Biochemical Bases for Behavior Disorders in Children

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The purpose of this study was to biochemically differentiate among diagnostic groups of psychotic, neurotic and control children using hair analysis. Forty out of fifty-seven behavior disturbed children were categorized into two groups: psychotic and neurotic (anxious-depressed), based on their behavior patterns on the Child Behavior Checklist (Kracke, 1979). All children in the control group failed to meet the criteria set for the psychotic or neurotic group. A hair sample of each child was analyzed to determine the biochemical levels of 21 elements. The results demonstrated that the psychotic, neurotic and control groups were significantly different in their levels of molybdenum, chromium, cobalt, vanadium, and lead and showed non-significant trends in copper, sodium and manganese. The control group had significantly elevated levels of cobalt and vanadium and significantly less molybdenum and chromium than the psychotic and neurotic groups. The neurotic group mean for lead

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was found to be significantly elevated when compared to the psychotic and control group means. The results suggest that children who demonstrate psychotic and , neurotic behavior patterns have unique biochemical processes which may be related to their disorder.

Recently it has been found that children with learning disabilities when compared to normals had significant differences in their hair element (major, trace elements and toxic metals) content (Pihl and Parkes, 1977). Other studies have demonstrated major element, trace element and toxic metal abnormalities have been associated with multiple behavior disturbances; depressed mood (Baker, 1971); elation and agitation (Bryce-Smith, 1972); psychotic behavior (Pfeiffer, 1978); CNS dysfunction-convulsions (Pfeiffer, 1978); hyperkinesis — irritability (Lesser, 1977); mental confusion (Newmark and Dluhy, 1975); fatigue -weakness (Hambidge, 1974); short attention span (Gordon, 1978). A more detailed account of the behavioral sequelae of major, trace element and toxic metal imbalances are reviewed in Kracke (1980).

What are the major elements, trace elements and toxic metals and what is their biochemical action? The major elements are

composed of substances that make up the essential ionic (electrolyte) environment of the cell: sodium, potassium, calcium and magnesium. These substances serve as structural components to both muscle and nerve cell, as well as having electro-chemical functions (Frieden, 1974). Trace metals by definition (Smith, 1978) are those substances that are essential for normal physiological functioning and whose concentration within the body is less than four grams. They include chromium, cobalt, copper, molybdenum, zinc, iron, selenium, nickel and vanadium. Trace elements have been shown to influence enzyme activity and may well serve to regulate enzymatic activity which in turn influences behavior through numerous biochemical processes. Most notably, those involved in maintaining proper integrity and functioning of the brain, neurotransmitters and neuronal transmission (Rosenbusch and Wever, 1971). Toxic metals are often implicated in behavior and learning disorders because of their pathophysiological effects. These substances when contained within the body at elevated levels disrupt neurochemical and biochemical functioning. If the toxic effects are severe it may relegate neurochemical and biochemical processes to partial or total dysfunction. The toxic metals examined in this study were: lead, mercury, cadmium, aluminum and arsenic. Toxic levels of these substances may render many neuro-biochemical systems inoperable.

Though a plethora of studies and findings document the importance of major and trace elements and toxic metals, no study has investigated the impact of these substances upon children who manifest psychotic and neurotic symptomatology. It is the intent of this study to investigate to what extent, if any, biochemical differences occur amongst groups of psychotic, neurotic, and control children using hair analysis (atomic absorption spectroscopy).

METHODOLOGY Subjects

The total sample was comprised of 37 boys and 20 girls. Twenty subjects each in the psychotic and neurotic groups were so designated out of a total population of 57 by using the Child Behavior Checklist (Kracke, 1980). Subjects in the psychotic and neurotic diagnostic groups were obtained out of special education classes from two local school districts and two local county mental health youth service programs. The 17 subjects of the control group were obtained from two regular classes from a local school district. The classes consisted of second and third graders, and fourth and fifth graders. All subjects met the established age limitations which were inclusive of 7 years 6 months and 12 years 6 months. The mean ages for the psychotic, neurotic and control groups were 8.8 years, 9.8 years and 8.9 years respectively. The ethnic composition of the total sample was 96 percent Caucasion, 3 percent Hispanic and 1 percent Oriental. The annual family income of the psychotic and neurotic subjects ranged from $7,000 to $10,000 and the control subjects annual family income fell between $19,000 and $25,000.

