Protein Needs And HIV

Adequate protein stores are vital for immune function and good health, especially for individuals who are challenged by the human immunodeficiency virus (HIV). Recently the focus has been on maintaining adequate lean body mass (LBM) and many people living with HIV infection (PLWHIV) have begun exercise regimes.

Some have initiated lifestyle changes that include a change in dietary habits as well. Inadequate dietary intakes and an inability of those living with HIV to meet increased dietary needs of calories and protein are well known. [1-3] Some HIV-positive individuals don't know they must strive to meet increased calorie and protein requirements. [4] Conversely, there are a great number of individuals who not only are aware of their increased needs but strive to meet those needs with the use of creatine or other supplements noted to increase body weight and muscle mass (see alternative focus article in this issue). Recommendations for PLWHIV to follow high-calorie and high protein diets are common. Currently there are no standard protein recommendations for HIV challenged individuals. Protein guidelines vary considerably among nutrition professionals and others who seek to improve the nutritional status of this population group. This review will serve to provide useful information on protein needs and recommendations for people living with HIV and AIDS. A focus on various issues to consider when recommending increased protein intakes are discussed as well.

BACKGROUND

In 1838 Mulder described protein as organic material that is the most important substance to life. [5] It seems as if ever since that time there has been a debate on the values and detriments of certain diets. Interestingly, in the late 1800's, a man named Dr. James H. Salisbury was the first to note the diet should be high in meat and low in starchy foods. [6] In 1928, another man claimed that diets low in starchy foods and high in meat caused "a host of bodily disorders". In present time, researchers are investigating the appropriateness of diets providing 12-20% protein for insulin-dependant diabetics. [7]

For review, proteins are the principle nitrogen-containing organic constituents in plant and animal tissues. [8] They are classified by biological function. Proteins are essential for the growth and maintenance of tissue and for the synthesis of many regulatory functions. [5, 8, 9] In fact, estimates place the number of functioning proteins in the human body at more than 100,000. [5] Proteins consist of over 50 amino acids* (AA's) in chains that are biochemically joined to form peptide links. There are 22 AA's that are widely distributed in proteins. Nine AA's are indispensable and can not be made in sufficient amounts by the body. Millward points out that after the first year of life human amino acid needs are almost entirely for maintenance. [10] Proteins that do not contain enough of the essential AA's to maintain body tissues or promote a normal rate of growth are considered incomplete. AA content greatly affects protein quality. High-biological proteins, such as egg, milk and meat, have enough essential AA's. Factors influencing the amount of protein required include tissue growth (as in pregnancy), diet, illness, and disease.

Body proteins have a wide variety of specialized functions and their synthesis depends on nucleoproteins*. Serum proteins, known as blood proteins, are vital in the maintenance of fluid balance. [5] Albumin plays a vital role in generating colloidal osmotic pressure, which facilitates fluid retention within blood vessels. Protein is involved in the maintenance of endocrine function. [9] It supplies AA's for the manufacture of protein substances like hormones and enzymes. Protein hormones are involved in regulating metabolism and immune response. Investigators from New Zealand recently found that lambs consuming a high protein diet displayed a significant interaction of dietary protein and immunization. [11] There are contractile proteins such as myosin that regulate muscle contraction and structural proteins like collagen and elastin. Nutrient transport proteins such as hemoglobin and retinol-binding protein (R-B protein) ferry nutrients to the tissues.

HIV-NEGATIVE PROTEIN NEEDS

The importance of adequate protein intake can not be overstated. Providing that the protein intake is of maximum biological availability, 0.45grams (gm) of protein per kilogram (kg) of body weight should cover the daily nitrogen losses that occur through urine, feces, sweat and minor losses in the healthy HIV-negative individual. To allow for loss of protein efficiency an allowance of 0.8 gm of protein per kg of body weight is suggested, for healthy HIV-negative adults. The recommended dietary allowance (RDA) for protein is 50 gm for HIV-negative females and 63 gm for HIV-negative males. Dietary protein use depends on total energy intake and protein quality. A measurement of protein quality called the protein efficiency ratio is used as the standard for U.S. RDA labeling. Proteins that have a biological value of 70 or more are capable of supporting growth as long as sufficient calories are ingested. Net protein utilization, which is the proportion of nitrogen consumed that's retained by the body under standard conditions, is the same as the biological value when food proteins are completely digested. Protein catabolism increases during injury, burns, immobilization, infection, fear, anxiety and anger. [5]

The effects of protein loss are widespread and include: decreased wound healing; respiratory muscle weakness; skeletal muscle weakness; and immunosuppression. [12] Protein deficiency is evident in several pathological conditions. Kwashiorkor is characterized by growth failure, skin lesions, edema, fatty liver, and changes in hair color. [5] Nutritional edema appears after serum albumin level is decreased and substantial depletion of tissue reserves has taken place. Marasmus, caused by a deficiency in calories and protein, is more severe but without edema. Besides protein-calorie malnutrition, decreased albumin synthesis can occur as part of the acute-phase response and as a result from redistribution of albumin pools or from albumin losses. [13] Dietary protein intake affects gastrointestinal (GI) bacteria and drug-metabolizing enzymes in the gut and liver. [14] GI tract villi are often flattened and atrophic and almost all digestive enzymes decrease in the severely malnourished person. [15] Even in the HIV-negative individual

this leads to poor digestion and absorption and may contribute to diarrhea.
A few studies have shown that the average adult male loses about 1.4 gm of nitrogen per day. [5] These losses increase in hot environments. Generally, obligatory nitrogen losses may range from 2-4 gm each day. Skin losses can be as much as 3.75 gm of nitrogen (23 gm of protein) with profuse sweating. Scrimshaw notes daily protein losses from infection such as flu wastes more than .5 gm of protein per kg. [16] Pneumonia wastes 1.2 gm per kg each day during illness. Health improving activity increases the need for protein and the more strenuous the exercise the greater the need is for more protein. [17] During physical exercise when carbohydrate is depleted protein may provide up to ten percent of the required energy. [18]

POOR PROTEIN STATUS

Wilmore reminds us body mass is critical for recovery from illness. [12] Without adequate stores of LBM complications arise that place the individual at increased nutritional risk. Rady and others study of cardiac patients found that all types of low albumin, except for malnutrition cachexia, increased the likelihood of postoperative organ dysfunction, GI bleeding, nosocomial infections, length of mechanical ventilation, stay in the CVICU, and hospital death. [19] Cachectic hypoalbuminemia increased the need for postoperative parenteral nutrition and prolonged the length of hospital stay. A body weight loss of 12% results in a 20% body protein loss. [12] Loss of body protein affects metabolism as well as the immune system. [20] Protein malnutrition is a principal factor of morbidity and mortality during infection and complications increase when serum albumin levels fall below 3.0g per deciliter (dL). [21, 22] Blackburn says during injury the amount of caloric expenditure (16%) that comes from protein sources is about two times that seen during non-stress. [21] At that time, reuse of body protein seems to be decreased. He suggests a level of 1.5 to 2.0 gm of protein per kg each day to produce positive nitrogen balance* in the presence of protein malnutrition.

