The Reflection of Hypoglycemia and Alcoholism on Personality: Nutrition as a Mode of Treatment

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Our era has seen formidable and unprecedented increase in mental illness. It has become a tidal wave which we have been powerless to stem. Alcoholism has played a major role—alcohol has the ability to cause acute and chronic psychosis. Henri Baruk. 1977.

Early reviews on the alcoholic personality (Armstrong, 1958) generally failed to establish any constellation of personality traits in alcoholics that would predispose a person to alcoholism. Sutherland et al. (1950) proposed that "No satisfactory evidence has been discovered that justifies a conclusion that persons of one type are more likely to become alcoholics than persons of another type." Lisansky (1960) later concluded that "we cannot reject the idea that personality factors play a very significant role in determining who will become an alcoholic and who will not." Barnes (1979) suggests that "there does appear to be a clinical alcoholic personality." He explains that, no doubt, this personality pattern exists as a cumulative result of a prealcoholic personality and the effects of a person's drinking history on that personality pattern. In contrast, the neurotic characteristics of alcoholics seem to be more a result of the disorder than of a prealcoholic personality trait (Krammeier et al., 1979).

In commenting on the psychiatric aspects of alcoholism, Dr. Ruth Fox (1965), Medical Director, National Council on Alcoholism, Inc., suggests that alcoholism is a chronic behavioral disturbance. Psychological characteristics found with a battery of tests on 300 consecutive private patients of Dr. Fox's showed the following character traits: "...inner battle between passivity and aggression, low frustration tolerance; inability to endure anxiety or tension; feelings of isolation, devaluation of self-esteem, sometimes with overcompensation; undue sensitiveness; impulsive; repetitive acting out of conflicts, masochistic; self-punitive behavior and extreme narcissism or exhibitionism, strong sense of guilt, hostility, either overt or covert; strong dependent needs; marked rebellion;. . ."

Dr. Carlton Fredericks (1976) tells us that the brain and nervous system are exquisitely sensitive to disturbances of body chemistry which may not noticeably affect other organs, and that a chronic defect in the utilization of sugar in the body may not cause a single physical symptom—though it often does, but it can make you claustrophobic or

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a hypochondriac, fill you with obsessive and unbased fears, or prod you into alcoholism or asthma.

There is a body of knowledge, admittedly not large but exciting, in both lower animals and man. which suggests that dietary factors might well represent some of the most serious primary proneness factors in the development of alcoholism and the alcoholic syndrome (Cheraskin and Ringsdorf. 1971). The etiology of malnutrition in alcoholics is quite complex. Dr. Abram Hoffer (1978) suggests that a number of physical diseases, depression, anxiety states, alcoholism and other addictions are the end product of quantity ingestion of sucrose. Dr. Roger Williams (1971) suggests that it is quite possible that malnutrition develops as a forerunner of alcoholism, and that it is only when malnutrition of the brain cells becomes severe that true alcoholism appears. He positively asserts that "no one who follows good nutrition practices will ever become an alcoholic." Alcohol may, if taken in large enough quantities, actively damage the brain cells as well as deprive the brain cells of the necessary items in the nutritional chain of life.

Dr. Williams expresses the idea that alcohol consumption, at high levels, undoubtedly acts in several ways to damage brain cells —stopping blood flow; direct or indirect poisoning of the brain cells; deprivation of the brain cells of minerals, amino acids and vitamins by substituting naked calories for good food. "Whatever the mechanisms are. the effect is the same. The brain cells are impaired and they die off with greater rapidity when the alcohol level in the blood is allowed to remain high." Dr. Williams suggests that from these facts it seems a reasonable deduction that alcoholism probably results from an impairment of the cells in the appetite-regulating mechanism in the hypothalamus region of the brain. He explains that in individuals who are prone to become alcoholics, these cells are vulnerable and may become so seriously damaged that the sight of food is nauseating and only alcohol has appeal.

Dr. Carlton Fredericks (1976) suggests: "All alcoholics are hypoglycemic, for it is an inevitable result of substituting whisky for food. Some alcoholics begin by becoming hypoglycemic, and at the point where the low blood sugar would ordinarily cause a craving for sweets, they pervert the craving into an appetite for alcohol. That' group in the alcoholic population can be 'cured' of alcoholism by adopting and staying on the hypoglycemic diet. The heavy consumer of sweets who becomes an alcoholic is suspect of being in this group. So is the drinker who, when drying out, eats large amounts of candy."

