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Nutritional assessment of alcoholics: Emphasis on vitamin A and selenium

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NUTRITIONAL ASSESSMENT OF ALCOHOLICS: EMPHASIS ON VITAMIN A AND SELENIUM

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NUTRITIONAL ASSESSMENT OF ALCOHOLICS: EMPHASIS ON VITAMIN A AND SELENIUM

(TITLE)

BY

KAREN M. McCAULEY, R.D.

THESIS

SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS
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ABSTRACT

The purpose of this study was to determine the nutritional status of patients admitted to the alcohol rehabilitation wards of the Danville, Illinois, Veterans Administration Medical Center. Selenium and vitamin A were specifically examined because reports have shown low levels of these nutrients in cirrhosis. Possible correlations between these nutrients and the results of liver function tests were also examined because the liver is an integral part of nutrient metabolism. Changes in vitamin A or selenium status during two weeks of hospitalization were examined because of expected change in dietary intake while in the hospital.

Nutritional assessment parameters included diet histories, height, weight, percent ideal body weight, anthropometrics, serum albumin, and total lymphocyte count, as well as serum vitamin A, retinol binding protein, and whole blood and plasma selenium.

Complete nutritional assessment data could be collected on 23 of the 27 subjects. Initial diet histories generally revealed inadequate caloric intake and adequate protein intake, although calories derived from alcohol were not included. Anthropometric data, serum albumins, and total lymphocyte counts were also generally adequate, indicating the absence of general malnutrition.

Initial retinol levels did deviate from the normal range, with the majority being elevated. Normal and depressed retinol levels were also found. Significant ($p \leq 0.01$) change in retinol levels were found after two weeks. Correlations between retinol and the results of liver function tests were significant ($p \leq 0.05$) only for albumin vs. retinol. Initial retinol binding protein levels were depressed for 10 of 24 subjects, but no significant change occurred after two weeks.

Whole blood and plasma selenium levels also deviated from the normal range, with initial levels usually being low. Normal and elevated levels were also found. No significant change was found in selenium status after two weeks, and no significant correlations were found between selenium levels and the results of liver function tests.

It was concluded that although protein-calorie malnutrition was not detected by the nutritional assessment, vitamin A and selenium abnormalities were found. The presence of both depressed and elevated vitamin A and selenium values suggests that a heterogeneous group was surveyed. The etiology of these abnormalities may be better understood if homogeneous groups could be defined and examined. The cause of the normalization trends found two weeks after admission also requires further investigation.

NUTRITIONAL ASSESSMENT OF ALCOHOLICS: EMPHASIS ON VITAMIN A AND SELENIUM

BY

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B.S., University of Illinois, 1979

THESIS

Submitted in partial fulfillment of the requirements for the degree of Master of Science in Dietetics from the Department of Home Economics in the Graduate College of Eastern Illinois University at Charleston, Illinois, 1983.

DEDICATION

This work is dedicated to my parents, Mary and Mel McCauley, for their guidance in the development of my basic premises, and also to my husband, Marc Rosen, for his understanding, patience, and his challenge to me to live according to my basic premises.

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CHAPTER I

INTRODUCTION

Background of Study

Alcoholism is a multidimensional disease, involving biochemical, physiological, psychological, and sociocultural factors. The relationship and significance of these factors change with the individual and with the severity and duration of alcohol abuse. The research concerning treatment of alcoholism must be a dynamic field if it is to be effective. Therefore, research into the interplay of each factor at various stages of alcoholism must continue. This study focuses on the interaction of several biochemical and physiological factors.

The body systems which are affected by alcoholism include the hepatic, pancreatic, gastrointestinal, neurological, and cardiovascular systems (Victor and Adams, 1977). Specific vitamin and mineral deficiencies, as well as protein-calorie malnutrition, have been associated with alcoholism (Simko et al., 1982). Alcohol can affect one's nutritional status in five ways: altering nutrient absorption or utilization, increasing nutrient needs or losses, and decreasing nutrient intake (Herbert, 1973).

Low plasma vitamin A levels reported in children with kwashiorkor (Trowell et al., 1954; Arroyave et al., 1959;

Gopalan et al., 1960) may be explained by low vitamin A intake, interference with absorption (Periera et al., 1967), and decreased synthesis of vitamin A transport proteins (Arroyave et al., 1961). This explanation may also be appropriate for the depressed retinol and retinol-binding protein (RBP) levels found in alcoholic cirrhosis (McClain et al., 1979).

Low plasma selenium levels have been found in cirrhotic alcoholics (Aaseth et al., 1980), which may indicate an increased need or a decreased intake of selenium. It has been suggested (Goldman and Kantrowitz, 1981) that alcoholic cardiomyopathies parallel those seen in association with selenium deficiency (Johnson et al., 1981; Collipp and Chen, 1981). Goldman and Kantrowitz (1981) also suggested that a selenium deficiency may accelerate the cardiac disease.

Statement of Problem

Evaluation of the alcoholic's nutritional status needs to be investigated using defined subgroups because of the diverse nature of the alcoholic population. As the liver is affected by alcohol, and the liver plays an important role in determining nutrient utilization, further investigation is needed concerning specific nutrient status in alcoholics. The purpose of this study was to determine the general nutritional status of patients admitted to the

alcohol rehabilitation wards of the Danville, Illinois, Veterans Administration Medical Center. Selenium and vitamin A were specifically examined because reports have shown low levels in cirrhosis. These nutrients were re-assessed after two weeks in the medical center to determine if the patient's status changed. Since the degree of liver impairment may affect the degree of nutrient utilization, possible correlations among nutrients and biochemical determinations of liver function were examined.

Research Questions and Hypotheses

The following research questions were investigated. Hypotheses are stated in the null form.

Research Question Number One

Are the alcoholics admitted to the rehabilitation wards at the Danville, Illinois, Veterans Administration Medical Center (DVAMC) malnourished?

Hypothesis

1. The nutritional status of alcoholics at DVAMC does not differ significantly from that of the general population as measured by anthropometrics, diet histories, and biochemical tests of visceral protein status and immunocompetence.

Research Question Two

Is the vitamin A status of alcoholics admitted to the rehabilitation wards at DVAMC abnormal?

Hypotheses

2. The vitamin A status of alcoholics at DVAMC does not differ significantly from published normal values of retinol, retinyl palmitate, retinoic acid, or retinol binding protein.

3. No significant change occurs in the vitamin A status of alcoholics at DVAMC during two weeks of hospitalization.

4. No correlation exists between vitamin A levels and results of liver function tests.

Research Question Number Three

Is the selenium status of alcoholics admitted to the rehabilitation wards at DVAMC abnormal?

Hypotheses

5. The selenium status of alcoholics at DVAMC does not differ significantly from published normal values of plasma and whole blood selenium.

6. No significant change occurs in the selenium status of alcoholics at DVAMC during two weeks of hospitalization.

7. No correlation exists between selenium levels and results of liver function tests.

Definition of Terms

Albumin: secretory liver protein which reflects visceral protein status.

Alcoholic: one who has been diagnosed as alcohol-dependent by a physician.

Alcohol: ethanol.

Anthropometrics: physical measurements of body composition, including height, weight, skinfolds, and arm circumference.

Diet History: record of food intake, either by a frequency of food eaten record, a twenty-four-hour recall, or a summation of food usually eaten in a given period of time.

Lean Body Mass: that part of the body which is not fat, as determined by subtracting body fat from body weight.

Liver Function Tests: those biochemical tests which indirectly describe the degree of liver function, including lactic dehydrogenase (LDH), gamma glutamyl transpeptidase (GGT), glutamic-oxaloacetic transaminase (SGOT), glutamic-pyruvate transaminase (SGPT).

Marasmus: nutritional disease typified by depressed anthropometrics, wasted appearance, and normal visceral protein status.

Mid-Arm Circumference (MAC): the circumference of the arm midway between the tip of the acromion and the olecranon process.

Mid-Arm Muscle Circumference (MAMC): the derivation of the arm muscle devoid of fat as determined by the following formula: MAMC=MAC-(.314 x triceps skinfold).

Kwashiorkor: nutritional disease typified by depressed visceral protein status, depressed immunocompetence, edema, and normal, elevated or depressed anthropometrics.

Nutritional Assessment: tests used in determining nutritional status, including dietary histories, anthropometrics, and biochemical determinations.

Nutritional Status: an estimation of one's nutrient reserves.

Protein-Calorie Malnutrition: a combination of marasmus and kwashiorkor.

Skinfold Measurements: determinations taken at four sites (biceps, triceps, subscapular, suprailiac) with calipers to assess body fat.

Total Lymphocyte Count: reflection of immunocompetence, determined by the following: white blood cells x percent lymphocytes.

Vitamin A: used to refer collectively to retinol, retinyl palmitate, and retinoic acid.

CHAPTER II

REVIEW OF LITERATURE

Alcohol

The host response to the ingestion of alcohol varies considerably. Thus the degree of organ injury, and the frequency and severity of complications due to alcohol abuse also vary. Factors which may be significant in determining the host response include genetic, environmental, psychological, social, nutritional, and metabolic factors. These factors may play a role in alcohol metabolism, absorption, or development of systemic complications. The systemic complications then may impair the nutritional status of the host.

Alcohol is absorbed primarily from the small intestine. High concentrations of alcohol have been found in the lumen of the gastrointestinal tract (Halsted et al., 1973). This elevated alcohol concentration can produce functional impairment (Baraona et al., 1974) and result in altered nutrient absorption (Israel et al., 1968; Israel et al., 1969). Structural changes in the small intestine have been reported, but the lesions heal rapidly (Pirola et al., 1969). Diarrhea may be due to an alcohol-induced decrease in brush border enzymes (Perlow et al., 1977). However, the malabsorption exhibited in many alcoholics may be secondary to malnutrition

(Baraona and Lieber, 1979). This conclusion is based on the results of a study in which alcohol administration failed to produce abnormal nutrient losses when subjects were fed adequate diets (Lindenbaum and Lieber, 1975).

Decreased food intake, or intake of inadequate diets, has often been attributed to an alcohol induced loss of appetite. This anorexia was attributed to high caloric intake from alcohol (Olsen, 1950) and also to specific vitamin and mineral deficiencies thought to be secondary to alcoholism (Roe, 1979).

The decreased intake has also been attributed to economic factors, in that money normally spent on food is instead spent on alcohol. However, poor economic status cannot be the causative factor for malnutrition in all alcoholics. It has been estimated that only 3-5% of alcoholics live on Skid Row, and that 70% live in respectable homes and are employed (Thomson et al., 1980). Recognition of this enlarging alcoholic population has led to the postulate that malnutrition is not the problem that it once was for alcoholics (Hurt et al., 1981). However, increasing the diversity and size of the alcoholic population, and thus lowering the percent considered to be malnourished, does not negate the problem of malnutrition.

Malnutrition can result for reasons other than impaired absorption, increased losses, or decreased intake. Metabolic and physiological changes can also result in malnutrition.

Hemorrhage from gastritis, esophageal varices, or ulcer may increase the severity of anemia already potentiated by alcohol's direct suppression of erythropoiesis (Lee, Bunn, and Wintrobe, 1979). Alcohol may affect the kidneys by increasing renal excretion. This increased excretion could result in low serum phosphate and magnesium levels (Victor and Adams, 1979). Pancreatitis is also associated with alcohol abuse, and when enzymatic secretions are inadequate, digestion and nutrient utilization is impaired (Snodgrass, 1977). However, it has been postulated that the combined effects of alcohol and malnutrition may be the etiological factors in alcoholic pancreatitis. Pancreatic symptoms in alcoholics have been shown to reverse upon adequate feeding (Mezey et al., 1970).

The hepatotoxicity of alcohol is now well recognized. Alcoholism may produce fatty liver, hepatitis, and cirrhosis. Most frequently associated with alcoholism is fatty liver, but liver biopsy is required to establish this diagnosis (Mezey and Santora, 1979; Maddrey, 1981) and the condition is reversible (Mezey, 1980). Hepatitis must also be diagnosed with biopsy and is strongly associated with the development of cirrhosis (Galambos, 1972). Reversibility becomes more unlikely as the liver damage progresses (Maddrey, 1981).

The controversy concerning malnutrition's role in the eitology of liver disease has continued. Data supporting alcohol's toxic effect independent of nutritional deficiencies

was reported in man, rats (Lieber, Jones and DeCarli, 1965) and baboons (Lieber and DeCarli, 1974). Patek (1979), however, has raised the question concerning the role of nutritional imbalances in the development of liver disease.

Regardless of the etiology of liver disease, alterations in the liver result in alterations in nutrient metabolism. Amino acid metabolism, lipid synthesis, cholesterol synthesis, and carbohydrate metabolism all occur in the liver. The liver is also a storage site for many nutrients. The interactions between the direct toxicity of alcohol, changes in liver structure, and alterations in liver functions all have nutritional implications.

Thus, changes in intake, absorption of nutrients, and utilization of nutrients may all be results of alcohol abuse. Increased nutrient losses may result from organ damage secondary to alcohol abuse. Therefore, the alcoholic may have nutrient needs above those of other individuals.

Vitamin A

Vitamin A is a term representing all compounds exhibiting retinol's biological activity, excluding the carotenoids. Retinol, retinoic acid, and retinyl palmitate are the three compounds of vitamin A with which this paper is concerned.

Retinol is absorbed in the intestine and is esterified to retinyl esters. Retinyl esters, including retinyl

palmitate, are incorporated into chylomicrons which travel via the lymphatic system to the liver. The retinyl esters, most of which are retinyl palmitate, are then stored in the liver (Goodman, 1980). Vitamin A is transported from the liver to circulation in the form of retinol bound to the transport proteins retinol binding protein (RBP) and pre-albumin (PA) (Kanai, Raz, Goodman, 1968; Peterson, 1971).

Retinoic acid is absorbed through the portal system but does not accumulate in the liver. The metabolism of retinoic acid occurs rapidly, and end products are excreted in the urine and bile (Ganguly, 1969).

The vitamin A status of an individual is of course dependent on dietary intake. The hypovitaminosis A seen in developing countries has been attributed primarily to poor intake secondary to poor economic status (Perisse, Polacchi, 1979). If intake is adequate, the next area of concern is intestinal absorption. Because vitamin A is fat soluble, factors affecting fat absorption also affect vitamin A absorption. Such factors include decreased pancreatic lipase, decreased bile salts, and alterations of the intestine (Arroyave et al., 1959). Absorption can be accelerated by emulsification, and vitamin E appears to accelerate absorption as well as promote vitamin A utilization (Ames, 1969).

