THE EFFECT OF INDIVIDUALIZED NUTRITION INTERVENTION IN MALNOURISHED, AMBULATORY PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

by

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ABSTRACT

Protein calorie malnutrition is prevalent among patients with chronic obstructive pulmonary disease (COPD). Nutritional interventions directed toward improving nutritional status in malnourished COPD patients appear warranted because malnutrition in these patients is associated with impaired respiratory muscle function and depressed immunocompetence. These impairments are associated with increased morbidity and mortality. One objective of this study was to identify factors contributing to weight loss in COPD patients. Another objective was to assess the effect of individualized nutrition counselling in the home environment on nutrient intake, body weight, body composition, pulmonary function and perceived well-being in COPD patients. Eleven malnourished, ambulatory COPD patients were randomized to a treatment group (n=6) or to a control group (n=5). The treatment group received individualized nutrition counselling from a dietitian and were provided with access to a nutritional supplement (Pulmocare™). The control group received no nutrition counselling or supplement. Nutritional status, pulmonary function, respiratory muscle strength, and quality of life were assessed at baseline and after six months. Evaluation of the nutrition education provided was completed at six months. There were no significant differences in body weight, body composition, nutrient intake, maximal expiratory pressure, maximal inspiratory pressure, or distance walked in six minutes between groups or over time. The treatment group had a significantly lower forced expiratory volume in one second percent

predicted ($FEV_1\%$) and forced expiratory volume in one second/forced vital capacity percent predicted ($FEV_1/FVC\%$) than the control group. There was no significant change in $FEV_1\%$ or $FEV_1/FVC\%$ over time. When analyzed as a whole (n=11), these COPD patients perceived their general well-being to be poorer than that of the general elderly population. Physical well-being was the component of well-being that was compromised. At six months, there was a significant decrease in overall perceived well-being and physical well-being. The majority of these COPD patients indicated that hospitalization for exacerbation of COPD was a time of significant weight loss. Dyspnea, fatigue, poor appetite, early satiety, gastrointestinal discomfort and intolerance to Pulmocare™ appeared to contribute to difficulties in augmenting intake to promote weight gain. All patients agreed that the dietitian provided useful information that was suited to their needs. All also, agreed that COPD patients should see a dietitian. Although individualized nutrition counselling was not successful in improving nutrient intake to promote weight gain, the patients perceived that it was beneficial. The patients' declining physical well-being limited the ability to intervene successfully. Earlier nutrition intervention and more aggressive nutrition support may be required for these patients.

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CHAPTER ONE

INTRODUCTION

An association between chronic obstructive pulmonary disease (COPD) and malnutrition has been well documented. Over the last decade, there has been increased interest in nutritional status as a determinant of the course and prognosis of COPD. Marasmic protein calorie malnutrition is common in COPD patients[1,2,3,4] and is associated with impaired respiratory function, depressed immunocompetence and increased morbidity and mortality. [5,6,7,8,9,10,11]

Improvements in nutritional status have been shown to result in improved respiratory muscle strength and endurance, and well-being in COPD patients.[12,13,14] Adequate nutrient intake to promote weight gain and improved nutritional status has been achieved in inpatients by short-term tube feeding (16 days)[13] and in outpatients by longer-term oral supplementation (eight weeks to three months).[12,14] Outpatient studies which supplied liquid oral supplements documented great variation in the patients' ability to consistently consume the supplements and increase energy intake. Over time, patients tended to decrease the amount of supplement consumed. After discontinuation of supplements, patients tended to return to inadequate levels of nutrient intake and lose weight.[12,15,16]

In the present study, it was anticipated that individualized nutrition counselling would facilitate a sustained increase in nutrient intake. Improvements in nutritional status were expected to enhance pulmonary function. Ultimately, the goal of nutrition intervention is to show that improvements in respiratory function and immunocompetence lead to reductions in morbidity and mortality and enhanced quality of life for COPD patients.

The goal of this six month study was to obtain information which would complement that of previous shorter nutrition intervention studies ($\leq$ three months) in COPD patients. This information would be beneficial for planning nutritional therapy for COPD patients.

STUDY OBJECTIVES

1. To identify dietary and personal factors which contribute to weight loss and decreased nutrient intake in malnourished, ambulatory patients with COPD.

2. To determine the effect of individualized nutrition counselling in the home environment on nutrient intake, body weight and body composition in malnourished patients with COPD.

3. To investigate whether changes in body weight and body composition result in changes in respiratory function as determined by spirometric, inspiratory and expiratory muscle strength tests and a six-minute walk test.

4. To determine the perceived-well being of malnourished ambulatory patients with COPD.

5. To evaluate the nutrition education provided to these patients.

6. To monitor the acceptance and usage of an oral nutritional supplement designed specifically for COPD patients, Pulmocare™.

NULL HYPOTHESES TESTED

1. (a) There will be no difference in the mean energy intake at baseline, two weeks, three months or six months within either the treatment group or the control group.

   (b) Mean energy intake at baseline, two weeks, three months and six months will not differ between the treatment and the control groups.

2. (a) There will be no difference in the mean protein intake at baseline, two weeks, three months or six months within either the treatment group or the control group.

   (b) Mean protein intake at baseline, two weeks, three months and six months will not differ between the treatment and the control groups.

3. (a) There will be no difference in the mean body weight at baseline, three and six months within the treatment or the control group.

   (b) Mean body weight at baseline, three months and six months will not differ between the treatment and the control groups.

4. (a) There will be no difference in mean fat free mass (FFM) at baseline, three and six months within the treatment group or the control group.

   (b) Mean FFM at baseline, three and six months will not differ between the treatment and the control groups.

5. (a) There will be no difference in the maximal static inspiratory pressure (MIP) at baseline and six months within either the treatment or the control group.

   (b) Mean MIP at baseline and six months will not differ between the treatment and the control groups.

6. (a) There will be no difference in the mean maximal static expiratory pressure (MEP) at baseline and six months within either the treatment or the control group.

   (b) Mean MEP at baseline and six months will not differ between the treatment and the control groups.

7. (a) There will be no difference in the mean distance walked in six minutes at baseline and six months within either the treatment or the control group.

   (b) Mean distance walked in six minutes at baseline and six months will not differ between the treatment and the control group.

8. (a) There will be no difference in the mean perceived well-being scores at baseline and at six months within the control group or the treatment group.

   (b) Mean perceived well-being scores at baseline and six months will not differ between the treatment and the control groups.

CHAPTER TWO

LITERATURE REVIEW

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

The term COPD encompasses a spectrum of diseases including emphysema, chronic obstructive bronchitis, asthma and bronchiectasis.[17] Predominantly diseases of smokers, these disorders are characterized by persistent bronchial airflow obstruction, leading to ventilation-perfusion mismatch with associated hypoxemia, hypercapnia, and compensated respiratory acidosis.[17] Of these diseases, chronic bronchitis and emphysema are the most predominant. In chronic bronchitis, several elements contribute to narrowed airways and increased airflow resistance. These include excessive intrabronchial secretions, mucosal edema and inflammation, hyperplasia and hypertrophy of mucus glands and goblet cells, and bronchial muscle spasm. In emphysema, destroyed alveolar septa, enlarged acini, and loss of lung recoil pressure cause limited airflow. Frequently, the pathological features of both emphysema and chronic bronchitis coexist in the same individual.[17,18] Definitions of terms relating to pulmonary function and pulmonary disease can be found in the glossary. Assessment of pulmonary function in COPD patients is discussed in Appendix 1.

The goals of the treatment of COPD are to prevent disease progression, reverse acute complications, optimize pulmonary function and functional status and prolong life. Treatment involves drug therapy and lifestyle modifications. Medications used to treat COPD include antiinflammatory agents, bronchodilators, and broadspectrum antibiotics. Lifestyle modifications include stopping smoking, exercising regularly, making activities of daily living as energy efficient as possible, using controlled breathing techniques and eating a nutritionally adequate diet.[17] Nutritional status is an important consideration for COPD patients.

Nutritional depletion is a more frequent finding in patients with emphysema than in patients with chronic bronchitis. Patients with emphysema or "Pink Puffers" typically are thin, tachypneic, and normoxic. Patients with chronic bronchitis or "Blue Bloaters" tend to be heavier, less distressed-appearing but cyanotic.[1]

PREVALENCE OF MALNUTRITION IN COPD

The prevalence of malnutrition and its severity varies according to the COPD population surveyed.[19] Vandenbergh and associates[11] observed weight loss of more than 10% of initial weight in 71% of COPD patients who were followed over a six year period. Patients with weight loss had a shorter survival time than those with stable weight. Five years after the onset of weight loss, weight-losing patients had a survival rate of 49%. This was significantly lower than the 76% survival rate of patients with stable weight.

Hunter and coworkers[2] found that 50% of their hospitalized COPD patients had weight measurements between 61 and 90% of their usual weight. Driver et al[20] reported that COPD patients who develop acute respiratory failure are likely to be malnourished. There were significantly greater deficits in percent ideal body weight, triceps skinfold thickness and arm muscle circumference in COPD patients who developed acute respiratory failure than in patients with stable COPD.

In outpatients with COPD, the prevalence and severity of nutritional depletion were found to be lower than reported in hospitalized patients.[21] Twenty-seven percent of an outpatient population reported weight loss of greater than five percent over the previous year. Overall, however, 48% of this population had a body weight loss of at least five percent, or a body weight less than 60% of predicted, or triceps skinfolds less than 60% of predicted.[4] Another study involving 64 outpatients with moderate to severe COPD found that the patients had a body weight range from 60% to 147% of standard weight for height, age and sex. Forty-seven percent of these patients were below 85% of their standard weight.[22] These studies document that malnutrition is a significant problem for patients with COPD. Several potential mechanisms have been proposed to explain the malnutrition observed in these patients.

POTENTIAL MECHANISMS OF MALNUTRITION IN PATIENTS WITH COPD

Adaptive Mechanism

Under normal circumstances, semistarvation and weight loss are associated with decreased total oxygen consumption.[23] Theoretically, therefore, individuals with severe COPD may lose weight as an adaptive mechanism to reduce ventilation requirements.[8] There is no scientific support for this theory.