Kepler and Moersch (1937) have described the symptoms of hypoglycemia as follows: "In attacks of any severity the attitude and general behavior of the patient are always disturbed. Any one of the following mental states, to mention the more common ones, may dominate the clinical picture: apathy, irritability, restlessness, fatigue, anxiety, incorrigibility, negativism, automatic behavior, somnambulism, confusion, excitement, disorientation, 'drunken behavior', fugue states, unconscious attacks, delirium, mania, stupor, coma. The motor activity may be decreased or increased. Speech is distorted: there may be garrulity, dyparth-ria or even aphasia. Emotional instability ranges from all forms of anxiousness to querulousness and violence. The character of thinking becomes confused and sluggish: the patient may be delirious. The trend of thought may remain within reasonable bounds, but obsessions, compulsions and even hallucinations or delusions frequently may be present. The mental group becomes distorted, the patient may become disoriented as to time, place and persons. There is loss of memory for events and the patient does not remember the attack. The mental symptoms may be associated with neurological disorder of varying type, as motor retardation or convulsive attacks of tonic or clonic type."

The behavioral disorders of hypoglycemia are quite varied; they are often misdiagnosed as some neurological or emotional disorder—hypoglycemics are often labeled as neurotics, psychotics or hypochondriacs. Doctors Cheraskin and Ringsdorf (1968) also note that the hypoglycemic symptoms

include anxiety, irritability, fatigue, mental confusion and uncontrolled emotional outbursts.

Dr. Roger Williams (1977) assures us: "That personality differences have such a firm, unquestionable basis is not however generally appreciated." Likewise, the similarity of the personality traits of the hypoglycemic and the "sober" alcoholic have largely been ignored. Following is a comparison of the traits commonly reported by the "sober" alcoholic and the hypoglycemic:

Dry Drunk	Hypoglycemia and Allergy
Irritability	Irritability
Depression	Depression
Aggressiveness	Aggressiveness
Insomnia	Insomnia
Fatigue	Fatigue
Restlessness	Restlessness
Confusion	Confusion
Desire to Drink	Desire to Drink
Nervousness	Nervousness

Worden and Rosellini. 1980

Doctors Hoffer and Osmond (1968) give us a comprehensive look at the personality of the alcoholic who is also hyperglycemic. "Some alcoholics long noted that although dry, they remained unhappy, tense, depressed or in many ways "neurotic." Some would remain dry for a while and then, out of sheer desperation, return to drink. It made sense that many of this group might be suffering from a biochemical malfunction instead of from ordinary varieties of neurosis, as was previously supposed."

To maintain a precarious balance is what our total being strives for. For example, if the concentration of sugar in the blood is too high, the blood will draw fluid from the tissues: this will dehydrate the cells and dilute the blood. If untreated, diabetes will develop. On the other hand, if blood sugar is too low for a prolonged period, the cells will not receive enough food or fuel and we have hypoglycemia (from the Greek, meaning "low sugar in the blood"). Our delicate balance of blood sugar is maintained by internal mechanisms. A group of cells of the islets of Langerhans (the 'alpha cells') in the pancreas secrete the hormone glucagon when there is a slight drop in the blood's normal level. This spurs the liver to release stored sugar. The pituitary gland secretes STH (somatotropic hormones), which keep the sugar from entering muscle and fat cells and conserve sugar for the nervous system, which absolutely requires it.

The adrenal medulla secretes adrenaline if blood sugar drops below a critical level. The secretion of glucagon, STH and adrenaline switches off as soon as the blood sugar level rises to a point above normal. The liver stops issuing sugar and rapidly starts absorbing it. The entry of glucose into muscle, liver and fat cells is promoted by a second group of cells (the 'beta cells') in the islets of Lang-erhans, which secrete the hormone insulin. The process is reversed if blood sugar becomes too low. Since hypoglycemia is low blood sugar and diabetes is high blood sugar they would seem to be opposites. However, it is more accurate to say that hypoglycemia and diabetes are sister illnesses which, in some instances, have a common pathway of development (Poulos, Stoddard and Car-ron, 1976). Hypoglycemia may be a forerunner of diabetes in some cases but all hypoglycemics are not prediabetic.

"Hypoglycemia," says Dr. Harold Harper (1978), "is an 'early warning system' for all the chronic degenerative diseases." Dr. Harper labels the syndrome Glucose Metabolism Dysfunction GMD).