Because transport of vitamin A from the liver to tissues is dependent on carrier proteins, factors affecting

RBP and PA also affect vitamin A status. Low plasma levels of RBP, vitamin A, and PA found in liver diseases (Smith, Goodman, 1971) are assumed to reflect a reduced rate of protein synthesis (Smith, Goodman, 1979). Vitamin A supplementation in cases of marginal vitamin A status have been shown to be either beneficial (Russell et al., 1978) or ineffectual (Valquist, 1978). Children suffering from kwashiorkor have been shown to have a high incidence of vitamin A deficiency (Rao, Khan, 1974; Zaklama et al., 1973) as well as depressed RBP (Ingenbleek et al., 1975) which improves upon feeding adequate calories and protein.

In addition to functional alterations or protein-calorie malnutrition, other factors have been implicated in vitamin A depletion. Liver and serum vitamin A levels have been shown to be significantly reduced in male Sprague-Dawley rats fed adequate basal diets and a moderate level of alcohol (Yew et al., 1981). Sato and Lieber (1981) concluded from their studies on baboons and rats that chronic alcohol consumption may deplete hepatic vitamin A by mechanisms other than malnutrition or malabsorption. Russell et al. (1978) estimated the incidence of vitamin A deficiency in alcoholic cirrhotic patients to be fifty percent. This incidence was attributed to protein malnutrition, poor vitamin A intake, impaired vitamin A transport, poor hepatic uptake of vitamin A, and possibly poor zinc nutritional status (Russell, 1980).

Thus, vitamin A status seems to be affected by a variety of functional and metabolic interactions. Unfortunately, serum vitamin A levels only reflect extreme conditions of tissue depletion or saturation. Although the liver would be the best indicator of vitamin A status, it is usually not possible to assay in humans.

Nevertheless, vitamin A deficiency and toxicity are both reported. Vitamin A deficiency can result in degeneration of the retina, reproductive organs, and mucous secreting membranes as well as bone malformation (DeLuca and Wolf, 1969). Glycoprotein biosynthesis (Goodman, 1980) and normal hematopoiesis (Hodges et al., 1978) may also be dependent on vitamin A. Night blindness has been associated with vitamin A deficiency, but association between night blindness and visible degeneration of the eye has been reported to be poor (Gopalan et al., 1960).

Symptoms of vitamin A toxicity include hair loss, dry skin, cheilosis, gingivitisis, headache, anorexia, fatigue, muscle and bone pain (Muenter et al., 1971) as well as hepatic changes (Farrell et al., 1977). Case reports of vitamin A toxicity are numerous and show wide variation in symptoms. Weber et al. (1982) reported a case of massive hepatic vitamin A accumulation without other signs of hypervitaminosis. Smith and Goodman (1976) reported three cases of symptomatic hypervitaminosis A. Increased retinyl ester concentrations in these cases led to the hypothesis that toxicity may result

when the RBP capacity is exceeded. Farris and Erdman (1982) reported a case of hypervitaminosis A in which the vitamin A intake was not consistent with the severity and duration of symptoms, suggesting a vitamin A sensitivity. Another case report (Hatoff et al., 1982) describes hypervitaminosis A as being precipitated by viral hepatitis after ingestion of supplemental A. The mechanisms for vitamin A toxicity may therefore be as numerous and complicated as those leading to vitamin A deficiency.

Selenium

Selenium has been demonstrated to be an essential nutrient in many species (Committee on Dietary Allowances, 1980). Its essential nature in man has been proposed based on the association of selenium deficiency with various diseases. Children with kwashiorkor were found to have low whole blood selenium levels (Burke et al., 1967) and to respond to selenium supplementation with increased weight gain (Schwartz and Folz, 1957) and improved reticulocyte counts (Hopkins and Majay, 1967). Kreshan disease, a congestive cardiomyopathy, has been primarily attributed to selenium deficiency. The disease appears to be confined to China, as a result of a selenium deficient zone of soil (Keshan Disease Research Group of the Chinese Academy of Medical Sciences, 1979).

However, congestive cardiomyopathy in prolonged parenteral nutrition use has recently been thought to be due to selenium deficiency (Johnson et al., 1981). Collipp and Chen (1981) described in a case report a New York child with cardiomyopathy who showed favorable response to selenium supplementation.

Decreased selenium levels have also been found in alcoholic cirrhosis (Aaseth et al., 1980). The association of these diseases with selenium deficiency has led to the hypothesis that selenium deficiency may accelerate the progression of cardiomyopathy as well as the progression of liver disease (Goldman and Kantrowitz, 1981).

The mechanism of such a progression, however, is unclear. Selenium's major biochemical role appears to be as part of the enzyme glutathione peroxidase (Rotruck et al., 1973) which aids in protecting cell organelles from peroxides (Hoekstra, 1975). Many peroxides, including hydrogen peroxide and lipid hydroperoxides, can be reduced by the enzyme. Thus, a deficiency of selenium may decrease glutathione peroxidase activity and increase the destructive action of peroxides on organ cells (Goldman and Kantrowitz, 1981).

The Recommended Dietary Allowance for selenium has not been established. Selenium deficiency in animals can be prevented by selenium concentrations of 0.1 μg/gm feed (RDA, 1980). This concentration is present in the average mixed diet of Americans (Morris and Levander, 1970). However,

variation of selenium content in food is extensive, and geographically high and low soil areas exist throughout the world. Thus individual selenium intake is dependent not only on the type of food ingested, but also on the area in which the food is grown. For example, blood selenium levels were shown to change in New Zealand with the importation of Australian wheat (Watkinson, 1981).

However, selenium can also be toxic at high levels. Animal feed grown in certain parts of South Dakota is toxic to livestock (Moxon, 1937). Human selenium toxicity has also been proposed to occur in this state (Howe, 1979). Symptoms of selenium toxicity include garlic breath odor, dyspnea, tetanic spasms, and respiratory failure as well as congestion of the liver and kidneys, endocarditis, myocarditis, epicardium hemorrhages, gastric and intestinal hemorrhages (Lo and Sandi, 1980). Because of the potential toxicity of selenium, supplementation must be prudent.

Furthermore, the margin between selenium requirement and toxicity seems to be relatively narrow, although definite ranges have not yet been established (Lo and Sandi, 1980).

Methodology

Nutritional Assessment

Dietary Intake

Methods for obtaining past dietary intake include those which extend over long periods of time, those which attempt

to determine usual or typical intake, and those which cover only the immediate past. The method used, as well as the extent of data obtained, will vary with differences in study objectives. The objectives in collecting dietary information in this study initially were to determine previous usual intake, and later to determine the intake of the immediate past.

A diet history attempts to measure average nutrient intake and eliminate day-to-day or personal variation (Tilve and Raganen, 1981). Usual patterns of intake and frequency of intake of certain foods are also often included. The reliability and validity of using each method has been investigated. Christenson (1973) concluded from a study of young women that the dietary history questionnaire was reliable in assessing food habits. Reshef and Epstein (1972) also found reliability to be fairly good. However, Rasanen (1979) found significantly different mean intakes on repeated diet histories of children. The diet histories also resulted in consistently higher mean values for nutrient intake when compared to the 24-hour recall method.

The 24-hour recall method for determining previous intake is often used because of time, cost, and personnel restraints (Beal, 1980). Rasanen (1979) found this method reliable when used with groups of children. Shuran (1981) found the 24-hour recall method valid for groups of elderly when evaluated by the Wilcoxen test. It was noted, however,

that this evaluation method was not a very powerful test of validity.

Both of these methods depend on the accuracy of recall. Shuran (1981) also noted that for the elderly, memory of foods eaten decreased with passing time. Beal (1980) states that middle and upper class groups tend to be more cooperative, women give better information than men, and persons with stable lifestyles can verbalize intake more easily. Additionally, Stunkard and Waxman (1981) repeated the results previously reported in that persons tend to over report intake when it is low and under report when intake is high.

Thus, dietary information reliability and validity will depend on the individual, the sample group, and the method. Therefore, although dietary information is a useful component of a nutritional assessment, the information must be viewed in light of the limitations involved and results of other nutritional assessment parameters.

Anthropometrics

Anthropometric data is included in nutritional assessments in order to assess the adequacy of weight, body fat, and lean body mass (LBM). Normal parameters to include are height, weight, actual weight as a percentage of ideal weight, and actual triceps skinfold and mid-arm muscle circumference (MAMC) as a percentage of the standard value (Blackburn et al., 1977).

Ideal body weight may be determined from insurance company tables, or by the guidelines used by the American Diabetes Association. Ideal body weights derived from tables are based on cross-sectional studies and actually represent norms rather than ideals (Society of Actuaries, 1982). The tables also reflect height with shoes and weight with clothing on. The guidelines used by the American Diabetes Association are based on increments in weight in proportion to increments in height, and are calculated individually (Davidson, 1976).

Skinfold measurements have long been used to determine subcutaneous fat reserves (Keys and Brozek, 1953). In a clinical setting, the triceps skinfold is assumed to reflect fat reserves (Frisancho, 1981). The standards with which actual measurements are compared have continued to be evaluated. Jelliffe's (1966) standards have been criticized for being set on data collected at inappropriate times and on inappropriate samples (Frisancho, 1974). Percentiles for triceps skinfold measurements, as well as for upper arm muscle size, were developed from the United States Ten State Nutrition Survey of 1968-1970 (Frisancho, 1974). These percentiles were criticized for representing lower income groups and not representing subjects older than 44 years. Frisancho (1981) then developed standards based on the United States Health and Nutritional Examination Survey of 1971-1974 to overcome the previous criticisms. Although all

of these standards are used as ideals, they actually represent norms.

The mid-arm muscle circumference (MAMC) is assumed to reflect muscle reserve. Standards for the MAMC were developed each time new standards for the triceps skinfold measurement were developed. Assumptions associated with the derivation of the MAMC values must be noted. The derivation assumes that the upper arm is cylindrical. This assumption may lead to an overestimation of male muscle area because flattening of the arm is more prevalent in males than in females. Estimates of humeral bone diameter are not included in the MAMC derivations, and this may also contribute to overestimation of male muscle reserves (Frisancho, 1974). Skinfold compressibility also varies between individuals, but further research is needed to determine if these variations make significant differences in estimating nutritional status (Himes et al., 1979). Of course, errors may also occur from individual technique variation.

The validity of using skinfold measurements to determine body fat or lean body mass has been investigated by other methods. Barter and Forbes (1963) compared anthropometric data to potassium-40 counting data. They concluded that lean body mass was not correlated with circumferential or skinfold measurements. The average of biceps and triceps skinfolds were, however, correlated with weight, total body fat, and circumferential measurements. Piechaczek (1975) found that

body density, and thus body fat and LBM, may be predicted from a series of anthropometric data.

The distribution of body fat has been shown to change with age, with changes in weight, and differences exist between sexes (Siervogel et al., 1982). Durnin and Rahaman (1967) found significant correlations between skinfold thicknesses and body density measurements. They also recommended the use of four skinfold sites to better represent fat distribution and to diminish any errors in single measurements. However, Roche et al. (1981) found weight/stature^2 to be the best indicator of body fat in men when compared to body density values. Unfortunately, this study examined individual skinfold sites, not cumulative values. The cumulative values of biceps, triceps, subscapular, and suprailiac skinfold thicknesses were used by Durnin and Womersley (1974) to develop a table relating the skinfold values to the percent body fat. This table was developed from the logarithm of the total skinfold measurements to achieve a linear relationship with body density.

When estimated from potassium-40 counting, lean body mass has been shown to decline with age (Allen et al., 1960; Forbes and Reina, 1970). In view of this, a linear regression equation was recently developed to be used with the table of Durnin and Womersley (1974). The equation adjusts for lean body mass loss with increasing age (Tauss, 1981).

Considering the limitations of the use of anthropometric measurements to determine the adequacy of body fat and lean body mass, the objective of the clinician must be kept in mind. The objective of performing anthropometrics is to add another parameter to the nutritional assessment, so that a picture of the whole person may be obtained and evaluated.

Biochemical Determinations

Serum albumin maintains osmotic pressure in the plasma, and serves as a carrier of metals, ions, fatty acids, bilirubin, enzymes, drugs, and hormones. Nutrition is important in regulating albumin synthesis by the liver, and malnutrition can decrease synthesis rates by half within 24 hours. This decreased rate will be maintained as long as the malnutrition continues. Redistribution of pools can maintain normal serum albumin levels with decreased synthesis rates (Rothschild et al., 1972).

Anderson and Wochos (1982) found depressed albumin levels significantly associated with a longer hospital stay for nephrology patients. The visceral proteins, albumin and transferrin, were found to be the most sensitive indicator of postoperative morbidity and mortality (Mullen et al., 1979), although the authors stressed the importance of not relying on a single malnutrition indicator. Albumin levels have been found to be reliable indicators of malnutrition in the elderly (Mitchell and Lipschitz, 1982). Albumin

levels were found to be a better predictor of malnutrition than transferrin levels in a study comparing healthy and malnourished subjects, young and old, of both sexes (Mitchell and Lipschitz, 1982). However, albumin levels may be depressed in a variety of conditions, such as acute inflammation, chronic liver disease, or myeloma (Wallach, 1978). These depressed values may be due to the condition, or be compounded by malnutrition which is secondary to the condition.

Immunocompetence is affected by malnutrition (Chandra, 1972). Immunocompetence may be measured by a total lymphocyte count or by delayed hypersensitivity skin testing (Blackburn et al., 1977). However, cost and time limitations favor using the total lymphocyte count. Skin testing can be affected by other conditions, as well as the technique of testing (Miller, 1978). Twomey et al. (1982) concluded from an extensive literature review that the use of delayed hypersensitivity skin testing seems unfounded. As the total lymphocyte count is derived from the white blood cell number and the percent lymphocytes, abnormalities of either may misrepresent the total count.

It must be reemphasized that depressed values for either albumin or total lymphocyte count do not alone classify a person as malnourished. All parameters of the nutritional assessment must be viewed as a whole.

Liver Function Tests

Leakage of enzymes from liver cells, or increased enzyme synthesis, can occur with liver injury. However, enzyme leakage may also occur with conditions which change cell permeability, such as hypoxia. No quantitative correlation between liver injury and elevation of enzymes exist, but generally, higher elevations are associated with more advanced injury. The higher elevations of enzymes may be missed, however, if the measurement occurs after initial injury. In this case, enzyme levels may be normal or even low (Alpert and Isselbacher, 1979). Enzymes usually assayed include alkaline phosphatase, 5'-nucleotidase, SGOT, SGPT, lactic dehydrogenase, and GGT.

Alkaline phosphatase is produced by many tissues besides the liver. Most serum alkaline phosphatase is from the bone (Isselbacher and LaMont, 1979). Thus, serum levels may also be elevated in diseases of the bone (Wallach, 1978).

