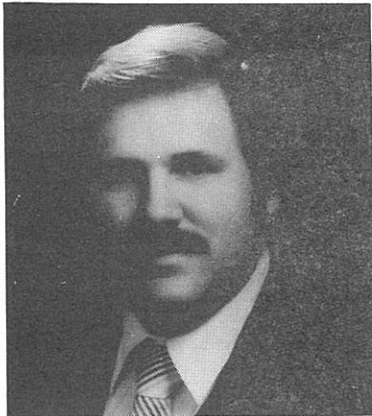


# Trace Element Patterning in Degenerative Diseases

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## Introduction

With all of the advances in modern nutrition, new analytical methods are constantly being focused upon in the total evaluation of the patient. The analysis of hair for trace elements has received much attention in recent years due to the trend of more physicians toward nutritional therapies and the need for a "cellular" source of biological samples for analysis of cellular level dysfunction.

Classically, physicians have been concerned with "monitoring the exhaust from the car" - an industrial analogy of the taking of venous blood for analysis. This usually only reflects pathology and occasionally reflects the opposite of what is happening in the cellular machinery. The analysis of iron gives us an example of the dichotomy of "cellular vs. effluent" analysis. There are many patients who have normal hemoglobins and serum iron levels but also have vertical ridging on their fingernails (a symptom of iron deficiency) as well as low hair levels of iron. Iron is intimately involved in the metabolic machinery of the cells as it forms an integral part of the cytochrome system. Hence, the lack of proper utilization of oxygen is impaired and the rapid growing nail matrix is synthesized in a defective manner.

Hair analysis has been around for many years but its recent resurgence in popularity has paralleled a growing emphasis on holistic medicine. In the past, hair has been used as biopsy material (Perkons, et al., 1966; Klevay, 1970), to

assess the zinc nutritional status of Iranian dwarfs (Halsted, et al., 1972), to assess zinc levels in post-burn patients (Pullen, et al., 1971; Piores, et al., 1967), to assess copper levels in chemical schizophrenics (Pfeiffer, 1972), to assess lead levels in hyperactive children (David, et al., 1972), and many more applications. More recently the focus of attention has been away from the toxicological approach and directed instead toward looking at a spectrum of the essential trace elements as well as several of the significant environmental toxins.

The final question that remains in our analysis of trace elements is the source of the specimen. Most of the selection requirements have been alluded to previously, but in summary the specimen (1) must be a cellular rather than an effluent source, (2) must be metabolically active yet one that is not rapidly changing, (3) should be relatively painless and simple to obtain, and (4) should be representative of the body's cellular machinery. Hair, perhaps better than any other tissue, satisfies most of these requirements in that: (1) it is a cellular source; (2) it is the second most metabolically active tissue in the body, second only to the bone marrow, yet it only grows at a rate of .5 to 1.0 mm per day; (3) it is painless and simple to obtain, and (4) it is probably as representative of cellular metabolism as any other tissue - whether visceral or ectodermal. Hence, this study of trace element levels in degenerative diseases was undertaken using hair as the standard for analysis.

**Methods**

In an attempt to obtain an adequate representative population, this initial study involved securing specimens from a number of physicians and having them indicate the nature of the pathology or degenerative processes present. Biochemical Concepts Laboratory in Albuquerque, New Mexico was kind enough to supply the patient information and the results of the trace mineral analyses for this study. Figure 1 shows the hair specimen envelope used by doctors for submission of the hair specimens. Doctors were asked to check up to 5 of the disease codes and upon completion of the analysis, the results were added to the data bank of an individual disease code. Only two criteria were used in rejecting data from the study. If more than 5 codes were checked, it was felt that either hypochondriasis or inaccurate diagnosis become significant and the patient is excluded from the study. Secondly, if the levels of any given trace element were greater than 4 times the mean, then external or internal contamination was suspected and the sample rejected. This was most often found with lead levels and the use of certain hair darkening agents was suspected. The hair specimens were collected from the suboccipital area and only the 1-2 inches proximal to the scalp were submitted to analysis. The hair is then washed, dried and chemically ashed with perchloric

*Trace Element Patterning*

acid and hydrogen peroxide prior to analysis. The analyses were performed a varian atomic absorption spectrophotometer.

Physicians Hair Specimen Envelope Instructions for Submissions of Hair Specime

**BIOCHEMICAL CONCEPTS  
SPECIMEN FOR ANALYSIS**

Patient's Name	Age	Sex	Wt.	Height	Hair Color	Previous Test	Hair Tint or Product	Date
NATURAL HAIR COLOR: Black = 1, Brown = 2, Red = 3, Blonde = 4, White = 5 PREVIOUS TESTS = Number of Previous Hair Analyses Given Patient. None = 0, One = 1, Two = 2, Three = 3, More Than Three = 4								

**DOCTORS INFORMATION**

Code No.: \_\_\_\_\_ City: \_\_\_\_\_  
 Name: \_\_\_\_\_ State: \_\_\_\_\_ Zip: \_\_\_\_\_  
 Address: \_\_\_\_\_ Phone: \_\_\_\_\_

**SAMPLE REQUIREMENT:** 3 Teaspoons of hair from nape of neck or nearest head hair possible  
 For office use only: \*Assigned By Laboratory LAB NO. \_\_\_\_\_