Progressive weight loss coupled with changes in LBM and decreases in circulating levels of export proteins present evidence for protein-energy malnutrition in AIDS. [23] Cachexia is distinguished by a major loss of lean body mass in muscle and major organs. [20] Metabolic changes of cachexia are thought to be controlled by cytokines*. An 'acute (short-term) phase response' is part of the changes seen in HIV infection with energy and nutrients going where they are needed the most. Kotler notes the liver produces great quantities of proteins during this period to meet body demands for tissue defense and repair. Levels of transport proteins decrease and other proteins such as C-reactive protein, ceruloplasmin, and fibrinogen increase. [24] These shifts in fluid lessen the concentration of visceral protein indexes. Conservation of fat and the wasting of protein stores takes place. [20] Results of a study led by Morlese suggest the repletion of nutrient transport protein pools, in severely malnourished children, is attained by a greater rate of synthesis but at different rates. [25] Investigators note rapid repletion of the pools of R-B protein and transthyretin takes place but repletion of the high density lipoprotein-apolipoprotein A1 pool occurs less quickly.

HIGH RISK BEHAVIORS

Many high-risk behaviors contribute to depressed protein stores. Inadequate nutrient intake is prominent in PLWHIV and may be due to depression or other controllable lifestyle factors. Getting some PLWHIV to increase food intake may be difficult especially if they are suffering from undiagnosed depression. Christensen and Somers found that some individuals experiencing a current episode of major depression consumed a diet containing less than the RDA in one or more nutrients due to a decrease in food intake. [26] Another study found that nondepressed subjects eat more protein than depressed subjects. [27] Depressed subjects ate more carbohydrates that came from an increase in sucrose consumption. Authors note the increased carbohydrate consumption may relate to the development or maintenance of depression.

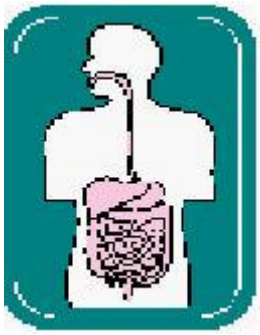
Caregaro and others note protein malnutrition and immunoincompetence are much more frequent in alcoholic and virus-related cirrhosis than energy malnutrition. [28] Alcohol consumption plays a large role in protein-energy malnutrition and unfortunately in the lives of many PLWHIV as well. As much as 50% of total caloric intake may come from alcohol in some individuals. [29] Research suggests alcohol affects protein nutrition by: 1) causing impaired digestion of proteins to AA's; 2) impaired processing of AA's by the small intestine and the liver; 3) impaired synthesis of proteins from AA's; and 4) impaired protein secretion by the liver. Heavy drinkers may develop meat and other protein aversion because of liver dysfunction. [30] Lieber notes AA abnormalities are common in people who consume large amounts of alcohol. [31] Cysteine and glutathione (GSH) liver requirements may be increased. In chronic alcoholism if alcohol intake exceeds about 1,100 calories for a number of years (~15) a great number of drinkers will develop structural liver abnormalities. [32] Baraona's study notes that, at least in rats, alcohol decreases the export of protein from the liver into the plasma. [33] Italian studies of 15 healthy volunteers serve to show that two alcoholic drinks slightly affect after meal hepatic protein metabolism. [34] The equivalent of about five drinks of wine profoundly impairs after meal hepatic protein metabolism by interfering with the fractional secretory rates of both albumin and fibrinogen. Investigators note acute intake of that much wine severely impairs the synthesis and secretion of liver proteins despite the absorption of a meal enriched with AA's.

Substance abuse is another risk factor for poor protein status. Individuals who must depend on pain medications, or those that use recreational drugs, may be unknowingly placing themselves at increased nutritional risk. Opiate addicts often replace high-protein foods with those that are high in sucrose and full of 'empty calories'. [35] Large numbers of addicts are HIV-positive. An HIV seroprevalence rate of 12.7% among 6,402 injection drug users and 7.5% among 3,383 crack smokers occurred during the years 1992 and 1993. [36] Another sample of 337 baseline HIV-negative gay men from the San Francisco Men's Health Study cohort helped to determine substance use behaviors. [37] Results show seroconverters are persistently more likely to report the use of marijuana, nitrite

inhalants, amphetamines, and cocaine than nonconverters. Amphetamines and cocaine can severely inhibit the urge to eat and cause unnecessary weight loss in the individual living with HIV. Cigarette smoking tends to decrease food intake as well. Mohs and others note during use or withdrawal from recreational drugs and nicotine significant changes in diet habits result in either weight gain or loss. [38] Most PLWHIV must use a variety of prescribed medications. The added burden of recreational drugs increases the chance that protein stores will be affected.

Protein synthesis and degradation, which is essential for maintaining protein mass during the aging process, are regulated nutritionally and hormonally. [39] Changes in AA and glucose concentrations, and either the level of or tissue response to insulin, insulin-like growth factor-I*, and growth hormone, occurs with aging. Protein status is affected by trauma and disease that impedes the interaction of these regulatory factors. Several effects occur including a difference in either protein synthesis or degradation and protein turnover. Recent symposium studies suggest this decline is partly because of a reduction in the synthesis of specific muscle proteins. A decline in the levels and actions of growth hormone, insulin-like growth factor-I, testosterone*, and DHEA lead some to believe that hormone replacement therapy may improve the sarcopenia* of aging. Indeed, hormone therapy is being increasingly used to treat PLWHIV who have protein-wasting diseases. Mooney reminds PLWHIV that higher protein intake is essential for the anabolic effect of anabolic steroids*. [40]

DIGESTION & ABSORPTION



The rate of protein turnover is greatest in the intestinal mucosa, liver, pancreas, kidney, and blood plasma. Growth hormone, gonadotropins such as testosterone, and adequate thyroxine play a role in anabolism*. Adrenal steroid hormones influence catabolism* by stimulating the breakdown of AA's. Protein absorption rate depends on: 1) the load of AA's released through digestion; 2) AA's proportion in the mixture to be dissolved; 3) carrier availability to transport the AA's into mucosal cells; and 4) the uptake of AA's by tissues. [5] Some food proteins such as those with increased amounts of fiber have a lower digestibility than others so net protein utilization is lower. Digestion in the HIV-positive person is often detrimentally affected. Many PLWHIV are at risk of protein-energy malnutrition, especially those with structural villus damage within the intestine. [23] Structural villus damage contributes to net negative balances of energy and protein. Gastric protein digestion may be affected by damaged mucosa within the stomach wall, which results in a lack of adequate hydrochloric acid secretion. The result of these malfunctions is an inability of the body to break protein down into AA's that can be readily absorbed and used for tissue synthesis. A vitamin B6 deficiency can add to the problem of assuring protein adequacy in PLWHIV by decreasing AA transport within the intestinal wall.

We know the liver regulates the outcome of incoming AA load but some break down also takes place in the kidney. Both of these organs are prone to dysfunction and disease in PLWHIV mainly because of needed medication regimes. Cimoch points out that a variety

of drugs are dependent on blood proteins for delivery and uptake into their target cells. [41] Markedly reduced transit times and generally poor bioavailability of orally administered drugs are present in many AIDS patients. [14, 41] Chemical changes that normally occur with enzymes in the stomach and intestine are often unable to function properly in the presence of HIV disease. Homeostasis may be unstable leading the way to negative protein balance.