The enzyme 5'-nucleotidase can be used to check the etiology of alkaline phosphatase elevation, as it is usually normal in bone disease (Isselbacher and LaMont, 1979). However, Wallach (1978) stated that 5'-nucleotidase is elevated only in obstructive hepatobiliary disease. Eastham (1978) includes hepatic biliary obstruction, hepatitis, and rheumatoid arthritis as possible causes of elevation.

Glutamic-oxaloacetic transaminase (SGOT) occurs in all tissues. Glutamic-pyruvate transaminase (SGPT) is primarily

in the liver. Isselbacher and LaMont (1979) state that elevated SGOT and SGPT are present usually in liver disease. However, previous leakage may result in normal or depressed serum levels. SGOT and SGPT levels may also be elevated in acute myocardial infarction and acute pancreatitis, as well as other conditions (Wallach, 1978).

Lactic dehydrogenase is present in all organs, and is released under a variety of conditions (Isselbacher and LaMont, 1979), including myocardial infarction, muscle damage, pneumonia, and carcinomas (Eastham, 1978). Abraira et al. (1980) also found elevated lactic dehydrogenase and SGOT levels in fasted obese males, who did not have liver disease and who were not alcoholics.

Gamma glutamyl transpeptidase (GGT) appears to be most sensitive to minimal liver damage, and may show elevation before any other enzymatic test in heavy drinkers (Isselbacher and LaMont, 1979). GGT levels were found to be twice as high in a group of chronic alcoholics than in a control group (Ivanov et al., 1980). Reyes (1980) reported a positive correlation between GGT activity and the amount of alcohol consumed. However, GGT activity has also been found to be elevated in patients with hyperthyroidism (Azizi, 1982).

CHAPTER III

METHODOLOGY

Study Design

A quasi-experimental, correlational ex-post facto design was used in this study. Each participant was used as his own control; that is, follow-up data were assessed in view of initial data for that subject. In addition, all data gathered on admission were grouped to survey the nutritional profile of the alcoholics treated in this study.

Population and Sample

All veterans admitted to the alcohol admitting wards at the Danville, Illinois, Veterans Administration Medical Center during a specified three-week period were eligible to participate in the study if they had a diagnosis of alcohol dependency. All participants were drinking prior to admission, and were not drinking while in the hospital.

Of the 45 admissions during the three weeks of the study, 27 patients were willing and able to participate. Of these 27 subjects, only 20 were available for the two-week follow-up. In the group of 27, all subjects were male, ranging in age from 20 to 68 years.

Instruments and Procedures

Each subject's weight was taken on a balance beam scale. Height was measured without shoes with an adjustable ruler attached to the scale. Skinfolds were taken with Lange calipers at four sites: triceps and biceps were taken midway between the olecranon and acromiun processes; subscapular skinfold was taken approximately one-half inch below the tip of the scapula; suprailiac skinfold was taken directly above the iliac crest. All skinfolds were taken in a series of three readings which were averaged to obtain one value for each site. To assure reliability, the series of three were repeated until the range of readings for each site was no greater than one millimeter. Arm circumference (MAC) was taken midway between the olecranon and acromiun processes. The mid-arm muscle circumference (MAMC) was derived from the equation MAMC=MAC-(.314 x triceps skinfold). Derivation of body fat and lean body mass was computed as described by Durnin (1967). Predicted body fat and lean body mass were assumed to be 15 and 85%, respectively. These predicted values for body fat and lean body mass were adjusted for age in persons over 50 years (Tause, 1981).

Records of usual intake and frequency of food eaten were taken by individual interview, and recorded on standard Veterans Administration (VA) forms (Appendix A). Anthropometrics and diet histories were taken by the investigator.

Albumin, glutamin-oxaloacetic transaminase (SGOT), glutamic-pyruvate transaminase (SGPT), and lactic dehydrogenase (LDH) determinations were made either with the CentriChem or the Dupont Automatic Clinical Analyzer using their respective manufacturer's methodology. Gamma glutamyl transpeptidase (GGT) determinations were made using the CentriChem analyzer. White blood cells were determined using the Model S Coulter and the Coulter Counting Principle. The percent lymphocytes were determined by a peripheral blood smear. All above laboratory determinations were performed by the medical center laboratory staff.

Retinol binding proteins were determined by radial immunodiffusion using the manufacturer's procedure (Calbiochem-Behring Corp., 1979). Vitamin A was determined using high performance liquid chromatography (Besner and LeClaire, 1980). Selenium levels were determined using gas chromatography (McCarthy et al., unpublished). These procedures were performed by the investigator and graduate students at the University of Illinois, Champaign-Urbana.

The investigator was notified of new admissions to the alcohol rehabilitation wards by the medical center gains and losses sheet each morning. All admissions to these wards during the specific three weeks were contacted by the investigator. If upon initial contact the patient was willing to participate in the study, appropriate consent forms were completed (Appendix B). Data were collected approximately

24-48 hours after admission to the medical center and again in approximately two weeks. Deviations from these time schedules were due to patient inaccessibility or staff schedule demands.

There was no alteration in the treatment of the patient because of participation in the study. All patients received multivitamins as prescribed by their physician, as well as other medications which their physician may have prescribed. All patients except two received a regular diet. One patient was receiving a sodium restricted diet and one patient was receiving a sodium and calorie restricted diet. All patients ate in the dining room after being served cafeteria style.

The data collected included dietary intake, liver function tests, anthropometrics, vitamin A levels, and selenium levels. Of the anthropometrics, only weight was reassessed after two weeks because skinfold determinations are not sensitive enough to detect changes within two weeks. Repetition of usual hospital laboratory tests was performed only at the discretion of the patient's primary physician, and therefore not included in the two week follow-up data. The physician did order selenium and vitamin A determinations after two weeks.

Patients were scheduled for initial laboratory tests either the morning after admission or the next working day, according to hospital procedures. Additional vials of blood were drawn by the hospital staff for vitamin A and selenium

determinations. These vials were appropriately labelled and frozen until transport to the University of Illinois laboratories.

Data Analysis

Diet histories and 24-hour recalls were evaluated for protein and caloric content using food composition tables (Pennington and Church, 1980; Adams, 1975). Food frequency lists were evaluated using the Basic Four Food Group Recommendations. All dietary information was recorded on VA forms (Appendix A).

Anthropometric data was recorded on a nutritional assessment survey form (Appendix C). Body weight was compared to ideal body weight (IBW) as recommended by the American Diabetes Association (Davidson, 1976) and expressed as a percentage of IBW. Triceps skinfold (TSF), mid-arm circumference (MAC), and mid-arm muscle circumference (MAMC) were compared to the standards of both Jelliffe (1966) and HANES (Frisancho, 1981) and expressed as a percentage of each standard. Body fat and lean body mass (LBM) as calculated from four skinfolds were compared to predicted values based on IBW. Fifteen percent was considered to be the predicted amount of body fat, with adjustments made for those over 50 years of age (Tauss, 1981). Body fat and LBM were expressed in kilograms as the difference from predicted values.

The normal range for serum albumin is 3.5-5.5 gm/dl for the Danville, Illinois, Veterans Administration Medical Center (DVAMA). Total lymphocyte counts below 1500 mm^3 were considered significantly depressed.

A subject was considered malnourished if all components classifying the type of malnutrition were met. Marasmus is defined as less than 80% IBW, less than 80% standard for TSF and MAMC, and having normal albumin and total lymphocyte count values. Kwashiorkor is defined as having normal anthropometrics, depressed albumin levels, and depressed total lymphocyte counts. Marasmus-kwashiorkor is defined as having depressed anthropometrics, depressed albumin levels, and depressed total lymphocyte counts (Weinsier et al., 1977).

The Student's T test was used to determine significant changes between initial and follow-up values for vitamin A, RBP, and selenium. The Pearson's correlation coefficient was used to determine significant correlations between vitamin A, RBP, and selenium and results of liver function tests.

Biochemical test normal ranges usually allow for 5% of the population to be outside of this normal range. Therefore, no more than 5% of the test results should be outside of the normal range if the sample is representative of the population. Significant deviation from the normal range was analyzed by the one-sided binomial exact test using Poisson approximation.

Assumptions

1. Alcoholics participating in this study represent the alcoholic population of Veterans in central Illinois who are not in acute medical distress.
2. The biochemical determinations used in this study are valid and reliable.
3. Anthropometrics taken in this study are reasonably valid and reliable.
4. Persons diagnosed as alcohol-dependent are truly alcoholics.
5. Published normal laboratory values and the medical center's normal values are reasonable standards to use for comparison in this study.

Limitations

1. Information obtained from the subject, e.g., diet history, may not be valid, reliable, or accurate because of poor memory, unwillingness to cooperate fully, or personal attitude. Also, if the subject is a binge drinker, eating habits while drinking and not drinking may be drastically different and difficult to describe.
2. All patients consecutively admitted for the duration of the study could not be included in the study because of either unwillingness to participate, inability to comprehend the study in order to sign consent, or inability of

the investigator to contact the subject within 24 hours of admission.

3. Some subjects could not be reevaluated after two weeks either because of lack of cooperation or discharge from the hospital.
4. Some evaluations could not be repeated exactly two weeks after initial evaluation because of staff and time constraints.
5. Weight could not be taken in the nude, but was taken with a minimum of clothing.
6. Actual body composition as determined by body density or labelled potassium counting could not be made because of a lack of necessary equipment. However, the method of body composition determination utilizing four skinfolds has been reported to have a good correlation with densiometric techniques.
7. Actual nutrient intakes of subjects while in the hospital could not be determined because of lack of time, personnel, and accepted hospital procedures.
8. Selection of subjects could not be randomized because each person had a choice in participating, time constraints for collecting serum for batch analysis, staff cooperation, and work schedules.
9. For patients seen twice, each subject served as his own control. However, for patients seen only on admission,

no control group exists and these persons must be compared to published or hospital norms.

10. Two weeks may not be the optimal time span to assess change in nutrient status. However, due to patient average length of stay, a longer time span could not be used.

CHAPTER IV

RESULTS AND DISCUSSION

Nutritional Assessment

Dietary Intake

Twenty-three dietary histories were obtained within 72 hours of admission to the medical center to determine previous usual intake. Caloric intake ranged from 651 kcal/day to 3860 kcal/day, with a mean intake of 1472 kcal/day. Protein intake ranged from 22.5 gm/day to 217.5 gm/day, with a mean of 88.8 gm/day. Protein intake per kilogram (kg) of actual body weight ranged from .25 gm/kg/day to 2.99 gm/kg/day. Only three subjects (6, 9, 10) met or exceeded the RDA range for caloric intake, but only three subjects (4, 18, 21) did not meet or exceed the RDA for protein intake. However, alcohol intake was not included in the caloric intake estimates (Table 1).

When food frequency lists were compared with the Basic Four Food Groups, the recommended intake for the fruit and vegetable group was frequently not met. Twenty-two subjects (1-20, 22, 23) failed to meet the recommended intake of four servings for this group. The recommended intake of two servings per day from the milk group was not met by 17 subjects (1, 2, 5-7, 10, 11, 13-23). The recommended intake of four servings per day from the bread group was not met by 8 subjects (3, 4, 14, 18-21, 23), and the recommended intake

Table 1

Body Weight and Calorie and Protein Intake, Initial and Follow-up

Subject	Weight lbs.	Weight kg	% IBW	Initial kcal/day	Initial Protein gm/kg/day	Follow-up kcal/day	Follow-up Protein gm/kg/day	Follow-up Weight lbs.	Follow-up Weight kg	Follow-up % IBM
1	134	60.9	79	1034.5	1.98	1781.5	1.65	137	62.3	81
2	95.5	43.4	79	1169.3	1.92	1869	1.84	103	46.8	85
3	115	52.3	83	1557.5	1.45	N/A*	N/A	N/A	N/A	N/A
4	137.5	62.5	88	789.3	.85	2012.5	.97	143	65	91
5	154	70.0	94	2103.3	1.90	1984.5	1.66	154	70	94
6	164.5	74.8	95	2321.6	1.28	2496.3	1.35	167.5	76.1	96
7	162	73.6	95	1197.3	1.30	N/A	N/A	N/A	N/A	N/A
8	134	60.9	97	1425.5	1.31	2031.5	1.65	136	61.8	99
9	181	82.3	98	2779.3	1.64	N/A	N/A	N/A	N/A	N/A
10	160	72.7	99	3859.8	2.99	2515	1.44	161	73.2	101
11	180.5	82.0	101	1320.3	1.09	N/A	N/A	N/A	N/A	N/A
12	158	71.8	103	1293	.99	1118	1.24	158	71.8	103
13	174	79.1	107	1427.8	.94	1313	.45	175.5	79.8	108
14	193	87.7	114	845.3	.67	2795.3	1.23	N/A	N/A	N/A
15	193	87.7	118	1691.8	1.14	2814.5	1.56	199	90.5	122
16	178	80.9	118	1019.8	.83	N/A	N/A	N/A	N/A	N/A
17	175	79.5	119	1204.5	.79	2587.5	1.45	182	82.7	124
18	187	85.0	124	651.3	.33	2729.5	1.49	190.5	86.6	126
19	206	93.6	124	1266	.68	1856.5	.99	208	94.5	125
20	181.5	82.5	126	1695.6	1.68	N/A	N/A	N/A	N/A	N/A
21	197	89.5	132	778	.25	2262	1.14	195	88.6	130
22	207	94.1	146	1359.8	1.06	1278	.65	207.5	94.3	146
23	280	127.3	178	1067.8	.59	N/A	N/A	N/A	N/A	N/A

*N/A = not available.

of two servings per day from the meat group was not met by subject 18. Questionable intake was found for subject 3 from the meat group and subject 18 from the milk group because of large variation in their usual intake.

Sixteen 24-hour recalls were obtained two weeks after admission to determine intake while in the medical center. Discharge or patient noncompliance precluded obtaining information from five subjects. Calculated estimated calorie and protein intakes are listed in Table 1. Calorie intake ranged from 1118 kcal/day to 2814 kcal/day, with a mean of 2090 kcal/day. Protein intake ranged from 36 gm/day to 141 gm/day, with a mean of 89 gm/day. Protein intake per kilogram body weight ranged from 0.45 gm/kg to 1.84 gm/kg.

Eleven subjects (1, 2, 4, 6, 8, 14, 15, 17-19, 21) increased their estimated caloric intake during these two weeks, while five (5, 10, 12, 13, 22) decreased caloric intake. Similarly, eleven subjects (2, 4, 6, 8, 12, 14, 15, 17-19, 21) increased their protein intake, while five (1, 5, 10, 13, 22) decreased protein intake. However, of those decreasing caloric intake, two (13, 22) were on restricted diets. Both were on sodium restrictions and one was also on a calorie restriction. Changes in protein intake generally paralleled changes in caloric intake, except in two cases. One subject's caloric intake increased with a decrease in protein intake, while another's caloric intake decreased with an increase in protein intake.