One must dichotomize hypoglycemia into two main types—fasting and reactive or functional—in considering the etiology of hypoglycemia. Fasting hypoglycemia can be traced to some specific organic defect; some of the known causative agents are endocrine diseases, liver diseases, pancreative islet-cell tumors and hyperplasia. Most cases of hypoglycemia are of the functional type (with which we shall be concerned) which occur as a reaction to the ingestion of food. This suggests that diet may be the cause of functional hypoglycemia.

Most hypoglycemics have a ravenous appetite for sweets. Since the overactive pancreas—which spurs the desire for sweets— will be restimulated when sugar is eaten, this illustrates an aberration of the 'wisdom of the body.' Characteristic of a group of alcoholics is this same desire for sweets; while sober they consume unbelievable quantities of candy, cookies and sugar in their coffee. The craving for sweets is a clue to low blood sugar. Meals are often made up of "junk"

foods that are high in refined carbohydrates, fats, and salt: low in high quality protein, and devoid of fresh fruits and vegetables and whole grains. This type of diet can lead to blood sugar instability in susceptible individuals and, indeed, it is claimed upwards of 95 percent of alcoholics suffer from low blood sugar (Meiers. 1973).

In response to a rapid fall in blood sugar, hypoglycemia produces the symptoms which result from the production of adrenaline. These symptoms include: sweating, weakness, rapid heart beat, inner trembling and hunger. When hypoglycemia appears in response to a slow fall (over a period of several hours) of blood sugar the symptoms include: mental confusion, blurred vision, headache, double vision, incoherent speech and sometimes convulsions. A variety of psychological and neurological symptoms may occur if hypoglycemia becomes chronic; one or all of the following could be included: personality changes, emotional instability, fatigue, suicidal intent, phobias, nervousness, depression, insomnia, antisocial behavior, mania, irritability, delirium, stupor, anxiety, "drunken behavior" and negativism.

For the orthomolecular practitioner, nutrition is a primary consideration in the treatment of hypoglycemia; likewise, it is the primary mode of treatment for alcoholism. The orthomolecular practitioner attempts to treat the hypoglycemic and the alcoholic by the provision of the optimal molecular environment for the mind, especially the optimal concentration of substances normally present in the human body.

Dr. Carlton Fredericks (1976) suggests that the necessary treatment for hypoglycemia, other than reducing sugar intake to the lowest level possible, consists of: "1. Nutritional help for the liver. . . (2) Relief for the pancreas from constant overstimulation by excessive intake of sugars and starches. . .(3) With the intake of sugar greatly reduced, and the starches held down to 60 grams daily—more for some patients, and sometimes less for others—we raise the protein intake—from meat, fish, fowl, eggs, cheese, milk and dairy products, and the intake of polyunsaturated (vegetable) fats. .
. (4) As important as the composition of the meals is their timing. Low blood sugar is most easily controlled with six small, rather than three large, meals daily. In each of these some protein is taken. (5) The diet is supplemented with multiple vitamins, multiple minerals and vitamin B Complex concentrate. Special purpose foods high in vitamins and minerals are also used: wheat germ, brewer's yeast and desiccated (dried) liver."

Dr. Harper suggests a modified high protein—low carbohydrate treatment diet which is followed for the first three months. Subsequently, he permits a gradual reintroduction of carbohydrate foods as tolerated.

The prime objective of the high protein — low carbohydrate diet is to minimize swift ascents and consequently sudden drops in blood glucose. It is necessary for the hypoglycemic and the alcoholic to eat six small meals a day with protein at each meal. This diet helps to regulate blood sugar. While medical men were neglecting the concept of hypoglycemia a few pioneers—Portis, Conn, Salzer, Rosenberg, Seltzer, Tintera, Nittler and many others—entered the arena and demonstrated the devastating effect of hypoglycemia on mind and body, and the almost miraculous recoveries which followed a high protein diet restricted in sugar and starch.