Procedure

Parents of potential subjects were contacted by a letter in which the research project was briefly described. In the letter it was specified that a small sample of hair from the child's head was required. The letter noted that this research was partly funded by a private research grant from Mineralab Incorporated in Hayward, California. After the parents notified their interest in participating in the project, they received a packet of information including a consent form, demographic inventory, Child Behavior Checklist (Kracke, 1979) which they completed and returned. Once the consent form was received from the parents, arrangements were made to obtain the hair sample from the child.

Based on the Child Behavior Checklist (Kracke, 1979) children in the experimental groups were divided into psychotic and neurotic classifications. Those 20 children having the highest psychotic behavior profile scores based on the Child Behavior Checklist (Kracke, 1979) were classified as psychotic, and those 20 having the highest neurotic (anxious-depressed) behavior profile scores were classified as neurotic. Those children not having significantly elevated psychotic or neurotic behavior profiles were not included in the study. No children in the

control group had elevated psychotic or neurotic profiles.

Instrumentation

The Child Behavior Checklist (Kracke 1979) was specifically developed by this author in order to systematically classify behaviorally disturbed children into two broad diagnostic classifications, psychotic and neurotic. Each of the scales contained symptom patterns and behavior that have been commonly associated with psychotic or neurotic symptomatology. The neurotic classification was composed of anxious and depressed symptom subscales. The checklist consisted of 24 behavioral items which were individually rated on their frequency of occurrence for each child. The behaviors on the Child Behavior Checklist were chosen for their objectivity and ease in being observed. The behavioral items were essentially taken from the Burks Behavior Rating Scale, Diagnostic and Statistical Manual of Mental Disorders (Third Edition), the Quay-Peterson Behavior Checklist, and the Devereux Child Behavior (DCB) Rating Scale. The use of these behavioral items by these instruments gives them face validity for their use on the Child Behavior Checklist. (The Child Behavior Checklist and Manual can be obtained from the author.)

The original version of the Child Behavior Checklist consisted of 31 items. This version was given to 40 graduate students in Clinical Psychology in order to establish which items were most indicative of psychotic and neurotic behavior patterns. Any item that did not have an overall agreement among raters of at least 70 percent was excluded from further analyses. A procedure described by Finn (1970) was used to obtain the estimates of reliability for the selected items and diagnostic clustering of symptoms. The 24 items that were eventually used had a mean reliability per item of .87 and an overall reliability of .99. Both of these coefficients were significant at the .001 level.

A test-retest (a week apart) reliability coefficient of the Child Behavior Checklist was found to be .40 which was significant at the $P < .05$ level. An inter-rater reliability coefficient of the scale was found to be .041, this was significant at the $P< .1$ level. Based on these findings the Child Behavior Checklist (Kracke, 1979) is reliable over time and fairly consistent among raters.

Atomic Emission Spectroscopy And Hair Analysis

Atomic emission spectroscopy was employed to assess the biochemistry of the hair samples. Atomic emission spectroscopy is a rather recent development of nuclear physics. This procedure entails removing approximately one gram of suboccipital hair from the subject, having it digested in acid and then analyzed by atomic absorption spectrophotometry. The spectrophotometry measures the distribution of atomic and subatomic particles in the hair. This technique assesses these elements within 3 to 4 percent margin of error of their actual molecular composition (Fassel, 1978). An excellent review, though somewhat esoteric explanation of atomic emission spectroscopy, has recently been reported in **Science** (1978) by Velmer Fassel. The atomic emission spectroscopy was performed at Mineralab Incorporated in Hayward, California, the world's largest mineral analysis laboratory.