Reduced Nutrient Intake

In COPD patients, reduced energy intake may be related to a flattened diaphragm impinging upon the stomach. This may potentiate early satiety and increased gastrointestinal discomfort.[8] COPD patients have been shown to be prone to nonspecific gastrointestinal disorders such as abdominal bloating and belching.[8] Additionally, anorexic effects of drugs used in COPD treatment such as theophylline, may contribute to depressed intake.[24] Toxic levels of theophylline cause nausea and vomiting which may also compromise nutrient intake.[6]

Several authors suggest that dyspnea, associated with chewing and swallowing food, impairs nutrient intake.[24,25,26,27] Wilson found that the most severely dyspneic patients had the lowest energy intakes.[27] Blood gas alterations themselves may contribute to reduced intake. Both dyspnea resulting from increased carbon dioxide (CO_2) production due to a high carbohydrate (CHO) diet and arterial oxygen (O_2) desaturation during eating may limit intake in patients with depressed respiratory reserve.[28] Some individuals with severe COPD experience significant arterial O_2 desaturation during meals. This desaturation occurs most commonly in patients with lower baseline O_2 saturation.[29]

Additionally, psychosocial factors may influence nutrient intake.[30] Common responses to increasing weakness, dyspnea or cough and sputum production are resentment, anger, anxiety, fear, and depression. Depressed individuals may lack adequate motivation or energy to attend to their nutritional needs.[31] Other factors could include inadequate nutrition knowledge, and undesirable living and eating arrangements.[8] Elderly people with lower levels of education and income, and those who are housebound, living alone, bereaved or disabled are more likely to become malnourished.[32]

Adequacy of Intake

The reported adequacy of dietary intake for COPD patients has been inconsistent. Factors contributing to variable results include food record accuracy and duration, and differences in the standards used to assess the adequacy of intake. Hunter and associates[2] found evidence in some COPD patients that reduced energy intake was at least partially responsible for development of protein calorie malnutrition. Overall, however, this study concluded that energy intake was not necessarily less than the recommended daily allowance (RDA). Vandenbergh[11] found that COPD patients with weight loss had normal energy intakes and patients with stable weight had energy intakes that appeared to be too high.

Braun and colleagues[4] indicated that based on predicted needs for a healthy population, energy intake was adequate in a sample of 60 outpatients with COPD. Patients with more severe nutritional depletion tended to ingest more calories per kilogram of body weight than those who were at more normal weight, normal percent triceps skinfolds, and normal percent midarm muscle circumference. Ryan and associates[33] found 10 COPD patients to have intakes of 135% of their measured resting energy expenditure (REE) and concluded that these patients consumed adequate energy to meet average energy requirements. Another study reported that malnourished patients with COPD had lower mean energy intakes than those who were well nourished.[12] Of 64 outpatients with stable COPD, the group of patients which had weights <75% of standard body weight consumed significantly more energy as a percentage of estimated daily energy expenditure than patients who were at normal weight.[22] Other investigations have also documented maintenance of underweight status with usual energy intakes of 150% basal energy expenditure (BEE).[15] If energy intake appears to be adequate, it may be that increased energy requirements can explain the weight loss observed.

Increased Energy Requirements

Several recent studies have examined energy expenditure in malnourished COPD patients.[33,34,35,36,37,38,39] In 10 COPD patients with a weight range of 64% to 100% of ideal body weight (IBW), REE was 117% of predicted values.[34]

Goldstein and associates[35] reported that the REE of malnourished patients with COPD was 16% greater than predicted values and it was also greater than that of a control group of malnourished patients without COPD. This study also reported that the thermic effect of nutrients administered either as a high carbohydrate (53%) or high fat (55%) diet was greater in COPD patients. The difference between the two groups was enhanced during feedings with the high CHO regime. Wilson et al[36] found a significantly elevated REE (115% predicted) in undernourished (<85% IBW) COPD patients. In contrast, the control group without COPD and COPD patients at normal weight had measured REEs similar to predicted energy requirements using the Harris Benedict equation.[40] Similarly, values of REE of 119%[37] and 125%[41] of predicted were reported in malnourished COPD patients (< 90% IBW and < 80% IBW respectively). These compared to 105%[37] and 115%[41] of predicted in adequately nourished patients with COPD and 94% in normal control subjects.[37]

Moore and associates[42] used four predictive equations, including the Harris Benedict equation, to predict REE in COPD patients with and without respiratory failure. All equations were found to underestimate REE as measured by indirect calorimetry.

In contrast, Ryan and associates[33] did not find evidence of increased REE in 10 moderately malnourished (78% IBW) patients with COPD. Measured REE was 94% of that predicted by the Harris Benedict equation. Thermogenesis did not appear to be abnormal. They concluded that although REE was normal, it was likely somewhat elevated given that in malnutrition energy expenditure tends to be reduced.[23]

Many factors could account for the variation in the results of these studies. Severity of COPD and degree of malnutrition were similar; therefore, variation in measurement of REE is likely a significant factor.[33] Insufficient time to allow steady state to occur,[36] lack of control in timing of measurements or incomplete fasting[35] would contribute to elevated REE measurements.[33]

Using a respiration chamber (5 x 2.5 x 2.5 meters), Hugi and associates[38] measured the 24 hour energy expenditure of 13 COPD patients and 8 control subjects matched for age. From this data, they estimated the 24 hour energy expenditure of these groups in free-living conditions. They concluded that although basal metabolic rate was increased in COPD, the measured 24 hour energy expenditure in a confined chamber and the estimated free-living 24 hour energy expenditure were not different between COPD and normal subjects. A reduced physical activity level in the COPD patients could explain these results. When comparing energy balance between weight stable and weight-losing patients with stable COPD, REE was found to be greater in weight-losing patients when values were normalized for predicted metabolic rate or kg fat free mass (FFM).[43] When compared to healthy control subjects, REE adjusted for FFM was significantly increased in weight-stable COPD patients. FFM explained 84% of the interindividual variation in REE in the healthy control group but only 34% of the variation in REE in COPD patients. This suggests that for patients with COPD other factors in addition to FFM are important determinants of REE.[44]

Hugi and associates investigated tumor necrosis factor (TNF) levels and energy expenditure in COPD patients. They found COPD patients to be hypermetabolic at rest in comparison to an age, body weight and fat free mass matched control group without COPD. Circulating TNF levels were not different between these two groups. In the COPD group, however, there was a statistically significant positive correlation between TNF level and REE expressed as percent predicted.[39] Another study found no measurable TNF activity in COPD patients. Hypermetabolism was not evident in this group.[45]

Compared to a normal control group, individuals with stable COPD showed a higher oxygen consumption ($\dot{V}_{O2}$) during sleep, when overnight $\dot{V}_{O2}$ was measured. This increase in $\dot{V}_{O2}$ may be related to increased respiratory work load during sleep.[46]

Increased respiratory muscle O_2 consumption has been observed in COPD patients.[37,47] Normally, $\dot{V}_{O2}$ of the respiratory muscles accounts for less than eight percent of total O_2 demand. Patients with COPD utilize a four to ten times greater portion of $\dot{V}_{O2}$ for respiration than do normal subjects.[48,49] Increased resistive load and decreased respiratory muscle efficiency, along with high total expired ventilation ($\dot{V}_E$) contribute to the increased cost of breathing.[7,37] Donahoe and associates[37] found that the oxygen cost of breathing was markedly increased in malnourished patients with COPD when compared to adequately nourished (>90% IBW) COPD patients. Hyperinflation and its associated respiratory muscle mechanical disadvantage combined with reduced respiratory muscle strength associated with malnutrition, likely result in decreased ventilatory efficiency. This may precipitate a vicious cycle of increased energy expenditure and weight loss.[37]

Alternately, if malnourished patients with stable COPD are not hypermetabolic, consume adequate energy and have normal thermogenesis, weight loss could occur in a step wise fashion during periods of clinical instability. Episodic exacerbations of chronic airflow obstruction or infection could result in anorexia and inadequate intake at a time of increased nutrient needs. As a result, a significant amount of weight could be lost over a short period of time. Upon recovery, patients may be able to maintain but not regain weight.[8,33] Although the mechanism of malnutrition in

COPD is not clear, evidence indicates that malnutrition has detrimental effects on respiratory function.

EFFECTS OF MALNUTRITION ON RESPIRATORY FUNCTION

The respiratory system essentially has three subdivisions. It is a composite of a drive mechanism, the central nervous system; a pump, the respiratory muscles; and a gas exchange organ, the lung. Nutritional status can influence the function of all three subsystems.

Nutrition and Ventilatory Drive (CNS)

When P_{O2} is < 55 mmHg, hypoxia becomes the main ventilatory drive for COPD patients. In contrast, in normal individuals increased ventilatory drive results primarily from increases in arterial carbon dioxide tension (Pa_{CO2}).[50] Generally, conditions which reduce metabolic rate decrease ventilatory drive. Starvation, for example, is associated with reduced metabolic rate, which returns to normal with refeeding.[23] Doekel and associates[51] demonstrated a 42% decrease in ventilatory response to hypoxia in persons subjected to 10 days of semistarvation. With refeeding, these changes were reversed. This study did not demonstrate a significant change in hypercapnic drive. Other studies indicate that high protein intake augments hypercapnic drive.[52] High carbohydrate intake has also been demonstrated to increase ventilatory drive in normal subjects.[52] This data indicates that both quality and quantity of food intake affect ventilatory drive.[53]

Nutrition and the Respiratory Muscles (Pump)

Adequate respiratory muscle strength and endurance are required for normal ventilation. Ventilation maintenance depends on the respiratory muscles' ability to generate the force needed to overcome the elastic and resistive properties of the lung. Lung resistance is increased in COPD and is associated with increased work of breathing.[5] Malnutrition impairs respiratory muscle function by decreasing the availability of energy substrates and altering the structure of muscle fibres.[53]

Reduced Substrate Availability

A reduction in muscle efficiency, (increased O_2 cost for equivalent power generation), or a reduction in the rate of energy supplied to a muscle, or reduced energy stores within a muscle will predispose that muscle to fatigue.[54] Inspiratory muscle fatigue is a factor which limits exercise capacity in patients with COPD and may contribute to respiratory failure.[7] Contractile muscle failure, one form of muscle fatigue, may occur when energy demands exceed energy availability.[54] Adenosine triphosphate (ATP) is essential for all muscle work; therefore, the muscle must generate adequate ATP for work performance. Skeletal muscles (and likely the diaphragm) have limited stores of ATP. Respiratory loading, especially with increased respiratory flow and resistive loads, challenges the diaphragm's capability to generate ATP. To meet this challenge, the diaphragm relies on a number of mechanisms including increased blood flow, increased utilization of intramuscular substrates (lipids and glycogen) and blood-borne substances (glucose and fatty acids).[55]