**MINERAL ANALYSIS PROGRAM**  Standard assay with consult and dist sheets  
 Standard - NO consult  
 Toxic Assay

**TYPE OF ASSAY CHECK (1) ONE**  
 AMOUNT ENCLOSED \$ \_\_\_\_\_

**DOCTOR: PLEASE CHECK NO MORE THAN 5 OF THE DISEASE CODES LISTED BELOW:**

<b>A. BONE AND JOINT DISORDERS</b> <input type="checkbox"/> A1. Osteoarthritis <input type="checkbox"/> A2. Rheumatoid Arthritis <input type="checkbox"/> A3. Gouty Arthritis <input type="checkbox"/> A4. Low Back Pain <input type="checkbox"/> A5. Somatic Dysfunction	<b>B. METABOLIC DISORDERS</b> <input type="checkbox"/> B1. Diabetes Mellitus, Adult Onset <input type="checkbox"/> B2. Diabetes Mellitus, Juvenile Onset <input type="checkbox"/> B3. Hypoparathyroidism <input type="checkbox"/> B4. Hyperparathyroidism <input type="checkbox"/> B5. Hypothyroidism <input type="checkbox"/> B6. Hyperthyroidism <input type="checkbox"/> B7. Adrenal Insufficiency <input type="checkbox"/> B8. Edema Fluid Retention <input type="checkbox"/> B9. Hypertension <input type="checkbox"/> B10. Obesity <input type="checkbox"/> B11. Weight Loss <input type="checkbox"/> B12. Hypotension	<b>C. HEMATOLOGICAL</b> <input type="checkbox"/> C1. Anemia <input type="checkbox"/> C2. Fatigue, Undetermined Origin	<b>D. ORGAN SYSTEMS</b> <input type="checkbox"/> D1. Respiratory Disorders <input type="checkbox"/> D2. Urinary Tract Disorders <input type="checkbox"/> D3. Gastrointestinal Disorders <input type="checkbox"/> D4. Otolith/Oryctolite Disorders <input type="checkbox"/> D5. Cardiovascular Disorders <input type="checkbox"/> D6. Neurological Disorders <input type="checkbox"/> D7. Ocular Disorders <input type="checkbox"/> D8. Musculoskeletal Disorders <input type="checkbox"/> D9. Dermatologic Disorders <input type="checkbox"/> D10. Lung Disorders	<b>E. DEGENERATIVE DISORDERS</b> <input type="checkbox"/> E1. Cancer <input type="checkbox"/> E2. Demyelinating Disease (MS) <input type="checkbox"/> E3. Arteriosclerosis	<b>F. EMOTIONAL AND MENTAL</b> <input type="checkbox"/> F1. Fatigue, Irritability, Depression <input type="checkbox"/> F2. Post Menopausal Syndrome <input type="checkbox"/> F3. Learning Disorders <input type="checkbox"/> F4. Schizophrenia <input type="checkbox"/> F5. Depression <input type="checkbox"/> F6. Nervousness and Anxiety <input type="checkbox"/> F7. Nervous Tics <input type="checkbox"/> F8. Psychotic Disorders	<b>G. MISCELLANEOUS</b> <input type="checkbox"/> G1. Chronic Infection <input type="checkbox"/> G2. Insomnia <input type="checkbox"/> G3. Cancer <input type="checkbox"/> G4. Drug Addiction <input type="checkbox"/> G5. Headache <input type="checkbox"/> G6. Hair Loss <input type="checkbox"/> G7. Allergies <input type="checkbox"/> G8. No Disorder Present	<b>H. OTHERS</b> 1. _____ 2. _____ 3. _____
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PLEASE FILL THE FORM OUT COMPLETELY FOR PROPER PROCESSING OF RESULTS.

PLEASE ENCLOSE REMITTANCE WITH SAMPLE.

Figure 1

**Results and Discussions**

The data presented is without the actual values as to avoid excess material on the graphs. Figure 2 is representative of the mean levels in mg% each trace mineral. The remainder of this section involves the discussion hair trace mineral profiles of several degenerative diseases along with possible biochemical explanations for the patterns found.

HAIR MINERAL AVERAGES (N=1400)			
Element	Total	Female	Male
Calcium	80.2	91.3	55.5
Magnesium	8.9	9.9	6.5
Sodium	53.5	54.2	51.9
Potassium	36.5	35.4	39.0
Copper	2.17	2.12	2.29
Zinc	16.6	16.7	16.4
Iron	1.56	1.59	1.49
Manganese	.079	.079	.080
Chromium	.059	.059	.059
Lithium	.019	.019	.019
Cadmium	.096	.086	.117
Lead	1.51	1.25	2.06

(all values expressed in mg%)

Figure 2

Population Averages

Figure 3 represents the variation of male and female averages from that of the total population. There are only minor variations in most of the trace elements with the exception of calcium, cadmium and lead, the former being elevated in the female and the latter two in the male. Calcium elevation in the female is thought to be indicative of the number of post-menopausal females in this study, many of which have osteoporotic tendencies. In the osteoporotic patient, calcium leaves its normal metabolic pathway and becomes deposited in the soft tissues, including the hair. Females with a higher propensity toward osteoporosis subsequently have higher hair calcium levels. The cadmium in the males is probably due to increased cigarette consumption in males whereas the increased lead in the males is most likely associated with the fact that it is still primarily the male who each morning drives to work in a cloud of lead contaminated exhaust fumes.

Part I - Cardiovascular and Metabolic Diseases

Diabetes Mellitus - Juvenile vs. Adult Onset

The juvenile diabetic differs very significantly from the adult onset diabetic as is noted by their hair patterns as depicted in Figure 4. The juvenile diabetic is not deficient in any of the trace elements other than potassium. This is predictable for two reasons; first, the juvenile form is thought to be a genetic rather than a nutritional disorder; secondly, potassium is known to be involved in glucose entry into the cell. The insulin dependent glucose cellular pump requires potassium for normal functioning (Schwartz, 1971). In patients that have an unnatural level of insulin and in whom diabetic keto-acidosis with its resultant potassium loss is a problem, one might expect lower than normal tissue reservoirs of potassium.

Conversely, the adult onset diabetic is a nutritional problem predominantly. The hair levels of magnesium, manganese, chromium and zinc are below normal. These four trace elements have characteristic roles in glucose metabolism. MAGNESIUM activates over 50% of the enzymes in the body including six of the nine glycolytic enzymes (Harper, 1973). With decreased levels of magnesium, the metabolic machinery of the body cannot function optimally. The role of ZINC is more specific than that of magnesium. Zinc is involved in the granulation and storage of insulin in the beta cells of the pancreas (Bollin, et al., 1964). It has also been established that the defect in

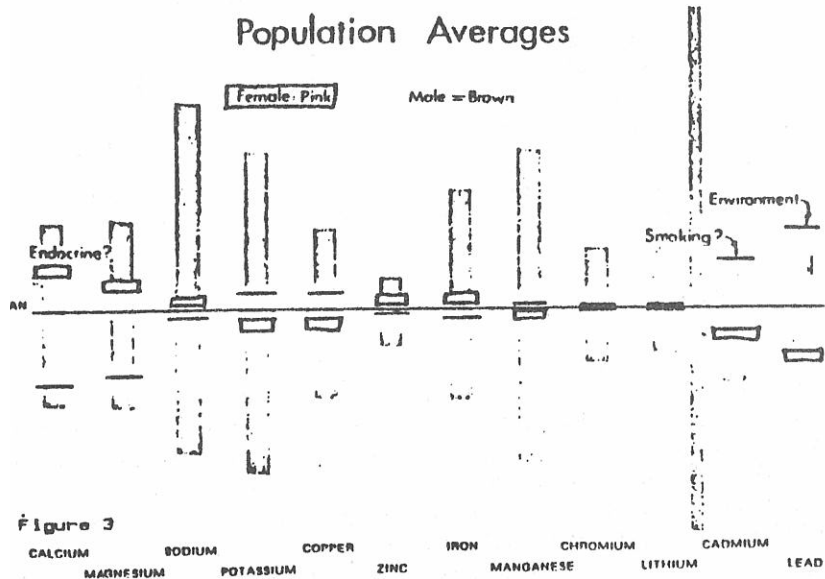
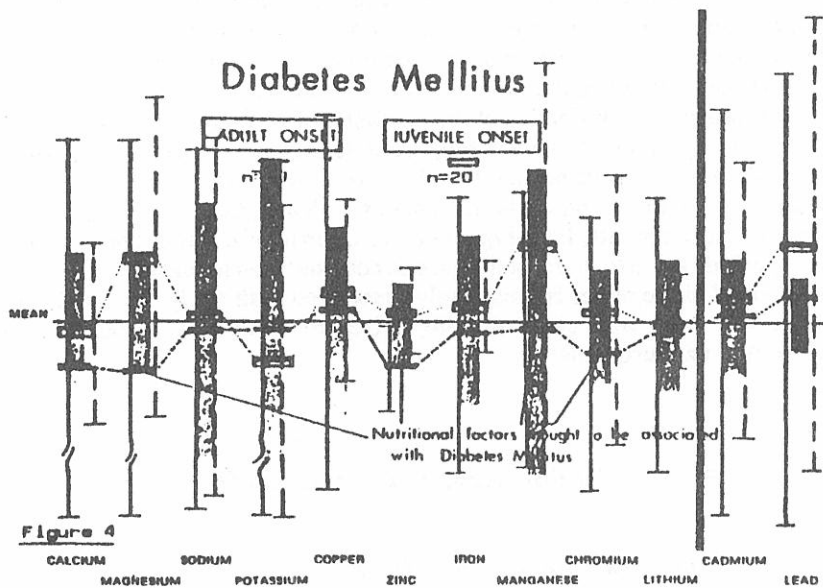