HIV-POSITIVE PROTEIN NEEDS

Many PLWHIV are not aware of their increased need for protein. Some of them may be unknowingly placing themselves at increased risk of nutritional complications by consuming diets that are critically low in protein. A New Mexico study of 59 AIDS patients with wasting showed a daily protein intake that ranged from 0.2 to 2.5 gm of protein per kg of body weight. Investigators note study participants were drastically short of meeting protein needs with 14% of them consuming less than 0.8 gm of protein/kg. [42] Decreases in circulating levels of export proteins and changes in LBM are common in AIDS. [23] The high mortality among AIDS patients with low serum albumin levels is well known. [3, 43] Kotler notes protein status is greatly influenced by HIV infection and although calories are important feeding is not enough to replete body cell mass. [3] HIV infection increases protein turnover and urinary nitrogen loss; decreases skeletal muscle protein synthesis; increases skeletal muscle breakdown; and increases hepatic synthesis.

Surprisingly poor nutritional status was evident in the study participants of one early investigative project led by Chlebowski and others. [43] At baseline, weight loss was evident in 98% of the 71 patients. Hypoalbuminemia was common in 83%. Most patients' albumin levels ranged from 3.4 to 2.5 g/dL. Subjects with albumin levels between 3.4 and 2.5 g/dL had an average survival of 103 days. Those with levels below 2.5 g/dL had only a 17 day median survival. Subjects with normal albumin levels at baseline had a rate of albumin decrease each day that was less than half that in the patients with low albumin. Investigators note the rate of albumin decrease may define a function limiting survival of the individual with AIDS. Selberg and colleagues sought to investigate nutritional status and protein metabolism in six AIDS patients with weight loss who were receiving total parenteral nutrition. [44] Although the investigators note poor nutritional status in this type of individual is associated with high whole body protein turnover, they report these patients can, at least for a short time, achieve positive nitrogen balance with adequate protein intake. This is possible mainly by way of an increase in whole-body protein synthesis.

Another study of 50 AIDS patients serves to remind us the diets of our patients may not always be well balanced. [2] We know protein intake must be adjusted to one's body weight and medical condition. People with health conditions such as HIV, AIDS, renal disease and liver dysfunction require individual and continued attention. Metabolic alterations in HIV disease increase nutrient needs. Individual's living with HIV require increased levels of protein just to maintain body protein stores. Some HIV challenged people are not able to use fat stores for energy when caloric intake is inadequate. [23, 3] Consequently, LBM is broken down to meet energy needs. Myers notes PLWHIV have

about a 44% increase in fat oxidation. [45] There's a 300% rise in fat storage and a 250% increase in glucose formation that's most likely from mainly protein sources. Futile cycling of fat contributes to the loss of LBM and very few PLWHIV regain lost protein as protein. Myers points out that the increased tendency for fat formation may be the result of a decrease in carnitine, which is a nonessential AA. Body protein changes increase with disease progression.

AMINO ACIDS

Some investigators note treatment of Herpes Simplex Type-I infection with L-lysine is often overlooked. [46] Cousins suggests that RDA's for lysine, methionine, tryptophan and threoine be established due to their likelihood of being limited in the human diet. [47] Specific AA's regulate functional changes in immune system response. [23, 41, 48] These AA's include: arginine; glutamine; branched-chain AA's; and sulfur-containing AA's like cysteine. Most of the body's proteins require cysteine. Methionine and cysteine, which may be low in PLWHIV, play a role in the synthesis of proteins for the immune system. [45, 49] Along with other functions, arginine plays a role in lymphocyte proliferation and wound healing. Increasing dietary intakes of n-3 polyunsaturated fatty acids, L-arginine, and ribonucleic acid increase body weight. [50] Researchers speculate this may occur by regulating the negative effects of tumor necrosis factor*.

Glutamine, another nonessential AA, is the most abundant free AA in the body. It's produced mainly in skeletal muscle and is a primary fuel for the mucosa of the small intestine. [51, 52] Glutamine may help maintain GSH, which is an important antioxidant for fighting disease and stress. [49] Suboptimal glutamine concentrations in skeletal muscle may result in muscle wasting and depletion of body cell mass. [51] Cimoch suggests that a deficiency of glutamine may increase the translocation of enteric pathogens into the bloodstream by decreasing the number of IgA-producing lymphocytes in the intestine. [41] Research studies show glutamine requirements increase during HIV infection and deficiency may develop if adequate glutamine is not in the diet. [51] When glutamine concentrations rise skeletal muscle protein is made and muscle mass is restored. Glutamine may prevent further muscle wasting and increase LBM. Several research studies report glutamine supplementation reduces myofibrillar protein breakdown and enhances skeletal muscle protein synthesis. [51, 52]

Low GSH levels are found in PLWHIV. Keusch points out that the stimulation of HIV replication is inhibited by GSH, N-acetyl cysteine (NAC) and GSH-esters in the test tube. [23] He notes experimental GSH deficiency reversibly impairs several immune responses in which CD4* T-cells participate. Low cysteine levels are associated with wasting. [45] Interesting data from rat studies shows that cysteine, methionine and GSH production increase with increasing protein intake. [53] Lark Lands, a popular health educator and consultant among those living with HIV says supplementation with an antioxidant called alpha-lipoic acid (also known as thioctic acid) may be the best way to raise GSH levels. [54] She notes other uses for this substance as well. German studies raise the possibility of its use as a antiviral and Lands suggests researchers in the U.S. may not be studying this acid because it's unpatentable. Although there are questions as to the possibility of

mineral interaction, a daily dose of 100 mg or more with meals is suggested. [54] Lands also notes even individuals with asymptomatic disease may need glutamine supplementation. [54] Anywhere from 10-40 gm per day may be useful for intestinal repair, weight gain or to restore wasted muscles. The powdered form is preferable and it's vital to remember that individuals on protein-restricted diets should consider the glutamine as part of their protein allowance. [49]

Essential AA deficiencies can cause a variety of problems. [55] Brown and others note tryptophan is necessary for the synthesis of proteins, serotonin and niacin. [56] Indoleamine 2,3-dioxygenase, which is initiated by infections, viruses, lipopolysaccharides, or interferons, results in significant catabolism of tryptophan. Brown's group thinks a systemic or localized depletion of tryptophan is at least partly the reason for anti-tumor effects of interferons and clinical side effects in the body. They note it's likely that tryptophan deficiency occurs in autoimmune diseases. This deficiency might be linked to degeneration, wasting and other symptoms. Increasing interferon levels are associated with HIV disease progression. These investigators suggest indoleamine 2,3-dioxygenase is not only induced by infection with HIV but increases with the incidence of opportunistic infections with a concomitant loss of tryptophan. This chronic tryptophan deficiency is said to begin the process responsible for cachexia, dementia, diarrhea and the immunosuppression of AIDS patients. Trypsin (an enzyme) has a significant amount of cysteine and is maintained at the expense of other proteins when cysteine is low. NAC is a cysteine precursor used for mucolytic treatment in bronchitis and for acetaminophen poisoning. [57] Supplementation with NAC is considered essential. [45, 58] Some clinicians note the AA should be supplied at a level of 2- 3 gm per day. [45] This is important because trypsin releases other enzymes needed for digestion from their precursors. Myers is quick to say when considering supplementation this AA should not be taken directly because excess cysteine can be toxic to the central nervous system. [49]

Some dietitians routinely recommend AA supplementation of L-Glutamine, and L-Carnitine. [58] L-Carnitine, which is sometimes low in HIV infection, is essential to letting the body burn fat for fuel. Constant AZT use is said to deplete L-Carnitine and result in the loss of muscle. Pharmaceutical strength products of L-Glutamine, and L-Carnitine can be bought with a physician's prescription. Williams notes over-the-counter supplemental AA preparations are an irritating and inefficient source of available nitrogen. [59] Besides the financial cost of protein supplementation an additional price may be paid by those who use AA supplements in large doses. Single AA supplements may cause imbalances and toxicity's. [17] Product quality is not standardized and their margin of safety is unknown. Beltz and Doering note there's no scientific evidence to support the use of purified AA's in uninfected athletes. [60] Research on the use of AA's for PLWHIV, such as L-Glutamine, must still address safety concerns.