Malnutrition will alter the availability of both local and blood derived energy substrates. Acute reduction in food intake leads to depleted glycogen stores, while prolonged starvation leads to decreased total body fat and lean muscle mass. This results in lower circulating levels of substrates (lipids and branch chain amino acids).[53]

COPD patients in acute respiratory failure were found to have significantly lower concentrations of ATP and creatine phosphate in intercostal and quadriceps muscles than COPD patients without respiratory failure. With refeeding, there was a significant increase in ATP, creatine phosphate and glycogen in these muscles.[56]

A further study investigating blood fuel metabolites in patients with advanced COPD found that patients with chronic respiratory failure had lower concentrations of free fatty acids (FFA), glycerol and 3-hydroxybutyrate, and higher concentrations of glucose and lactate, than those patients without chronic respiratory failure. Arterial FFA, glycerol and 3-hydroxybutyrate concentrations correlated positively with arterial P_{O2} and negatively with arterial P_{CO2} at rest as well as during exercise and recovery. These results suggest that lipolysis is reduced in patients with advanced COPD with chronic respiratory failure and that these patients may have altered glucose metabolism.[57]

Altered Structure of Muscle Fibres

Previously, it was believed that respiratory muscles were spared any loss in muscle protein during starvation because of their constant activity.[8] Both animal and human studies show that this is not the case. Rats starved for 10 days experienced a significant reduction in lung tissue mass that was proportional to the decrease in total body mass.[58] Similarly, significant linear correlations between body weight and diaphragm mass have been found in humans.[59,60] Arora and associates measured diaphragm muscle mass, thickness, area and length at necropsy. In the nonobese, very muscular group (>120% IBW) all diaphragm muscle dimensions were significantly greater than those of the normal weight group (85-115% IBW). The diaphragm dimensions of the underweight group (<85% IBW) were significantly lower than normal.[60]

Arora and Rochester[61] demonstrated significantly reduced respiratory muscle strength, maximal voluntary ventilation and vital capacity in nutritionally depleted patients without lung disease. The loss of muscle strength was shared equally between inspiratory and expiratory muscles and was directly proportional to the degree of weight loss. Respiratory muscle strength was diminished to a greater extent than the diaphragmatic muscle mass.

Kelly and colleagues used total exchangeable potassium and sodium to estimate body cell mass and nutritional status of 59 surgical patients requiring TPN. There was a positive correlation between body cell mass and inspiratory muscle strength. After receiving TPN, the majority of these patients demonstrated improvement in both body cell mass and inspiratory muscle strength.[62]

There is a curvilinear relationship between vital capacity (percent predicted) and respiratory muscle strength (percent predicted). Vital capacity was 65% of predicted at one half normal respiratory muscle strength and 50% of predicted at one third normal respiratory muscle strength. In patients with myopathic respiratory muscle weakness, no CO_2 retention occurred when respiratory muscle strength fell from normal to half normal. However, arterial partial pressure of carbon dioxide (Pa_{CO2}) increased after respiratory muscle strength decreased below half normal. CO_2 retention became clinically significant when respiratory muscle strength was one third to one quarter normal.[63]

The strength and endurance of a muscle depends on its fibre composition. The diaphragm and intercostal muscles contain a mix of all three muscle fibre types, type I (slow twitch-oxidative), type IIA (fast twitch-oxidative) and type IIB (fast twitch-glycolytic).[64] Starvation is associated with diaphragm muscle atrophy and a reduction in muscle fibre output. Reduction in diaphragm mass results from a decrease in muscle fibre cross sectional area. The greatest magnitude of loss occurs in fast twitch fibres.[64,65,66] Atrophy of fast twitch muscle fibres exerts a considerable impact on both the contractile and fatigue properties of the diaphragm.[67] Fast twitch muscle fibres generate larger levels of force more rapidly than slow twitch fibres but they are more susceptible to fatigue. Because fatigue is related to the level of force generated during breathing, fast twitch fibre atrophy predisposes the diaphragm to fatigue.[64] Mineral and electrolyte imbalances in malnutrition may also contribute to reduced muscle contractility.[5,24]

Additionally, drugs used in COPD treatment have an impact on respiratory muscles. Long-term steroid use promotes protein catabolism and gluconeogensis. Consequently, generalized muscle weakness and negative nitrogen balance may result.[6]

To assess the clinical value of nutritional repletion, it is important to determine whether it can promote increased respiratory muscle strength to a level where patients can be weaned from mechanical ventilation. Several studies indicate that this is possible. Combined results from two retrospective studies show that of 70 patients investigated, 42 patients did not receive adequate nutritional support. Of these 42 patients, only 48% were successfully weaned from mechanical ventilation. A significantly greater percentage (89%) of the 28 patients who received adequate nutritional support were weaned successfully.[63] Further well controlled refeeding studies are required to establish the relationship between nutritional support, respiratory muscle strength and the ability to wean from a ventilator.

Nutrition and Lung Structure (Gas Exchange Organ)

Keys and associates observed a reduction in respiratory efficiency (expressed as O_2 extraction/1 minute ventilation) with 12 weeks of semistarvation and a further reduction after 24 weeks. It returned to normal with refeeding.[23]

Morphologic and ultrastructural changes in lungs of starved rats are similar to changes seen in emphysema. Adult rats partially deprived of food for 10 days, developed airspace enlargement with a minimal degree of alveolar wall destruction.[58] Kerr and Riley[68] compared rats that were partially deprived of both energy and protein to a control group and to a group deprived only of energy. Only the rats that were protein deprived displayed emphysema like changes in the lung. They concluded that protein was the major dietary component contributing to nutrition induced emphysema in rats.

When starved for three weeks, rats lost 40% of their body weight and displayed increased lung surface forces and decreased lung tissue elasticity.[69] It was hypothesized that increased surface forces were related to surfactant loss. Additionally, alterations in tissue elasticity likely resulted from reduced connective tissue components. In starved rats, lung proteolytic activity has been shown to be significantly increased and total lung collagen content to be decreased.[69]

One study where rats were deprived of food or water showed a reduction in minimal surface tension of the lung extract in the water deprived group but an increase in minimal surface tension in the food deprived group. Lecithin content was reduced in the food deprived group but increased in the water deprived rats.[70] From these observations, it was concluded that starvation and dehydration will adversely affect the balance between production and degradation in surface active materials.

Glucose is an important carbohydrate substrate for phosphatidylcholine (PC) synthesis. In the lung, the fatty acid component of PC can be synthesized from glucose or taken from plasma.[8] Starvation reduces the availability of precursors for phospholipid synthesis. This likely results in reduced surfactant synthesis.[49]

Schols and associates[71] demonstrated a relationship between impaired nutritional status and disturbed gas exchange in COPD. Mean arterial partial pressure of oxygen (Pa_{O2}) was significantly decreased and Pa_{CO2} increased in COPD patients with impaired nutritional status. Another study found that body weight was inversely related to diffusing capacity after adjusting for forced expiratory volume in one second (FEV_1)[9]. In malnourished patients with COPD, decreased capacity to synthesize and secrete surfactant, along with malnutrition induced structural changes in the lung may have deleterious effects on pulmonary mechanics. Malnutrition may contribute to the development of atelectasis and significantly delay recovery from acute exacerbations of chronic lung disease.[8]

PULMONARY DEFENSE MECHANISMS

Pulmonary infection is a major complication of malnutrition.[7] Additionally, it is well recognized that impaired nutritional status results in compromised immunocompetence.[72,73] The primary aspects of immune system impairment in protein calorie malnutrition include cell mediated immunity, secretory IgA antibody response, complement system and bactericidal capacity of neutrophils. By impairing local defenses, it is likely that malnutrition increases the incidence, severity and duration of respiratory infections.[65]

Impaired cell-mediated immunity has been reported in COPD patients.[2,3,26] Hunter found 38% of hospitalized COPD patients were anergic.[2] Yoneda reported an anergy incidence of 60% in COPD patients.[3]

Respiratory tract colonization had a direct relationship with nutritional status parameters in COPD patients. Patients with significant nutritional impairment had more tracheal bacterial cell adherence and were more frequently colonized by Pseudomonas species. Improvement in nutritional status lead to a reduction in bacterial cell binding to tracheal epithelial cells.[5] Bacteria clearing from the lung is depressed in hypoxia and malnutrition.[74] After refeeding a small group of COPD patients for 21 days, it was found that increased energy intake and weight gain were associated with an increase in reactivity to skin test antigens and an increase in absolute lymphocyte count.[75]

The high prevalence of malnutrition among patients with COPD and its association with impaired respiratory function, depressed immunocompetence and increased morbidity and mortality suggest that nutritional intervention should be an integral part of therapy.

NUTRITIONAL CONCERNS FOR PATIENTS WITH PULMONARY DISEASE

The goal of nutritional intervention is to improve nutritional status with minimal stress on the respiratory system. Diet composition has been shown to affect carbon dioxide production and respiratory drive. For patients with impaired respiratory function, these effects may have clinical significance.

Respiratory Quotient and Gas Exchange

The process of converting fat, CHO, and protein to energy results in O_2 consumption and CO_2 production. The ratio of CO_2 produced to O_2 consumed is the respiratory quotient (RQ). The RQs for CHO, protein, and fat are 1.0, 0.8, 0.7, respectively. The RQ of a typical mixed diet is .85.[50]

Fat yields the least CO_2 production for the amount of O_2 consumed. Much debate has developed over the provision of CHO verses fat calories to patients with respiratory insufficiency.

CHO versus Fat

Diets administered with increased fat calories and reduced CHO calories can decrease CO_2 production and RQ. Consequently, ventilatory requirements are diminished. Gieseke and colleagues[76] observed increased minute ventilation, RQ, CO_2 production and O_2 consumption after a single CHO load. This increased endogenous CO_2 load, however, was well tolerated even in patients with advanced COPD. Askanazi and associates[77] compared the effect of a high fat TPN feed (nonprotein calories 50% fat, 50% CHO) with a high CHO TPN feed (nonprotein calories 100% CHO). The high CHO diet resulted in a 20% increase in CO_2 production and a 26% increase in minute ventilation when compared to the CHO/fat regime. The RQ increased from .87 to 1.0.