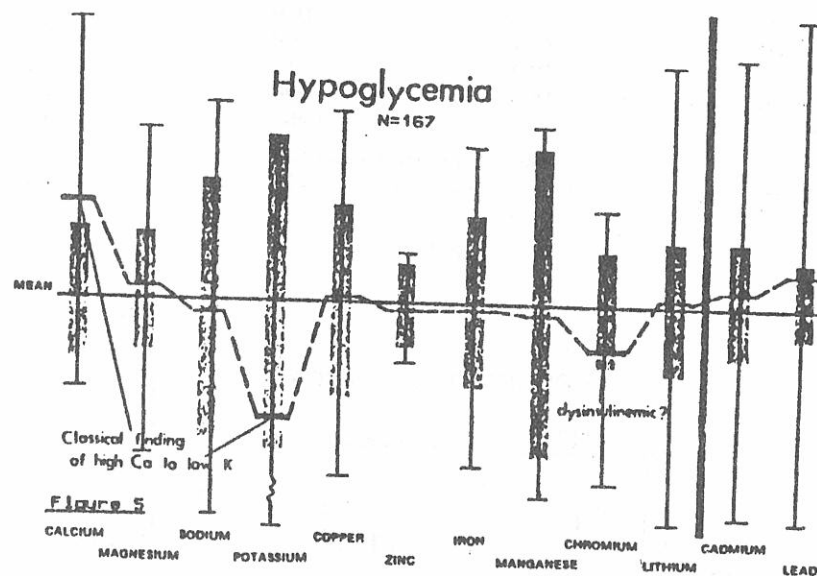
Figure 3



dysinsulinemia, a pre-diabetic state, is one of the beta cells being unable to store and granulate insulin (Zilva & Pannal, 1972). Hence, a zinc deficiency state could be a precursor to dysinsulinemia and adult onset diabetes. The role of CHROMIUM is at yet another location in the metabolism of glucose. Chromium acts at the cell membrane facilitating the entry of glucose into the cells. It has been referred to as the "glucose tolerance factor." Studies with the adipose tissue of experimental animals indicate that chromium is ineffective without insulin and that the effectiveness of a given dose of insulin can be enhanced by 50-100% by the addition of chromium to the system (Mertz, 1967). Chromium has been postulated to act as a disulfide bridge between the A-chain of insulin and membrane sulphydryls allowing insulin to be effectively bonded to the membrane of the target tissues (Mertz, 1967). The role of MANGANESE in glucose metabolism is the least understood. Biochemically, it is important in the functioning of isocitric dehydrogenase, an important control enzyme in the regulation of the Krebs cycle (Harper, 1973). Rats deficient in manganese exhibit severely diabetic glucose tolerance curves which revert to normal with the inclusion of manganese in the diet (Everson & Shrader, 1968). In conclusion, the four trace elements above appear to synergistically affect the body's ability to handle glucose and may be of significant value in the prevention of adult onset diabetes mellitus.

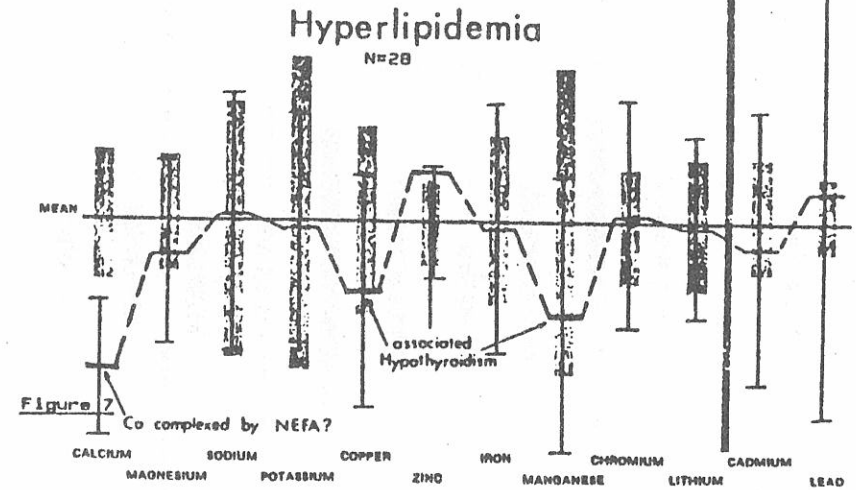
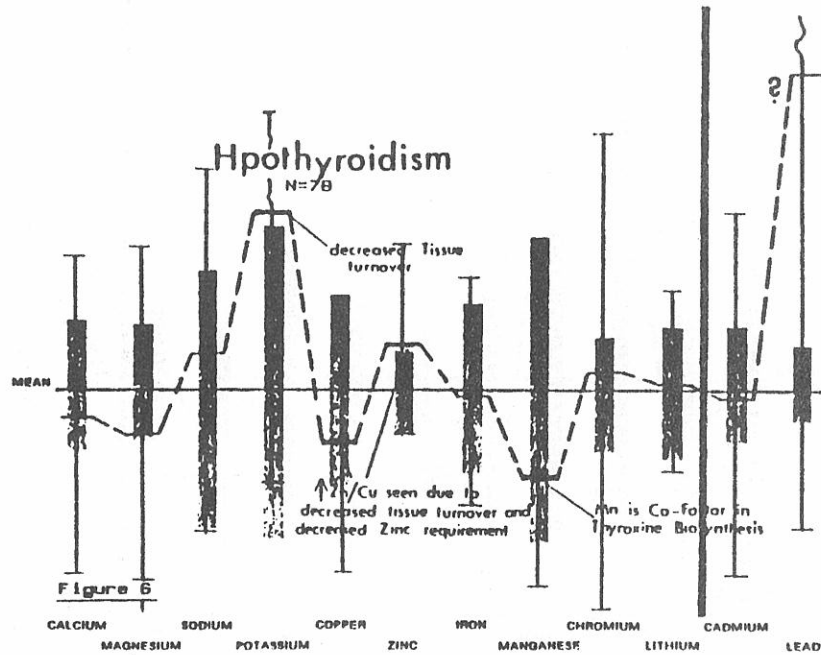
### Hypoglycemia

The hypoglycemic patient exhibits a characteristic increase in the C:RATIO as noted in Figure 5. Perhaps even more significant, however, is low CHROMIUM level found in these patients since dysinsulinemia commonly misdiagnosed as hypoglycemia and many pre-diabetic states can be overlooked.