Generally, initial histories revealed inadequate caloric intake and adequate protein intake. Caloric intake improved in the medical center during the two week interval.

Anthropometrics

Initial body weights ranged from 79-178% ideal body weight, with a mean of 109% for 23 subjects. Weights taken approximately two weeks later ranged from 81-146% ideal body weight (IBW) for 15 subjects, with a mean of 109% (Table 1). However, body weights generally increased, with the exceptions of three subjects (5, 12, 22) who remained the same, and one subject (21) who lost weight. The two subjects who were initially 79% IBW may be considered deficient in weight (1, 2).

Triceps skinfold values ranged from 40-212% of Jelliffee's standard and 45-241% of the HANES standard (Table 2). Means were 98.1% and 111.8%, respectively. Using 80% standard as the value above which is adequate, and below which is inadequate, nine subjects compared to Jelliffe's standard and seven subjects compared to the HANES standard were evaluated as inadequate in fat reserves.

Mid-arm circumference values ranged from 81-165% of Jelliffe's standard and 74-114% of the HANES standard (Table 2). Means were 106% and 96%, respectively. Using 80% as the value above which is adequate, and below which is inadequate, subject 2 was below 80% of the HANES standard.

Table 2

Anthropometric Values and Percent Standard

Subject	TSF (mm)	HANES %	JELLIFFE %	MAMC (cm)	HANES %	JELLIFFE %	MAC (cm)	HANES %	JELLIFFE %	Body Fat (kg)	LBM * (kg)
1	10.3	94	82	22.2	80	88	25.4	80	87	+2.0	-17.9
2	6.8	62	54	21.5	77	85	23.6	74	81	-3.6	-8.0
3	6.7	61	54	25.4	91	100	27.5	87	94	-1.7	-9.2
4	10.2	93	82	22.8	82	90	26.0	82	89	+1.3	-10.2
5	6.8	62	54	28.1	101	111	30.2	95	103	+8.0	-12.5
6	8.3	75	66.4	28.1	101	111	30.7	96	105	+15.8	-20.1
7	13.7	125	110	26.6	95	105	30.9	97	105	+4.7	-8.6
8	6.8	62	54	24.0	86	95	26.1	82	89	-0.9	-0.7
9	10.0	91	80	32.0	115	126	35.1	111	120	+5.7	-7.0
10	9.2	84	74	27.9	100	110	30.8	97	105	+3.3	-4.0
11	12.5	114	100	28.2	101	111	32.1	102	110	+11.8	-10.7
12	9.0	82	72	22.8	82	90	25.6	81	87	+3.2	-1.4
13	5.0	45	40	27.6	99	109	29.2	92	100	+1.8	-1.9
14	25.3	230	202	27.4	98	108	35.3	110	120	+9.9	+1.0
15	11.3	103	90	29.7	106	117	33.2	104	113	+10.1	+3.5
16	10.7	97	85.6	28.5	102	113	31.9	101	109	+10.4	+1.9
17	16.5	150	132	27.0	97	107	32.2	101	110	+13.6	-0.7
18	7.7	70	61.6	30.6	110	121	33.0	104	113	+11.0	+5.4
19	15.8	144	126	28.8	103	114	33.8	107	115	+13.1	+5.0
20	12.7	115	102	25.6	92	101	29.6	93	101	+10.7	+6.6
21	14.5	132	116	25.2	90	100	29.8	94	102	+14.8	+6.7
22	25.8	235	206	25.7	92	102	33.8	107	115	+22.3	+7.3
23	26.5	241	212	27.9	100	110	36.2	114	165	+38.4	+17.2

*LBM, body fat = kg above or below predicted.

Mid-arm muscle circumference (MAMC) values ranged from 85-126% and 77-115%, using the Jelliffe and the HANES standards, respectively. Means were 105% using the Jelliffe standard and 96% using the HANES standard. Again, using 80% as a marker of significance, subject 2 was considered inadequate in muscle reserves, using the HANES standard.

Comparison of actual body weight in kilograms, body fat, and lean body mass (LBM) with predicted body weight, body fat, and LBM was made based on the four skinfold method. Thirteen subjects (11-23) had body weights above the predicted, or ideal, weight. All thirteen also had more body fat than was predicted for their age and weight. Four of these (11, 12, 13, 17) had LBM values below the predicted values, indicating that fat was replacing lean. Ten subjects (1-10) had body weights below the predicted value, and all ten had LBM values below the predicted value. Three (2, 3, 8) also had body fat below the predicted value (Table 2). This indicates that for most of those below IBW, the deficit was in lean, not in fat.

Biochemical Data

Serum albumin levels were all within normal limits of 3.5-5.5 gm/dl. Total lymphocyte counts were within normal limits, being above 1500 mm^3, with the exception of subject 20, in which it was significantly depressed. That reading was 1170 mm^3.

Vitamin A

Initial retinol values ranged from 22-412 µg/100 ml, with a mean of 159.6 µg/100 ml for 26 subjects. Retinol values from 19 subjects taken approximately two weeks later ranged from 33-259 µg/100 ml, with a mean of 95 µg/100 ml. The normal range for retinol is 50-100 µg/100 ml. Three initial values (subjects 14, 12, 21) and two follow-up values (subjects 19, 21) were below this range. Eighteen initial sample values and four follow-up values were above this range (Table 3). For the initial values, 21 of 26 subjects' values were outside of the normal range. Using the one-sided binomial exact test with Poisson approximation, there was a 99.2% probability that no more than four subjects would be outside of the normal range.

Initial RBP values ranged from 0.6-6.3 mg/100 ml, with a mean of 3.7 mg/100 ml for 24 subjects. Follow-up on 17 subjects gave a range of 0.9-5.25 mg/100 ml, with a mean of 3.6 mg/100 ml. The normal range for RBP is 3-6 mg/100 ml. Ten initial values and four follow-up values were below this range. One initial value was above this range (Table 3). For the initial values, 10 of 24 subjects' values were outside of the normal range. Using the one-sided binomial exact test with Poisson approximation, there was a 99.2% probability that no more than four subjects would be outside of the normal range.

Table 3

Initial and Follow-up RBP and Retinol Levels, and Liver Function Tests

Subject	1st (mg/100 ml) RBP	2nd RBP	1st (µg/100 ml) ROL*	2nd ROL	alb. (gm/dl)	SGOT	SGGT	LDH	SGPT
1	4.92	3.12	98	67	4.2	36	79	90	17
2	3.96	2.46	112	92.5	4.3	29	38	83	11
3	N/A**	N/A	114	N/A	5.0	19	9	66	9
4	N/A	3.3	245	80	5.0	182	164	143	33
5	4.92	4.92	257	72	4.8	72	414	72	28
6	2.64	2.4	153	69	5.0	60	156	82	17
7	N/A	N/A	N/A	N/A	4.5	23	24	87	9
8	4.92	N/A	163	124	4.5	48	32	87	26
9	5.28	N/A	293	N/A	4.9	N/A	N/A	N/A	N/A
10	2.34	3.3	224	79	5.3	43	40	89	25
11	5.16	4.5	328	93	4.9	12	23	72	10
12	2.16	0.9	35	53	3.8	87	463	92	19
13	6.3	4.92	203	64	4.7	66	24	107	18
14	3.24	3.3	49	74	4.6	222	309	186	54
15	5.64	N/A	412	223	4.8	90	403	166	46
16	4.6	N/A	113	N/A	4.9	169	27	116	62
17	3.6	5.04	183	259	5.1	22	39	77	10
18	2.7	3.84	61	85	5.1	15	26	68	10
19	2.1	N/A	51	40	3.9	66	94	157	13
20	2.1	N/A	161	N/A	4.7	35	18	90	13
21	2.94	1.38	22	33	3.6	79	233	104	28
22	2.4	4.92	113.5	129.5	4.3	34	24	114	17
23	4.68	5.25	318	93.5	4.7	26	34	92	13
24	0.6	N/A	110	N/A	4.9	67	90	128	22
25	1.94	N/A	113	N/A	4.8	26	29	82	9
26	4.56	N/A	88	N/A	4.7	62	698	69	45
27	4.92	3.84	107	75	4.8	50	N/A	90	19

*ROL = retinol **N/A = not available

Retinoic acid and retinyl palmitate was found in several subjects. However, actual values are not reported because the analytical technique for quantification is not sensitive enough to yield high reproducibility.

Correlations between vitamin A and the liver function tests are listed in Table 4. Significant correlation ($p \leq 0.05$) was found for initial retinol values vs. albumin ($r=.333$), follow-up retinol values vs. albumin ($r=.4006$), and follow-up RBP values vs albumin ($r=.5688$). Correlations between retinol and albumin levels may be a reflection of the stoichiometric relationship between RBP and retinol. Although this relationship does not exist for albumin and retinol, albumin and RBP production and secretion are similarly regulated by the liver. RBP has a greater sensitivity to changes in normal metabolism because of a shorter half life than albumin. This may explain the lack of correlation between initial RBP and albumin.

Lack of correlation between the liver function tests and vitamin A levels may be due to the sample size and its diversity. Correlations may have been found to be significant if subgroups of low, normal, and elevated vitamin A levels could have been compared to the liver function test results.

The Student's T test was used to analyze differences between initial and follow-up values for retinol and RBP. Retinol T_0 was significant at 2.64, but RBP was not

Table 4

Retinol and RBP Correlations with
Liver Function Tests

	p	r
Initial retinol samples		
vs SGOT	.4786	-.0995
vs SGPT	.6850	.0576
vs LDH	.9417	.0106
vs GGT	.9496	.0092
vs albumin	.0158	.333
Follow-up retinol samples		
vs SGOT	.269	-.1839
vs SGPT	.6581	.0752
vs LDH	.8268	.0367
vs GGT	.7439	-.0564
vs albumin	.0127	.4006
Initial RBP samples		
vs SGOT	.7921	.0568
vs SGPT	.1825	.2816
vs LDH	.5699	-.1221
vs GGT	.3703	.2008
vs albumin	.5291	.1351
Follow-up RBP samples		
vs SGOT	.2961	-.2786
vs SGPT	.7968	-.07
vs LDH	.6799	-.119
vs GGT	.0718	-.4775
vs albumin	.0215	.5688

significant at 2.7 ($p \leq 0.01$). Thus, the major changes seen during the two weeks were in the retinol levels.

Selenium

Initial blood selenium values ranged from 0.032-0.283 ppm, with a mean of .135 for 27 samples. Follow-up blood selenium values ranged from 0.07-0.305 ppm, with a mean of 0.158 ppm for 20 samples. Initial plasma selenium values ranged from 0.051-0.234 ppm with a mean of 0.92 ppm for 25 samples. Follow-up plasma selenium values ranged from 0.054-0.136 ppm, with a mean of 0.089 ppm for 15 samples (Table 5).

The normal range for blood selenium is 0.106-0.175 ppm. Ten initial values and six follow-up sample values were below this range. Six initial sample values and five follow-up sample values were above this range. For the initial values, those of 15 of 27 subjects' values were outside of the normal range. Using the one-sided binomial exact test with Poisson approximation, there was a 99.2% probability that no more than four subjects would be outside of the normal range. The normal range for plasma selenium is 0.097-0.142 ppm. Seventeen initial values and ten follow-up sample values were below this range. Two initial sample values were above this range. For the initial values, 18 of 27 were outside of the normal range. Using the one-sided binomial exact test again, there was a 99.2% probability that no more than four subjects would be outside of the normal range.

Table 5

Initial and Follow-up Plasma and Whole Blood Selenium Levels and Liver Function Tests

Subject	1st bl. Se.	2nd bl. Se.	1st pl. Se.	2nd pl. Se.	alb.	GGT	SGOT	SGPT	LDM
1	.129	.121	.126	N/A*	4.2	79	36	17	90
2	.079	.097	.051	.134	4.3	38	29	11	83
3	.113	N/A	.234	N/A	5.0	9	19	9	66
4	.251	.085	.067	.115	5.0	164	182	33	143
5	.102	.305	.130	.069	4.8	414	72	28	72
6	.078	.117	.087	.136	5.0	156	60	17	82
7	.121	N/A	.077	N/A	4.5	24	23	9	87
8	.136	.112	.052	.074	4.5	32	48	26	87
9	.045	N/A	.105	N/A	4.9	311	N/A	N/A	N/A
10	.110	.070	.089	.065	5.3	40	43	25	89
11	.15	.099	.158	.129	4.9	23	12	10	72
12	.190	.157	.066	.088	3.8	463	87	19	92
13	.032	.173	.077	N/A	4.7	24	66	18	107
14	.099	.146	.097	.086	4.6	309	222	54	186
15	.185	.178	.101	.112	4.8	403	90	46	166
16	.156	N/A	.079	N/A	4.9	27	169	62	116
17	.121	.290	.086	.055	5.1	39	22	10	77
18	.093	.245	.077	.069	5.1	26	15	10	68
19	.141	.098	.056	.077	3.9	94	66	13	157
20	.283	N/A	.074	N/A	4.7	18	35	13	90
21	.096	.138	N/A	N/A	3.6	233	79	28	104
22	.087	.084	.096	.067	4.3	24	34	17	114
23	.122	.176	.067	N/A	4.7	34	26	13	92
24	.219	.285	N/A	N/A	4.9	90	67	22	128
25	.163	N/A	.099	N/A	4.8	29	26	9	82
26	.219	N/A	.077	N/A	4.7	698	62	45	69
27	.093	.187	.079	.054	4.8	N/A	50	19	90

*N/A = not available.

Correlations between selenium and the liver function tests were not significant ($p \leq 0.05$) (Table 6). However, this lack of correlation may again be due to the sample size and its diversity. Correlations may have been found if subgroups of low, normal, and elevated plasma and whole blood selenium values could have been compared to the results of the liver function tests. The Student's T test was used to analyze differences between intial and follow-up samples. Neither whole blood nor plasma selenium differences were significant ($p \leq 0.01$).

Hypothesis One

THE NUTRITIONAL STATUS OF ALCOHOLICS AT DVAMC DOES NOT DIFFER SIGNIFICANTLY FROM THAT OF THE GENERAL POPULATION AS MEASURED BY ANTHROPOMETRICS, DIET HISTORIES, AND BIOCHEMICAL TESTS OF VISCERAL PROTEIN STATUS AND IMMUNOCOMPETENCE.