In ambulatory COPD patients, consumption of a low CHO (28% CHO, 55% fat) or moderate CHO (53% CHO, 20% fat) liquid diet resulted in lower CO_2 production, RQ and Pa_{CO2} than a high CHO (74% CHO, 9.4% fat) liquid diet. Oxygen consumption, blood pH, arterial P_{O2} and minute ventilation were not different between the diets.[78]

In comparison to a placebo drink (< 10 kcal), after consumption of a single large liquid CHO load (920 kcal), $\dot{V}_{CO2}$ and $\dot{V}_E$ were increased at rest, in a group of patients with stable COPD. Forty minutes after CHO load ingestion, walking performance was tested. At this time, the average total 12-minute walking distance decreased 5.1%. However, interindividual variability in the reduction of distance walked was great. It ranged from 0.2 to 15.2%.[79]

Mean maximal work load performed by patients with chronic airflow obstruction was lower after a high CHO (53% CHO, 30% fat) liquid meal than after a high fat (55% fat) low CHO (28%) liquid meal or a noncaloric placebo liquid meal. This finding suggests that meals with high fat and low CHO content may be less likely to impair work performance, in patients with chronic airflow obstruction in the post absorptive phase, than meals with a high CHO content.[80]

CHO consumed in excess of energy requirements is converted to fat. The theoretical RQ of glucose conversion to triglyceride is 8.67. Clinically, however, the RQ never reaches these levels. An RQ greater than 1.0 indicates net lipogenesis.[50]

When metabolic pathways are diverted from oxidation to lipogenesis, CO_2 production decreases by 68% and O_2 consumption decreases by 96%. This disproportionate decrease in O_2 consumption explains the increase in RQ despite the decrease in CO_2 production. This decrease in CO_2, however, is undesirable because it is not associated with the concomitant production of significant amounts of ATP.[81]

The clinical significance of increased CO_2 production with high CHO diets appears to be most critical for patients in respiratory failure or for those who are being weaned from mechanical ventilation.[50] Stable patients with COPD appear to be able to consume diets containing 50-55% CHO with no adverse effects.[5,8]

Protein and Micronutrients

Protein intake has little effect on CO_2 production but has been demonstrated to augment the ventilatory drive mechanism.[82] Consequently, adequate protein to meet individual needs should be provided, but overfeeding of protein should be avoided.[8] It has been suggested that increased ventilatory drive is the result of an altered ratio of tryptophan to amino acid competitors at the blood brain barrier. The resulting decrease in central serotonin production could lead to increased ventilatory sensitivity.[83]

Patients with COPD may be predisposed to having increased micronutrient requirements. If COPD patients have increased energy requirements, they may also have increased needs for water soluble vitamins involved in energy metabolism. Also, applicable to the predominantly elderly COPD population are concerns with potential depressed water soluble vitamin storage and altered nutrient absorption.[22] Special attention should be given to magnesium, potassium and phosphate as these nutrients can affect respiratory muscle function.[24] In hypophosphatemia, myopathic changes occur. This may be caused by severe red cell 2,3-diphosphoglycerate depletion which impairs O_2 transfer capability from hemoglobin to myoglobin. Additionally, an intracellular phosphate deficit results in decreased high-energy substrate availability and impaired energy dependent processes including muscle contraction.[84] There is increasing evidence that nutritional intervention results in improved nutritional status and respiratory function.

NUTRITIONAL INTERVENTION IN AMBULATORY PATIENTS WITH COPD

Attempts to refeed malnourished COPD patients have had variable results. Only studies which achieved adequate energy and protein intake to promote significant weight gain and improved nutritional status were able to demonstrate improvement in respiratory function.[12,13,14,15,16]

Knowles and associates[16] conducted a randomized, observer blind crossover trial to investigate the effects of oral nutritional supplementation on respiratory function in 25 ambulatory patients with severe COPD. Patients were supplemented for eight weeks. There was considerable variation in the patients' ability to increase energy intake (range -5 to +86%). No weight gain occurred after four weeks of supplementation and only slight weight gain occurred after eight weeks. No improvement in respiratory muscle strength or endurance was reported.

Lewis and colleagues[15] fed 21 malnourished COPD patients an enteral formula in addition to their usual diet for eight weeks.

Patients did not tolerate the supplement well and tended to decrease their usual intake while receiving the supplement. As a result, no significant changes in anthropometric measures, pulmonary function or respiratory muscle function were observed.

In contrast, Wilson and associates[14] documented increased body weight, and increased respiratory muscle function in six patients with emphysema who were admitted to a metabolic ward for refeeding. They were fed a diet with an energy level of 1.5x their REE for two weeks. A prospective randomized control trial involving 14 malnourished COPD patients which provided oral supplementation for three months to achieve an energy intake of either 2300 calories (for women) or 2500 calories (for men) per day also documented improvement in body weight and nutritional status.[12] Respiratory muscle and hand grip strength increased in parallel with nutritional status. There were no changes in pulmonary function or arterial blood gas tension. Improved nutritional status also correlated with improved sternomastoid muscle contractility and fatigability. General well-being, breathlessness scores and six-minute walking distances also significantly improved with supplementation.

Twenty-eight malnourished patients with emphysema participated in a placebo controlled, randomized, double blind study.[85] For 13 weeks, 13 patients in the fed group received a 1 kcal/ml nutritional formula (20% protein, 30% fat and 50% CHO). These patients were instructed to consume 400 ml/day of supplement in addition to their usual diet. The control group received 400 ml/day of a liquid which had the same consistency and taste but only contained 0.1 kcal/ml. The fed group had a mean weight gain of 1.5 kg which was significantly greater then the 0.16 kg weight gain of the control group. No differences were observed in pulmonary function tests, FVC, FEV_1, maximal voluntary ventilation, or immunological status.

Yoshikawa and associates[86] examined the effect of long-term supplementary oral nutrition in malnourished patients with COPD. The abstract reported that 11 ambulatory patients consumed a branch chain amino acid enriched enteral formula in addition to their usual diet for 12 months. The amount of additional energy consumed was not reported. After three months of supplementation, there was significant improvement in nutritional status, increased body weight and skinfolds, and increased maximal inspiratory mouth pressure. Arm-muscle circumference and hand grip strength were also increased at 12 months. Self-perceived quality of life and level of dyspnea scores also improved over the study period.

Another study, to date reported only in abstract form, investigated the effects of supplemental nutrition alone or in combination with an anabolic steroid (nandrolone deconoate) in a double blind randomized trial involving a group of COPD patients enrolled in an inpatient rehabilitation program.[87] Dietary intake was 160% of measured REE in the treatment group and 140% in the placebo group. The treatment group experienced a significant increase in weight and fat free mass. Additionally, there was a significant exercise x treatment interaction for weight and fat free mass and percent fat mass. This study concluded that daily consumption of a nutritional supplement in addition to a normal dietary intake as part of a general physical training program gradually increases fat free mass in depleted COPD patients. Also, supportive treatment with nandrolone deconoate may enhance the repletion of fat free mass.

A pilot study involving seven malnourished COPD patients who received a nutritionally adequate diet for one week followed by three weeks of diet plus daily subcutaneous injections of recombinant methionyl human growth hormone (GH) documented no increase in weight during the week of diet alone. Significant weight gain, however, did occur during the first week of GH treatment. Weight gain continued over the following two weeks. Nitrogen balance and maximal inspiratory pressure also improved with GH treatment.[88]

Increases in REE, fat oxidation, nitrogen and glucose balance, and decreases in protein and glucose oxidation were attributed to administration of GH to six malnourished COPD patients receiving eucaloric TPN for 12 days. The ability of GH to promote positive nitrogen balance with a eucaloric diet which would otherwise require a significant increase in energy intake may be beneficial for COPD patients who have limited tolerance for excess nutrition.[89]

The effects of an alternate approach to refeeding, short-term tube feeding, was investigated in a prospective randomized control study involving 10 hospitalized, malnourished patients with COPD.[13] Patients were fed enterally an additional 1000 calories to their usual intake. After 16 days of refeeding, there was significant increase in weight, and improvement in respiratory muscle endurance and expiratory muscle strength. These studies indicate that nutrition in the form of either short-term tube feeding or longer-term ($\leq$3 months) oral supplementation alone or in combination with steroids or growth hormone can achieve improved nutritional status and respiratory function in ambulatory COPD patients. Studies which monitor intake, nutritional status, pulmonary function and well-being for over longer intervention periods are required. Additionally, none of these studies provided nutrition counselling in the patients' homes. Assessment of personal factors affecting nutrient intake need to be further investigated. Additionally, no information is available on the patients' perceptions of the value of nutrition education in COPD.

WELL-BEING IN INDIVIDUALS WITH COPD

Changes in health may be regarded as a series of impacts on daily life and well-being. Treatment of COPD is primarily directed toward relief of symptoms, prevention of exacerbations of disease, improvement of functional ability and quality of life. Quality of life measurement attempts to quantify the impact of disease and the response to interventions on a patient's life and perceived well-being. [90] Subjective well-being is a general term referring to the affective reactions of individuals to their life experiences along a positive-negative continuum. There are three primary aspects of subjective well-being. First, it is subjective and dwells within the experience of the individual. Second, it includes positive measures and not just the absence of negative measures. Finally, it is a global assessment of all aspects of a person's life.[91]

There are two components to perceived well-being, physical well-being and psychological well-being.[92]

Braun and associates[21] used a psychiatric test (SCL-90R) to determine general well-being, and indices of depression, and anxiety in outpatients with COPD. This study concluded that psychiatric factors appeared to only play a minor role in nutritional depletion. Openbrier and colleagues [93] used the Zung self-rating depression scale to determine the depression level in malnourished and adequately nourished COPD patients. Both groups showed a moderate degree of depression.