Maugh (1978) outlined the use of hair tissue analysis as an adjunctive medical procedure that complements blood serum and urine analysis. He went on to state that hair tissue analysis provides a continuous and historical record of nutritional status and exposure to heavy metal pollutants. Hair tissue analysis has certain advantages over blood serum and urine analysis in that measures of blood serum and urine are dynamic in nature. That is, they both rapidly fluctuate within hours depending on one's physiological requirements and needs (Hopps, 1976). Hair tissue, however, is an exception in that it is a static measure of the prevailing physiological constituents and processes over the past six months (Flynn, 1977).

The following are several advantages of using hair as opposed to blood as a specimen: (1) biologically stable (stores easily, ships well, and will not deteriorate); (2) easy to obtain (non-traumatic and non-intrusive); (3) not subject to rapid physiological changes as are blood and urine; (4) reflects actual systemic (body) mineral levels; (5) easy and accurate to measure; (6) is in

equilibrium with systemic activities and reflects metabolic balance over a period of time (Hopps, 1976).

Numerous researchers have demonstrated the efficiency of hair tissue analysis and its applicability to clinical states (Banta and Markesbery, 1977; Creason, Hinners, Baumgarner and Pinkerton, 1975; Hopps, 1976; Maugh, 1978; Smith, 1978). Maugh (1978) has reported high correlations between chemical concentrations in the hair and internal organs. The hair generally has major and trace element concentrations 10 times higher than those present in serum or urine. Hopps (1976) reviewed the literature and found impressive support that the hair is representative of internal organ and tissue levels of major and trace elements. Klevay (in press) has demonstrated a linear correlation between hair copper levels and liver copper levels in animals. This relationship was found to be statistically significant at the 99.9 percent confidence limits.

Some confusion still remains concerning the relationship between serum and hair tissue levels for the trace elements. However, this controversy is not the focus of this study, and therefore, can not be dealt with in any detail. Bland (1979) writes of this confusion; "Hair tissue levels may more adequately reflect intra-cellular element concentrations whereas serum levels may better reflect membrane transport and extra-cellular phenomena" (p. 27). In conclusion, the biochemistry of the hair is a more stable, reliable and representative medium of the essential elements. These advantages enhance its use in the present investigation exploring the biochemical anomalies in behavior disturbed children. The biochemical elements studied in this investigation were: Calcium, magnesium, phosphorus, sodium, potassium, iron, copper, molybdenum, manganese, zinc, chromium, selenium, lithium, nickel, cobalt, vanadium, lead, mercury, cadmium, aluminum and arsenic.

Statistical Procedures

One-way analyses of variance were computed across the biochemical elements for the diagnostic groups (psychotic, neurotic and control). The student-Newman-Keuls statistic was employed to test the

Table 1

Summary of Analysis of Variance of Biochemical Elements Across Psychotic, Neurotic and Control Groups

Source dfT*	SST	MS B/W	F	P
Calcium	44,426	275 812	.34	n.s.
Magnesium	4,247	54 76	.71	n.s.
Sodium	73,599	2,513	1.98	.15 n.s.
Potassium	19,126	1,269 376 340	1.11	
Iron	378	6.63 6.77	.98	n.s.
Copper	949	45 15	2.85	.07
Molybdenum	.013	.002 .0001	17	.00001
Manganese	.33	.011 .006	2.02	.14
Zinc	2,907	56 51	1.1	n.s.
Chromium	.004	.0004 .0001	5.8	.005
Selenium	.78	.012 .014	.87	n.s.
Cobalt	.0025	.0005 00001	15	.00001
Vanadium	.05	.01 .0006	19	.00001
Lead	31	3.6 .45	7.99	.0009
Mercury	1.73	.039 .03	1.29	n.s.
Cadmium	1.17	.025 .02	1.23	n.s.
Aluminum	114	2.87 2.01	1.43	n.s.
Arsenic	3.68	.055 .066	.83	n.s.

*dfT = 56 for all statistical analyses

significance of the mean biochemical differences. Due to the number of analyses of variances performed, 5 percent or approximately 1 out of 20 could have occurred by chance alone.

