassium depletion due to the administration of low potassium dextrose or sodium chloride solutions. Psychiatric symptoms are quickly relieved with nor-malization of these electrolytes.[44] [45]

Personality Characteristics Reflected in Tissue Mineral Analysis (TMA) Patterns

The endocrine and nervous systems' response to stress, either physical or emotional, is well known and has been described in the "fight or flight" mechanism. As stated by Dunbar, "It has been said that the endocrine glands translate the tempo of the nervous system into the tempo of metabolism and vice-versa. But sometimes the glands are the pacemakers for emotion ..."[46] Emotions can trigger endocrine responses, which in turn affect nutritional status. Studies have shown that the emotional status of an individual can affect the absorption and excretion of minerals.[47] Henkin reported that "Trace metals have been shown to influence hormones at several levels of actions ... Similarly, hormones have been shown to influence trace metal metabolism ... including excretion and transport."[48]

Since it is well known that psychic factors can trigger an increase in mineral excretion and/or absorption, it is reasonable to assume that chronic emotional stress may be reflected in TMA patterns.

The relationship between mental disorders and mineral patterns through tissue mineral analysis of hair is in its early stages of research. Thus far TMA research has revealed significant findings on the relationship of toxic heavy metals such as lead, mercury and cadmium as well as other nutrient elements.[51 52 53 54] Blood and hair levels of lead have been correlated with continuing exposure in individuals who are occupationally exposed.[55] However, since heavy metals normally depart from the blood stream for deposition into tissue, blood levels do not accurately reflect past exposures. This has been illustrated in studies of children with former lead exposure. Alterations in CNS functions are found to be present even when the serum level of lead is well below the upper limits of 30 mcg. per dl. Therefore, TMA is probably the most valuable screening tool for assessing previous heavy metal burdens.[56] Toxic metal exposure occurs in utero, and TMA has been found useful in assessing maternal exposure in neonate hair.[57 58]

Observation of thousands of TMA patterns over many years has led to the recognition of associated personality traits. In order to simplify this discussion, the categorization of individuals according to TMA patterns will be reviewed. Eight distinctive TMA patterns are identified as sympathetic (S) types 1 through 4 and para-sympathetic (PS) types 1 through 4.[59] Categorization of nutrients including vitamins, minerals, foods and drugs into sympathetic and parasympathetic categories has been reported elsewhere.[60] The S type 1 is considered to have an elevated metabolic rate and a relative increase in the tissue retention of sympathetic minerals, which include phosphorus, sodium, potassium and iron. The PS type 1 has a reduced or sedated metabolic rate with a relative increased tissue retention of parasympathetic elements calcium, magnesium and copper. The subtypes have variations of sympathetic and parasympathetic mineral retention.

Aggressive Behaviour

William Walsh was probably the first researcher to report the correlation of TMA patterns to aggressive or violent behaviour.[61] He reported significant TMA mineral variations in subjects who had committed violent crimes compared to those who were less violent. We have found that elevated tissue sodium and potassium levels are related to aggressive personality traits, particularly in the S type 1 individuals. TMA patterns of S types show low calcium/phosphorus (<2.63) and calcium/potassium (<4.0), and elevated sodium/magnesium (>4.0) ratios. The S type 1 is analogous to the type A personality: highly competitive, aggressive, constantly in a hurry and easily angered or hostile.

The elevated tissue sodium and potassium levels in S types can be clinically associated with increased adrenal and thyroid activity.[62] Aggressive behavior is associated with hyperadrenal conditions such as Cushing's syndrome, as well as induced hyperadrenal states due to the use of corticosteroid therapy.[63 64] Increased sympathetic neuro-endocrine activity contributes to the retention of sodium and potassium,[65 66 67] which helps to explain the increased incidence of heart disease in the S types.

Elevated tissue iron is also associated with hostility. This is perhaps due to the accumulation of iron in the hippocampus region of the brain. Excess iron accumulation can affect neurological function by increasing lipid peroxidation or by displacing other minerals normally present in the region of or within the hippocampus. Hyperactivity is often seen in children with elevated tissue iron levels.[68]

Anxiety

Aggressiveness, hyperactivity and anxiety are all associated with the S type 1 pattern, which is associated with low tissue calcium and magnesium relative to sodium and potassium. Decreased tissue calcium is associated with neuromuscular hyperexcitability. Many symptoms of anxiety neurosis are identical to those seen in patients with hypocalcemia.[69] Patients suffering from anxiety neurosis show excessive serum lactate levels. Lactate infusion, which readily brings on anxiety symptoms in patients, can be prevented by calcium infusion. Apparently the mechanism behind anxiety

neurosis is overactivity of the central nervous system and adrenal function,[70] both of which increase calcium and magnesium losses from the body[71] and increase tissue retention of sodium, potassium and phosphorus.