When life quality was evaluated in a large sample of COPD patients (n=985) results showed that quality of life was severely compromised. These patients' scores for the Sickness Impact Profile (SIP) indicated that the patients' illness had a very substantial effect on psychosocial and physical function. Their scores for the Profile of Mood States (POMS) showed relatively high levels of anxiety, depression, hostility, fatigue and confusion.[94]

Parsons measured well-being in 38 ambulatory patients with severe COPD.[91] At both the time of initial testing and one year later, these patients saw themselves as neither having a very good nor very poor general well-being. Physical well-being scores tended to fall toward the lower end of the scale. Therefore, it appeared that psychological well-being contributed significantly to the average overall well-being scores. Problem-focused coping strategies (ie efforts to deal with the sources of stress, either by changing behaviour or environmental conditions) were positively correlated with psychological well-being. The perception of well-being appeared to be enhanced for those who used problem-focused coping.

The goal in health care is to assist patients to cope with existing problems, while maximizing the patients' potential for health and self-care. Directing nutritional interventions in a way that emphasizes the patients' ability to problem solve and focus on strengths rather than deficits should enhance the patients' perception of well-being.

CHAPTER 3

METHODS

EXPERIMENTAL DESIGN

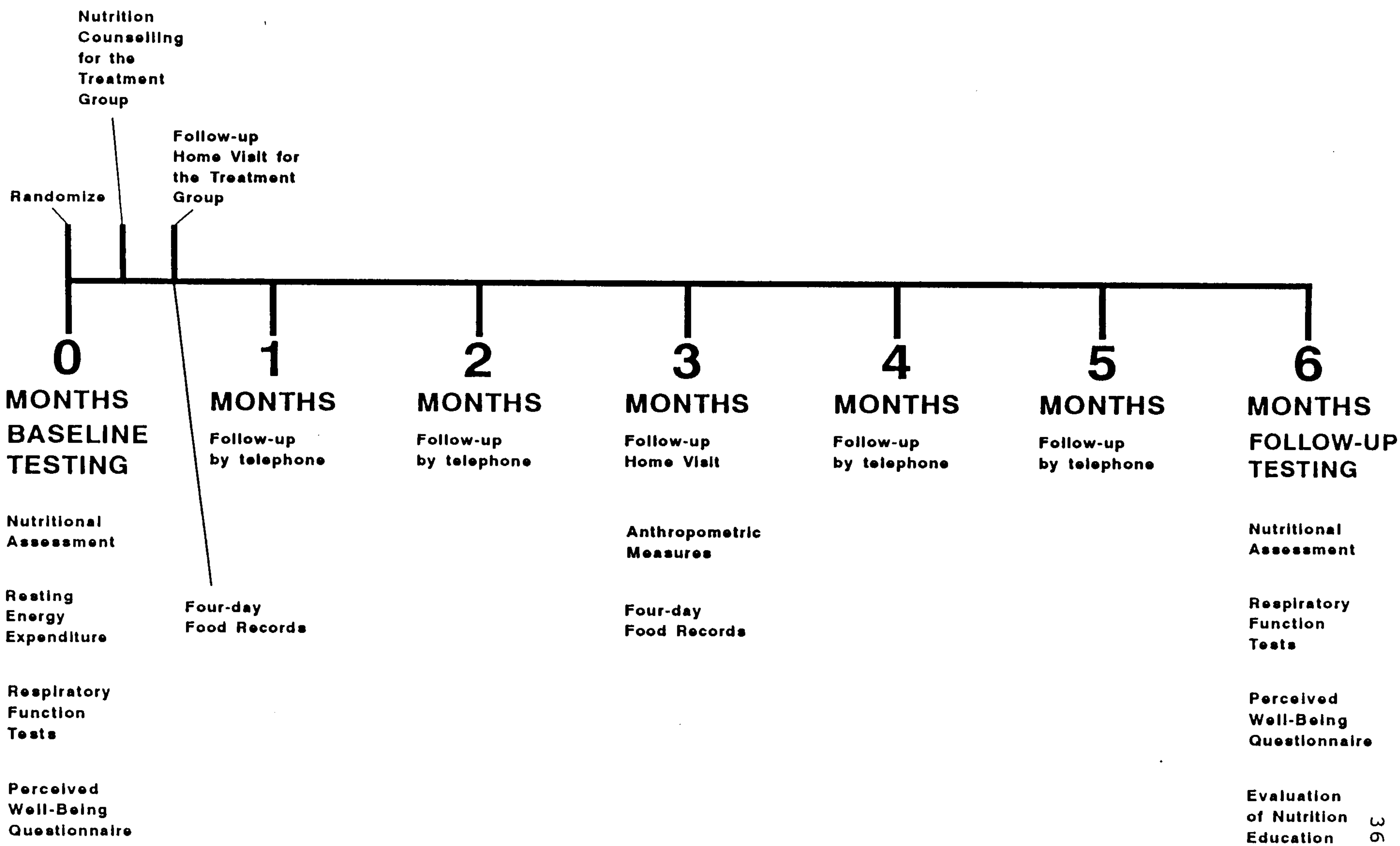
The study was a randomized control design. Fourteen ambulatory, malnourished individuals with stable, moderate to severe COPD were randomized to a treatment or control group. At baseline, the treatment group received individualized nutrition counselling from a Registered Dietitian Nutritionist (RDN) in their homes to establish a diet with a minimum energy intake goal of 150% of REE. An oral supplement specifically designed for respiratory patients (Pulmocare™) was provided for the treatment group. Use of the supplement, although optional, was encouraged by the RDN. The control group received written guidelines to increase energy and protein intake but no individualized counselling was given. Both groups received the same testing and follow-up protocol except for a two-week, follow-up home visit only given to the treatment group. Nutritional status, pulmonary function and well-being were monitored over the six month study period.

Diet history, four-day food records, anthropometric measures and biochemical parameters were used to assess nutritional status. REE was assessed using indirect calorimetry. Spirometry, arterial blood gases, maximal inspiratory and expiratory mouth pressures, and distance walked in six minutes were used to assess pulmonary function. The Perceived Well-Being Scale-Revised (PWB-R)[92] was implemented to assess self perceived well-being. A questionnaire developed by Hauchecorne[95] was administered to evaluate the nutrition education provided. This study received approval from the University of British Columbia Clinical Screening Committee for Research and Other Studies Involving Human Subjects and the University Hospital Research Committee (Appendix 2). Figure 1 outlines the study protocol.

SUBJECT RECRUITMENT

Physician referral was used to recruit subjects from either the University Hospital respiratory clinic (Vancouver, BC), St Mary's Hospital respiratory rehabilitation program (New Westminster, BC) or from a private practice of respirologists in New Westminster, BC. Patients were eligible for the study if they had a) moderate to severe COPD, (ie forced expiratory volume in one second/forced vital capacity (FEV_1/FVC) < 70% of predicted), b) no exacerbation of their disease two months prior to the study and c) if they were free of other chronic diseases. Other subject criteria included an age between 45-80 years and body weight of <90% of ideal body weight according to the Metropolitan height and weight tables (1983).[96] Subjects were required to be living independently, able to purchase food and prepare meals for themselves or have someone available to do it for them. Non-smoking subjects were preferred but those smoking less than six cigarettes/day were allowed into the study. Alcohol consumption was set at < 30 gm alcohol/day. Before enrolling in the study, the subjects provided written consent (appendix 3). Thirty-five patients were referred to the study. Twenty of these met the baseline criteria and were interviewed in their homes.

Figure 1: Outline of the Study Protocol



Subsequently, 14 patients were enrolled into the study. For personal reasons, the other six chose not to enter into the study.

MEASUREMENTS OF NUTRITIONAL STATUS

Diet Assessment

Diet histories were obtained from 20 COPD patients, 14 enrolled in the study as well as six others who were referred to the study but were unable to participate fully. Patients were interviewed in their homes. The diet history questionnaire (appendix 4) included questions concerning personal data, weight history, appetite level, gastrointestinal concerns, chewing and swallowing ability, food allergies and intolerances. Additional information regarding social factors relevant to purchasing food, preparing and consuming meals was also obtained. A recall of a typical day's intake and a food frequency questionnaire (appendix 5) were used to collect qualitative, descriptive information about usual food consumption patterns.[97]

Validity of a food intake record refers to the degree to which it measures what it purports to measure. It is difficult to validate food intake records because the 'truth' about dietary intake is never known with absolute certainty.[97] When a food intake record purports to measure usual intake over an extended period of time, revealing the true intake presents onerous practical difficulties. If the definition of 'usual' has no time limit, then it is impossible to determine the true 'usual' intake.[98] Because the 'truth' is not known, researchers have taken the approach of assessing relative rather than absolute validity. This approach involves evaluating one method against another method which has wider acceptance, or demonstrating that a method elicits a usual intake by showing that it produces similar results on two different occasions.[98] Researchers have used both approaches in trying to validate food records as a tool for determining average daily intake of energy, protein, and vitamins and minerals.[97,99] Reliability/reproducibility, of a dietary intake method is another consideration. A dietary assessment method is considered precise if it gives very similar results when used repeatedly in the same situation.[97] The acceptable level of precision depends on the intended use of the data.[100] In general, studies investigating the precision of seven-day weighed intake records have found good agreement between group mean values obtained for energy and most nutrients on two separate occasions. An exception to this is when subjects have been on special diets.[97] However, seven-day intake records place a high burden on respondents and compliance may be a problem. In one study, regression analysis showed that records from the first two days of record keeping were more valid for assessing group comparison than those from the last three days. Accuracy of recording deteriorated toward the end of the recording period.[101] A four-day food record has been shown to estimate usual protein intake within 10% with 95% confidence for a small group. For the same level of confidence, an intake record of a minimum of three days is required for estimating usual energy intake.[100] For this study, therefore, the use of four-day food records seemed appropriate.