Results

A one-way analysis of variance was performed on each of the elements and the results are summarized in Table I.

Based on these data molybdenum, chromium, cobalt, vanadium and lead were found to be significantly different across diagnostic groups. Copper (P< .07), sodium (P < .15), and manganese (P < .14) all demonstrated trends towards differing significantly across groups. The student-Newman-Keuls statistic was used to differentiate among the means of the psychotic, neurotic and control groups for those elements that had significant and near significant F-ratios. Table 2 contains a summary of the findings of the student-Newman-Keuls statistical analysis. The mean differences showed the control group to have significantly elevated levels of cobalt and vanadium and significantly less molybdenum and chromium than either the psychotic and neurotic groups. Finally, the neurotic group mean for lead was found to be significantly elevated when compared to the psychotic and control group means. No differences were found between groups for copper, sodium or manganese.

Table 2

A Summary of the Mean Biochemical Differences Across Diagnostic Groups using the Student-Newman-Keuls Statistic

M	**Psychotic**	**Neurotic**	**Control**
	24.02	37.21	14.01
Sodium			
SD	23.59	52.14	19.90
M	4.07	4.97	1.88
Copper			
SD	4.13	5.00	1.92
M	.06	.07	.04*
Molybdenum			
SD	.016	.012	.005
M	.07	.11	.06
Manganese			
SD	.055	.109	.038
M	.030	.033	.024*
Chromium			
SD	.008	.010	.005
M	.014	.012	.022*
Cobalt			
SD	.005	.003	.007
M	.027	.018	.065*
Vanadium			
SD	.018	.007	.038
M	.995	1.465*	.582
Lead			
SD	.502	.999	.198

* indicates that this mean is significantly different from the other 2 means at the P < .05 level.

In summary, it was found that diagnostic groups were significantly different across the following elements: molybdenum, chromium, cobalt, vanadium and lead; with trends in copper, sodium and manganese. Within group differences were found in each except copper, sodium and manganese. Thus diagnostic groups were found to differ in regards to their biochemical make-up.

Discussion

The present study has demonstrated that controls had significantly elevated cobalt and vanadium levels and depressed molybdenum and chromium levels as compared to the psychotic and neurotic groups. The neurotic group had significantly more lead than the psychotic and control groups.

At the present still little is known regarding the behavioral effects of cobalt, molybdenum, vanadium and chromium. However, the biochemical influence of these substances appears to be more clearly delineated (Pfeiffer and Iliev, 1972). Pfeiffer et al. (1972) have found that cobalt and chromium act to stimulate the brain whereas vanadium and molybdenum act to sedate it. The control group in the present study had increased levels of cobalt and vanadium and decreased levels of molybdenum and chromium when compared to the psychotic and neurotic groups. Thus, the control group had a particular ratio of counter balancing of biochemicals that act as

stimulants and sedatives within the brain. Possibly this particular ratio and arrangement of antagonistic biochemicals may be influential in moderating normal behavior.

Increased levels of lead have been associated with multiple behavioral and biophysical disturbances. Lead as well as disrupting enzymatic and cellular bio-systems causes central nervous system damage (Campbell, Williams and Barltrop, 1970; Chisolm and Barltrop 1979; Gordon, 1978). As a neuro-toxin, lead is known to block neuronal transmission and acetylcholine (Gordon, 1978); decrease by 20 percent the brain levels of dopamine (Sauerhoff and Michaelson, 1973); cause demyelination of the motor-nerve (Campbell et al., 1970; Chisolm et al. 1979); and inhibit calcium-mediated release of neurotransmitters (Granick, Sassa and Kappas, 1978).