Findings of the study included generally inadequate caloric intake and adequate protein intake. Although the initial diet histories revealed inadequate caloric intake, alcohol's contribution to the total caloric intake was not included. Inclusion of alcohol may have raised caloric intake to acceptable levels. However, evidence exists to support the hypothesis that alcohol's caloric value is not constant at 7 kcal/gm. Despite isocaloric diets, Lieber et al. (1965) found less weight gain in animals fed ethanol containing diets. It was proposed that this difference may be due to an effect of alcohol on food utilization, or

Table 6

Plasma and Whole Blood Selenium Correlations with Liver Function Tests

	p	r
Initial blood selenium		
vs SGOT	.3436	.1896
vs SGPT	.4392	.1553
vs LDH	.3479	.1879
vs GGT	.3336	.1975
vs albumin	.772	-.0573
Follow-up selenium		
vs SGOT	.6223	-.1173
vs SGPT	.8077	-.0581
vs LDH	.4352	-.1849
vs GGT	.2445	.2806
vs albumin	.2094	.2934
Initial plasma selenium		
vs SGOT	.4049	-.1782
vs SGPT	.5664	-.1204
vs LDH	.2571	-.2355
vs GGT	0	0
vs albumin	.1463	.2992
Follow-up plasma selenium		
vs SGOT	.6085	.1441
vs SGPT	.8658	.0477
vs LDH	.7393	.0939
vs GGT	.8184	.0676
vs albumin	.9244	-.0268

decreased availability of alcoholic calories. Lieber (1975) later used hypotheses on hepatic cellular metabolism of alcohol to further explain alcohol's caloric contribution: increased activity of the microsomal enzymes may lead to heat production without conservation of chemical energy.

The validity of the histories themselves may also be responsible for apparent inadequacies. Under-reporting of alcohol intake has been a concern of other investigators (Hurt et al., 1981), occasionally to the extent of incorrectly classifying alcoholics as nonalcoholics (Armor et al., 1976). However, Eagles and Longman (1963) found no significant difference between self-reported dietary intakes and those reported by the families of the participants.

Comparison of reported caloric intake and body weight in this study revealed a discrepancy due either to errors in reported intake or exclusion of alcohol's caloric contribution. While only two subjects initially met or exceeded the RDA for caloric intake, only two subjects were below 80% ideal body weight. This discrepancy may also be partially due to the standards used. The RDA is not meant to be used for individual evaluation, and ideal body weight may not reflect usual weight.

Increased reported caloric intake two weeks later did agree with generally increasing weight. This change may be due to actual caloric increase, substitution of nutrients for alcohol, or decreased activity while in the medical

center. However, caloric intake for ten subjects remained below the RDA range for calories.

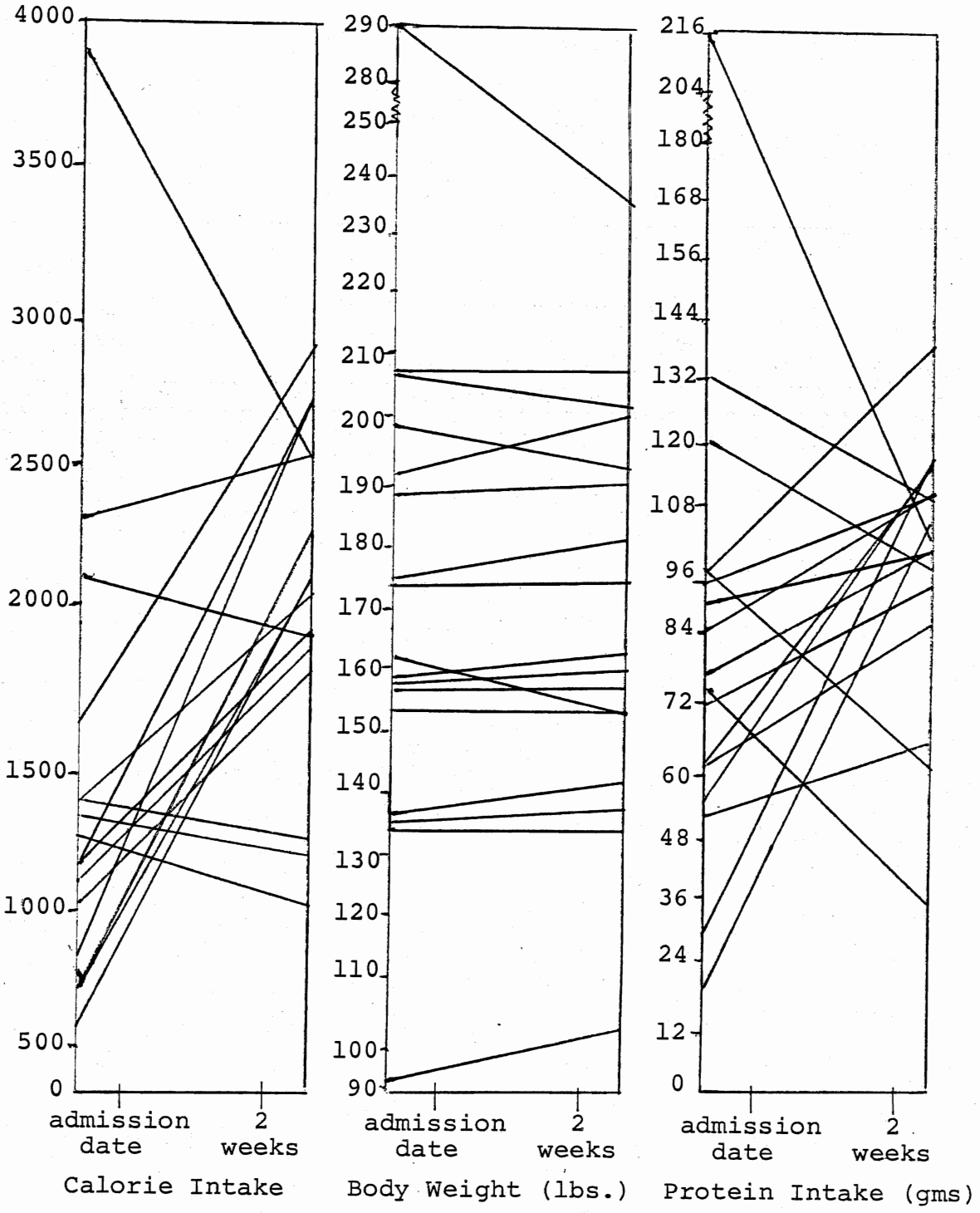
All subjects except five (subjects 5, 10, 12, 13, 22) increased their reported caloric intake (Figure 1). However, subjects 10 and 13 increased in body weight, indicating errors in reporting. Of those increasing their caloric intake, only subject 21 lost weight. This case also indicates errors in reporting intake. Of the six subjects more than 15% above IBW for whom follow-up was possible, four increased in body weight (Table 1).

Protein intake was generally adequate on initial and follow-up reports, with mean intakes differing only by 0.6 gm proten/day. Initial food frequency reports substantiated this, with only one subject not meeting the recommended intake from the meat group.

Protein intake reported per kilogram of actual body weight decreased for six subjects. However, four of these had increased body weight (subjects 1, 2, 10, 13). Protein intake for subject 1 decreased from 120.8 gm to 102.5 gm, increased for subject 2 from 83.5 gm to 86.3 gm, decreased for subject 10 from 217.5 gm to 105.3 gm, and decreased for subject 13 from 74 gm to 36 gm. Subjects 5 and 22 maintained their body weight despite decreasing protein intake. Protein intake per kilogram actual body weight increased for all other subjects with concurrent increases in body weight except for two subjects. Subject 12 maintained his weight

Figure 1

Changes in Reported Calorie Intake, Body Weight, and Reported Protein Intake With Time



at 103% IBW. Subject 21 lost two pounds in two weeks, decreasing his weight from 132% IBW to 130% IBW.

Although examination of calories and nutrients besides protein may have been enlightening, exactness of foods consumed could not be recalled. Nutrient variation in specific foods is also large, and may have negated the value of such an examination. Dietary recall methods have been shown to have low predictive value of biochemical measures for several nutrients, including vitamin A (Kerr et al., 1982). Therefore, dietary intake information was only examined for general adequacy, not for specific adequacy.

Seven or nine subjects could be classified as having inadequate fat reserves as measured by the TSF, depending on which standard was used. When four skinfolds were used to assess body fat, only three subjects were below their predicted body fat reserves. Subjects 2, 3, and 8 were -3.6, -1.7, and -0.9 kg body fat, respectively. The TSF for these subjects were 54% of the Jelliffe standard. Subjects 5, 6, 9, 10, 12, and 13 were 80% or below Jelliffe's standard for TSF, but body fat assessed by four skinfolds were above the predicted values. These differences may be due to variation in body fat distribution (Table 2).

Perhaps one of the subjects could be classified as having inadequate somatic muscle reserves as measured by MAMC, depending on which standard is used. However, fourteen subjects were below predicted LBM using four skinfolds.

Barter and Forbes (1963) found LBM not to be correlated with circumferential or skinfold measurements when compared to potassium-40 data. However, only two skinfold site measurements were averaged by these authors. Densiometric and skinfold measurements have been highly correlated (Piechaczek, 1975) and found to be predictive of LBM, although six skinfold sites were found to be most predictive of LBM. Lack of correlation between MAMC values and LBM in this study may reflect a confusion in terminology. MAMC is reported to reflect somatic muscle. LBM includes somatic muscle as well as all body parts, excluding subcutaneous fat.

Serum albumin levels were normal in all subjects. This further substantiates adequate protein intake. Depressed serum albumin levels have been found to be significantly associated with longer hospital stay (Anderson and Wochos, 1982), malnutrition (Mitchell and Lipschitz, 1982), and postoperative morbidity and mortality (Mullen et al., 1979). Although reliance on one indicator of malnutrition is not advisable, lack of albumin depression in the subjects indicates a lack of general protein malnutrition.

Although the total lymphocyte count was within normal range for all but one subject, lymphocyte counts alone have lacked predictive value for determining malnutrition (Mullen et al., 1979).

When the nutritional assessment of each subject was analyzed as a whole, none of the subjects could be classified

as having kwashiorkor. A subject would have been classified as having kwashiorkor if protein intake was poor, albumin was depressed, and total lymphocyte count was depressed (Weinsier et al., 1977). One subject, 2, was classified as moderately marasmic on admission. Body weight was less than 80% IBW, TSF was below 80% of both standards, MAMC and MAC were below 80% of the HANES standard only, albumin and total lymphocytes were within normal limits. However, this subject's weight increased above 80% IBW within two weeks. This subject's absolute protein intake had increased from 83.5 gm to 86.3 gm protein/day. However, protein/kilogram body weight decreased from 1.92 to 1.84. This may have been responsible for the fall in RBP from 3.96 to 2.46 mg/100 ml.

As only this one subject could be classified as malnourished, the null hypothesis could not be rejected: the nutritional status of alcoholics from the rehabilitation wards at Danville, Illinois, Veterans Administration Medical Center (DVAMC) does not differ significantly from that of the general population as measured by this nutritional assessment.

These findings are consistent with, although more favorable than, reports of other investigators. Tomaiolo and Kraus (1980) found only 13% of patients admitted with a primary diagnosis of alcoholism to be malnourished. Hurt et al. (1981) also found the majority of alcoholic patients to be adequately nourished. However, Simko et al. (1982) found

poorer nutritional status in alcoholics with and without liver disease than in abstinents. Kirby and Iber (1980) found 18% of 300 alcoholics studied to be 20% below ideal body weight and 25% of subjects to have deficits in albumin, transferrin, and creatinine/height values. Bienia et al. (1982) found no statistical difference between alcoholics and nonalcoholics in the incidence of malnutrition, death rate, or incidence of infection.

While it seems logical to conclude that the incidence of malnutrition is not great in this alcoholic population, incidence variability may parallel that of nonalcoholic populations. The nutritional assessment used, and those reviewed, are directed toward identifying cases of gross malnutrition: marasmus and kwashiorkor. Vitamin, mineral, and trace element deficiencies may also constitute malnutrition, although they are not generally included in nutritional assessments.

Hypothesis Two

THE VITAMIN A STATUS OF ALCOHOLICS AT DVAMC DOES NOT DIFFER SIGNIFICANTLY FROM PUBLISHED NORMAL VALUES OF RETINOL, RETINYL PALMITATE, RETINOIC ACID, OR RETINOL BINDING PROTEIN.

Findings of the study with respect to vitamin A status included three subjects (12, 14, 21) who were below the normal range of 50-100 µg/100 ml for retinol. These were consistent with reports of serum retinol values in cirrhotic

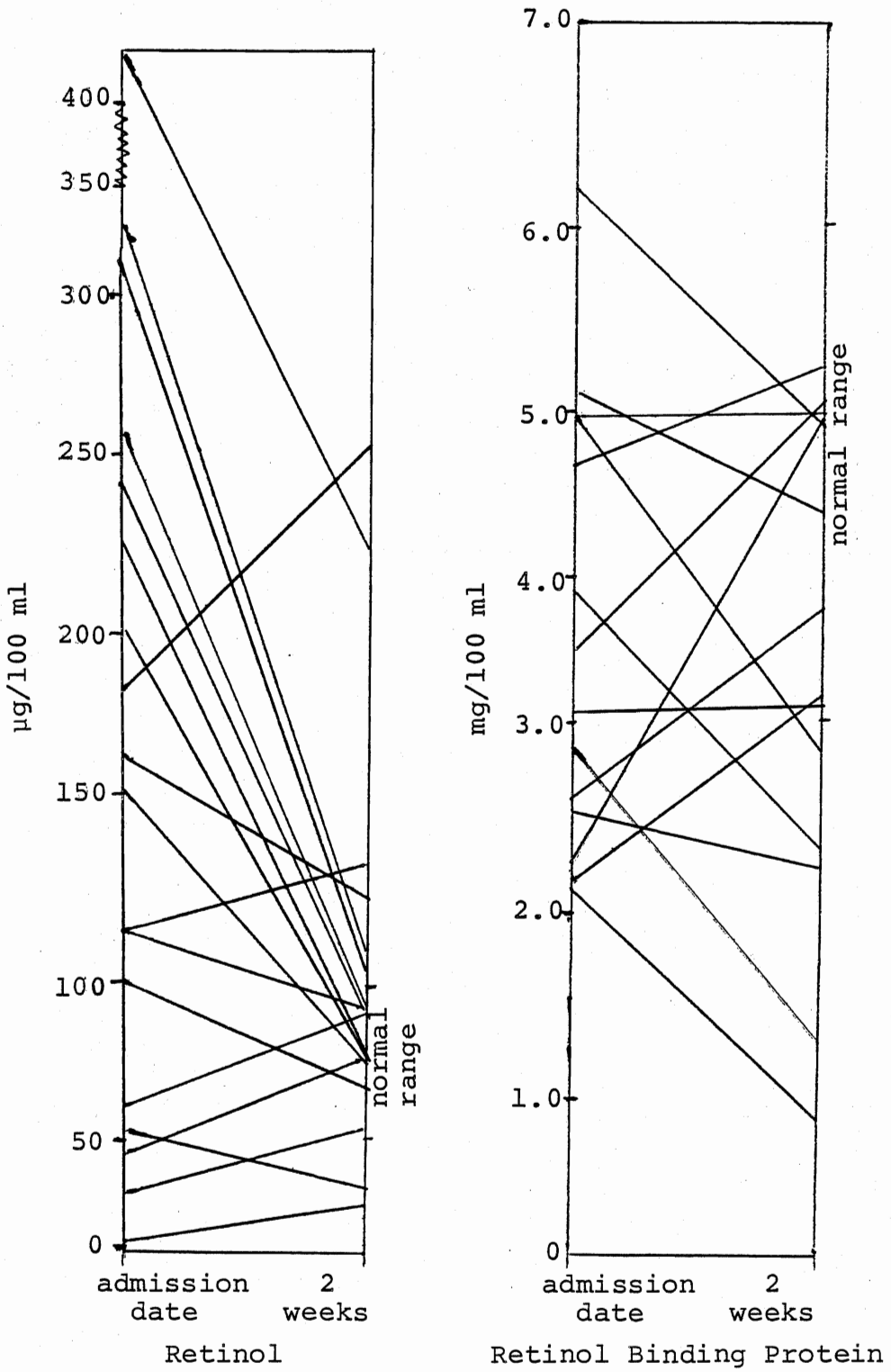
subjects (McClain et al., 1979; Mobarhan et al., 1981). Low serum retinol values in cirrhotic subjects have been attributed to poor dietary intake of vitamin A, poor dietary intake of protein, general malnutrition, fat malabsorption, impaired hepatic storage of retinyl esters, decreased synthesis or release of RBP, and zinc deficiency. Intake of vitamin A, absorptive ability, hepatic vitamin A status, and zinc status were not assessed in this study.