The study participants completed four four-day food records over the study period. These were collected at baseline, two weeks, three months and six months. The food intake record form (appendix 6) was adapted from University Hospital, UBC Site, Dietary Department Dietary History Food Intake Form and the food intake form developed for a previous clinical nutrition study[102] All subjects received written and verbal instruction in their homes on how to record foods eaten, describe cooking methods used and estimate portion sizes in common household measures. Subjects recorded the time, place, and amount of all foods and beverages consumed on four consecutive days. One weekend day was included in the recording period to account for potential day of the week effects on food and nutrient intake.[97] To reduce errors and omissions, a RDN reviewed the completed food records with the subjects. Food records were analyzed for total energy, protein, carbohydrate, and fat using the Food Perfect nutrient analysis program (Intelligent Software Engineering, Victoria, BC). Nutrient values used in analysis were those contained in the Canadian Nutrient File database (Health and Welfare Canada) and supplementary food values obtained from "Bowes and Church's: *Food Values of Portions Commonly Used*."[103]

<u>Anthropometry</u>

To eliminate inter-examiner error, one person took all the anthropometric measurements. Height measurements were determined to the nearest .5 cm and were taken while the subjects were standing erect without shoes. Subjects were weighed in light clothing with no shoes. Baseline and six month body weights were measured to the nearest .1 kg with a triple beam balance (Healthometer, Continental Scale Corporation, Bridgeview Illinois). At three months, a portable scale (Seca 760, Vogel and Halke GmbH and Co, Hamburg Germany) calibrated to the triple beam scale was used to take weight measurements in the subjects' homes. Ideal weight was calculated from the midpoint of the weight range for a given height and frame size according to the Metropolitan Height and Weight Tables (1983).[96] To determine frame size, elbow breadth measurements were taken and compared to the standards adapted from Frisancho and Flegel.[104] Percent ideal body weight, percent usual body weight and body mass index (BMI) were calculated.

Skinfold thickness provides an estimate of subcutaneous fat stores and hence total body fat. Lange calipers (Cambridge Scientific Industries Inc, Cambridge Maryland) were used to take skinfold measurements, on the left side of the body, at four sites: triceps (TSF), biceps, subscapular and suprailiac. Skinfold thickness was measured in triplicate according to published techniques.[97,105] The mean of the closest two values was recorded. Percent body fat was estimated from the sum of the four skinfolds calculated from published tables.[106]

Mid-upper-arm muscle circumference (MAMC) and mid-upper-arm muscle area (AMA) correlate with total muscle mass.[97] Mid-upper-arm circumference was measured with a flexible, nonstretch tape. TSF and mid-upper-arm circumference were used to calculate MAMC and

AMA using the following equations[97] (p202,203):

$$MAMC\ cm = (C) - (\pi \times TSF\ cm)$$

$$AMA\ cm^2 = \frac{(C - (\pi \times TSF\ cm))^2}{4\pi}$$

Where MAMC = mid-upper-arm muscle circumference, C = mid-upper-arm circumference, TSF = triceps skinfold, AMA = mid-upper-arm muscle area

Values obtained for MAMC and AMA were compared to age and sex specific percentile distributions developed from data collected during the Nutrition Canada Survey (1970-72).[105]

Bioelectrical Impedance Analysis (BIA)

BIA can be a reliable and valid approach for estimation of body composition[107] and is a useful body composition measure in patients with severe COPD.[108] BIA is based on electrical current conductance through body fluids.[107] In the body, lean tissues represent a low resistance, high conductance, electrical pathway because they contain large amounts of water and conducting electrolytes. In contrast, fat and bone which contain small amounts of fluid and conducting electrolytes are low conductance, high resistance, electrical pathways. Resistance is the voltage drop of an applied electrical current as it passes through a substance. Reactance is the opposition to the flow of an electrical current caused by capacitance or the storage of charge during the introduction of a current. When exposed to an electrical current, cell membranes in the body act as capacitors. Theoretically, therefore, reactance is a measure of the quantity of cell membrane capacitance. Body fat, total body water and extracellular water offer resistance to electrical current; only cell membranes have capacitance. Impedance is the apparent resistance in an alternating electrical current and is the sum of all resistance and reactance in the body.[109] Theoretically, FFM is linearly related to body height^2/body resistance (ht^2/res) or height^2/impedance.[110]

A tetrapolar impedance plethysmograph (RJL Systems, Detroit, MI) with a 800uA, 50kHz current[107] was used to determine body resistance(R). Measurements were made within 30 minutes of voiding and at least two hours after eating. The subjects lay supine on a bed clothed but with socks and shoes removed. Spot electrodes were placed on the right hand dorsal surface at the distal metacarpal and on the right foot at the distal metatarsal. Electrodes were also placed between the distal prominence of the radius and the ulna and between the medial and lateral malleoli at the ankle.[110] FFM was estimated using the following equation:[108]

$$\text{FFM kg} = 2.38 + 0.58\ (\text{ht m})^2/\text{Res} + 0.23\ (\text{wt kg})$$

where FFM=fat free mass, ht=height, Res=resistance, wt=weight

Total body fat and percent body fat were calculated by difference from total body weight and FFM.

Biochemical Parameters of Nutritional Status

At baseline and six months fasting blood samples were drawn by venipuncture from the subjects at the Department of Laboratory Medicine, University Hospital, UBC site. Blood samples were analyzed for complete blood count (Coulter Stacker), albumin (Kodak Ektachem 700, dye binding-BCG), total protein (Kodak Ektachem 700, Biuret), magnesium (Kodak Ektachem 700, colormetric), phosphorus (Ektachem 700, phosphomolybdate-reduction) and calcium (Ektachem 700, Arsenazo III dye).

RESTING ENERGY EXPENDITURE

Indirect calorimetry was used to determine REE. The Sensormedics MMC Horizon™ metabolic cart and Beckman Canopy System (Beckman Instruments Inc, Anaheim CA) located in the Pulmonary Function Exercise Lab at University Hospital, UBC site were used to determine CO_2 production ($\dot{V}_{CO2}$), oxygen consumption ($\dot{V}_{O2}$), REE and RQ. The MMC Horizon System is described in detail elsewhere.[111] Use of a plexiglass canopy avoided the discomfort associated with a mouthpiece and nose clips. The equation used to calculate REE was:

$$REE = 3.94 \times \dot{V}_{O2} + 1.11 \times \dot{V}_{CO2}$$ [112]

where REE = resting energy expenditure, $\dot{V}_{O2}$ = oxygen consumption, $\dot{V}_{CO2}$ = carbon dioxide production

REE was not corrected for nitrogen loss (protein disappearance). Twenty-four hour urine samples were not collected; therefore, urinary nitrogen losses could not be quantified. The error introduced by ignoring protein disappearance, however, has a negligible effect on the measurement of energy expenditure.[113] Gas, temperature and volume calibrations were checked prior to each test.

Indirect calorimetry was performed in the morning after a 12 hour overnight fast. Subjects took their usual morning medications. Before measurements began, subjects rested for 30 minutes. The test was performed in a darkened laboratory held at room temperature. Subjects lay on a bed in a supine position. They were relaxed with their eyes closed but not asleep. Taped soft music was played to enhance relaxation.

During the test, subjects were continuously monitored with an oximeter. Once the canopy was in place, the multiple speed fan was adjusted to maintain mixed expired CO_2 between 0.65% and 0.85%.[114] Measurements were taken at 30 second intervals. The test continued until at least 10 measurements were taken at steady state (variations in minute volume (V_E) and V_{O2} of < 10% and RQ of < 5%). The mean of the five lowest steady state measures was taken to represent the REE.[33] Gas analyzer calibration was confirmed at the beginning and end of each test.

REE obtained from indirect calorimetry was compared to energy expenditure predicted from the Harris Benedict equation (BEE).[40]

Males

BEE = 66 + 13.7 x (wt kg) + 5 x (ht cm) - 6.8 (age yr)

Females

BEE = 655 + 9.6 (wt kg) + 1.8 (ht cm) - 4.7 (age yr)

where wt = weight, ht = height

MEASUREMENTS OF PULMONARY FUNCTION

Arterial Blood Gases

Arterial blood gases are essential to the assessment of lung function impairment in individuals with moderate to severe COPD.[115,116] A respiratory technician drew all radial artery blood samples. These samples were taken while subjects were at rest and breathing room air. Once drawn, the samples were immediately placed on ice and then analyzed. A radiometer blood gas analyzer (ABL 300 Radiometer, Copenhagen Denmark) was used to determine arterial oxygen tension (Pa_{O2}), arterial carbon dioxide tension (Pa_{CO2}) and pH.

Spirometry

Spirometric tests were performed using a computerized spirometry system (Moose spirometer, Cybermedic, Louiseville Colorado) following the standard procedures of the American Thoracic Society.[117] Subjects performed the forced vital capacity (FVC) test, while seated and wearing nose clips. Subjects were verbally coached to maintain maximal effort throughout the test.[117] A minimum of three acceptable FVC tests were performed.[117] Best effort FVC, forced expiration in one second (FEV_1) and FEV_1/FVC% were automatically computed and compared to the normal values of Morris.[118]

Respiratory Muscle Performance

Maximal static inspiratory (MIP) and maximal static expiratory (MEP) pressures were used to assess respiratory muscle strength.[119]

Tests were performed while subjects were seated and wearing nose clips. Subjects performed the test while breathing through an occluded valve (Model 2700; Hans Rudolph Inc, Kansas City MO) containing a small air leak (internal diameter 0.6 mm) to minimize artifact resulting from facial muscle contraction. Pressures were measured using a differential pressure transducer (±350 cm H_2O) (Model DP45-16; Validyne CO., Northridge CA), recorded on a thermal chart recorder (Model 30000; Gould Inc., Cleveland OH) and read directly from the tracing. Prior to each test, the system was calibrated with a mercury manometer. A factor of 1.36 was used to convert mmHg to cm H_2O.[120]

MIPs were measured at residual volume (RV) and MEPs at total lung capacity (TLC). Patients performed one or two practice MIPs and MEPs. Then a minimum of three consistent MIPs and MEPs were performed. The highest value obtained for a pressure held for more than one second was recorded. Results were compared to reported normal values of McElvaney.[121]

Six-Minute Walk

A twelve-minute walking test is a useful and reproducible exercise tolerance measure which provides a practical guide to everyday disability in COPD.[122,123] A high correlation coefficient (r=.955) between a six-minute and twelve-minute walking test indicate that they are similar exercise tolerance measures.[124]

Due to the potential for a training effect, each subject completed a practice walk at baseline and if possible at six month retesting. The tests were performed along a 60 m level enclosed hospital corridor. The investigator walked with the patients for the first lap and for the last 30 seconds of the walk. Standard verbal encouragement was given at the completion of every second lap. Patients were also informed of the time remaining until the end of the test. Walking distances were recorded to the nearest 0.5 m.