As a consequence of the aforementioned bio-physiological anomalies, increased levels of lead may manifest itself behaviorally in aggression, destructiveness, impulsive behavior (Bryce-Smith, 1972); hyperirritability, temper tantrums, isolation, frequent crying, fearfulness, listlessness, loss of affection (Gordon, 1978); and manic-depressive psy-chosis (Stern, 1969). However, most notably lead has been found to be associated with hyperactivity in children (David, 1972; Duva, 1977; Maclssac, 1977; Oliver, 1977; Oliver, Hoffman, Sverd, Clark and Voeller, 1976; Rummo, 1974); and deficits in cognitive and intellectual functioning (Barrett, 1978; Duva, 1977; Maclssac, 1976; Needleman, Gunnoe, Leviton, Reed, Peresie, Maher and Barrett, 1979; Perino and Ernhart, 1977; Rummo, 1975; Sachs, McCaughran, Krall, Rozenfeld and Yongsmith, 1979; Yamins, 1977).

According to data received by the Center for Disease Control, between 3 and 20 percent of all pre-school age children have increased lead absorption. Bryce-Smith (1972) reported that children are more susceptible to lead induced central nervous system impairment than are adults. Lin-Fu (1972) and Hardy (1971) have postulated that current levels of lead pollution in the environment are producing significant brain damage in children.

It is worthy of note that Pihl and Parkes (1977) found a similar biochemical pattern with their control group when compared to children who were learning disabled. In both studies the control group had elevated cobalt and depressed lead and chromium levels as compared to the pathological groups. The elevated lead levels in the pathological groups in both studies may be the primary cause of the behavioral disturbances noted. Quite possibly lead interacts with cobalt and chromium to compromise their action and level of concentration. It seems imperative that environmental levels of lead be monitored in order to prevent toxic or behavioral manifestations of overexposure. As well, children who may be a high risk for elevated lead levels (toxicity) should be screened and evaluated as a course of treatment.

Copper, sodium and manganese all demonstrated trends of being elevated in the psychotic and neurotic diagnostic groups. Though nothing conclusively can be stated some speculation is warranted in light of the significance of these biochemical substances on behavior. Increased levels of copper and ceroloplasmin (copper containing enzyme) inhibits the functioning of the enzyme hydroxytryptophane decarboxylase which decreases the production of the neurochemical transmitter serotonin. A state of hypercopremia (elevated blood copper) appears associated with depression and perceptual disturbances in schizophrenics (Pfeiffer and Iliev, 1972). Lesser (1977) has noted that excess copper can cause depression, feelings of alienation, irritability, and even paranoia. Pfeiffer (1978) has documented that elevated copper levels could be associated with paranoid and hallucinatory schizophrenia, autism, childhood hyperactivity, and depression.

Elevated sodium levels (if extreme, hyper-natraemia) may change the electrical excit-ability of neurons in the brain and thus alter neuronal transmission and the production of monoamines (norepinephrine, serotonin) (Becker, 1974; Faragella, 1974; Mendels, Frazer and Secunda, 1972). The following symptoms are associated with hypernat-raemia: irritability, sleeplessness, convulsions, confusion, brain damage and hyper-kinesis (Chambers, 1975; Feig and McCurdy, 1977; Macaulay and Watson, 1967; Morris-Jones, Houston and Evans, 1967; Park, Meacham and Netsky, 1976).

The production of the neurochemical transmitter acetycholine is dependent upon enzymes that require manganese. Banta and Markesbery (1977) found that manganese intoxication mimicked schizophreniclike psychoses, dementia, and impaired higher cortical functioning. Possibly some causal link exists between these elements and psychotic and neurotic behavior patterns in children. These disturbances in behavior may result when the proper regulatory systems fail or when imbalances occur. However, these biochemical disruptions may only be the remnants of a deeper or more complex biochemical disruption. Unfortunately, except on a rather rudimentary level it is difficult to speculate the direct action of these minute biochemical elements and toxins on neurochemical and brain-behavior relationships. The direct action and functioning of many of these biochem-icals is simply not known at this time.