However, two of the three subjects with initial low retinol levels reported taking multivitamins daily prior to admission (subjects 12, 21). RBP levels were depressed in these two subjects, and may account for the depressed retinol levels despite supplemental vitamin A intake. Protein intake was below the RDA for subject 21 and may have been responsible for depression of RBP synthesis. All liver function tests were elevated in subject 14. However, GGT levels were elevated in all three subjects with depressed retinol levels. Nutritional assessments did not reveal malnutrition. Limited data concerning the other possible reasons for the depressed serum retinol levels prevents a conclusive statement concerning the etiology of these values.

Five of the initial 26 serum retinol values were within the normal range of 50-100 μg/100 ml (Figure 2). Dietary intake information was available for three of these subjects, the other two having been discharged prior to the interview. Protein intake was adequate in two subjects (1, 19) and

Figure 2

Changes in Retinol and Retinol Binding Protein With Time



inadequate for subject 18. Caloric intake was initially adequate for all three subjects. RBP was below normal limits for two of these subjects, including the subject whose protein intake was inadequate. This inadequate protein intake may be the cause for the depressed RBP in the one subject, as his liver function tests were all within normal limits. Failure for the nutritional assessment to identify this protein malnutrition may mean that the deficiency was of short duration. RBP is more sensitive to protein deprivation than in albumin or anthropometrics. The case in which the RBP was depressed, although protein intake was adequate, may be due to inaccuracies in reported intake, stress, or decreased liver function. GGT, SGOT, and LDH were all above normal limits in this subject.

Eighteen subjects had initial serum retinol values above the normal range. None of these subjects reported taking vitamin supplements at home. Four of these subjects had RBP levels below the normal range. Of these four, two had at least two abnormal liver function tests. Both of those with abnormal liver function tests, low RBP levels, and high retinol levels had adequate reported protein intake. As the binding of retinol to RBP takes place within the cytoplasm of the hepatocyte, liver dysfunction may have interrupted this process, allowing retinol to circulate unbound. Plasma vitamin A has been reported to be increased in ethanol-fed baboons with fatty livers as compared to

controls, but not in baboons with fibrosis or cirrhosis (Sato and Lieber, 1981). Ethanol increases the fluidity of membranes, which, if continued chronically, changes membrane composition. This changed composition then increases membrane rigidity and impairs a variety of membrane-bound functions (Rubin and Rottenberg, 1982).

Smith and Goodman (1976) suggested that vitamin A toxicity may result when RBP's capacity to transport vitamin A is exceeded, and vitamin A circulates in a form other than bound to RBP. Cases of hypervitaminosis A on which they based this suggestion were characterized by increased circulating retinyl esters. However, these were cases of increased vitamin A supplementation. The vitamin A supplement is normally in the form of a retinyl ester, retinyl palmitate or acetate. Furthermore, ethanol treatment in the rat has been shown to enhance vitamin A hepatotoxicity (Leo et al., 1982).

Ten of the initial RBP levels were below the normal range of 3-6 mg/100 ml (Figure 2). Diet histories could not be obtained for two of these because of hospital discharges. Only two subjects had diet histories revealing previous inadequate protein intake. Thus, protein malnutrition probably was not the major cause of the depressed RBP levels. RBP levels were not significantly correlated with the liver function tests, although only two subjects had normal liver function tests and depressed RBP bevels. However, the liver

the liver function tests may not correlate with changes in liver pathology and morphology. Changes of either may affect RBP synthesis and secretion.

Retinoic acid and retinyl palmitate were found in initial serum samples. When these samples were tested again, approximately six months later, levels were significantly different because the analytical techniques are not sensitive to quantification. Reproducibility of retinol and RBP values was much higher. Retinol and RBP may be metabolized more slowly than retinoic acid and retinyl esters.

Therefore the null hypothesis may be partially rejected. Sufficient data could not be collected to reject the hypothesis concerning retinyl palmitate and retinoic acid. However, the null hypothesis that no difference exists between normal retinol values and this sample may be rejected based on the one-sided binomial exact test using Poisson approximation. Twenty-one subjects were outside of the normal range, although a 99.2% probability existed that no more than four would be outside of the normal range. The null hypothesis that no difference exists between normal RBP values and this sample could be rejected based on the one-sided binomial exact test using Poisson approximation. Ten of the 24 subjects were outside of the normal range, although a 99.2% probability existed that no more than four would be outside the normal range.

Hypothesis Three

NO SIGNIFICANT CHANGE OCCURS IN THE VITAMIN A STATUS OF ALCOHOLICS AT DVAMC DURING TWO WEEKS OF HOSPITALIZATION.

Findings of this study with respect to change in vitamin A status included one (subject 21) of three initially low retinol values which remained low after two weeks. The follow-up retinol value in this subject was an improvement over the initial value, 22 µg/100 ml vs. 33 µg/100 ml. However, RBP in this case decreased in the two week interval. Initial RBP of 2.94 mg/100 ml decreased to 1.38 mg/100 ml, although reported protein intake increased from 22.5 gm to 101 gm protein. Body weight in this subject decreased two pounds, although reported intake increased from 778 kcal to 2262 kcal/day. This suggests errors in dietary intake reporting.

Subjects 12 and 14, who initially had low retinol levels, had normal retinol levels upon follow-up. Subject 14 had normal RBP levels both initially and upon follow-up. Normalization of retinol levels in this case may reflect better dietary intake and/or vitamin supplementation. (All subjects received vitamin supplementation while in the medical center.) The other subject's RBP level was below normal range both initially and upon follow-up (Table 3).

All but four of the initial 18 elevated retinol levels normalized upon follow-up (Figure 2). This may reflect the abstinence from alcohol and a normalization of retinol

metabolism. Of the four subjects whose retinol levels did not normalize, two did reflect a relative normalization in that levels fell. However, two subjects (17, 18) had increased retinol levels. In these two cases RBP also increased, although RBP was still within normal range.

Normal vitamin A metabolism is dependent on many factors. It is not surprising then that abnormal vitamin A metabolism be related to many factors. Absence of significant correlation in this study exemplifies the need to look at multiple variable correlations, and to examine data on each individual as well as on the group.

Significant data on individuals were found. Two subjects had initially low retinol levels, despite reported multivitamin intake prior to admission. RBP was depressed in these two subjects, although reported protein intake was inadequate for one of these subjects. Of the subjects with normal initial retinol levels, two subjects had RBP levels below normal limits. One of these had reported inadequate protein intake.

None of the subjects with elevated initial retinol levels reported taking vitamins prior to admission. Four of these subjects had elevated retinol levels despite RBP levels below normal. Of all initial RBP levels, ten were below normal range. Only two of these RBP levels could be explained by inadequate protein intake. Low and elevated

retinol levels seemed to normalize after two weeks. However, only 50% of initially low RBP levels improved.

The null hypothesis that no change in vitamin A status occurs during two weeks of hospitalization could be partially rejected. Significant changes ($p \leq 0.01$) in retinol levels were found. Significant changes in RBP were not found. Retinyl palmitate and retinoic acid could not be analyzed.

Hypothesis Four

NO CORRELATION EXISTS BETWEEN VITAMIN A LEVELS AND RESULTS OF LIVER FUNCTION TESTS.

Findings of the study were that correlations between vitamin A and liver function tests were not significant for tests other than albumin. This suggests that either the liver function tests were not truly representative of liver function and/or morphology, serum vitamin A levels were not related to liver function, or that factors other than that of liver function were at least partially responsible for vitamin A levels. Other factors, such as zinc and absorptive capability, do affect vitamin A status, the latter possibility seeming most plausible. However, sample size and diversity may also be partially responsible for this lack of significant correlation. Nevertheless, the null hypothesis is not rejected.

Hypothesis Five

THE SELENIUM STATUS OF ALCOHOLICS AT DVAMC DOES NOT DIFFER SIGNIFICANTLY FROM PUBLISHED NORMAL VALUES OF PLASMA AND WHOLE BLOOD SELENIUM.

Findings of this study included ten of twenty-seven initial whole blood selenium values which were below normal range. Seventeen initial plasma selenium values were below normal range. These findings are consistent with decreased selenium levels found in cirrhosis (Aaseth et al., 1980). However, subjects with depressed whole blood selenium levels did not necessarily have depressed plasma selenium levels. Six of the ten with low blood selenium levels also had low plasma levels. Stable isotope studies have found erythrocyte selenium content to exceed plasma or serum selenium levels. Although whole blood contains plasma and erythrocytes, findings similar to the isotope studies would be expected. Indeed, initial whole blood selenium levels usually exceeded plasma levels when initial whole blood selenium levels were normal or elevated. However, less than half of the depressed whole blood selenium samples followed this pattern (Table 5). This finding may reflect a defect in heme metabolism due to selenium deficiency. Selenium deficient rat liver has been found to produce heme metabolism defects, including increased heme synthesis and degradation and decreased formation of some heme proteins (Burk et al., 1980).

Glutathione peroxidase contains selenium. Changes in glutathione have been reported in acute and chronic alcohol

abuse (Guerri and Grisolia, 1980). Lower glutathione levels may be related to increased GGT levels, as the major function of GGT is to degrade intracellular glutathione. There was no significant correlation in this study between GGT and selenium levels. However, it may be the frequency and duration of GGT elevation which is significant in affecting glutathione metabolism. Also, glutathione peroxidase in the red cell accounts for only 25% of the total selenium. Therefore, factors other than glutathione may be involved.

Six initial whole blood selenium levels (subjects 4, 12, 15, 20, 24, 26) and two plasma selenium levels (subjects 3, 11) were above normal ranges. However, the subjects with the increased plasma levels did not have increased whole blood selenium levels. As plasma is a component of whole blood, it may be that whole blood levels increase before the increase is exhibited in the plasma. However, if the subjects were in the process of replacing selenium stores, plasma levels may first be increased, and then normalize. Although a follow-up plasma level was not available for one subject with an initially elevated plasma level, the other case did show plasma selenium levels falling to within normal range (subject 11) (Table 5).

Based on the one-sided binomial exact test using Poisson approximation, there was a 99.2% probability that no more than four subjects' values would be outside the normal range. Fifteen whole blood selenium values and 18 initial plasma

selenium values were outside of the normal range. Thus, the null hypothesis could be rejected: selenium status of alcoholics from the rehabilitation wards did differ from normal values.

Hypothesis Six

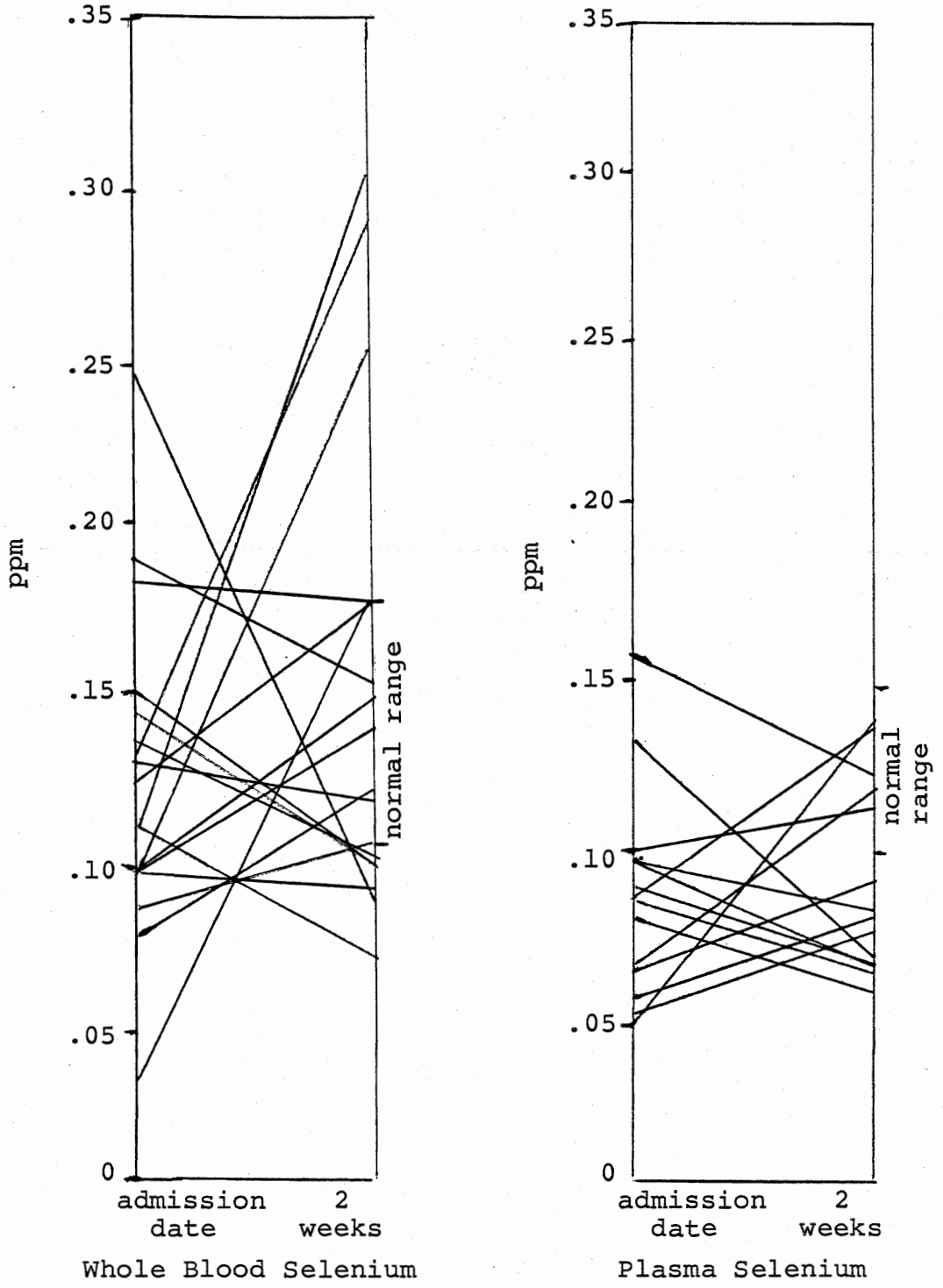
NO SIGNIFICANT CHANGE OCCURS IN THE SELENIUM STATUS OF ALCOHOLICS AT DVAMC DURING TWO WEEKS OF HOSPITALIZATION.