The alcoholic is among the individuals who have high nutritional risk—risks of protein energy malnutrition and vitamin depletion. Chronic protein-energy malnutrition characterized by wasting is caused by inadequate diet, maldigestion, malabsorption and alcoholic liver disease (Blackburn and Bist-rian, 1977). The vitamin requirements of alcoholics exceed those of nonalcoholics. Multiple causes of vitamin depletion may coexist in individual alcoholics, including dietary insufficiency, malabsorption, hyperex-cretion, and impaired synthesis of the active or coenzyme form of the vitamin (Fenerlein, 1977). Alcohol damage to the gut reduces absorption of some vitamins, including fola-cin and thiamin (Sinclair, 1972; Tomasulo et al., 1968). Alcohol damage to precursor blood cells in the bone marrow increases the need for folacin, vitamin B6 and vitamin

B12 (Hines, 1975). It has been shown by Eichner (1973) that if alcoholic subjects are placed on a low folate diet they will develop megaloblastic hemopoiesis more rapidly than nonalcoholic subjects. Chronic alcoholics have depleted body folate stores. Roe (1978) found that the hemopoietic response was suppressed in those patients who were folate deficient and also receiving alcohol. This effect could be reversed by eliminating alcohol. Tomasulo (1968) found a significant impairment of thiamin absorption in the alcoholic. The Wernicke-Korsakoff syndrome, peripheral neuritis and pellagrous psychosis are neurological disorders associated with primary or conditioned nutritional deficiencies in alcoholics(Fenerlein, 1977). In Wernicke-Korsakoff (the most common) brain damage is due to thiamin deficiency. The acute state of the Wernicke-Korsakoff syndrome, that is, Wernicke's encephalopathy, can be effectively treated with intravenous thiamin. In the early stages, the greatest nutritional risk is that the disease will not be recognized and the patient may develop an irreversible chronic brain syndrome characterized by memory loss and an inability to function independently (Victor, Adams and Collins. 1971: Riggs and Boles, 1944, 1945).

It is known that deficiencies of the neurotropic vitamins Bl, B6, and B12 play a role in the development of certain psychotic states: for example, the deficiencies of vitamin Bl in Wernicke-Korsakoff psychosis (Freedman, Kaplan and Sadovic. 1975) and some depressive states (Dickerson and Lee, 1978). Alcoholics are often depleted in folic acid, ascorbic acid, vitamin B6. vitamin B12, magnesium, zinc and often quite depleted in protein (Frederick. 1976).

Intracellular magnesium depletion and hypomagnesium occur commonly in chronic alcoholics and appear to arise through the combined effects of poor diet and hyperexcretion of magnesium in the urine (Lim and Jacob, 1972A). Magnesium deficiency is associated with neuromuscular dysfunction characterized by tetany, seizures, ataxia, muscle weakness, tremors, behavioral disturbances and lowered magnesium levels in the serum. Hypomagnesemia combined with alkalosis are believed to be the factors which induce seizures and other symptoms of delirium tremens associated with alcohol withdrawal (Victor. 1973).

Chronic alcoholics often experience night blindness. The night blindness responds to administration either of vitamin A or vitamin A and zinc. Zinc stimulates synthesis of ret-inol binding protein and is also necessary for optimal activity of alcohol dehydrogenase activity in converting retinol to retinal (Russell et al., 1978).

The orthomolecular treatment for alcoholism requires the same high protein-low carbohydrate diet—as for hypoglycemics'— with megadoses of vitamins and minerals, especially ascorbic acid, niacin, vitamin B6, vitamin Bl, vitamin E, a high potency multivitamin and mineral, a high potency B complex, L-glutamine and sometimes B12 injections.

Dr. Williams has demonstrated that alcoholics who are treated with super nutrition may spontaneously stop drinking—or even recover the ability to take a single drink and stop. Doctors Hoffer and Osmond recommend a diet devoid of junk foods and, in the appropriate instances, a regime of nutritional supplements. Dr. Williams has repeatedly noticed that nutritional support reduces the physiologic craving for alcohol. He recommends a high-protein diet supplemented with therapeutic amounts of the available vitamins and minerals. Dr. Williams made the following assertion: "It is our opinion that in the great majority of individual alcoholics, the practical elimination of alcoholic craving can be assured provided the recommendations which we have made are followed. Whether they are followed is a question which must be answered separately in each individual case."