It should be acknowledged that possibly some of the aforementioned findings could have resulted from some unavoidable methodological flaws that were incurred. In this study there were a disproportionate number of males and females across the experimental and control groups. In the diagnostic groups there was a higher percentage of males to females than in the control group. There was unequal representation across the diagnostic and control groups in regards to annual family income. The control group had a higher percentage of higher income families than the diagnostic groups. Finally, it was found that nutritional status was unequally represented across the psychotic, neurotic and control groups. Essentially, the control group had a larger percentage of its members having nutritionally adequate diets as compared to the psychotic and neurotic groups. These inequalities in the subject sample certainly compromise the implications and generalizability of this study. Well controlled studies are needed in order to either substantiate or refute these findings.

It is important to keep in mind that due to the nature of this study causality cannot be directly inferred from the data. Further research is needed in order to empirically investigate the exact mode of action of these biochemical substances and toxins on disturbed behavior.

References

BAKER, E.F.W.: Sodium Transfer to Cerebrospinal Fluid in Functional Illness. Canadian Psychiatric Association Journal 16, 2,167-170,1971.

BANTA, R.G. and MARKESBERY, W.R.: Elevated Manganese Levels Associated with Dementia and Extrapyramidal Signs. Neurology 27,213-216,1977.

BARRETT, R.P.: Auditory-Visual Integration, Intelligence, and Reading Achievement in Normal and Subclinically Lead Poisoned Children. Doctoral Dissertation, University of Pittsburgh. Dissertation Abstracts International 39, 1451B-1452B, 1978. University Microfilms No. 7816777.

BECKER, J.: Depression: Theory and Research. New York: John Wiley and Sons, 1974.

BLAND, J.: Hair Tissue Mineral Analysis: An Emergent Diagnostic Technique. Washington. Bellevue Redwood Medical Laboratories, Inc., 1979.

BRYCE-SMITH, D.: Behavioral Effects of Lead and Other Heavy Metal Pollutants. Chemistry in Britain 8, 6, 240-243, 1972.

CAMPBELL, A.M.G., WILLIAMS, E.R. and BARLTROP, D.: Motor Neurone Disease and Exposure to Lead. Journal of Neurology, Neurosurgery and Psychiatry 33, 877-885, 1970.

CHAMBERS, T.: Hypernatremia: A Preventable Cause of Acquired Brain Damage. Developmental Medicine and Child Neurology 17, 91-94, 1975.

CHISOLM, J.J. and BARLTROP, D.: Recognition and Management of Children with Increased Lead Absorption. Archives of Disease in Childhood 54, 249-262, 1979.

CREASON, J.P., HINNERS, T.A., BAUMGARNER, J.E. and PINKERTON, C: Trace Elements in Hair as Related to Exposure in Metropolitan New York. Clinical Chemistry 21, 603, 612, 1975.

DAVID, O.: Lead and Hyperactivity. Lancet 2,900-903, 1972.

DUVA, N. A.: Effects of Asymptomatic Lead Poisoning on Psychoneurological Functioning of School-age Urban Children: A Follow-up Study. Doctoral Dissertation, Fordham University, 1977. Dissertation Abstracts International 38, 168A, 1977. University Microfilms No. 7-14,863.

FARAGELLA, F.F.: Mineral Metabolism. In: Flach and Draghi (eds.), The Nature and Treatment of Depression. New York, John Wiley and Sons, 1975.

FASSEL, A.V.: Quantitative Elemental Analysis by Plasma Emission Spectroscopy. Science 202, 13, 183-191, 1978.

FEIG, P.V. and McCURDY, M.G.: Hypernatraemia. New England Journal of Medicine 297, 1444-1452, 1977.

FINN, R.H.: A Note on Estimating the Reliability of Categorical Data. Educational and Psychological

Measurement 30, 71-76, 1970.

FLYNN, A.: Hair Elemental Analysis as a Measure of Mineral Status. Journal of Applied Nutrition 29,167-173,1977.

FRIEDEN, E.: The Evolution of Metals as Essential Elements. In: M. Friedman (Ed.), Protein-Metal Interactions. New York, Plenum Press, 1974.

GORDON, G.F.: Insight Preventive Medicine. Osteopathic Medicine 3,5,5-9,1978.

