Functional Diagnosis in Nutritional Medicine

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Functional Diagnosis is one of the most important cornerstones of Nutritional Medicine. Likewise, clinical nutrition is one of the most important foundations of holistic medical treatment. Tracking down possible shortcomings and imbalances is an important first step in evaluating the nutritional status of any patient. This should be the first step in the process of attempting to achieve a complete rehabilitation and optimization of health.

Although minerals and trace elements were the last substances to be "discovered" as important contributors to a smoothly-operating biological system, the methods suggested for measuring and monitoring their presence or absence is still a subject of controversy.

The basic question is: what part of the human body may provide the most reliable information about the body's mineral status? There are a number of different options. The most frequently used sources include blood, urine, hair, teeth, nails. Others, like liver, kidney or spleen biopsy, may give more direct insight into the mineral status. However, they may be discounted for general use because one has to apply quite invasive methods to obtain them. The use of teeth for analytical purposes does not seem to be practical as they are not readily available. Urine contains whatever the body has excreted and not what it has retained; and little is known about nails - neither the normal range of elements present, nor to what extent they are contaminated. We are left with two options: blood and hair. Before the relative merits of either are considered, it must be emphasized that, in analyzing both, one is not using different methods for assessing the same thing. The two methods reflect body mineral status over different scales of time. Blood mineral concentrations are transient, depending on the supply of elements in the previous hours or days. Hair, in contrast, while offering no information on immediate levels, fixes the elements, providing a lasting record of levels over the previous few weeks to months.

The most often asked question is, whether hair mineral really reflects the level of minerals at the cell level (in the tissues)? This question was addressed by different research groups. Jacob and his group indicated that hair copper of adult rats was found to correlate directly with copper of the whole liver.[1] The comparative study of the copper content of the liver and hair of African children with kwashiorkor suggested a direct correlation between the reduced levels of copper in the hair and the liver of these subjects.[2] A completely opposing view was expressed by Epstein.[3] "In primary biliary cirrhosis, hair copper does not reflect liver content and is of no value as a biopsy material for copper analysis." As it is defined, cirrhosis is an inflammatory disease in which the normal cells are replaced by fibrous tissue. Passage of blood through this tissue may be obstructed by the cirrhosis. In biliary cirrhosis, the distribution of copper (and other elements) will not proceed as in a non-obstructed, "normal" liver. This is usually the case for all tissues, including hair.

What Can We Learn from Hair Mineral Analysis?

The often heard opinion of mainstream medicine is that the only significant use for hair mineral analysis (HMA) is for detecting the body burden of toxic elements. However, it appears from the scientific literature that HMA is providing very significant information about deficiency of zinc,[4] chromium,[5] copper,[6] potassium,[7] calcium and magnesium,[8] to name a few. It

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also can indicate excess of minerals. These elevated levels of minerals do not always mean increased ingestion. Rather, in many cases they reveal certain patterns caused by metabolic changes. These changes, as we will see in the following examples, can contribute to an understanding of the underlying biochemical changes, which are very difficult to detect by other analytical means.

Energy production is one of the major functions of our metabolism. Normally, the breakdown products of carbohydrate, protein and fat enter the Krebs cycle and 'burn down' to carbon dioxide and water. Deficiencies in minerals, trace elements and certain vitamins, being present in different stages of the Krebs Cycle as coenzymes, or catalyzers, can stall the normal oxidation/reduction process. NADH (Nicotinamide adenine dinucleotide) and its oxidized form, NAD+, play a significant role in catalyzing the oxidation/reduction of the different metabolites of the Krebs Cycle. If there are deficiencies in these essential elements and/or vitamins, the loop of NADH/NAD+ will be substituted by a breakdown product of glucose–pyruvate, which will oxidize NADH and, in turn, will itself be reduced to lactic acid. Lactic acid usually is the end product of fermentation. The accumulation of lactic acid in the cells will produce an acidic state, metabolic acidosis (MA), which is incompatible with normal homeostasis. The self-defense mechanism of the body activates the parathyroid gland and its hormone will be actively involved in transferring calcium (and later magnesium) to the tissues (cells) in an attempt to neutralize the lactic acid[9] by salt formation (calcium lactate, magnesium lactate).