WELL-BEING

The Perceived Well-Being Scale (PWB)[92] allows for psychological and physical well-being assessment. The composite score of these two subscales provides an index of an individual's overall well-being. Psychological well-being in this scale is defined as "the presence of positive emotions such as happiness, contentment, joy, and peace of mind and the absence of negative emotions such as fear, anxiety, and depression."[92] p.24 The definition of physical well-being is "self-rated physical health and vitality coupled with perceived absence of physical discomforts."[92] p.24 General well-being is the composite of physical and psychological well-being. The original 14 item PWB scale was revised to a 16 item scale (PWB-R) (GT Reker personal communication). The revised scale allows assessment of eight physical and eight psychological well-being items (appendix 7). To allow comparison of scores with PWB scores previously published for COPD patients,[91] one statement (#17) "I have a good appetite for food" was added from the original PWB to the PWB-R. The item "I think I have a heart condition" was changed to "I think I have a lung condition" to more appropriately reflect the physical condition of this patient group.[91] Respondents were asked to rate each item on a seven point Likert-type scale with choices from strongly agree to strongly disagree. The possible score ranges for both the psychological and physical well-being scales are 8-56. For general well-being, the possible range is 16-112 (GT Reker personal communication). For 121 elderly individuals (60-90 yr), the reported alpha coefficients of .74 for psychological well-being, .85 for physical well-being and .86 for general well-being (GT Reker personal communication) indicate the reliability of this assessment tool.

NUTRITIONAL INTERVENTION

Control Group

The control group received no individual counselling and were not offered the oral nutritional supplement, Pulmocare™. At baseline, however, this group received written guidelines to increase energy and protein intake and a list of easy to prepare meals and snacks (Appendix 8). This group was encouraged to consume their usual diet.

Treatment Group

A RDN provided the treatment group with individualized nutrition counselling in their homes within one week of baseline testing. Nutrition counselling was based on the information obtained from the diet history, baseline food frequency, four-day food record and REE measurement. The goal energy level of the diet was at least 1.5X REE. To obtain this intake level, written guidelines for increasing energy and protein intake and a list of easy to prepare meals (Appendix 8) were discussed with the subjects. The RDN encouraged maximal intake of nutrient dense foods. Additionally, a general meal plan including three meals and one to three snacks per day was created for each subject (appendix 9). Also, a shopping list of recommended foods was generated (appendix 9). As appropriate, recipes for milkshakes and other nutrient dense foods were provided. Two weeks after the initial counselling session, the RDN made a home follow-up visit to review and revise the initial diet plan.

At baseline, each subject in the treatment group received 24 cans (235 ml) of Pulmocare™. The RDN provided suggestions for flavouring this product. The patients were encouraged to take the supplement or have a snack between meals. The use of Pulmocare™ was optional. The patients' Pulmocare™ supply was repleted as necessary throughout the study. Use of the supplement was monitored in two ways. First, the subjects were asked record the number of cans of supplement consumed on a daily record form (appendix 10). Second, they were asked to return empty cans for recycling.

Both the treatment and control groups received follow-up phone calls at one, two, four, and five months. At three months, they received a home visit. Both groups were asked to record weekly weights as well as medication changes and illness over the study period.

EVALUATION OF NUTRITION EDUCATION

A nutrition education evaluation tool (appendix 11) developed by Hauchecorne[95] was used to assess the patients' perceptions of the nutrition education provided. This questionnaire was based on an outcome measurement approach. The Value-Added Ambulatory Encounter (VAE) conceptual framework of Bopp[125] was adapted to produce the Value of Nutrition Education (VNE). Questions were developed to address components of the revised (VAE). Included in the evaluation are the patients' perceptions of the credibility of the dietitian, the usefulness of information received, the impact of nutrition counselling on physical and emotional well-being and the patients' abilities to make dietary changes. This instrument is considered to be a valid measure of effective practice because it reflected an expert panel's collective experience as health professionals. The panel considered it to be an instrument that they would find useful in evaluation of nutrition education in their own work settings. Comparable initial and one week returns indicate the reliability of this questionnaire. Spearman correlation coefficients calculated for these two times were positively correlated. Only questions which had a minimum significant R of .40 were included in the questionnaire.[95]

This instrument was issued to the treatment group at the end of the study. At the completion of the study, the control group also received individualized nutrition counselling. After at least one follow-up contact with the RDN, the patients in the control group were asked to complete the nutrition education evaluation questionnaire.

ANALYSIS OF THE DATA

The Systat PC statistical program (Systat Inc, Evanston Il) was used to perform all statistical tests. The acceptable level of significance was set at $p <.05$. Descriptive statistics were used for baseline characteristics and factors contributing to weight loss and reduced nutrient intake. Differences between the treatment and control group in baseline characteristics were assessed using the independent t test. A 2X2 (group x time) RM ANOVA (with repetition on the time factor) was used to analyze pre and post intervention mean values for pulmonary function tests (FEV_1, FVC, FEV_1/FVC, MIPs, MEPs and six-minute walking distance). A 2x3 (group x time) RM ANOVA (with repetition on the time factor) was used for analysis of anthropometric measures (wt, FFM). 2x4 (group x time) RM ANOVA (with repetition on the time factor) was used to analyze nutrient intake (energy and protein). Post Hoc differences between the means over time were analyzed with the Tukey test. The Pearson coefficient was calculated for FFM calculated from BIA and FFM calculated from skinfolds. Nonparametric statistics, the Wilcoxon signed rank test for two dependent groups, and the Mann-Whitney U test for two independent groups, were used for the PWB-R score analysis. Frequency distributions were tabulated for the evaluation of nutrition education questionnaire.

CHAPTER 4

RESULTS

STUDY PARTICIPANTS

Eleven of the 14 patients enrolled in the study completed the six month trial. Two patients, one in the treatment group and one in the control group died before the study was completed. Another subject in the control group dropped out of the study due to illness. One of the patients in the treatment group completed the post intervention tests at four months because she was leaving the country for a prolonged vacation. Over the study period, five (three in the treatment group and two in the control group) of the 11 participants were hospitalized for exacerbations of their disease. Three of these five were hospitalized two or three times. All of the subjects were taking bronchodilators: B_2 agonists, theophylline containing compounds and Atrovent. Two subjects were on low dose prednisone (both in the control group), and five subjects (three in the treatment group and two in the control group) required short periods of higher doses of prednisone during the study period. Two subjects (one in the treatment and one in the control group) who required thyroid replacement therapy had thyroid hormone levels within the normal range. Three subjects (two in the treatment group and one in the control group) were on continuous oxygen therapy and one (control group) required oxygen at night. Eight patients lived with a spouse, three were single (two in the treatment and one in the control group). Eight patients had quit smoking. Three participants, all in the control group continued to smoke 0-6 cigarettes per day.

BASELINE DATA

Group means and standard error for baseline subject characteristics, nutritional status parameters, pulmonary function and respiratory muscle function measures are shown on Table 1. Both groups had a similar number of males and females. There was no difference in age, height, weight, percent ideal body weight (%IBW) or percent usual body weight (%UBW). The average %IBW for all 11 subjects was 78.2%±2.1%. This indicates a moderate degree of malnutrition.[126] All were greater than 10% below their IBW (range 69%-88%) in accordance with inclusion criteria. There were no differences between groups in triceps skinfolds (TSF), sum of skinfolds (SSF) or mid-upper-arm muscle circumference (MAMC) or mid-upper-arm muscle area (AMA). All but two subjects were below the 25^{th} percentile for both TSF and MAMC. The majority of subjects were below the fifth percentile for both.

Biochemical parameters including albumin, total protein, magnesium, phosphorus, calcium and total lymphocytes for both groups were within the normal range. There was no difference between groups for any of these measures.

In agreement with entrance criteria, the average FEV_1/FVC percent predicted was 43.8%±3.2% (range 31%-62%). This indicates severe COPD. Although there were no differences between groups in FVC, FEV_1 or FEV_1/FVC, the treatment group had significantly lower FEV_1% predicted (24.1%±2.4%), p=.04 and FEV_1/FVC% predicted

(37.7%±2.6%), p=.03 than the control group (FEV_1% predicted 40.2%±6.8%, FEV_1/FVC% predicted 51%±4.8%). Baseline maximal inspiratory and expiratory pressures did not differ between groups.

ENERGY EXPENDITURE AND NUTRIENT INTAKE

The mean measured resting energy expenditure (REE) for the 10 subjects who were able to complete this test was 1243 ± 61 kcal/day. It was not different than that predicted by the Harris Benedict equation: 1182±40 kcal/day. On average, REE was 104%±2% of predicted (range 93-115%). Mean baseline energy intake for the all subjects was 1941±103 kcal/day (38kcal/kg). The mean energy intake was 1.6x (range 1.4 - 2.2x) the mean REE for the 10 subjects whose REE was measured.

The mean energy and protein intakes for the two groups are shown in Table 2. There was no significant difference at baseline between the two groups for energy or protein intake.

There was no significant group effect, for energy or protein intake. Averaged over group, there was a significant time effect for energy intake F = 3.00 p=0.048; however, when analyzed with the Tukey test there was no significant difference between mean energy intake at any of the time points. Averaged over group, protein intake did not differ over the study period. There was no interaction observed for either energy or protein intakes.