### Hypothyroidism

This metabolic anomaly is most characteristically recognized by the increased Zn/Cu RATIO. The increased ZINC and POTASSIUM are due to decreased tissue turnover and subsequently a decreased need for these elements. Zinc is an activator of the enzyme collagen synthetase (McClain, et al., 1972). If the body is metabolically underactive, the tissue requirement for zinc decreases and tissue levels remain elevated. In contradistinction COPPER and MANGANESE are involved in thyroxine biosynthesis. Manganese is involved in the interconversion of phenylalanine to thyroxine (Seven, 1960) whereas copper is involved in the oxidation of iodide to elemental iodine in the thyroid acinar cell. Figure 6 is the profile for the hypothyroid.



**Hyperlipidemia**

The characteristic hair pattern of patients with hyperlipidemia is given in figure 7. The pattern is very similar to that of the hypothyroid individual (figure 6) probably because of the association of hyperlipidemia with hypothyroidism. The serum cholesterol usually parallels the thyroid hormone in an inverse manner. The low CALCIUM in these individuals is of uncertain etiology but may reflect the complexation of calcium by increased levels of non-esterified fatty acids or perhaps the chronic pancreatitis, sometimes associated with hyperlipidemia, particularly Fredrickson types I and V. Either reason is very acceptable however.

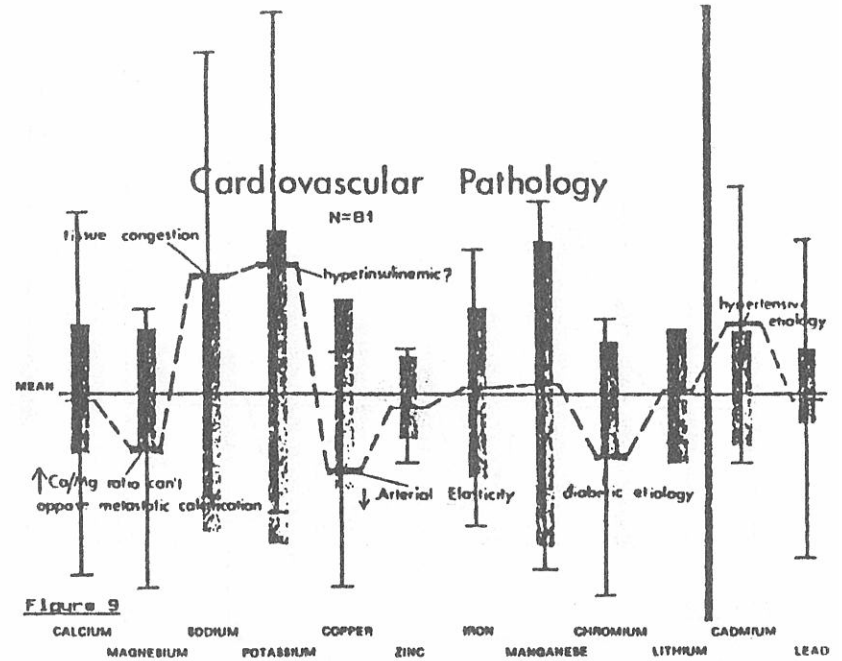
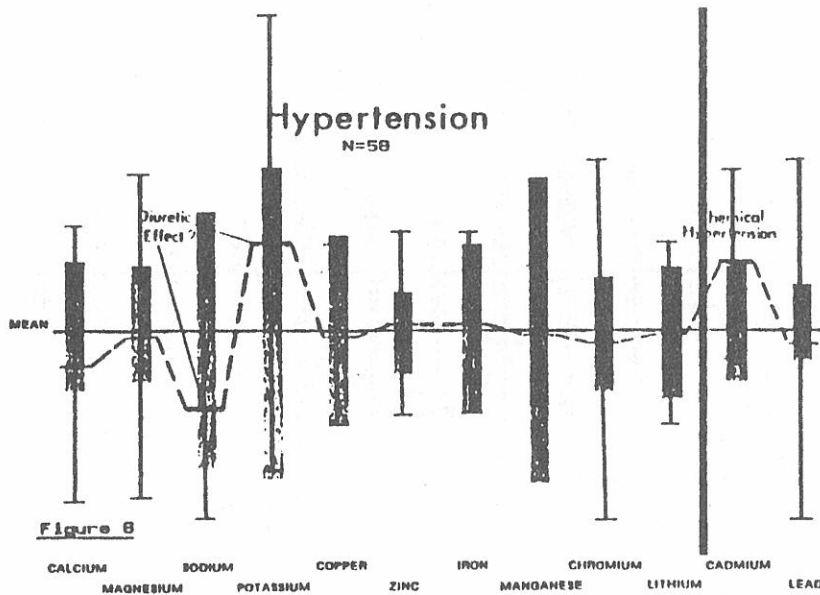
doubling the systolic blood pressure, a phenomenon not capable of being induced even by renal artery constriction and the renin-angiotensin system (Schroeder, et al., 1966). Studies have shown that hypertensive individuals excrete 5 to 10 times more cadmium than their normotensive counterpart while there was no difference in the excretion of any other trace mineral (McKenzie & Kay, 1973). When the kidneys of individuals succumbing to accidents vs. those suffering hypertension related deaths were assayed for cadmium and its natural biological antagonist, zinc, it was found that the Cd/Zn RATIO was 30-40% higher in those with hypertension associated deaths (Schroeder, 1965). The low tissue SODIUM in these individuals is perhaps a reflection of diuretic therapy, the first line of therapy in the conventional medical treatment for hypertension. The data suggests that perhaps essential hypertension is not really "essential" at all but rather low grade reno-toxicity - a curable entity just as the other causes of metabolic hypertension (i.e., pheochromocytome, Conns syndrome, etc.).

**Hypertension**

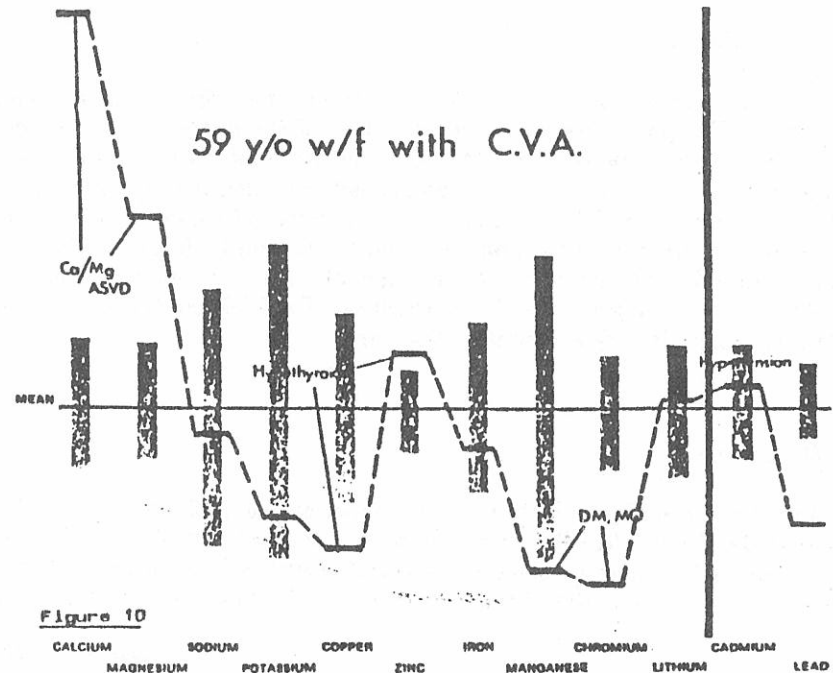
The most significant finding in the hypertensive individuals is that of increased cellular CADMIUM levels as illustrated in Figure 8. Cadmium has very potent hypertensive effect at low levels in the tissues (Schroeder, 1967). When included in the diet of experimental animals, cadmium was capable of