BIOCHEMICAL ASSESSMENT

The biochemical assessment of protein status is vital to establish nutritional care plans and replete low protein stores. A 1995 review notes in the nutritional assessment of patients who are critically ill there's no single indicator that's consistent. [61] Body measurements are inaccurate and imprecise in individuals or are at risk of non-nutritional influences. Jensen reminds us it's important to focus on realistic indexes that are relative to the patient's metabolic status. [62] Biochemical assessment of PLWHIV is all the more difficult due to several factors. Laboratory parameters may be altered because of metabolic derangement, fluid shifts, nutrient deficiency, and medication use. [24] In critically ill patients data is more accurate when collected after fluid resuscitation and peak metabolic response to injury. [62] Fields-Gardner notes visceral protein measurements are the most helpful indicators of nutritional status. [24] Some laboratory parameters used in the assessment of protein stores are in table one. These test results may be altered because of stress or injury.

Since these indicators have different synthesis rates and half-lives, some of them reflect changes in nutritional status more quickly than others. One must remember, protein levels are influenced not only by their rates of synthesis and half-lives, but also by catabolism, loss into interstitial spaces, and abnormal external loss. Blood albumin helps to evaluate nutritional status and oncotic pressure. [63] It's often used to indicate body protein reserves and is the most common abnormality related to nutrition in individuals with infection. Although albumin has a half-life of 14-21 days, most clinicians still use it as an indicator of protein depletion. Several factors that affect albumin and other indicators of nutritional status are in table two. Albumin levels will decrease for patients lying on their back and are slightly decreased in those on oral contraceptives. Benjamin reminds us we should always consider some assessment of inflammation such as erythrosedimentation rate because it's value affects the interpretation of other tests. [64] Kotler only looks at the albumin level as an inflammatory response marker. [3] Total blood protein is used to evaluate nutritional status and liver function, and to diagnose malabsorption, neoplasia, and nephrotic syndromes. [63] Low total protein can be caused by a wide variety of conditions and patients should be in a fasting state before the blood draw. Conditions most likely to affect results in PLWHIV include: protein losing enteropathies; starvation; malabsorption; m alnutrition; cirrhosis or other liver diseases; prolonged immobilization; heart failure; nephrotic syndromes; and neoplasia.

Parameter	Half-life
Albumin	~19 days
Transferrin	~9 days
Prealbumin	~2 days
R-B protein	~11 hours
Creatinine-height index (somatic)	

TABLE 1. COMMONLY USED PROTEIN/NUTRITIONAL STATUS INDICATORS

Manning and Shenkin report that in the critically ill plasma concentrations of hepatic proteins lack specificity because they're affected by the individual's disease state and therapeutic interventions. [61] Some clinicians look at prealbumin as a baseline noting that in many cases the first value will be low. Prealbumin is not as affected by exogenous factors as transferrin. [65] Mears found prealbumin to be a sensitive measure of nutritional

status that allowed for earlier assessment and intervention. [66] Serial measurement of plasma pre-albumin is useful in monitoring the response to nutritional support. [65] It is being increasingly used to assess nutritional status because of its short half-life and high tryptophan content. [67] It's two roles in the blood are to bind retinol and transport thyroxine. [63] Prealbumin values in adult males have a slightly higher value than comparative females. Values decrease during the acute phase response caused by infection or tissue injury. Transferrin is used to differentiate iron deficiency anemia from hypochromic microcytic anemias. It's more reflective of acute changes in nutritional status. Transferrin may not be increased in iron-deficient states where there's a severe protein malnutrition.

The nitrogen balance technique is a classic way to determine human AA and protein requirements in patients with normal renal function. [5] Nitrogen balance is said to be of great importance in assessing the nutritional status of the critically ill. [61] A 24-hour urine urea nitrogen measure is valuable when appraising utilization of dietary intake. [21] Fields-Gardner notes positive nitrogen balance may be achieved by another 20-30 gm of protein each day for those individuals who need to restore protein stores. [24] This test is inexpensive but the ammonia in urine is said to interfere with the method and in some facilities the test is rarely performed. [63] There are other limitations to the use of this test as well. Not only is it difficult to obtain a complete 24 hour urine collection but it's not valid in renal failure or in instances where fecal and skin losses of nitrogen occur. An

Parameter	Conditions Affecting Value
Albumin	* Trauma; congestive heart failure; body losses; hypercatabolic states; severe hepatic insufficiency; infection; stress; poor protein intake; burns; fluid overload; inadequate liver production; nephrotic syndrome; zinc deficiency; small bowel bacterial overgrowth; oral contraceptives; IV fluids; Protein losing enteropathies; neoplasia; peptic ulcer; thyroid disease; burns; severe skin disease: prolonged immobilization; heart failure; chronic inflammatory disease;inadequate body protein reserves + Dehydration; infusion of albumin; infusion of whole blood
Total protein	* Protein losing enteropathies; starvation; malabsorption; malnutrition; cirrhosis or other liver diseases; prolonged immobilization; heart failure; nephrotic syndromes; neoplasia; pregnancy; IV fluids; glomerulonephritis; hyperthyroidism; burns; severe skin disease; chronic diseases + Dehydration; chronic liver disease; tropical diseases; macroglobulinemia of Waldenstr"m; granulomatous diseases; neoplasms; collagen diseases; chronic infection; chronic inflammation; hemolysis
Prealbumin	* Malnutrition; inflammatory processes; hepatic disease + Corticosteroids
Transferrin	* Acute catabolic states; increased iron stores; iron overload; chronic inflammatory states; hereditary atransferrinemia; acquired liver disease; neoplasia; hemolysis; renal disease; chronic debilitating illness; Kwashiorkor; nephrotic syndromes; overhydration + Oral contraceptives; blood loss; iron deficiency anemia; viral hepatitis; dehydration
R-B protein	* Liver disease; acute catabolism; vitamin A deficiency + Renal failure

TABLE 2. FACTORS AFFECTING THE INTERPRETATION OF PROTEIN STORES

Key: * =Decrease + =Increase

insufficient intake of calories can result in negative nitrogen balance and anabolic/catabolic hormones can skew results too. Manning and Shenkin note direct measurement of urine nitrogen is the preferred test. [61] Creatinine-height index enables estimation of somatic protein stores but as with the other tests has it's limitations. It's interesting that in this era of managed care there are some insurance companies that restrict reimbursement of chemistry batteries. [68] Some clinicians note nutritional status can be evaluated in hospital patients by asking specific questions related to weight change, oral intake, current diet, and reason for admission or diagnosis. Caregaro and others report anthropometry is currently the most reliable method for nutritional assessment in clinical practice. [28] Myers reminds us most common methods of lean body mass assessment are inaccurate in those with HIV. [45] Bioelectrical impedance analysis (BIA) is useful in gauging LBM and therefore protein stores. It's a recommended method but must be calibrated for HIV. [45, 69] Protein stores can also be measured by in-vivo neutron activation analysis. [70]