GRANICK, J.L., SASS, S. and KAPPAS, A.: Some Biochemical and Clinical Aspects of Lead Intoxication. Advances in Clinical Chemistry 20,287-339, 1978.

HAMBIDGE, K.M.: The Clinical Significance of Trace Element Deficiencies in Man. Proceedings of the Nutrition Society 33,249-259,1974.

HARDY, H.L.: Review Article: Lead as an Environmental Poison. Clinical Pharmacological Therapeutics 12, 982-1003,1971.

HOPPS, H.C.: The Biologic Basis for Using Hair and Nail for Analyses of Trace Elements. Trace Element Substances in Environmental Health 8, 174-188, 1976.

KLEVAY in Press.

KRACKE, K.R.: Child Behavior Checklist. Unpublished Manuscript. California School of Professional Psychology, 1979.

KRACKE, K.R.: Biochemical Abnormalities Related to Childhood Depression and Behavior Disorders. Unpublished Doctoral Dissertation, California School of Professional Psychology, Fresno, 1980.

LESSER, M.: Mental Health: Its Not Just in our Heads. Select Committee on Nutrition and Human Needs. Washington, D.C.: U.S. Government Printing Office, 1977.

LIN-FU, J.: Undue Absorption of Lead Among Children — A New Look at an Old Problem. New England Journal of Medicine 286, 702-710, 1972.

MacAULEY, D.: Hypernatraemia in Infants as a Cause of Brain Damage. Archives of Disease in Childhood 42,485-501,1967.

MacISSAC, D.S.: Learning and Behavioral Functioning of Low Income, Black Preschoolers with Asymptomatic Lead Poisoning. Doctoral Dissertation, Fordham University, 1976. Dissertation Abstracts International 37,2747B, 1976.

MAUGH, T.H.: Hair A Diagnostic Tool to Complement Blood Serum and Urine. Science 202, 1271-1273,1978.

MENDELS, J., FRAZER, A. and SECUNDA, S.K.: Intra-erythrocyte Sodium and Potassium in Manic/ depressive Illness. Biological Psychiatry 5, 2, 165-171,1972.

MORRIS-JONES, P.H., HOUSTON, I.B. and EVANS, R.C.: Prognosis of the Neurological Complications of Acute Hypernatremia. Lancet 2,1385-1390,1967.

NEEDLEMAN, H.L., GUNNOE, C, LEVITON, A., REED, R., PERESIE, H., MAHER, C. and BARRETT, P.: Deficits in Psychologic and Classroom Performance of Children with Elevated Dentine Lead Levels. The New England Journal of Medicine 300,13, 689-895,1979.

NEWMARK, S.R. and DLUHY, R.G.: Hyperkalemia and Hypokalemia. Journal of the American Medical Association 231,6, 631-633,1975.

OLIVER, D.J., HOFFMAN, S.P., SVERD, J., CLARK, J. and VOELLER, K.: Lead and Hyperactivity. American Journal of Psychiatry 133,10,1155-1158, 1976.

OLIVER, D.J.: Lead and Hyperactivity. Journal of Abnormal Child Psychology 5, 4,405416,1977.

PARK, B.E., MEACHAM, W.F. and NETSKY, M.G.: Hypernatraemia and Brain Disorders. Journal of Neurosurgery 44, 409420,1976.

PERINO, J. and ERNHART, C.B.: The Relation of Subclinical Lead Level to Cognitive and Sensorimotor Impairment in Black Preschoolers. Journal of Learning Disabilities 7,10,616-620,1977.

PFEIFFER, *C.C.:* Zinc and Other Micro-nutrients. Connecticut, Keats Publishing, Inc., 1978.

PFEIFFER, C.C. and ILIEV, V.: A Study of Zinc Deficiency and Copper Excess in the Schizophrenias. In *C.C.* Pfeiffer (Ed.), Neurobiology of the Trace Metals. New York, Academic Press, 1972.

PIHL.R.O. andPARKES.M.: Hair Element Content in Learning Disabled Children. Science 198, 204-206, 1977.