**Cardiovascular Pathology**

The patients with actual cardiovascular symptoms reflect combinations of the pathology presented in previous examples as depicted in Figure 9. The role of hypertension in cardiovascular disease is well established. The role of CADMIUM in essential hypertension has also been suggested earlier in this



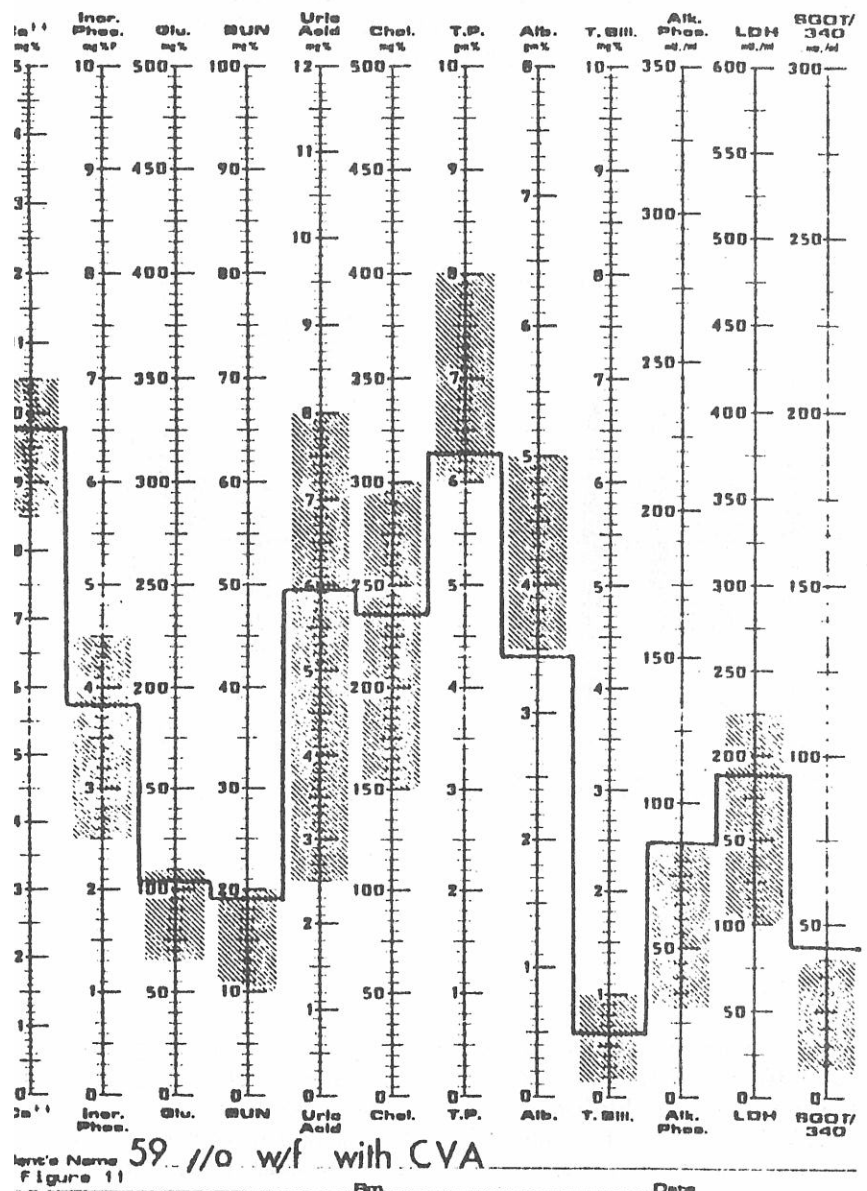
manuscript (Figure 8). The association of adult onset diabetes mellitus with vascular pathology has also been well established by the medical profession. Likewise the role of CHROMIUM to adult onset diabetes mellitus has also been previously alluded to in this paper (Figure 4). It has also been established that patients with atheromatous disease at autopsy had lower levels of chromium in their aortas than did the individuals without atheromatous involvement (Schroeder, 1974). The role of COPPER in cardiovascular disease is thought to be associated with its activation of the enzyme lysyl oxidase. This enzyme oxidizes four lysine molecules and after multiple schiffs base formation, the molecules cyclize into the amino acid, desmosine. Desmosine is found only in the elastic tissues of the body and chicks grown on a diet with a deficiency of copper have 30-40% less elastin in the great vessels, a condition when extrapolated to humans might indicate a predisposition to aortic and other arterial aneurysms (Hill, et al., 1967). Increased SODIUM in the tissues may be due to the secondary hyperaldosteronism found in patients with diminished effectiveness of the heart as a pump. The Ca/Mg RATIO approaches 16:1, approximately twice the normal ratio of 8:1. When this ratio becomes altered in favor of CALCIUM, the deposition of calcium in the tissues can result predisposing the patient to such varied conditions as nephrolithiasis, arteriosclerosis, etc. MAGNESIUM therapy may be of therapeutic value in these patients (Varo, 1974). Figure 9 offers an excellent example of how varied pathology in the trace elements affects individual



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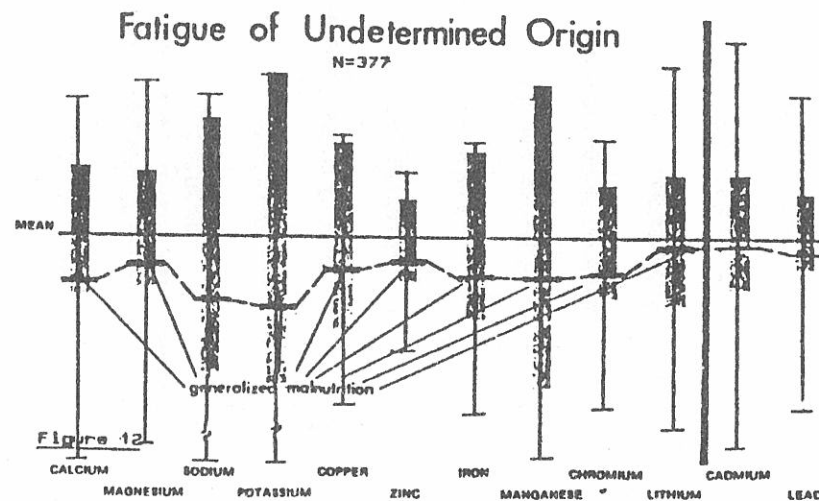


body systems and ultimately results in a degenerative symptom complex such as coronary artery disease. Figure 10 is the hair profile of a 59 year-old white female that suffered a cerebrovascular accident. The profile is described in the figure. In contrast, the SMA-12 (See Figure 11) on the same individual is supplied for comparison. From which can the most useful information be gleaned?