HIGH PROTEIN DIETS

Studies investigating the interaction between diets high in protein and various minerals are prominent in the literature. [71-77] Associations between various cancers and protein intake are evident as well. [78] High protein levels provide a protective effect against malignant hepatic tumors. On the other hand, specific AA deficiencies or imbalances inhibit or suppress various types of tumor growth. Now that protease inhibitors have given many PLWHIV a new lease on life we need to be more concerned with the long-term effects of treatments. The risks for osteoporosis, gout, and uric acid kidney stones increase with the use of excess protein. Thirty-five percent of the serum calcium is bound to protein and high protein intake can cause a profound increase in urinary calcium. [79] Each increase in protein intake results in a further increase of urinary calcium. Studies show that a protein intake of 142 gm results in markedly negative calcium balances even if daily calcium intake is as high as 1,400 milligrams. This is due to the metabolism of sulfur- containing AA's. [80] Phosphorus lowers urinary calcium but increases endogenous calcium loss through the intestine. This interaction between calcium and protein can be offset by an increase in dietary calcium. Although they may not have a diagnosed lactose intolerance, many PLWHIV avoid dairy products, hence the nutrient-nutrient interaction remains. Without an increase in dairy products, which have the highest calcium to protein ratio, dietary intake is usually not sufficient to compensate for the protein-induced loss of this mineral. Therefore the threat of increased calcium loss through urine and feces is a real concern for those PLWHIV. Long term skeletal effects that result from the constant ingestion of high-protein diets are unknown. [79] It seems prudent to note since many PLWHIV have expanded lifespans the chance of these diets causing detrimental changes to skeletal structure is increased.

The importance of including adequate amounts of carbohydrate and fat must be understood. Some individuals may attempt to replace carbohydrates with protein. Reports suggest a diet low in carbohydrates and high in protein and fat can cause a number of disorders. Some individuals note undesired increases in lipoproteins, fatigue, constipation or amenorrhea. Coleman notes a low carbohydrate intake can cause liver and muscle

glycogen depletion, which causes water losses. [81] Diets too low in carbohydrate may also result in diminished kidney function that leads to increased sodium excretion. Besides the possibility of these problems, a low carbohydrate diet can cause electrolyte loss, ketosis, weakness, and nausea.

There are other factors that may be considered when deciding to increase protein intake. Studies show that increased intakes of protein may require additional vitamin B-6 to maintain adequate levels. [82] Excessive protein supplementation can cause GI effects. Some note a high protein intake occasionally promotes loss of appetite and diarrhea. [83] High protein diets that are low in carbohydrates can lead to not only bone calcium loss, but hyperuricemia, and hyperuricosuria, as well. [60, 84] Excessive protein supplementation can lead to dehydration and liver and kidney damage. Fluid intake must be increased to allow for the elimination of nitrogenous wastes. [83] This entails more work for the liver and kidneys. People with hepatic encephalopathy or renal failure have less ability to excrete these wastes. Diets that are too high in protein may affect the status of other minerals as well. Some study results suggest that the response to dietary protein on mineral status varies with age. [71, 75] Along with protecting muscle mass, increased protein diets may help the person living with HIV by increasing levels of zinc. [73] Many proteins including stomach enzymes require zinc. [47] There's some indication that reduced serum albumin may contribute to low levels of serum zinc. [47, 70] Also, the absorption of zinc and copper is in some degree controlled by metallothionein, which is a small metalloprotein that's rich in cysteine. [47] Myers notes metallothionein may be low in PLWHIV because cysteine is known to be low in this population group. [45] This impairs the absorption of zinc and copper and when there's a zinc deficiency the body breaks down other metalloproteins namely those found in muscle and soft tissues. Hunt and colleagues study served to show that high protein diets may not only increase zinc retention but may reduce indexes of iron status as well. [77]

PROTEIN NEED SUGGESTIONS

A high protein diet and light anabolic exercise helps PLWHIV to prevent muscle mass loss and build up protein stores. [48] Early protein recommendations were between 1.2-1.8 gms/kg of body weight daily. [85] Current suggestions for optimal protein intake vary based on individual needs and sources (see table three on page 13). [24, 42, 48, 58, 86-93] A number of practicing professionals routinely recommend their clients consume between 1.2-1.5 gm of protein per kg of body weight each day. [87, 90- 93] One program designed to build and maintain lean body mass includes protein guidelines that recommend one or more grams of protein for each pound of current body weight. [94] Donna Tinnerello, founding member of the Nutritionists in AIDS Care, a New York group, notes unless individuals require specialized nutrition support they rarely require more than two grams of protein per kg. [89] In her book, Romeyn tells HIV-positive readers' to take their total number for energy needs and multiply it by 3% (.031) to determine protein maintenance needs. [48] The amount required for anabolism is found by multiplying total energy needs by 4% (.042). Fields-Gardner suggests 1-1.4 gm of protein per kg of body weight to maintain protein stores and 1.5-2.0 gm of protein per kg to replete and build protein stores. [24] Positive nitrogen balance is desirable to blunt severe catabolic response to

stress. Individuals receiving specialized nutrition support may require up to 2.5 gm/kg/day. Keep in mind those individuals with renal disease or hepatic dysfunction may require decreased protein levels. They need to be increasingly aware of high-biological protein sources.

Information Source/Facility	Clinician	Protein Per Kg of Body Weight	Year
University of New Mexico+	Nickell, RD, LD, CNSD	2.0-2.5	1994
Nutritional Guide ^	HRSA (Various)	1.25	1996
A Clinician's Guide... ^	Fields-Gardner, MS, RD	1.0-2.5	1997
Fenway Health Ctr.* (MA)	Smigelski, RD	0.75 (per pound)	1997
Project Open Hand* (OH)	Bell, RD	1.2-1.5	1997
Mercy Hospital* (FL)	Vasquez, RD	1.2-1.5	1997
Nutrition Poweri (CA)	Jensen, MS, RD, CNSD	1.0 (per pound)	1997
Presentation	Muurahainen, MD, PhD	1.2-1.5	1997
Presentation	Tinnerello, MS, RD	1.5-2.0	1997
Nutrition and HIV...^	Romeyn, MD	.031-.042 x total energy needs	1998

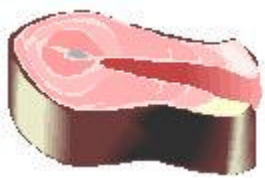
TABLE 3. PROTEIN GUIDELINES FOR HIV-POSITIVE ADULTS

Information Source: Book=^ Personal Correspondence Facility RD=* Journal=+ Private Practice=i

Martin notes it's harder to replenish albumin stores in the presence of a selenium deficiency. [95] Champredon and others report high protein and energy content of the diet must be coupled with specific AA supply to patients with AIDS. [96] So how much protein is enough for the person living with HIV? We're not positive yet but we do know that PLWHIV may need an increase in protein between 10-70% of normal requirement. [95, 97] It's vital these individuals know that in adults eight essential AA's must be supplied in adequate amounts, proportion, and timing for tissue protein to be made. Most importantly, PLWHIV must know that muscle proteins are broken down and used as energy when total calorie and protein intake is not enough to meet body needs. If the diet is too low in carbohydrate or fat up to 58% of the total dietary protein consumed is broken down to yield energy. [8] This basic information helps the person living with HIV to understand why diets low in carbohydrate and fat are not appropriate.

