

# Editorial

## Vitamin Dependency

*“Man is a food-dependent creature. If you don’t feed him, he will die. If you feed him improperly, part of him will die.”*

—Emanuel Cheraskin, M.D., D.M.D.

Dependency is a fact of life. The human body is dependent on food, water, sleep, and oxygen. Additionally, its internal chemistry is absolutely dependent on vitamins. Without adequate vitamin intake, the body will sicken; virtually any prolonged vitamin deficiency is fatal. Surely this constitutes a dependency in the generally accepted sense of the word.

Nutrient deficiency of long standing may create an exaggerated need for the missing nutrient, a need not met by dietary intakes or even by low-dose supplementation. Recently,<sup>1</sup>Robert P. Heaney, M.D., used the term “long latency deficiency diseases” to describe illnesses that fit this description. He writes:

“(I)nadequate intakes of many nutrients are now recognized as contributing to several of the major chronic diseases that affect the populations of the industrialized nations. Often taking many years to manifest themselves, these disease outcomes should be thought of as long-latency deficiency diseases. . . (I)nadequate intakes of specific nutrients may produce more than one disease, may produce diseases by more than one mechanism, and may require several years for the consequent morbidity to be sufficiently evident to be clinically recognizable as “disease.” Because the intakes required to prevent many of the long-latency disorders are higher than those required to prevent the respective index diseases, recommendations based solely on preventing the index diseases are no longer biologically defensible.”

There are at least two key concepts presented here: The first is, “Inadequate intakes of specific nutrients may produce more than one disease.” This exactly sup-

ports Dr. William Kaufman’s statements to this effect 55 years ago, when he wrote that, in considering “different clinical entities one cannot exclude the possibility that they may be caused by the same etiologic agent, acting in different ways. For example, in experimental animals, it has been shown that the lack of a single essential nutrient can produce a variety of dissimilar clinical disorders in different individuals of the same species. . . (O)ne might not suspect that the same etiologic factor, lack of a specific essential nutrient, was responsible for each of the various clinical syndromes of the same tissue deficiency disease which is permitted to develop at different rates in different individuals of the same species.”<sup>2</sup>

While amyotrophic lateral sclerosis, progressive muscular atrophy, progressive bulbar palsy, and primary lateral sclerosis are not all the same illness, they and the other neuromuscular diseases may have a common basis: unacknowledged, untreated long-term vitamin dependency. Therefore, each may respond to an orthomolecular approach such as that successfully used by Dr. Frederick R. Klenner<sup>3</sup> for multiple sclerosis and myasthenia gravis, half a century ago.

The second key point Heaney makes is that vitamin “intakes required to prevent many of the long-latency disorders are higher than those required to prevent the respective index diseases.” This confirms Dr. Abram Hoffer’s observations to this effect some 40 years ago, when he treated prisoners of war presenting severe, protracted nutrient deficiencies. Hoffer wrote<sup>4</sup> that when released, after as long as 44 months of captivity, “only 75 percent had survived. They had lost about one-third of their body weight. In camp they suffered from classical scurvy, beri-beri, pellagra, many infections, and from protein and calorie deficiency. They were rehabilitated in hospitals and were given doses of vitamins that were then considered high. Since then these Hong Kong veterans have suffered from a variety of physical and psychi-

atric conditions.” However, “the history of a small sample, about 12, is much different, for they have been taking nicotinic acid (niacin) 3 grams per day. These 12 have recovered and remain well as long as they take this quantity of vitamin regularly.

“About 35 years ago (in the 1930s and 1940s) it was reported that some chronic pellagrins required at least 600 milligrams per day of vitamin B<sub>3</sub> to prevent the return of pellagra symptoms. This was astonishing then and unexplainable since pellagra as a nicotinic acid deficiency disease should have yielded to vitamin (small) doses. Today the concept of vitamin-dependency disease has developed. It is based upon the realization that there is a much wider range of need for nutrients than was believed to be true then.

“A person is said to be vitamin dependent if his requirements for that vitamin are much greater (perhaps 100-fold greater or more) than is the average need for any population. The optimum need is that quantity which maintains the subject in good health, not that quantity which barely keeps him free of pellagra. From this point of view the Hong Kong veterans have become vitamin B<sub>3</sub> dependent as a result of severe and prolonged malnutrition. It is likely that any population similarly deprived of essential nutrients for a long period of time will develop one or more dependency conditions.”

Thirty years ago, in another paper,<sup>5</sup> Hoffer made this statement: “The newer concept of vitamin-dependent disease changes the emphasis from simply dietary manipulation to consideration of the endogenous needs of the organism. It comes within the field of orthomolecular disease. The borderline between vitamin deficiency and vitamin-dependency conditions is merely a quantitative one when one considers prevention and cure.” (p. 251)

The differentiation between deficiency and dependency is dose. Every patient that was ever helped by high-dose nutrient

therapy lends support to the concept of vitamin dependency. By the same token, symptoms resulting from inappropriate and abrupt termination of large doses of nutrients provide equally good evidence for vitamin dependency. While deprivation of low doses of vitamin C causes scurvy, abrupt termination of high maintenance doses may cause its own set of problems. Called “rebound scurvy,” this includes classical scorbutic symptoms, as well as a predictable relapse of illness that had already responded to high-dose therapy.