Part II - Neuro-psychiatric Disorders

Fatigue of Undetermined Origin

Figure 12 depicts the hair profile of patients with generalized fatigue. The pattern shows no specific trends but rather a generalized trend of trace mineral malnutrition. The elements present in the highest relative concentrations are the toxic elements, CADMIUM and LEAD. These patients suffer not only from low grade toxic metal poisoning but also generalized metabolic incompetence.

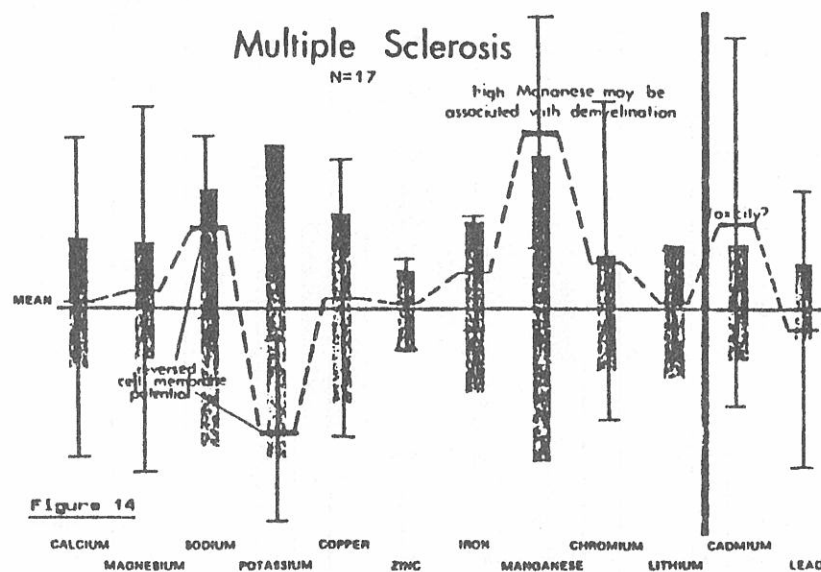
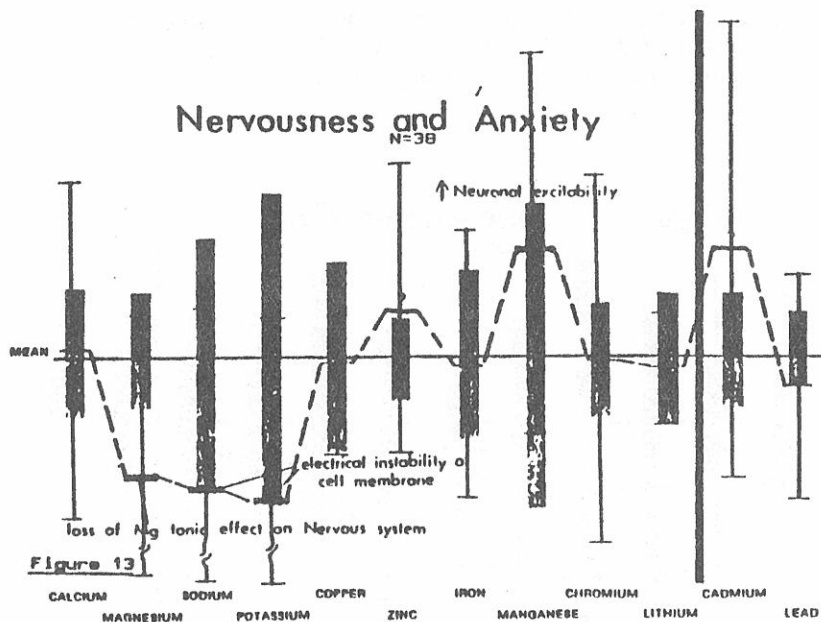


Nervousness and Anxiety

The pattern of nervousness and anxiety as depicted in Figure 13 reflects much variation in the trace element levels. No explanation is offered for the

elevated zinc and cadmium levels. The characteristic finding is the low Mg/Mn RATIO. MANGANESE, when in excess, can produce Parkinsonian-like tremors perhaps through some alteration in basal ganglion function (Cotzias, 1958). Conversely, MAGNESIUM has a tonic action on the nervous system. Magnesium delays synaptic transmission by interfering with the calcium mediated degeneration of acetylcholine in the synaptic cleft (Guyton, 1972). When the Mg/Mn RATIO is low, symptoms related to nervousness and anxiety result; when the ratio becomes elevated, nervous system depression results. The extreme deficiencies of SODIUM and POTASSIUM result in abnormal functioning of the cell membrane. The low intracellular sodium may reflect a generalized increase in efficiency in the sodium pump enabling neurons to repolarize and discharge at an elevated rate.

fatigued depolarized cell, one that might experience difficulties transmitting an impulse. Hence, the pathology of multiple sclerosis may be related to a defect in the membrane contained sodium pump. The manganese elevation is also of interest because high manganese in the drinking water has been associated with neurological abnormalities (Cotzias, 1958).



### Multiple Sclerosis

The pattern of multiple sclerosis is the only pattern so far encountered with reversed SODIUM and POTASSIUM levels as noted in Figure 14. This reversal from the normal cellular level with sodium levels high in the cell and potassium levels low, when extrapolated to a nerve, would be indicative of a

### Schizophrenia

The schizophrenic pattern, as depicted in Figure 15, is very simple showing only elevations in COPPER and CADMIUM. The elevation of copper in the hair has been well established by Pfeiffer (1974) in his description of chemical schizophrenia. Hair analysis should be performed on any patient with a mental aberration as a trace mineral imbalance can be the cause and hence would be curable through nutritional alteration, such as the avoidance of copper and the inclusion of zinc into the diet of a high copper schizophrenic.