ROSENBUSCH, J.P. and WEVER, K.: Localization of the Zinc Binding Site of Aspartate Transcarbo-moylass in the Regulatory Subunit. Proceedings National Academy of Science. United States Printing Office 68,1019,1971.

RUMMO, J.H.: Intellectual and Behavioral Effects of Lead Poisoning in Children. Doctoral Dissertation, 1974. Dissertation Abstracts International 35,3035B, 1974. University Microfilms No. 74-26, 930.

SACHS, H.K., McCAUGHRAN, D.A., IRALL, V., ROZENFIELD, I.H. and YOUNGSMITH, N.: Lead Poisoning Without Encephalopathy. American Journal of Diseases of Children 133, 786-790,1979.

SAVERHOFF, M.W. andMICHAELSON, LA.: Hyperactivity and Brain Catecholamines in Lead-exposed Developing Rats. Science 182,1022-1024,1973.

SMITH, L.H.: Trace Elements. The Western Journal of Medicine 128,3, 223-227,1978.

STERN, H.: Lead, Lithium and Manic-depressive Psychosis. The Chicago Medical School Quarterly 28, 87-95,1969.

YAMINS, J.G.: The Relationship of Subclinical Lead Intoxication to Cognitive and Language Functioning in Preschool Children. Doctoral Dissertation, Hofstra University, 1976. Dissertation Abstracts International 37, 4176B, 1977. University Microfilm No. 77-2802.

BOOK REVIEW

Living And Working With Schizophrenia
Seeman, M.V., Littmann, S.K., Plummer, E., Thornton, J.F., Jeffries, J.J.
University of Toronto Press, Toronto, Ontario 1982,146 pages.

In the past few months, this reviewer has received two books, one American, one Canadian, aimed at promoting greater understanding of schizophrenics among those who work and interact with them. While two books do not constitute a significant trend, it is to be hoped that such informational discourses will become the leitmotiv of the 1980s. For it is only through enlightenment that the general populace will begin to treat schizophrenics with equity and empathy; two elements mandatory for the integration of the patient into his community.

As with Mona Wasow's **Coping With Schizophrenia** (1982), a "survival manual" written in a similar vein, **Living And Working With Schizophrenia** is alternately a treasure and a disappointment. Indeed, the book does provide an electrifying glimpse of the psychotic's netherworld, by noted schizophrenic authoress Margaret Gibson. And, in its question-and-answer format, it supplies valuable information concerning: a clear description of schizophrenia; the belief that the illness may have a biochemical etiology; the justifiable concern that schizophrenics have been unfairly maligned; inpatient and outpatient treatment procedures; family therapy (though, like Wasow, the authors deny the concept of the schizogenic family); an erudite section on pharmaco-therapy; advice on how family and friends can aid the schizophrenic; knowledge regarding the schizophrenic in the work-place; suggestions on how to change public attitudes surrounding the malady; personal accounts by schizophrenics and the parents of schizophrenics; plus a reasonably comprehensive (international) index of organizations and self-help groups upon which the families of schizophrenics can rely for assistance.

Living And Working With Schizophrenia is the literary offspring of five clinicians from Toronto's renowned Clarke Institute of Psychiatry. It was their dedicated involvement with the institute's Aiding Families of Schizophrenics program, that resulted in their being awarded the American Psychiatric Association's "Certificate of Significant Achievement" last year.

This book has considerable overall value as an introduction to schizophrenia and Canada's mental health system. Yet, it is seriously deficient in one crucial area. Since its authors are traditional therapists espousing traditional treatment modalities, ortho-molecular supporters will not find any surprises. In fact, this text devotes only one (unfavorable) paragraph to megavitamin therapy. Nutrition as an etiological and therapeutic factor is similarly glossed over.

The writers claim: "Families should be wary of novel, untested treatments because they may delay the beginning of effective treatment."

There is nothing untested concerning the fundamental tenets of orthomolecular psychiatry. But how do we convince the orthodox of that?

G. Charles Brown