This transfer activity occurs through the blood system with the help of the parathyroid hormone. Because the blood has an extremely effective buffering system, the level of calcium is very well controlled (9-11 mg/%). The excess will be excreted and will show up simultaneously in the urine and the hair tissue. The elevated level of calcium in a 24h urine may indicate the presence of MA, but without any information as to the period during which this process was occurring. Elevated levels of calcium and magnesium in the hair tissue, on the other hand, can indicate an approximate time of this metabolic impairment by observing how far the levels of these two elements are from the mean of their reference values.

Under normal conditions (aerobic phosphorylation) the energy is produced at about 80% efficiency by breaking down glucose to carbon dioxide and water. In the case of metabolic acidosis, the process of fermentation (glucose> pyruvate>NADH> NAD+ >lactate), will yield, with the same calorie intake, only about 20% of energy. It is no wonder that people experiencing MA are complaining about fatigue, headaches, migraines, lack of concentration and forgetfulness. The only common denominator is lack of energy.

There is another avenue leading to MA. In populations where the consumption of protein is increased, the high phosphorus content in the protein will produce an acid ash end-product (phosphoric acid). In North America, this problem is compounded by the consumption of large quantities of phosphoric acid contained in soft drinks, and canned foods buffered by phosphate buffers, all of which increase the production of acid ash. The metabolic consequence is an attempt to neutralize the phosphoric acid (and sulfuric acid produced from sulfur-containing amino acids) by removing and transferring calcium from the bones and teeth to the mitochondria. "The increased incidence of osteoporosis with age", as it is postulated by Wachman,[10] "may present in part, the result of a lifelong utilization of the buffering capacity of the basic salts of bone for the constant assault against pH homeostasis. The loss of as little as two meq of calcium per day would, over a decade, as-

suming a total body content of one kg, account for 15% loss of inorganic bone mass in an average individual."

Mineral Interaction

The observation in nature that soils rich in certain minerals can cause a deficiency in others has incited many nutritionists to study this interrelationship among the different elements. In the last 50 years, many studies were conducted in order to find the explanation for these antagonisms. It was found that ions whose valance shell electronic structures[11] or electronic configurations[12-14] were similar, would be antagonistic. These studies were conducted in animals, and the survival time and body weight gain or loss were used to measure the biological effects of the interaction of the different elements. Impressive data was compiled by the Task Group on Metal Interaction in 1978[15] and also on nutritional elements interaction with toxic elements.[16] These studies of element interaction can assist health practitioners in providing a well-informed program of food supplements. As long as people were able to receive all the necessary nutrients from food, the concentration of each ingredient was at a physiological level with minimal interference among the different elements. As we learned more recently, however, the food we eat does not contain the quantities of nutrients that we once took for granted. The mass production of food results in foodstuffs which may be tempting to our senses but which are deprived of essential vitamins, minerals, trace elements and other nutritious ingredients. Some of these are added to the end product, but in quantities and qualities that can't be compared with the 'originals'. This is the rationale behind building up a supplementation program. This kind of program attempts to provide nutrients in quantities that are sufficient in supporting the metabolic needs of the body for achieving optimum performance. These quantities are many times more than those present under normal physiological conditions (mega doses). This of course is a very relative statement. For instance, the RDA for ascorbic acid is 60 mg/day. Human, primates and guinea pigs are missing an enzyme which transforms glucose into ascorbate. Dr Levine[17] studied animals capable of producing ascorbic acid and found that the mg/kg production of ascorbic acid - calculated on a human weighing 70 Kg is:

	without stress	under stress
Rabbit	1547	15,870
Rat	2737	13,860
Mouse	2352	19,250

Based on this information one can conclude that by taking 37 times the RDA for ascorbic acid, only the necessary quantity for a non-stress situation is satisfied. When using megadoses of minerals and trace elements, their interaction and antagonism should be taken into consideration. Instead of interfering at the absorption site, we can supplement the competing elements at a different time of the day (this kind of interaction chart is available at Anamol Labs).