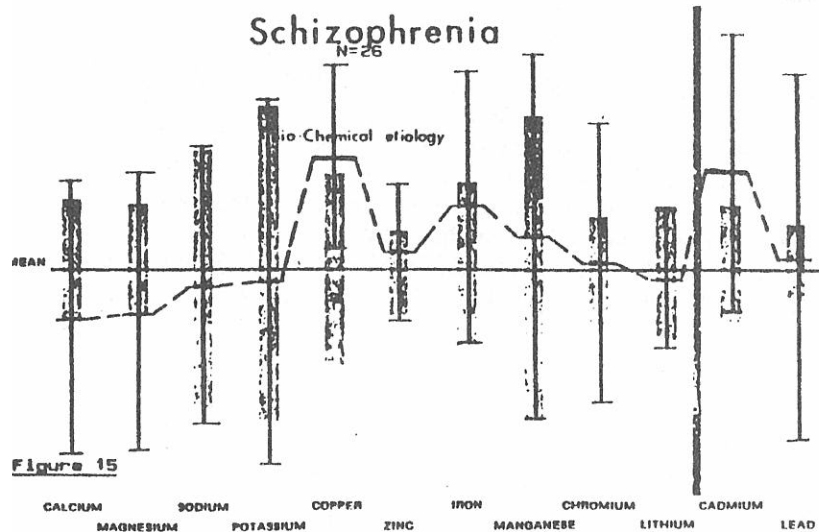


Figure 15

Learning Disorders

The learning disorder profile presented in Figure 16 summarizes much of the material previously presented in this section. One of the more obvious findings is the decreased Mg/Mn RATIO. This would be manifest as nervousness in an adult whereas in a child it is more indicative of a hyperactive personality disorder. The tonic effect of magnesium on the nervous system is lost whereas manganese's effect on the primitive motor system is stimulated. The copper levels in the hair of these children is extremely high reflecting a tendency toward personality disorders. Zinc, which is necessary for RNA biosynthesis (Astead, et al., 1974), levels are depressed. ZINC is also a biochemical antagonist of copper. There may be a true physiological barrier to learning since RNA has been implicated as the molecule in the brain responsible for information storage. LEAD may be the biggest problem in these children. Chronic iron lead poisoning alone can cause symptoms of hyperactivity, a fact well established in the literature (Lansdown, et al., 1974; Kopito, et al., 1967). When all of these factors are combined into one child, a hyperactive disturbed child may result. Even though Ritalin is conventionally the drug of choice in these conditions, a more logical initial approach might be to correct the underlying nutritional problems and let the body heal itself.

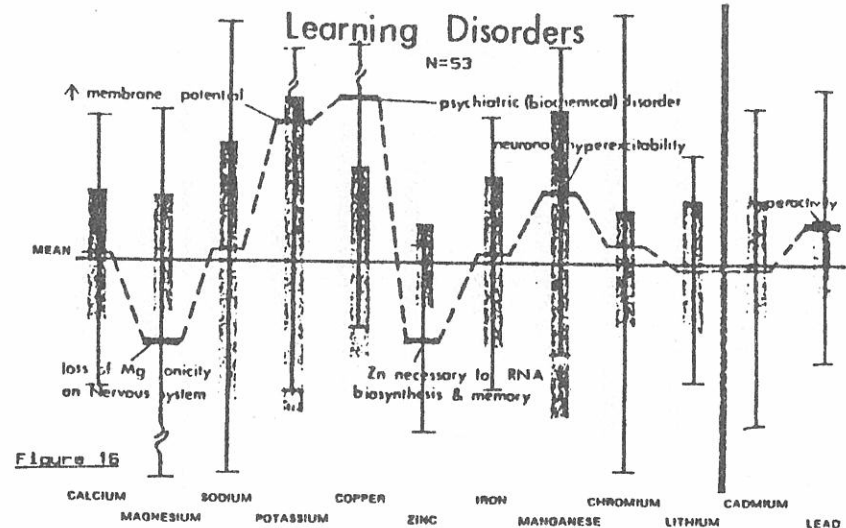


Figure 16

Part III – Miscellaneous Disorders

The last remaining disorders are unrelated but each has one or more characteristic finding associated with a trace mineral imbalance.

Dermatological Disorders

The principal significant finding in patients with dermatological disorders was a low hair ZINC level as shown in Figure 17. Zinc, along with ascorbic acid, is necessary for collagen biosynthesis. Zinc is involved in the assembly of the proteinaceous backbone of the collagen molecule. Without zinc, there will be delayed healing as well as problems maintaining the health of tissue rich in collagen such as skin.

Chronic Low Back Discomfort

The pattern of chronic low back discomfort is relatively simple, as noted in Figure 18, with the only significant finding being a low POTASSIUM. Low tissue potassium may predispose to muscle fatigability, the probable origin of the low back discomfort.

Genito-Urinary Disorders

Figure 19 represents the hair profile of patients with genito-urinary disorders. The principal pathology in these patients is the low ZINC levels. The highest levels of zinc in the body are found in the genital tissues and the kidneys. Chronic prostatitis is almost always associated with low hair zinc levels and chelated zinc therapy can be beneficial in these patients. The zinc level, while absolutely low, is relatively lower when one considers the high tissue levels of CADMIUM found in these patients. Cadmium is the toxic biological antagonist of zinc. Cadmium can cause deficiencies in the formation of the genital organs of lower animals, a condition fully reversible with zinc therapy (Parizek, 1957). The high levels of SODIUM and POTASSIUM may be a reflection of altered renal function with the retention of these two monovalent cations.