PROTEIN BOOSTING TIPS

Most of us are aware that there's a difference between knowing your nutritional needs and taking the steps to meet those needs. Many of us have experience with clients who do not follow recommendations exactly as suggested. This often causes undue harm to the individual who actually seeks to improve their nutritional status. An anecdotal report notes undesired increases in lipoproteins and fatigue when consuming diets that are too high in protein. [98] The client's alternative diet called for eating between 16- 20 ounces of meat a day with zero-to-minimal carbohydrate. This type of diet is not recommended. Information on how to increase



dietary protein is welcome by most people seeking to improve nutritional status. Several protein-boosting tips to help PLWHV maintain adequate protein stores can be offered. Because egg is very high in quality Romeyn suggests the addition of egg whites to other foods to increase their protein value. Cooked egg can be sprinkled on a variety of dishes to increase protein content. Obviously clients must understand that raw egg whites are not desirable and Romeyn does a good job in conveying this information. Casein or whey proteins are usually high in quality as well. However, casein, which is the major component of cow's milk, is not readily digested. [47] Myers notes partially hydrolyzed protein may be used when digestion is suboptimal. [45] Soy-based products may provoke GI problems and colitis. Glycinin and conglycinin, major storage proteins which are allergens in soy, are often contained in drinks that rely on soy as their protein source. A wide variety of high protein supplements are easily available to help boost protein intake when necessary.

People who are challenged by HIV must maintain adequate protein stores for immune function and good health. We know they're not always able to use fat stores for energy and LBM is broken down to meet energy needs. A necessary course of action is to intervene before the futile cycling of fat begins. People who live with this virus need to be aware of their nutritional needs. A focus on increased calorie and protein intake should be a part of every HIV-positive individual's care. The era of managing HIV as a long-term chronic disease has arrived and we must do everything we can to improve nutritional status and overall well-being. Lifestyle changes that include a positive change in dietary habits will help PLWHIV to maintain valuable LBM and avoid unnecessary complications. Yet, to insure more effective nutrition interventions we need uniform protein recommendations that are appropriate.

Bibliography

1. Gorbach SL, Knox TA, Roubenoff R. Interactions Between Nutrition And Infection With Human Immunodeficiency Virus. Nutr Rev. 1993;51(8):226-34.
2. Zambelli A, Comi D, Meraviglia P, Martini F, Cargnel A. Effect of Two Different Diets on AIDS Patients' Nutritional Status. Int Conf AIDS. 1996 Jul 7-12;11(2):293 (abstract no. Th.B.4243). Milano, Italy.
3. Kotler D. Whey Protein and L-Glutamine Use in HIV-associated Weight Loss. Presentation at The First International Conference on AIDS Wasting, Fort Lauderdale, FL. Nov 1997.
4. Meyer SA. Nutrition Knowledge of People With HIV Infection in Broward County. AIDS Pt Care. 1994;8(5):279-281.
5. Robinson CH, Lawler MR. Proteins and Amino Acids. In: Beck JJ, ed. *Normal and Therapeutic Nutrition,* 16th Edition. New York, NY. Macmillan Publishing Company, Inc.; 1982:48-73.
6. Brillat-Savarin JA. *The Physiology of Taste: Or Meditations on Transcendental Gastronomy.* Translator: M. F. K. Fisher, George Macy Companies Inc., 1949.
7. Charlton M, Sreekumaran Nair K. Protein Metabolism in Insulin-Dependant Diabetes Mellitus. J Nutr. 1998;128(2):323S-327S.
8. Williams SR. Proteins: The Problem of Building Tissue. In: Hirsch CK, ed. *Essentials of Nutrition and Diet Therapy.* Third Edition. St. Louis, Missouri: C.V. Mosby Company;1982:35-48.
9. Worthington- Roberts BS. Proteins and Amino Acids. In: *Contemporary Developments in Nutrition.* St. Louis, Missouri: The C.V. Mosby Company; 1981:109-134.
10. Millward DJ. Human Amino Acid Requirements. J Nutr. 1997;127(9):1842-1846. World Wide Web: http://nutrition.org (assessed 17 Sep 1997).
11. Kambara T, McFarlane RG. Changes in T Cell Subpopulations of Sheep Due to Age and Dietary Protein Intake; Association With Protective Immunity to Trichostrongylus Colubriformis. Vet Immunol Immunopathol. 1996;51(1-2):127-35.

12. Wilmore DW. Growth Hormone Promotes Protein Anabolism and Recovery. Presentation at The 62nd Annual Meeting of The Florida Dietetic Association, Marco Island, FL. July 1997.
13. Kaysen GA, Rathore V, Shearer GC, Depner TA. Mechanisms of Hypoalbuminemia in Hemodialysis Patients. Kidney Int. 1995;48(2):510-6.
14. Knapp HR. Nutrient-drug Interactions. Brown ML, ed. *Present Knowledge in Nutrition*, Sixth Edition. Washington, DC: International Life Sciences Institute, Nutrition Foundation;1990:451-456.
15. Latham MC. Protein-energy Malnutrition. Brown ML, ed. *Present Knowledge in Nutrition*, Sixth Edition. Washington, DC: International Life Sciences Institute, Nutrition Foundation;1990:39-46.
16. Scrimshaw NS. Effect of Infection on Nutrient Requirements. J Paren Enter Nutr 1991;15:589-600.
17. De Luise K. Sports Nutrition: Fuel For Active People. World Wide Web: http://www.cheshire-med.com/services/dietary/nutrinew/sportn1.html (accessed 21 Dec 1996).
18. Lemon PW, Mullin JP. Effect of Initial Muscle Glycogen Levels on Protein Catabolism During Exercise. J Appl Physiol. 1980;48(4):624-629.
19. Rady MY, Ryan T, Starr NJ. Clinical Characteristics of Preoperative Hypoalbuminemia Predict Outcome of Cardiovascular Surgery. JPEN. 1997; 21:81-90. World Wide Web: http://www.clinnutr.org (accessed 17 Sept 1997).
20. Kotler D. Malnutrition in HIV Infection: What Causes It? Lifetimes2, Magazine. 1997;1. World Wide Web: http://www.iapac.org (accessed 17 Sept 1997).
21. Blackburn GL. Nutritional Assessment and Support During Infection. Am J Clin Nutr. 1977;30(9):1493-1497.
22. Mullen JL, Gertner MH, Buzby GP, Goodhart GL, Rosato EF. Implications of Malnutrition in The Surgical Patient. Arch Surg. 1979;114:121-125.
23. Keusch GT, Thea DM. Clinical Nutrition. Malnutrition in AIDS. Med Clin North Am. 1993;77(4):795-814.
24. Fields-Gardner C, Thomson C, Rhodes SS. *A Clinician's Guide to Nutrition in HIV and AIDS*. Chicago, IL: American Dietetic Association; 1997.
25. Morlese JF, Forrester T, Del Rosario M, Frazer M, Jahoor F. Repletion of The Plasma Pool of Nutrient Transport Proteins Occurs at Different Rates During The Nutritional Rehabilitation of Severely Malnourished Children. J Nutr. 1998;128(2):214-219.
26. Christensen L, Somers S. Adequacy of The Dietary Intake of Depressed Individuals. J Am Coll Nutr. 1994;13(6):597-600.
27. Christensen L, Somers S. Comparison of Nutrient Intake Among Depressed and Nondepressed Individuals. Int J Eat Disord. 1996;20(1):105-109.
28. Caregaro L, Alberino F, Amodio P, Merkel C, et al. Malnutrition in Alcoholic and Virus-related Cirrhosis. Am J Clin Nutr 1996;63:602-9.
29. US Department of Health and Human Services. Alcohol and Nutrition. In: Alcohol Alert, Newsletter. Rockville, MD:National Institute on Alcohol Abuse and Alcoholism. 1993; 22(346):1-3.
30. Zamula E. Bodily Harm: The Health Effects of Excessive Drinking. FDA Consumer, Magazine. 1986;20(4):13-15.
31. Lieber CS. Alcohol and Nutrition An Overview. Alcohol Health and Research World. 1989;13(3):197-205.
32. Rudman D, Feller AG. Liver Disease. In: Brown ML, ed. *Present Knowledge in Nutrition*, Sixth Edition. Washington, D.C: International Life Sciences Institute- Nutrition Foundation; 1990:385-394.
33. Baraona E; Leo MA; Borowsky SA; Lieber CS. Pathogenesis of Alcohol-induced Accumulation of Protein in The Liver. J Clin Invest. 1977;60(3):546-54.
34. Volpi E, Lucidi P, Cruciani G, Monacchia F, et al. Moderate and Large Doses of Ethanol Differentially Affect Hepatic Protein Metabolism in Humans. J Nutr. 1998;128(2): 198-203.
35. Morabia A, Fabre J, Chee E, Zeger S, et al. Diet and Opiate Addiction: A Quantitative Assessment of The Diet of Non-Institutionalized Opiate Addicts. British J Addiction. 1989;84(2):173-180.
36. Kral AH, Bluthenthal RN, Booth RE, Watters JK. HIV Seroprevalence Among Street-Recruited Injection Drug and Crack Cocaine Users in 16 US Municipalities. Am J Public Health. 1998;88(1):108-113.
37. Chesney MA, Barrett DC, Stall R. Histories of Substance Use and Risk Behavior: Precursors to HIV Seroconversion in Homosexual Men. Am J Public Health. 1998;88(1):113-116.
38. Mohs ME, Watson RR, Leonard-Green T. Nutritional Effects of Marijuana, Heroin, Cocaine, and Nicotine. J Am Diet Assoc. 1990;90(9):1261-1267.
39. Davis TA, Reeds PJ. The Roles of Nutrition, Development and Hormone Sensitivity in The Regulation of Protein Metabolism: An Overview1,2,3. J Nutr. 1998;128(2): 340S-341S.