Toxic elements most frequently occurring are: lead, cadmium, aluminum and mercury. A flood of information is available on their toxic effect on different body functions. It is postulated that most of their toxic effect is caused by the fact that they are substituting the nutritional ones, like calcium, magnesium, zinc, manganese, copper. By so doing, all the enzyme activities depending on the presence of these nutritional elements will be impaired or completely eliminated. Moreover, studies comparing the absorption of lead by children having a low calcium diet with children on a high calcium diet, demonstrate that the children on a low calcium diet absorbed significantly more lead than the high calcium intake group. This occurred even though both groups were exposed to the

same environment. Extrapolation of this experiment may indicate that, exposed to the same environment, the well-nourished individual has a better chance to withstand the hazards of toxic environmental exposure. In cases of confirmed body burden of toxic elements, the usual answer is chelation therapy, using different chelating agents, such as EDTA. These chelating agents remove elements indiscriminately, toxic or nutritional; the latter having to be replaced after the treatment. Using the understanding of antagonistic interaction of elements, one could use "replacement therapy." For instance, calcium and zinc, elements in which we are usually deficient, can remove lead, cadmium, aluminum and copper from the system in cases where their levels were elevated.

Conclusion

It can be said that hair mineral analysis can detect deficiencies or excesses of nutritional elements, recognize certain patterns indicating metabolic changes, like metabolic acidosis, and can also indicate the presence of toxic elements. Using HMA to monitor a supplementation program, the health professional is able to introduce the necessary changes according the patient's biochemical individuality. Using "replacement therapy," the toxic elements can be removed with hair analysis serving as a follow-up assessment method for effectiveness of detoxification.

References

1. Jacob RD, Klevay LM, Logan GM,: Hair metal as an Index of Hepatic Metal in Rats: Copper and Zinc. *Am J Clin Nutr,* 1978; 31: 477-480.
2. MacDonald I, Warren PJ: The Copper Content of the Liver and Hair of African Children with Kwashiorkor. *Brit J Nutr,* 1961; 15: 593-596.
3. Epstein O, Boss AMB, Lyon TDB, Sherlock S: Hair copper in Primary biliary cirrhosis. Am *J Clin Nutr,* 1980; 33: 965-967.

4a. Bollin SE et all: *The structure and metabolism of the Pancreatic Inlets.* Oxford. Pergamon Press. 1964.

4b. Contiero E, Folin M: Trace elements nutritional status. Use hair as a diagnostic tool. *Biol Trace Elem Res,* 1994; 40: 151-180

4c. Olcucu A, Caglar P: Zinc levels in human hair and serum of infants and children and their relationship to various diseases in the upper Euphrates basin. *J Trace Elem Exp Med*, 1993; 6:141-145.

5. Merz W: Biological role of chromium. *FASEB* 1967; 26: 186-192.
6. Klevay LM: Hair as a Biopsy Material. Assessment of copper nutriture. *Arch Intern Med* 1978; 138: 1127-1128.
7. Kapito L, Elian E, Schwachman H: Sodium, potassium, calcium and magnesiumin hair from neonates with cystic fibrosis and in amniotic fluid from mothers of such children. *Pediatrics,* 1972; 49: 620-624.
8. Kopito L, Schwachman H: Alterations in the elemental composition of hair in some diseases. In: eds. Brown AC: *First Human Hair Symposium,* New York. Medcoin Press 1974: 83-90. Cotton D, Porters J, Spruit D: Magnesium content of the hair in Alcopecia Areata Atopica. *Dermatologica,* 1976; 162: 60-62.
9. Tamari GM, Rona Z: Hair mineral levels and their correlation with abnormal glucose tolerance. *Cytobiol Rev,*1985; 4: 191-196.
10. Wachman A et al: Diet and osteoporosis. *Lancet,* 1968; May 4: 958-959.
11. Hill CH, Mairone H: Chemical parameters in the study of in vivo and in vitro interactions of transition elements. *Fed Proc, Amer Soc Biol* 1970; 29: 1474-1481.
12. Hariman RH, Matrone G, Wise GH: Effect of high dietary manganese on hemoglobin formation. *J Nutr,* 1955; 55: 429-439.
13. Thompson ABR, Olatunbosun D, Valberg LS: Interaction of intestinal transport system for manganese and iron. *J Lab Clin Med,* 1971; 78: 642-655.
14. Chetty KN: *Interaction of cobalt and iron in chicks.* Ph.D. Thesis. North Carolina University,1972.
15. Nordberg GF et al: Factors influencing metabolism and toxicity of metals: a concensus report. *Environ Hlth Persp,* 1978; 25: 3-41.
16. Mahaffey KR, Rader JI: Metabolic interactions: lead, calcium and iron. *Ann NY Acad Sci,* 1980; 355: 285-297.
17. Levine M: New concepts in the biology and biochemistry of ascorbic acid. *New Engl J Med,* 1986; 314: 892-902.