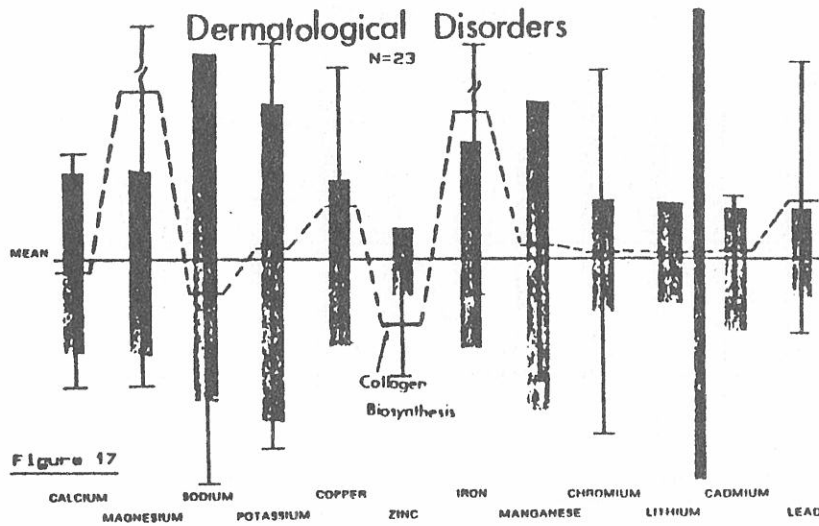


Figure 17

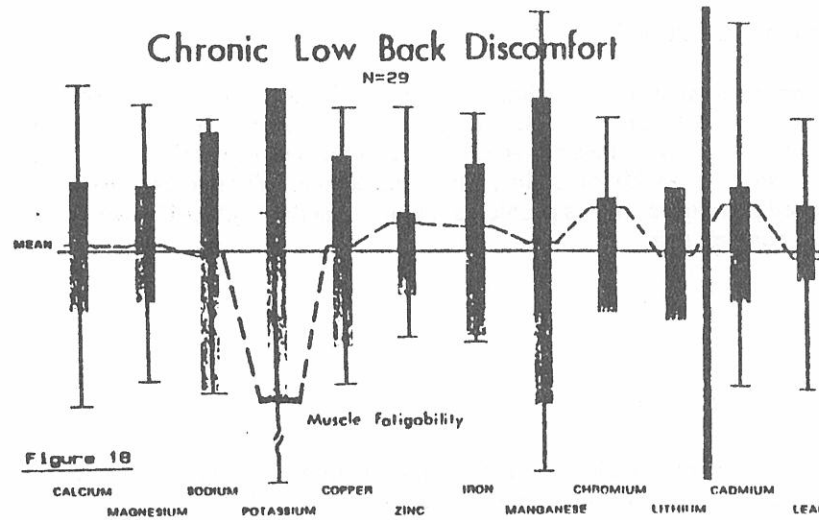


Figure 18

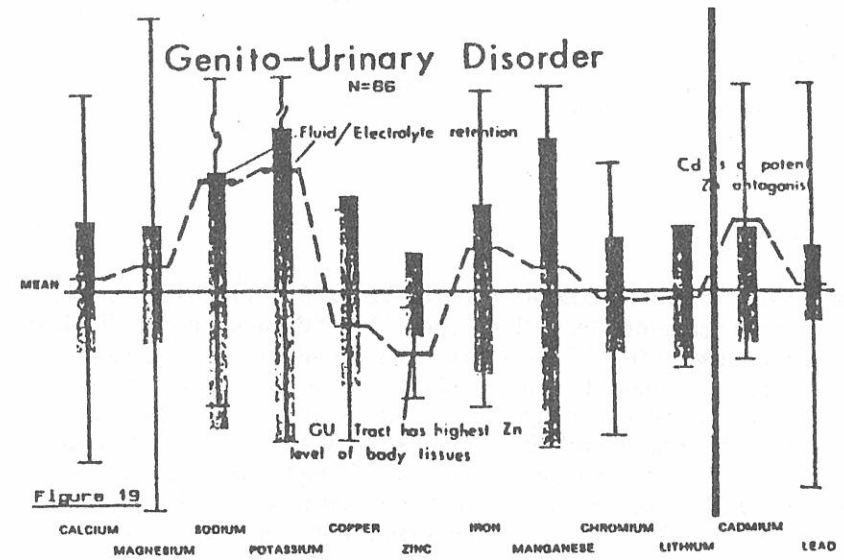
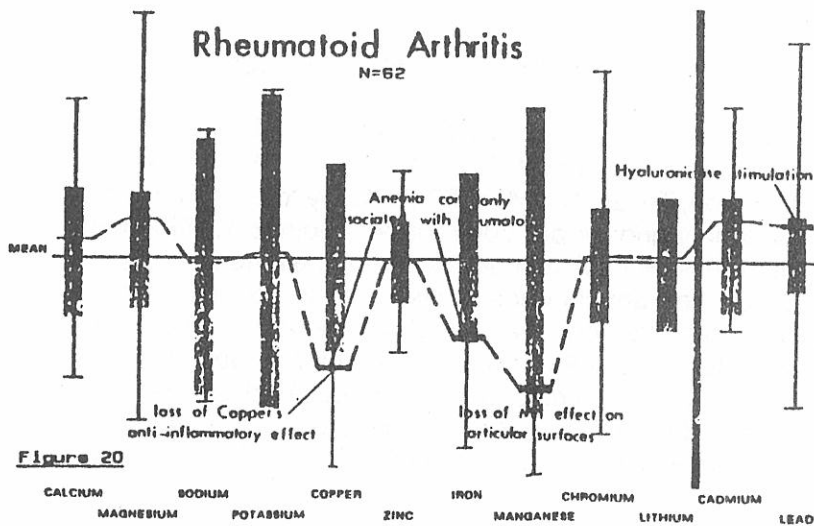


Figure 19

Rheumatoid Arthritis

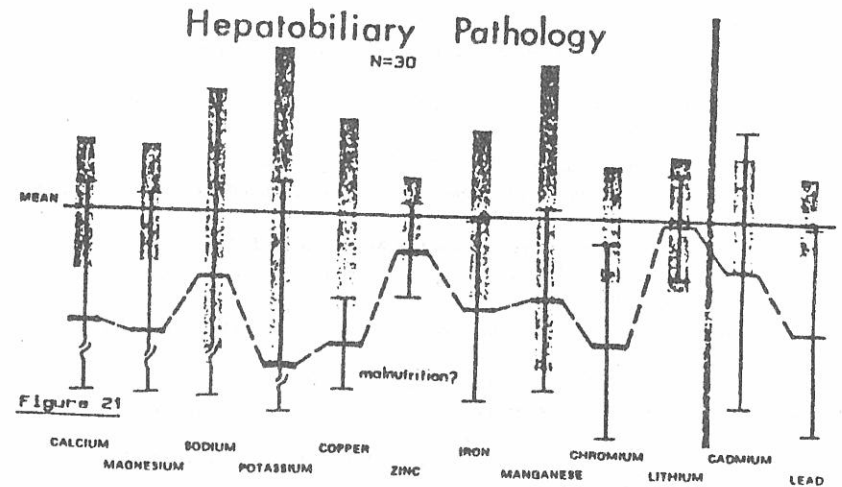
The rheumatoid patient (Figure 20) has several characteristic trace mineral disorders, the most significant being a MANGANESE deficiency. The Mn/Pb RATIO is also usually very depressed in these patients and the interplay of these two elements may be significant in the joint pathology.

Manganese is necessary for the biosynthesis of chondroitin sulphate, a mucopolysaccharide found in the articular surface of joints. Manganese deficient chicks synthesize 30-40% less of this compound than normal chicks (Leach, 1967). There may also be an aberration in the processing of manganese by the rheumatoid patients. When rheumatoid arthritics are fed larger doses of radioactive manganese, there is very little turnover in the manganese stores. Conversely, the normal individual turns over manganese at the rate of 25% of the total body stores daily (Cotzias, et al., 1968). When coupled with the fact that LEAD stimulates the enzyme hyaluronidase, which in turn stimulates the breakdown of hyaluronic acid, a component of the joint lubricating fluid, then the joint has received a double insult and is perhaps more susceptible to auto-immune attack (Blackburn, 1949). The IRON is low in these individuals and most rheumatoids have a chronic, refractory low grade anemia with hemoglobins in the range of 10-11 g%. The low COPPER level may also contribute to the anemia as copper is necessary for the proper utilization of iron (Cartwright & Wintrobe, 1964). Copper may also possess anti-inflammatory action. People have worn copper bracelets for years for their arthritis. Recent evidence suggests that aspirin, upon reaching the stomach, may chelate copper from the mucosal wall and it's the aspirin-copper chelate responsible for the well documented anti-inflammatory action of aspirin. The clinical response in patients treated with 1 mg of copper chelated with 300 mg of aspirin has been promising.



**Hepatobiliary Disorders**

The last profile, shown in Figure 21, is that of hepatobiliary disorders. The pattern is very non-specific, similar to the fatigued patients presented in Figure 12. The pattern is one of generalized malnutrition, with regard to trace elements. The liver is perhaps the organ most sensitive to inadequate nutrition. It comes as no surprise that patients with liver problems are malnourished and would benefit from a diet higher in natural unprocessed foods or by taking supplements.



**Conclusion**

The conclusions have been included in the results section of each individual disorder. One need keep an open mind to any new diagnostic laboratory procedure and realize that neither blood, serum nor any single method of testing is a panacea in and of itself.

**Acknowledgments:**

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