40. Mooney M. Best Protein - The Real Deal. Medibolics, Newsletter. West Hollywood, CA: Program for Wellness Restoration. May 1997; 2(1):3.
41. Cimoch PJ. Nutritional Health Prevention and Treatment of HIV-associated Malnutrition: A Case Manager's Guide. Inter Assoc Phy AIDS Care. May 1997. World Wide Web: http://www.iapac.org (accessed 16 Aug 1997).
42. Nickell DM, Butler-Sanchez LM, Waters DL, Danska JM, et al. Calorie and Protein Intake of AIDS Patients Compared to Recommended Guidelines. J Am Diet Assoc. 1994; 94(9S): A-25.
43. Chlebowski RT, Grosvenor MB, Bernhard NH, Morales LS, Bulavage LM. Nutritional Status, Gastrointestinal Dysfunction and Survival in Patients With AIDS. Am J Gastroenterol. 1989;84(10):1288-1293.
44. Selberg O, Suttmann U, Melzer A, Deicher H, et al. Effect of Increased Protein Intake and Nutritional Status on Whole-body Protein Metabolism of AIDS Patients With Weight Loss. Metabolism. 1995;44(9):1159-1165.
45. Myers C. Information Relating to HIV and Nutrition: HIV and Liquid Food Supplements. Toronto, Canada: Resource Centre. 1997 February. World Wide Web: http://www.catie.ca (accessed 31 Jan 1998).
46. Ferroli CE, SerVaas C, Kagan C. Treatment of Herpes Simplex Type-I Infection With The Amino Acid L-Lysine is Overlooked. J Am Diet Assoc. 1996; 96(9S): A-106.
47. Cousins RJ, Hempe JM. Zinc. In: Brown ML, ed. *Present Knowledge in Nutrition.* Sixth Edition. Washington, DC: International Life Sciences Institute- Nutrition Foundation; 1990:251-260.
48. Romeyn M. Appendix II. Diet Diary and Nutritional Assessment. In: *Nutrition and HIV- A New Model For Treatment*. San Francisco, CA: Jossey-Bass Publishers; 1998:361.
49. Myers C. Information Relating to HIV and Nutrition: HIV and Diet Revisited. 1997 February. Toronto, Canada: Resource Centre. World Wide Web: http://www.catie.ca (accessed 31 Jan 1998).
50. Suttmann U, Ockenga J, Schneider H, Selberg O, et al. Weight Gain and Increased Concentrations of Receptor Proteins For Tumor Necrosis Factor After Patients With Symptomatic HIV Infection Received Fortified Nutrition Support. J Am Diet Assoc. 1996;96(6):565-9.
51. Summary of Selected Research Publications on The Role of Glutamine Supplementation in The Management of Patients With HIV Infection and AIDS, Including a Discussion on Antioxidants and Their Precursors. Boston, MA:Cambridge Nutraceuticals, Insight Resources, Inc.; 1996.
52. Winslow- Stoltz C. Glutamine as a Supplement in Individuals With HIV. IFARA News, Newsletter. June 1996;1(1):15.
53. Hum S, Koski KG, Hoffer LJ. Varied Protein Intake Alters Glutathione Metabolism in Rats. J Nutr. 1992;122(10):2010-2018.
54. Lands L. NAC, Glutamine, and Alpha Lipoic Acid. AIDS Treatment News, Newsletter. April 4, 1997;268. World Wide Web: http://www.aegis.com (accessed 16 Feb 1998).
55. Millward DJ, Price GM, Pacy PJH, Halliday D. Maintenance Protein Requirements: The Need For Conceptual Revaluation. Proc Nutr Soc. 1990;49(3):473-487.
56. Brown RR, Ozaki Y, Datta SP, Borden EC, et al. Implications of Interferon-induced Tryptophan Catabolism in Cancer, Auto-immune Diseases and AIDS. Adv Exp Med Biol. 1991;294:425-35.
57. Sanford JP, Gilbert DN, Moellering RC, Sande MA. Alternative Therapies For HIV. In: Wickwire C, ed. *The Sanford Guide To HIV/AIDS Therapy.* Sixth Edition. Vienna, VA: Antimicrobial Therapy, Inc.; 1997:123.
58. Smigelski C, RD, Fenway Community Health Center: Cambridge, MA. Personal correspondence; Nov 1997.
59. Williams SR. Nutrition For Adults: Aging and The Aged. In: Hirsch CK, ed. *Essentials of Nutrition and Diet Therapy.* Third Edition. St. Louis, MO: C.V. Mosby Company;1982:211.
60. Beltz SD, Doering PL. Efficacy of Nutritional Supplements Used by Athletes. Clin Pharm. 1993;12(12):900-908.
61. Manning EM, Shenkin A. Nutritional Assessment in The Critically Ill. Crit Care Clin. 1995;11(3):603-634.
62. Jensen TG. Determination of Nutritional Status in Critical Care. J Am Diet Assoc. 1984;84(11):1345-1348.
63. *Holy Cross Hospital Laboratory User's Guide.* Hudson, OH: Lexi-Comp, Inc; 1993.
64. Benjamin DR. Laboratory Tests and Nutritional Assessment. Protein-energy Status. Pediatr Clin North Am. 1989;36(1):139-161.
65. Ament ME. Enteral and Parenteral Nutrition. In: Brown ML, ed. *Present Knowledge in Nutrition*, Sixth Edition. Washington, DC: International Life Sciences Institute, Nutrition Foundation;1990:444-450.