Writes Robert F. Cathcart, M.D.: “There is a certain dependency on ascorbic acid that a patient acquires over a long period of time when he takes large maintenance doses. Apparently, certain metabolic reactions are facilitated by large amounts of ascorbate and if the substance is suddenly withdrawn, certain problems result such as a cold, return of allergy, fatigue, etc. Mostly, these problems are a return of problems the patient had before taking the ascorbic acid. Patients have by this time become so adjusted to feeling better that they refuse to go without ascorbic acid. Patients do not seem to acquire this dependency in the short time they take doses to bowel tolerance to treat an acute disease. Maintenance doses of 4 grams per day do not seem to create a noticeable dependency. The majority of patients who take over 10-15 grams of ascorbic acid per day probably have certain metabolic needs for ascorbate which exceed the universal human species need. Patients with chronic allergies often take large maintenance doses.

“The major problem feared by patients benefiting from these large maintenance doses of ascorbic acid is that they may be forced into a position where their body is deprived of ascorbate during a period of great stress such as emergency hospitalization. Physicians should recognize the consequences of suddenly withdrawing ascorbate under these circumstances and be prepared to meet these increased meta-

bolic needs for ascorbate in even an unconscious patient. These consequences of ascorbate depletion which may include shock, heart attack, phlebitis, pneumonia, allergic reactions, increased susceptibility to infection, etc., may be averted only by ascorbate. Patients unable to take large oral doses should be given intravenous ascorbate. All hospitals should have supplies of large amounts of ascorbate for intravenous use to meet this need.”<sup>6</sup>

This need is especially serious for the cancer patient, whose exceptionally positive response to mega-ascorbate therapy, and dramatically negative response to ascorbate deprivation, is the very picture of vitamin dependency. Linus Pauling’s colleague Ewan Cameron, M.D., wrote:

“Ascorbate, however administered, is rapidly excreted in the urine, so that administration should be continuous or at very frequent intervals. Furthermore, exposure to high circulating levels of ascorbate induces over-activity of certain hepatic enzymes concerned with its degradation and metabolism. These enzymes persist for some time after sudden cessation of high intakes, resulting in depletion of circulating levels of ascorbate to well below normal unsupplemented values. This is known as the rebound effect. It causes a sharp decrease in immunocompetence and must be avoided in the cancer patient. Clinical experience has shown that the best responses are observed when vitamin C is administered intravenously, so insuring a high plasma level. However, because long-term continuous intravenous administration is impractical, we recommend an initial intravenous course of ten days duration, followed by continuous maintenance oral regimen.”<sup>7</sup>

In short, the body only misses what it needs. That is dependency. The destructive consequences of alcohol and other negative drug dependencies are taught in elementary schools. At the same time, the consequences of ignoring our positive nu-

trient dependencies go largely undiscussed even in medical journals. Vitamin dependencies induced by genetics, diet, drugs, or illness are most often regarded as medical curiosities. The Hoffer-Osmond discovery that schizophrenics, forming about one to two percent of the population, are dependent on multi-gram doses of niacin, remains a psychiatric heresy. The Irwin Stone-Linus Pauling idea of population-wide, genetically-based hypoascorbemia has received negative attention, when it has received any attention at all. Yet, writes Dr. Emanuel Cheraskin, “hypovitaminosis C is a very real and common, probably epidemic, problem which clearly has not been properly viewed and surely not adequately reported.”<sup>8</sup>

This is not a total surprise. It took decades for medical acknowledgement that selenium and vitamin E are actually essential to health.

Simple cause-and-effect micronutrient deficiency, a doctrine long held dear by the dietetic profession, is not always sufficient to explain persistent physician reports of megavitamin cures of a number of diseases outside the classically accepted few. Perhaps it is a law of orthomolecular therapy that the reason one nutrient can cure so many different illnesses is because a deficiency of one nutrient can cause many different illnesses.

If nutrient deficiency is basically about inadequate intake, then dependency is essentially about heightened need. As a dry sponge soaks up more milk, so a sick body generally takes up higher vitamin doses. The quantity of a nutritional supplement that cures an illness indicates the patient’s degree of deficiency. It is therefore not a megadose of the vitamin, but rather a megadeficiency of the nutrient that we are dealing with. Orthomolecular practitioners know that with therapeutic nutrition, you don’t take the amount that you believe ought to work; rather, you take the amount that gets results. The first rule of building a brick wall is that you must have enough bricks.

A sick body has exaggeratedly high needs for many vitamins. We can either meet that need, or else suffer unnecessarily.

Until the medical professions fully embrace orthomolecular treatment, “medicine” might well be said to be “the experimental study of what happens when poisonous chemicals are placed into malnourished human bodies.”

—Andrew W. Saul  
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