Addiction

TMA patterns suggest that it is important to make a distinction between physiological and/or psychological addictions.

A physiological addiction is associated with the S type 1, which is analogous to Watson's "fast Oxidizer".[72] Their metabolic machinery is in high gear, so to speak, which requires constant fueling. In review, their TMA pattern is dominant in the stimulatory minerals, phosphorus, sodium, potassium, etc., which indicates a high rate of metabolism and sympathetic neuroendocrine dominance. In order to keep their high level of energy, they may develop addictive personality reactions to stress such as being late for appointments, waiting until the last minute to meet deadlines and starting several projects at once. This stress continuously stimulates the neuroendocrine system, which in turn helps to maintain their high energy levels. The result of this type of behaviour if left unchecked is ultimately the "burnout syndrome". We have found that the S type 1 is often found to have a strong allergy or sensitivity to grains, which leads to an addiction, frequently to alcohol. The grains lead to increased losses of calcium and magnesium with increased retention of phosphorus, sodium, potassium and iron.

Psychological addiction is seen in PS types, which are analogous to Watson's "slow oxidizer". Due to the sedative mineral and parasympathetic neuroendocrine dominance, decreased energy production is evident. Along with this biochemical pattern, fatigue, depression and low self-esteem can develop. In an effort to feel better generally and psychologically, they may yield to food cravings such as binging on refined sugars and carbohydrates in order to increase their energy levels. Eventually the use of recreational chemicals or alcohol may develop due to their immediate energy-elevating and mood-altering effects.

Mania and Depression

Generally speaking the S type 1 pattern has been observed in patients with manic-depressive illness. It has long been known that manic and excited paranoid patients have increased rates of neurotransmission while depressed patients have a decreased rate of neurotransmission.[73] Increased membrane permeability at the synaptic level is associated with increased sodium retention. Studies have reported that adrenal hormones fluctuate concomitantly in patients experiencing manic-depressive cycles[74] and depression.[75]

Such disorders are more specifically related to the sodium-to-potassium ratio. Diagnosed manic depressive patients usually show an elevated tissue sodium and potassium but a much lower sodium/potassium ratio. This may explain the mechanism behind the effectiveness of lithium in that lithium salts temporarily improve the metabolic ratio of these electrolytes.

Depression

Depression is commonly seen in the PS types. The TMA pattern consists of elevated tissue calcium and magnesium levels relative to phosphorus, sodium and potassium. This pattern is associated with adrenal and thyroid insufficiency, a condition which contributes to a reduction in metabolic rate and energy production.

Fatigue is a fundamental aspect of depression that occurs alone or in combination with other symptoms including anxiety, fear and sadness.[76] As mentioned previously, hypercalcemia is universally associated with depression. Increased magnesium levels have similar effects as calcium. Sodium deficits are related to abnormalities in membrane permeability. Lowered membrane permeability is associated with decreased synaptic impulse transmission and has been found in underactive or depressed groups.[77] Fluctuations in adrenal steroids have also been noted in patients during mood alterations.[78 79 80] In addition hypothyroidism has been noted in affected patient groups compared to normal control groups.[81]

Conclusion

These are only a few of the many observations that have been made between personality and

tissue mineral patterns. Further development of TMA in this area may help to uncover many more relationships between emotions and disease. That most disease conditions have an emotional counterpart is well known. It is becoming more recognized that emotions are linked to biochemical changes that in turn can lead to the development of disease. The most recognized at this time is the relationship of Type A behaviour and an increased incidence of heart disease. Psychic and emotional factors can trigger an increase in mineral excretion and absorption. It is reasonable to assume that chronic or strong emotional reactions will produce neurological, hormonal and ultimately dynamic nutritional changes, which will eventually lead to metabolic changes that can be measured. For example, an increase in circulating stress hormones, as well as changes in mineral concentrations, will be reflected in the hair. The theory put forth in this paper is that tissue mineral patterns can reflect these changes. TMA may eventually be developed as a tool for assessing not only psycho-somatic but also somato-psychic relationships.

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