Findings of the study included the observation that a follow-up of initially depressed selenium levels revealed normalization in four subjects (6, 13, 14, 21). Three initially depressed whole blood selenium levels were elevated above normal upon follow-up (subjects 5, 18, 27). Subjects 2 and 22 retained depressed whole blood selenium levels. Follow-up information was not available for subject 9 (Figure 3).

Of those normalizing their previously low whole blood selenium levels, plasma levels fell below normal range for subject 14, and increased to normal range for subject 6. Follow-up plasma levels were not available for two subjects in this group. Follow-up plasma levels of subjects with sequentially elevated follow-up whole blood selenium values (subjects 5, 18, 27) all had decreased to below normal range. This may indicate that the plasma levels serve as a pool for the erythrocyte to draw upon for normalization.

Figure 3

Changes in Whole Blood and Plasma Selenium With Time



Initially normal whole blood selenium levels rose above normal range upon follow-up for two subjects (17, 23). The follow-up plasma selenium level was not available for subject 23, but levels did fall below normal range for subject 17. Initially normal whole blood selenium levels fell below normal range upon follow-up for three subjects (10, 11, 19). Follow-up plasma levels in these subjects revealed one which fell but remained in normal range, one which fell below normal range, and one which rose but remained below normal range.

Although 65% of follow-up whole blood selenium levels and 67% of follow-up plasma selenium levels remained outside of normal ranges, 53% of follow-up plasma levels and 40% of follow-up whole blood selenium levels showed normalization trends. However, statistically significant changes ($p \leq 0.01$) between initial and follow-up whole blood and plasma selenium levels were not found. Therefore the null hypothesis could not be rejected.

Hypothesis Seven

NO CORRELATION EXISTS BETWEEN SELENIUM LEVELS AND RESULTS OF LIVER FUNCTION TESTS.

Findings showed no correlation existing between plasma or whole blood selenium and the results of liver function tests. Changes in liver morphology, dependence on alternate pathways of alcohol degradation, and vitamin E status

may all be responsible for the individual changes in selenium status. While there was no significant correlation between selenium levels and liver function tests, these tests may not be significant markers of liver morphology change. Also, the degree of elevation in this study may not be an important factor. Rather, frequency of previous elevations and duration may be more significant.

Increased dependence on the microsomal ethanol oxidizing system with increased lipoprotein synthesis and endoplasmic reticulum proliferation may follow chronic alcohol use (Hannah and Soares, 1979). Alcohol microsomal oxidation involves interaction with hydroxyl radicals. Lipid peroxidation forms free radicals and is inhibited by vitamin E and glutathione peroxidase (Tappel, 1980). Therefore, length of alcohol abuse may determine relative dependence on alternate alcohol degradation pathways and influence vitamin E and selenium metabolism.

CHAPTER V

SUMMARY AND CONCLUSIONS

Nutritional assessment of 27 alcoholic hospitalized Veterans included diet histories, anthropometric data, serum albumin, and total lymphocyte counts. Initial diet histories generally revealed inadequate caloric intake and adequate protein intake. Exclusion of alcohol calories may be responsible for the low caloric intakes recorded for some subjects. Caloric intake generally improved as assessed by 24-hour recalls two weeks after admission. Two subjects were below 80% IBW upon admission, and both gained weight during the two week interval. Seven subjects were below 80% of the HANES standard for TSF, while nine subjects were below 80% of the Jelliffe standard for TSF. One subject was below 80% of the HANES standard for MAMC.

Serum albumin and total lymphocyte counts were generally within normal range. When all assessment components were analyzed for the individual, only one subject could be classified as moderately marasmic. Therefore, the null hypothesis could not be rejected: the nutritional status of alcoholics from the rehabilitation wards at DVAMC does not differ significantly from that of the general population as measured by this nutritional assessment. However, this nutritional assessment identifies only protein and/or calorie malnutrition. Other nutrient deficiencies were not examined.

Eighty-three percent of initial retinol values were outside the normal range. Forty-eight percent of initial RBP values were outside the normal range. Retinyl palmitate and retinoic acid levels could not be adequately analyzed. Therefore the null hypothesis could be partially rejected: the vitamin A status of alcoholics from the rehabilitation wards at DVAMC did differ from normal values of retinol and RBP as measured by the one-sided binomial exact test using Poisson approximation.

Abnormal initial retinol levels were both below and above normal ranges, although the majority were above normal ranges. Low retinol levels existed despite reported vitamin supplementation prior to admission. No vitamin supplementation was reported in those subjects with initially elevated retinol levels. However, ethanol treatment has been shown to enhance vitamin A hepatotoxicity in the rat (Leo et al., 1982). The presence of elevated retinol levels in these subjects is a significant finding. Seventy-nine percent of retinol levels showed normalization trends after two weeks. This suggests an influence of ethanol on vitamin A metabolism, although other variables are probably involved.

While ten initial RBP levels were below the normal range, only two subjects had diet histories revealing previous inadequate protein intake. Thus, protein malnutrition probably was not the major cause of the depressed RBP levels.

Half of the depressed initial RBP levels, for which follow-up was available, did improve after two weeks.

Despite this improvement in RBP levels the change after two weeks was not significant ($p \leq 0.01$). There was significant change found in retinol levels after two weeks. Therefore, the null hypothesis of no significant change occurring in vitamin A status after two weeks of hospitalization could be partially rejected.

Although retinyl palmitate and retinoic acid levels could not be quantitated, their presence in the serum may be significant. Further investigation is required concerning these vitamin A metabolites in alcoholism.

No correlation existed between vitamin A levels and results of liver function tests, except for albumin levels. Either these liver function tests were not truly representative of liver function and/or morphology, or serum vitamin A levels were not related to liver function. Other factors may also influence both vitamin A levels and liver function tests, and suggests the need for multiple variable correlations. Also, division of the sample into subgroups of low, normal, and elevated vitamin A levels may be necessary to provide homogeneousness. Nevertheless, the null hypothesis of no correlation existing between vitamin A levels and the results of liver function tests could not be rejected.

Fifty-six percent of initial whole blood selenium levels and 67% of initial plasma selenium levels were outside the

normal range. Thus, the null hypothesis could be rejected: selenium status of alcoholics from the rehabilitation wards at DVAMC did differ from the normal values as measured using the one-sided binomial exact test and Poisson approximation.

Although 65% of follow-up whole blood selenium levels and 67% of follow-up plasma levels remained outside of the normal ranges, 53% of follow-up plasma selenium levels and 40% of follow-up whole blood selenium levels showed normalization trends. This may also indicate an influence of ethanol on liver morphology, which in turn affects selenium metabolism. However, the null hypothesis of no significant change occurring during two weeks of hospitalization could not be rejected.

The null hypothesis of no correlation existing between selenium levels and the results of liver function tests also could not be rejected. This may again indicate a need to examine low, normal, and elevated plasma and whole blood selenium groups individually.

Implications

This study did reveal several important points to consider when evaluating the nutritional status of the alcoholic. Usual nutritional assessments may not be adequate tools, except for identifying protein and calorie malnutrition.

Malnutrition may be prevalent, but the alterations may be of vitamins, minerals, and trace elements. These areas of nutritional status are not usually examined, but may have important implications in alcoholism. The presence of both low and elevated retinol, and plasma and whole blood selenium levels indicates that requirements may be quite different in alcoholics.

Length and degree of alcohol abuse may play a significant role in nutritional status. While this type of information may be difficult to obtain, attempts should be made to define the degree of abuse.

Although fatty liver is reversible, morphological changes in the liver may also be important in assessing nutritional status. Changes in liver composition as well as liver function may change nutrient requirements and toxicity levels. Therefore, normal nutrient supplementation may not be adequate or advisable.

Nutrient interaction is a complex area to study, but need for such study becomes more apparent when studying nutrient imbalances. Studies of vitamin A status may include vitamin A, zinc, protein, fat, and vitamin E. Studies of selenium status may include selenium, vitamin E, and protein.

In conclusion, this study has shown that while nutritional assessments may not identify malnutrition in alcoholics, vitamin and trace element malnutrition may exist.

Abnormal vitamin A and selenium levels seemed to normalize with abstinence from ethanol and improved dietary intake. The presence of both low and elevated retinol and selenium levels indicates that a heterogeneous sample was examined. Etiology of these alterations requires further investigation.

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APPENDIX A

NUTRITIONAL CARE INFORMATION FORM

NUTRITIONAL CARE INFORMATION

PATIENT'S NAME	WARD	IDENTIFICATION NO.

PATIENT'S PHYSICIAN	PATIENT'S ADDRESS	BED	AGE	SEX

DIAGNOSIS (ES)

PATIENT'S OCCUPATION

CURRENTLY PRESCRIBED MEDICATIONS

PRESENT DIET RX

SELF-PRESCRIBED MEDICATIONS AND DIETARY SUPPLEMENT

PREVIOUS DIET COUNSELING

PREVIOUS DIET RX

KIND OF DIET

RECEIVED INDIVIDUALIZED DIET INSTRUCTION ☐ YES ☐ NO

RECEIVED PRINTED DIET INFORMATION ☐ YES ☐ NO

SELF-IMPOSED DIET REGIMENS

INSTRUCTOR

LOCATION

RECOLLECTION OF DIET INSTRUCTION ☐ GOOD ☐ POOR

PHYSICAL DATA

HEIGHT	PRESENT WEIGHT
IDEAL WEIGHT	RECENT CHANGE
MAX. WT.	MIN. WT.

TRICEPS SKINFOLD	MM
MID UPPER ARM CIRCUMFERENCE	CM
MID UPPER ARM MUSCLE CIRCUMFERENCE (DERIVED)	CM
TRICEPS SKINFOLD AS % OF STANDARD	
MID UPPER ARM MUSCLE CIRCUMFERENCE AS % OF STANDARD	

PHYSICAL HANDICAPS WHICH INFLUENCE FOOD INTAKE (✓)

☐ VISUAL ☐ AUDITORY

☐ MOBILITY ☐ DEXTERITY

☐ DENTAL ☐ OTHER

FACTORS INFLUENCING FOOD INTAKE

YES	NO	*(Items 1 through 13, check yes or no)*
		1. Has a good appetite
		2. Resides alone
		3. Prepares own food
		4. Has cooking facilities
		5. Has refrigeration
		6. Has adequate funds for food
		7. Shops in supermarket for food
		8. Buys dietetic/Health foods
		9. Follows 3-meal pattern daily
		10. Eats at normal pace
		11. Is a smoker
		12. Has elimination problems
		13. Has food allergies

(14 through 17, check applicable items and note frequency when indicated)

14. Type of food preparation		Conventional cooking
		Convenience foods
		Snack foods
15. Location of Meals		Home
		Restaurant
		Other
16. Food cravings		Sweets
		Fried
		Salty
17. Food preferences *(Check & identify*		Ethnic
		Regional
		Religious

(Use space below for additional information and for comments or explanations about answers to questions 1-17. Reference number of question if comment relates to questions 1-17)

SIGNATURE	TITLE	DATE

NUTRITIONAL CARE FINDINGS: After reviewing diagnostic procedures, laboratory values, clinical findings, physical impairments, and nutritional care information, summarize pertinent Subjective and Objective findings and make an Assessment of patient's nutritional care needs.

SUBJECTIVE:

OBJECTIVE:

ASSESSMENT:

NUTRITIONAL CARE PLAN: After this is formulated, the plan should always be documented in the patient's medical record. Plan should include:

Dx (Diagnostic) — Such as laboratory work-up, consultations.

Rx (Therapeutic) — Such as diet modifications and adjustments, recommendations for improving dietary habits, goals in relation to nutritional problems, referrals to other health care team members.

Pt. Ed. (Patient Education) — Such as diet instruction (individual and group) including family member and/or caretaker, handout materials to be given, and follow-up visits for continuing care.

PLAN

DX:

RX:

PT. ED.:

SIGNATURE *(Dietitian)*	TITLE	DATE

FOOD INTAKE AND ACTIVITY PATTERNS

NAME

IDENTIFICATION NO.

DIRECTIONS: Under Usual Pattern record time, activities engaged in throughout day and while eating meals and snacks, and customary eating pattern with portion sizes. Give frequency per week of major foods not eaten daily. Include all food, condiments, alcoholic and carbonated beverages. Under Alternative Pattern record time, activity, and eating pattern which differs from usual routine, e.g. weekends, different work shifts, etc.

USUAL PATTERN			ALTERNATIVE PATTERN		
HOUR	ACTIVITY	FOOD INTAKE	HOUR	ACTIVITY	FOOD INTAKE

(Continue on reverse side if more space is needed.)