Dr. William Shrive (1965), looking at the problem of alcoholism in a broad biological perspective, became interested in gluta-mine—an amino acid in proteins but often destroyed when proteins are broken down. He argued that this substance might have something to do with alcoholism. Dr. Aaron Prigot and co-workers (1962) found that intravenous amino acid supplements consistently

reduce the morbidity, severity of seizures and the necessity for ancillary sedation in patients with delirium tremens. These investigators explain that: ". . .amino acid preparations were employed prophylactically with a high degree of success to forestall delirium in patients whose clinical status or low amino acid levels indicated their susceptibility. Assay of serum from these patients, by two dimensional paper chromatography, revealed that depletion of amino acids was characteristic of delirium tremens. The values for the amino acids reverted to higher levels as the clinical manifestations of delirium abated following the administration of amino acid medication."

Two of the essential amino acids, leucine and isoleucine, are involved in the etiology of pellagra and may be involved in alcoholism since pellagra is a neurological disorder associated with nutritional deficiencies in alcoholics. Pellagra is caused by a monotonous diet which depends primarily on corn. Corn is low in L-tryptophane. low in vitamin B3 which is present in a tightly bound form from which the human body can extract little, and is too rich in leucine compared to isoleucine. Leucine increases the loss of vitamin B3 into the urine. Dr. Hoffer (1980) explains that pellagrins made normal by giving them B3 quickly become psychotic by giving them leucine. If isoleucine is given, the psychosis is promptly reversed; isoleucine shuts off the loss of B3. Dr. Hoffer asserts that the combination of isoleucine and vitamin B3 would be powerful—the isoleucine would enhance the effect of the vitamin so that lower doses could be given.

At a large prestigious hospital in New York City, the author treated, individually, a group of physicians who had been referred by their peers because of excessive drinking. The orthomolecular approach to treatment was quite successful with these patients who were alcoholics with hypoglycemia symptoms. I had read the papers of Hoffer, Williams, Pauling, Osmond and others in the field of orthomolecular psychiatry. Following their lead I treated the nutritional deficiencies of these men with the prescription of a diet high in protein, low in carbohydrates and megadoses of certain vitamins and minerals. In accordance with Dr. Williams' "biochemical individuality," each regime was unique for that person. I gave these men megadoses of vitamin C complex with bioflavanoids (8 to 12 grams daily in divided doses); 2 to 4 grams of niacin (time release capsules 400 mg); a super B complex (100 mg) twice and sometimes three times a day: a high potency multivitamin and mineral (Willner's Super-Vite 75); L-glutamine (not glutamic acid) 2 to 3 grams a day; Tryptophane 1500 mg to 3,000 mg each day, taken at bedtime; magnesium and calcium: Bl, 500 mg to 1,500 mg each day.

This nutritional support reduced the craving for alcohol in every case where the recommendations were completely followed. These men were able to "dry out" without the devastating effects of hallucinations— the imaginary pink elephants of the alcoholic with delirium tremens. To give them support and to encourage them to stay on their regime I saw these patients two to three times a week at first—it was very important that they have success immediately. In several weeks many of them found they had lost their craving for alcohol. Perhaps this was because they did not substitute their craving for alcohol (at the early stages of the regime) with candy, coke, cake, coffee, etc. They substituted protein—almonds, seeds, cheese, plain yogurt, cottage cheese, a chicken leg or a slice of turkey. Eventually this became a habit with them. After six weeks to three months, those patients who had stayed on their regime were able to stay sober with none of the "drying-out" effects. Those who were still on their regime at the end of a year were sober and reported phenomenal ability to concentrate, to remember names and to relax; an ability to quit smoking and a feeling of well-being which they had not experienced in years.

Drinking becomes less rewarding when megadoses of niacin and L-glutamine are used. "Overdosing" with vitamins is actually beneficial for alcoholics—usually, because of substituting alcohol for food, their body is depleted in most vitamins. There is ample evidence for this (Pauling, Hoffer, Osmond, Williams); views supported by these scientists

hold that great benefits may be gained from massive "overdosage" of various vitamins. Orthomolecular psychiatry was founded on this premise.

In alcoholism, if within six weeks on the hypoglycemic diet the alcoholic's blood sugar remains low, it would be advisable for him to have some intravenous feedings of protein. An alcoholic who is put into the hospital and given protein intravenously with intensive vitamin therapy responds rapidly to this regime. Dr. Williams positively asserts that "when alcoholics, as a group, are familiar with the story of nutrition, they will be among those most anxious to learn what their own peculiar individual nutritional needs are."

From all the facts relating alcoholism to biological factors and nutritional deficiencies, it becomes clear that whatever measures may be taken to prevent alcoholism, the neglect of the nutritional approach cannot be justified. Roger J. Williams. 1971.

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