66. Mears E. Outcomes of Continuous Process Improvement of a Nutritional Care Program Incorporating Serum Prealbumin Measurements. Nutrition. 1996;12:(7-8): 479-84.
67. Malave I, Vethencourt MA, Pirela M, Cordero R, et al. Serum Levels of Prealbumin in Undernourished Children and Controls With or Without Associated Clinical infection. (Article in Spanish) Arch Latinoam Nutr. 1996;46(3):203-209.
68. Kuhnert KA, RD. Initial Screening. Dietetics Listserv (www.eatright.org), Internet Post. American Dietetic Association. Feb 3, 1998.
69. Fields-Gardner C. Clinical Applications of Bioelectrical Impedance Analysis and Interpretation of Currently Available Reports. Presentation at The First International Conference on AIDS Wasting, Fort Lauderdale, FL. Nov 1997.
70. Lustig JR. Nutrition and HIV Infection. Asia Pacific J Clin Nutr. 1993:2, 3-14.
71. Takeda T, Kimura M, Yokoi K, Itokawa Y. Effect of Age and Dietary Protein Level on Tissue Mineral Levels in Female Rats. Biol Trace Elem Res. 1996;54(1):55-74.
72. Johnson WT, Evans GW. Effects of The Interrelationship Between Dietary Protein and Minerals on Tissue Content of Trace Metals in Streptozotocin-Diabetic Rats. J Nutr. 1984;114(1):180-190.
73. Greger JL, Snedeker SM. Effect of Dietary Protein and Phosphorus Levels on The Utilization of Zinc, Copper and Manganese by Adult Males. J Nutr. 1980;110(11):2243-2253.
74. Mahalko JR, Sandstead HH, Johnson LK, Milne DB. Effect of a Moderate Increase in Dietary Protein on The Retention and Excretion of Ca, Cu, Fe, Mg, P, and Zn by Adult Males. Am J Clin Nutr. 1983;37(1):8-14.
75. Sherman AR, Helyar L, Wolinsky I. Effects of Dietary Protein Concentration on Trace Minerals in Rat Tissues at Different Ages. J Nutr. 1985;115(5):607-614.
76. Wallwork JC, Johnson LK, Milne DB, Sandstead HH. The Effect of Interactions Between Dietary Egg White Protein and Zinc on Body Weight, Bone Growth and Tissue Trace Metals in The 30-day-old Rat. J Nutr. 1983;113(7):1307-1320.
77. Hunt JR, Gallagher SK, Johnson LK, Lykken GI. High- versus Low-meat Diets: Effects on Zinc Absorption, Iron status, and Calcium, Copper, Iron, Magnesium, Manganese, Nitrogen, Phosphorus, and Zinc Balance in Postmenopausal Women. Am J Clin Nutr. 1995;62(3):621-632.
78. Cheney C, Worthington- Roberts BS. Diet and Cancer. In: *Contemporary Developments in Nutrition.* St. Louis, Missouri: The C.V. Mosby Company; 1981:367-387.
79. Worthington- Roberts BS. Calcium and Sodium. In: *Contemporary Developments in Nutrition.* St. Louis, Missouri: The C.V. Mosby Company; 1981:240-272.
80. Heaney RP. Protein Intake and The Calcium Economy. J Am Diet Assoc. 1993;93(11): 1259- 1260.
81. Coleman E, RD, MA, MPH. Atkin's Diet Information. Dietetics Listserv (www.eatright.org), Internet Post, American Dietetic Association. Feb 23, 1998.
82. Hansen CM, Leklem JE, Miller LT. Vitamin B-6 Status of Women With a Constant Intake of Vitamin B-6 Changes With Three Levels of Dietary Protein. J Nutr. 1996;126(7):1891-1901.
83. Worthington-Roberts BS. Diet and Athletic Performance. In: *Contemporary Developments in Nutrition*. St. Louis, Missouri. The C.V. Mosby Company; 1981:570.
84. Blackburn GL. Management Of Obesity. Presentation at The 80th Annual Meeting of The American Dietetic Association, Chicago, IL. October 1995.
85. Sherman C. Dealing With The Challenge of AIDS. Top Clin Nutr. 1989;4(1):37.
86. Health Care and HIV: Nutritional Guide For Providers and Clients. Vienna, Va: Health Resources and Services Administration, Bureau of Primary Health Care, US Department of Health and Human Services; May 1996.
87. Muurahainen N. Nutritional Therapies in HIV Related Wasting. Presentation at The 1st International Conference on AIDS Wasting, Fort Lauderdale, FL. Nov 1997.
88. Jensen J, RD, Santa Monica, CA: Nutrition Power. Personal correspondence;July 1997.
89. Tinnerello D, MS, RD. Nutritional Management of HIV/AIDS From The Beginning. Presentation at The 62nd Annual Meeting of The Florida Dietetic Association, Marco Island, FL. July 1997.
90. Silvana Vasquez, RD, Miami, FL:Mercy Hospital, Special Immunology Services. Personal correspondence; October 1997.
91. Connor C, RD, Huntington Station, NY:Long Island Association for AIDS Care. HIV/AIDS Nutritional Care Survey, Written Response; September 1997.
92. Melissa J, RD, Los Angeles, CA:Project Angel Food. HIV/AIDS Nutritional Care Survey, Written Response; September 1997.

93. Bell T, Dietitian, LP, Columbus, OH: Project Open Hand. HIV/AIDS Nutritional Care Survey, Written Response; September 1997.
94. Vergel N, Mooney M. PoWer Program For Wellness Restoration Protocols. Medibolics, Newsletter. Houston, TX: PoWer Program for Wellness Restoration. 1995;1(1).
95. Martin BM, RD, LD, CNSD. HIV/AIDS Nutrition. Presentation at The 78th Annual ADA Meeting, Chicago, IL. October 1995.
96. Champredon C, Laurichesse H, Tauveron I, Gourdon F, et al. Rate Limiting Amino Acids For Protein Synthesis in AIDS. Int Conf AIDS. 1996 Jul 7-12;11(2):101 (abstract no. We.B.3260).
97. O'Sullivan P, Linke RA, Dalton S. Evaluation of Body Weight and Nutritional Status Among AIDS Patients. J Am Diet Assoc. 1985;85:1483.
98. Fowler K. High Protein Diets. Dietetics Listserv (dietetics-L@www.eatright.org), Internet Post, American Dietetic Association. March 3, 1998.

About the author: Sharon Ann Meyer AS, AA, DTR, Certified HIV Counselor is the President of HIV ReSources, Inc., and Editor-In-Chief of the HIV ReSource Review. Contact her at: sharon@hivresources.com.

Acknowledgements: Many thanks to the U.S. Registered Dietitians (table three) who added their information and to Chester Myers of St. Lawrence Technologies, Inc. in Tiverton, Ontario, Canada for reviewing this article and adding his insights.