SUMMARY OF FOOD INTAKE

FOOD		AMOUNT *(Per day &/or week)*	FOOD		AMOUNT *(Per day &/or week)*
1. EGGS			6. FRUITS	CITRUS	
				OTHER	
2. MEAT AND SUBSTITUTES			7. MILK AND DAIRY PRODUCTS		
3 BREAD AND CEREALS			8. FATS		
4. POTATOES AND SUBSTITUTES			9. SWEETS		
5. VEGET-ABLES	YELLOW/DK. GREEN		10. OTHER		
	OTHER				

CHECK (✓) APPROPRIATE VALUE	ESTIMATED LEVEL OF NUTRITIONAL INTAKE														
	CAL	PRO	FAT	CHO	VIT. A	THIA	RIBO	NIAC	ASCOR ACID	VIT. D	CA	FE			
HIGH															
ADEQUATE															
LOW															

SIGNATURE	TITLE	DATE

USUAL PATTERN			ALTERNATIVE PATTERN		
HOUR	ACTIVITY	FOOD INTAKE	HOUR	ACTIVITY	FOOD INTAKE

ADDITIONAL INFORMATION *(Use this space for new information or to update, information on pages 1 and 2, e.g., changes in diet prescription, supplementary feeding, weight, nutrional care plan etc.)*

PATIENT NAME	IDENTIFICATION NO.	PAGE 4 OF 4

☆U.S. G.P.O. 1977–240-992/566

APPENDIX B

CONSENT FORMS

Statement of Information

Project Title: Nutritional Status of Alcoholics: Emphasis on Vitamin A and Selenium

I, _____, give my permission to be enrolled as a participant in this study. The reason for this study has been explained to me. I understand that it is to obtain information about nutritional status, particularly body composition, vitamin A and selenium, in relation to the use of alcohol. Thus this study might be of importance in the management of persons using alcohol.

As a participant in this study, I understand that I will be asked questions about my diet, that I will be weighed, measured for height and upper arm circumference, and measured with Lange skinfold calipers. It is also my understanding that additional blood will be drawn when my blood is drawn for my initial routine laboratory work and that my blood will be drawn again approximately two weeks after admission for the determination of vitamin A and selenium.

I have been told that by participating in this study I will not be exposed to any unusual risks, that there are no expected side effects, and that I will receive no compensation for my participation. I also understand that I may withdraw from the study at any time, and that my participation or withdrawal will in no way affect the quality of my medical care and/or veterans benefits.

I have had the opportunity of discussing the study with the principal investigator. I understand that confidentiality will be maintained and that anonymity will be preserved by using codes in case of any publication resulting from this study.

_____ _____

Signature Investigator's Signature

_____ _____

Date Date

_____

Witness

_____

Date

PART II - AGREEMENT BY SUBJECT'S REPRESENTATIVE TO ALLOW SUBJECT TO PARTICIPATE IN RESEARCH BY OR UNDER THE DIRECTION OF VETERANS ADMINISTRATION

DATE

1. I, _____, am authorized to give consent
(Type or print name of subject's representative)

for _____ by virtue of _____
(Type or print subject's name) *(Relationship, legal appointment, etc.)*

I voluntarily consent for this person to participate as a subject in the investigation entitled _____
(Title of study)

2. I have signed one or more information sheets with this title to show that I have read the description including the purpose and nature of the investigation, the procedures to be used, the risks, inconveniences, side effects, and benefits to be expected, as well as other courses of action open to me and my right to withdraw the subject from the investigation at any time. Each of these items has been explained to me by the investigator in the presence of a witness. The investigator has answered my questions concerning the investigation and I believe that I understand what is intended.

3. I understand that no guarantees or assurances have been given me since the results and risks of an investigation are not always known beforehand. I have been told this investigation has been carefully plannned, that the plan has been reviewed by knowledgeable people, and that every reasonable precaution will be taken to protect the well-being of the subject.

4. In the event the subject sustains physical injury as a result of participation in this investigation, if the subject is eligible for medical care as a veteran, all necessary and appropriate care will be provided. If the subject is not eligible for medical care as a veteran, humanitarian emergency care will nevertheless be provided.

5. I realize I have not released this institution from liability for negligence. Compensation may or may not be payable, in the event of physical injury arising from such research, under applicable federal laws.

6. I understand that all information obtained about the subject during the course of this study will be made available only to doctors who are taking care of the subject and to qualified investigators and their assistants where their access to this information is appropriate and authorized. They will be bound by the same requirements to maintain the subject's privacy and anonymity as apply to all medical personnel within the Veterans Administration.

7. I further understand that, where required by law, the appropriate federal officer or agency will have free access to information obtained in this study should it become necessary. Generally, I may expect the same respect for the subject's privacy and anonymity from these agencies as is afforded by the Veterans Administration and its employees. The provisions of the Privacy Act apply to all agencies.

8. In the event that research in which the subject participates involves certain new drugs, information concerning the subject's response to the drug(s) will be supplied to the sponsoring pharmaceutical house(s) that made the drug(s) available. This information will be given to them in such a way that the subject cannot be identified.

I _____
NAME OF SUBJECT'S REPRESENTATIVE

HAVE READ THIS CONSENT FORM. ALL MY QUESTIONS HAVE BEEN ANSWERED, AND I FREELY AND VOLUNTARILY CHOOSE THAT THE SUBJECT PARTICIPATE. I UNDERSTAND THAT THE SUBJECT'S RIGHTS AND PRIVACY WILL BE MAINTAINED. I AGREE TO THE SUBJECT'S PARTICIPATION AS A VOLUNTEER IN THIS PROGRAM.

9. Nevertheless, my consent for the subject's participation in the investigation is limited as follows:

ADDRESS OF SUBJECT'S REPRESENTATIVE (Print or type)	SIGNATURE OF SUBJECT'S REPRESENTATIVE
WITNESS'S NAME AND ADDRESS *(Print or type)*	WITNESS'S SIGNATURE
SUBJECT'S NAME *(Print or type)*	SUBJECT IS NOW A PATIENT AT *(Name of VA Facility)*
INVESTIGATOR'S NAME *(Print or type)*	INVESTIGATOR'S SIGNATURE

☐ Signed information sheets attached.

☐ Signed information sheets available at:

SUBJECT'S IDENTIFICATION *(I.D. plate or print name - last, first, middle)*	SUBJECT'S I.D. NO.	AGE	WARD

AGREEMENT BY SUBJECT'S REPRESENTATIVE TO PARTICIPATE IN RESEARCH BY OR UNDER THE DIRECTION OF THE VETERANS ADMINISTRATION

☆ U.S. Government Printing Office: 1979—311-645/9

PART I-AGREEMENT TO PARTICIPATE IN RESEARCH BY OR UNDER THE DIRECTION OF THE VETERANS ADMINISTRATION

DATE

1. I, _____, voluntarily consent to participate as a subject
(Type or print subject's name)

in the investigation entitled _____
(Title of study)

2. I have signed one or more information sheets with this title to show that I have read the description including the purpose and nature of the investigation, the procedures to be used, the risks, inconveniences, side effects and benefits to be expected, as well as other courses of action open to me and my right to withdraw from the investigation at any time. Each of these items has been explained to me by the investigator in the presence of a witness. The investigator has answered my questions concerning the investigation and I believe I understand what is intended.

3. I understand that no guarantees or assurances have been given me since the results and risks of an investigation are not always known beforehand. I have been told that this investigation has been carefully planned, that the plan has been reviewed by knowledgeable people, and that every reasonable precaution will be taken to protect my well-being.

4. In the event I sustain physical injury as a result of participation in this investigation, if I am eligible for medical care as a veteran, all necessary and appropriate care will be provided. If I am not eligible for medical care as a veteran, humanitarian emergency care will nevertheless be provided.

5. I realize I have not released this institution from liability for negligence. Compensation may or may not be payable, in the event of physical injury arising from such research, under applicable federal laws.

6. I understand that all information obtained about me during the course of this study will be made available only to doctors who are taking care of me and to qualified investigators and their assistants where their access to this information is appropriate and authorized. They will be bound by the same requirements to maintain my privacy and anonymity as apply to all medical personnel within the Veterans Administration.

7. I further understand that, where required by law, the appropriate federal officer or agency will have free access to information obtained in this study should it become necessary. Generally, I may expect the same respect for my privacy and anonymity from these agencies as is afforded by the Veterans Administration and its employees. The provisions of the Privacy Act apply to all agencies.

8. In the event that research in which I participate involves certain new drugs, information concerning my response to the drug(s) will be supplied to the sponsoring pharmaceutical house(s) that made the drug(s) available. This information will be given to them in such a way that I cannot be identified.

I_____
NAME OF VOLUNTEER

HAVE READ THIS CONSENT FORM. ALL MY QUESTIONS HAVE BEEN ANSWERED, AND I FREELY AND VOLUNTARILY CHOOSE TO PARTICIPATE. I UNDERSTAND THAT MY RIGHTS AND PRIVACY WILL BE MAINTAINED. I AGREE TO PARTICIPATE AS A VOLUNTEER IN THIS PROGRAM.

9. Nevertheless, I wish to limit my participation in the investigation as follows:

VA FACILITY	SUBJECT'S SIGNATURE
WITNESS'S NAME AND ADDRESS *(Print or type)*	WITNESS'S SIGNATURE
INVESTIGATOR'S NAME *(Print or type)*	INVESTIGATOR'S SIGNATURE

☐ Signed information sheets attached. ☐ Signed information sheets available at:

SUBJECT'S IDENTIFICATION *(I.D. plate or give name - last, first, middle)*	SUBJECT'S I.D. NO.	WARD

AGREEMENT TO PARTICIPATE IN RESEARCH BY OR UNDER THE DIRECTION OF THE VETERANS ADMINISTRATION

VA FORM SEP 1979 10-1086

SUPERSEDES VA FORM 10-1086 JUN 1975, WHICH WILL NOT BE USED.

APPENDIX C

NUTRITIONAL ASSESSMENT WORKSHEET

JULY 1981

DATE: _____

NUTRITIONAL ASSESSMENT WORKSHEET

NAME:	height		inches		cm.		
	weight		lbs.		kg.		
DIET:	IBW		lbs.		kg.		% 1 BW
	BEE		KCal.				
AGE:	MAC		cm.		% Std.		
SEX:	MAMC		cm.		% Std.		
UNIT:	TSF		mm.		% Std.		
	biceps		mm.				
	subscapular		mm.				
	suprailiac		mm.				
	Total		mm.		% Body Fat		
		albumin		gm/dl			
Dx:	+ lymphocyte			mm^3			
		BUN		mg/dl			
	creatinine			mg/dl			
Rx:							
				Body Composition (kg.)			
					actual	pred.	difference
				body wt.			
				body fat			
				LBM			

☆U. S. Government Printing Office: 1979—629-457

NUTRITIONAL ASSESSMENT SURVEY

NAME_____ DATE_____

AGE_____ SEX_____ UNIT_____

DIET ORDER_____ PHYSICIAN_____

_____ DIAGNOSIS_____

NUTRITIONAL MEDICATIONS_____ _____

_____ _____

BLOOD PRESSURE_____

HEIGHT - (ft)_____ CM_____

WEIGHT - (lb)_____ KG_____

IDEAL BODY WEIGHT_____ % OF IDEAL BODY WEIGHT_____

MID-ARM CIRCUMFERENCE (cm)_____

MID-ARM CIRCUMFERENCE % OF STANDARD_____

MID-ARM MUSCLE CIRCUMFERENCE (cm)_____

MID-ARM MUSCLE CIRCUMFERENCE % OF STANDARD_____

TRICEPS SKIN-FOLD (mm)_____

TRICEPS SKIN-FOLD % OF STANDARD_____

SKIN-FOLD MEASUREMENTS

BICEPS (mm)_____ % OF BODY FAT

TRICEPS (mm)_____ _____

SUBSCAPULAR (mm)_____

SUPRA-ILIAC (mm)_____

TOTAL _____

MISCELLANEOUS INFORMATION:_____

_____

_____

_____

Surveyor

APPENDIX D

OPERATIONAL DESIGN

Operational Design

Hypothesis	Working Hypothesis	Objectives	Instruments	Test Statistic
1. The nutritional status of alcoholics at the DVAMC does not differ significantly from that of the general population as measured by anthropometrics, diet histories, and biochemical tests of visceral proteins and immunocompetence.	The dietary histories of the study group are adequate. No difference exists between study group and general population for anthropometric values or biochemical tests of visceral protein status and immunocompetence.	Determine the approximate dietary intake. Determine height, weight, TSF, MAMC, body composition, albumin, and total lymphocyte count.	Interview administered food frequency checklist; RDA; Basic Four Food Groups; food composition tables. Beam scales, Lange calipers, measuring tape, Jelliffe and HANES standards for anthropometrics; calculated predicted standards for body composition; hospital listing of normal ranges for biochemical tests.	One-sided binomial exact test using Poisson approximation.

(continued)

Operational Design
(continued)

Hypothesis	Working Hypothesis	Objectives	Instruments	Test Statistic
2. The vitamin A status of alcoholics at DVAMC does not differ significantly from published normal values of retinol, retinyl palmitate, retinoic acid, and RBP.	Same.	Measure retinol, retinyl palmitate, and RBP, and compare to normal ranges.	HPLC; published normal ranges; immunodiffusion plates.	One-sided binomial exact test using Poisson approximation.
3. No change occurs in the vitamin A status after two weeks of hospitalization.	Same.	Assess change in vitamin A status after two weeks.	Retinol, RBP, retinyl palmitate, retinoic ester values.	Student's T test.
4. No correlation exists between vitamin A levels and the results of liver function tests.	No correlation exists between retinol, retinyl palmitate, retinoic acid, RBP and LDH, SGOT, SGPT, GGT, or albumin.	Correlate vitamin A and results of liver function tests.		Pearson's correlation coefficient.

(continued)

Operational Design
(continued)

Hypothesis	Working Hypothesis	Objectives	Instruments	Test Statistic
5. The selenium status of alcoholics at DVAMC does not differ significantly from published normal values of plasma and whole blood selenium.	Same.	Measure plasma and whole blood selenium and compare to normals.	Gas chromatography; published normal values.	One-sided binomial exact test using Poisson approximation.
6. No change occurs in selenium status after two weeks of hospitalization.	Same.	Assess change in selenium status after two weeks.	Whole blood and plasma selenium values.	Student's T test.
7. No correlation exists between selenium levels and results of liver function tests.	No correlation exists between whole blood, plasma selenium and LDH, SGPT, SGOT, GGT and albumin.	Correlate selenium levels with liver function tests.		Pearson's correlation coefficient.

VITA

Karen McCauley was born in Collinsville, Illinois, on February 9, 1958. She received her elementary education and secondary education in Collinsville, Illinois. She received her Bachelor of Science degree in home economics, with a major in dietetics, from the University of Illinois, Champaign-Urbana, Illinois in 1979. She began graduate work in nutritional sciences and completed an internship in dietetics at the University of Alabama in Birmingham, Alabama, in 1980. She also became a registered dietitian in 1980.

She began work as a clinical dietitian at the Veterans Administration Medical Center in Danville, Illinois in 1980. She has been the nutrition support team dietitian at this facility since 1981.

Her professional affiliations include the American Dietetic Association, Illinois Dietetic Association, Eastern Illinois Dietetic Association, American Society for Parenteral and Enteral Nutrition, American Home Economics Association, and Illinois Home Economics Association.