Table 1:

Comparison of baseline characteristics, nutritional status parameters, pulmonary function and respiratory muscle performance in malnourished patients with COPD

Characteristic	Treatment	Control
Patients (n)	6	5
Sex	2M:4F	2M:3F
Age yr	59.7±4.4*	63.8±5.6
Height cm	168.2±4.9	168.1±4.5
Weight kg	50.7±4.1	50.4±4.0
% IBW	77.4±3.6	79.1±2.1
% UBW	87.8±2.7	86.6±4.1
BMI	17.8±0.8	17.7±0.6
TSF mm	8.5±1.8	6.8±0.9
SSF mm	22.4±3.4	18.9±2.1
MAMC cm	19.7±0.73	19.8±0.88
AMA cm^2	31.2±2.3	31.5±2.7
Albumin g/L	41.3±1.0	40.6±0.93
Total Lymph $x10^9/L$	1.8±0.1	1.7±0.2
FVC L	2.3±0.3	3.0±0.5
FEV_1 L	0.69±0.1	1.16±0.2
FEV_1% predicted	24.1±2.4	40.2±6.9**
FEV_1/FVC	0.30±0.02	0.40±0.04
FEV_1/FVC% predicted	37.8±2.6	51.0±4.8**
MIP cm H_2O	43.3±5.9	58.7±9.7
MEP cm H_2O	76.4±13.3	83.2±12.0

* mean ± std error
** p = <.05 treatment vs control group

Table 2:

Mean energy and protein intake of malnourished patients with COPD over a six month period*

	Treatment				Control			
Month	0	.5	3	6	0	.5	3	6
Energy** (kcal)	1937 ±190§	2433 ±271	2103 ±105	2129 ±235	1946 ±67	2092 ±138	2173 ±37	1897 ±94
Protein (gm)	75±9	90±9	80±7	93±14	76±6	73±6	78±3	77±4

* based on four day food records
** signficant time effect F=3.00 p=0.048
§ mean ± std error

Although not significant, by ANOVA and post hoc testing for all subjects combined, the greatest difference in energy intake from baseline occurred at .5 months for the treatment group. Over the first two weeks of the study period, all subjects in the treatment group but not all subjects in the control group had an increase in energy and protein intake. At this time point, the mean difference from baseline for energy intake for the treatment group was 497±108 kcal/day (range +202 - +867 kcal/day). For protein, the mean difference was 15±5.5 gm protein/day (range +3 - +40 gm pro/day). (By paired t test, these increases in energy (t=4.6, p=.006) and protein (t=2.8 p=.04) intake for the treatment group were significant) For the control group the mean differences were 145±123 kcal/day (range -291 - +446/day) and -3±7.0 gm protein/day (range -30 - +7.0 gm pro/day). The treatment group showed a trend for energy intake to decrease at three months and then stabilize at six months. The control group showed a trend toward increased energy intake over the first three months and a decrease in intake at six months. There appeared to be a trend for the treatment group to have a higher protein intake than the control group throughout the study period (F=.64 p=.44). Protein intake at baseline for both groups was 1.5 gm/kg. In the treatment group, it increased to 1.8gm/kg at .5 months and 6 months.

ANTHROPOMETRIC MEASURES

The mean values for anthropometric measures at baseline, three and six months are shown in Table 3. Fat free mass calculated from BIA (FFMBIA) correlated positively with mid-upper-arm area (AMA) (r=.7, p=.017) and fat free mass calculated from sum of skinfolds (FFMSF) (r=.94, p <.001). The equation to calculate FFMBIA was developed for patients with COPD,[108] while the tables to predict body fat from the sum of skinfolds[106] were derived from a healthy population. Additionally, these tables had limited data for elderly populations. Therefore, FFMBIA was chosen to represent further analysis involving FFM.

There was no significant difference in body weight, triceps skinfold (TSF), sum of skinfolds (SSF), mid-arm-muscle circumference (MAMC), or AMA between the groups or over time. Also, there was no significant interaction observed for any of these variables. Averaged over group, there was a significant time effect for FFMBIA (F = 5.98 p = .01). Follow up multiple comparison analysis with the Tukey test, however, showed no significant difference in FFMBIA between any two time points. Values for FFMBIA resulted in no group by time interaction.

BIOCHEMICAL PARAMETERS

All mean biochemical parameters were within the normal range. There was no significant difference in any of the biochemical parameters between groups or over time.

Table 3:

Comparison of anthropometric measures for the treatment and control groups over a six month period

	Treatment			Control		
Month	0	3	6	0	3	6
Wt kg	50.7 ± 4.1 *	52.2 ± 4.5	51.0 ± 4.5	50.4 ± 4.0	51.3 ± 4.5	50.6 ± 4.1
TSF mm	8.5 ± 1.8	8.8 ± 1.8	9.5 ± 2.0	6.8 ± 0.9	7.6 ± 0.9	7.6 ± 1.3
SSF mm	22.4 ± 3.4	24.7 ± 4.7	25.0 ± 4.5	19.0 ± 2.1	20.3 ± 2.7	20.4 ± 2.8
MAMC cm	19.3 ± 0.7	19.8 ± 0.9	19.6 ± 0.8	19.8 ± 0.9	19.9 ± .0.9	19.6 ± 1.0
AMA cm^2	31.2 ± 2.3	31.6 ± 2.9	30.9 ± 2.7	31.5 ± 2.7	31.8 ± 2.8	30.7 ± 3.0
FFMBIA** kg	40.0 ± 3.4	41.6 ± 3.8	39.8 ± 3.4	39.4 ± 3.5	40.5 ± 3.6	39.4 ± 3.1

* mean ± std error
** Significant time effect F=5.98 p=.01

PULMONARY AND RESPIRATORY MUSCLE FUNCTION

The mean pre and post intervention values for pulmonary and respiratory muscle function tests are shown in Table 4. There was no significant group effect, time effect or interaction for FEV_1, FVC or FEV_1/FVC. Averaged over time, the control group had significantly higher FEV_1 percent predicted (F=5.08 p=0.05), and a higher FEV_1/FVC percent predicted, (F=6.2 p=0.03) than the treatment group. There was no significant change in FEV_1 percent predicted or FEV_1/FVC percent predicted over time and no interaction. Body weight did not correlate significantly with FEV_1. Change in body weight did not correlate with changes in FEV_1, FEV_1/FVC, MIP, or MEP. Despite no significant group or time effect for MIP, there was a significant group x time interaction for MIP ($F=8.04$, $p=.02$). This interaction is shown in Figure 2. Over the study period, there was a trend for MIPs to increase (+21%) for the treatment group and decrease (-24%) for the control group. MEPs were not significantly different between groups. There was no significant change over time and no interaction. The treatment group showed a trend towards improved MEP. Mean MEP at retest was 17% higher than baseline. MEP for the control group decreased one percent at retest.

There was no significant difference between groups or over time for distance walked in six minutes and no interaction was observed. There was a trend for the treatment group to have a greater increase in distance walked than the control group at retest. The difference from baseline for the treatment group was an increase of 41 m (11%) while the control group had an increase of 8 m (2%).

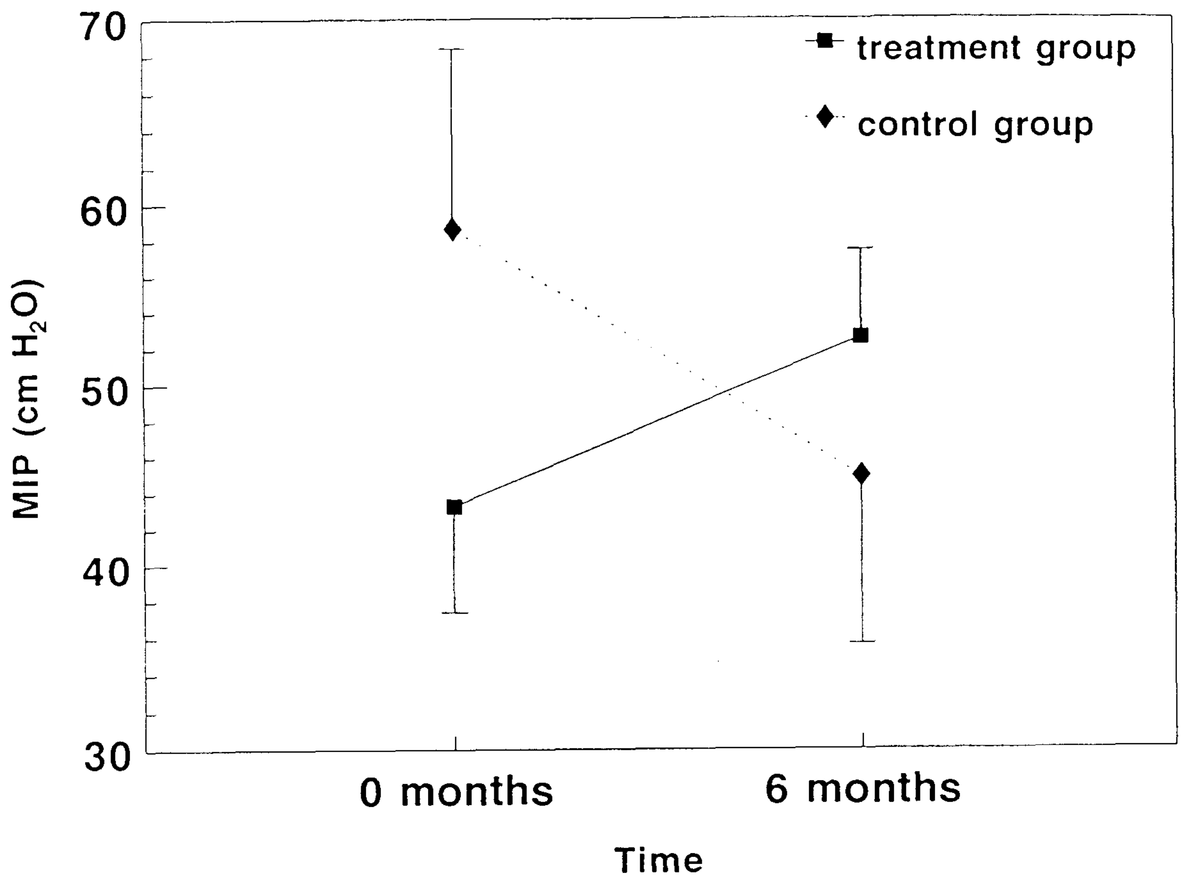
Table 4:

Comparison of pre and post-intervention pulmonary function, respiratory muscle function tests, and six-minute walk test for the treatment and control groups

	Treatment		Control	
Month	0	6	0	6
FVC L	2.3±0.26*	2.1±0.25	3.0±0.52	2.8±0.38
FEV_1 L	0.69±0.10	0.62±0.07	1.20±0.22	1.07±0.22
FEV_1%** predicted	24.2±2.4	21.1±1.9	40.2±6.9	37.6±8.1
FEV_1/FVC L	0.30±.02	0.30±.02	0.40±0.04	0.37±0.04
FEV_1/FVC%§ predicted	37.8±2.6	38.5±1.6	51.0±4.8	46.8±4.8
MIP cm H_2O§§	43.3±5.9	52.6±4.9+	58.7±9.7	44.9±9.3
MEP cm H_2O	76.4±3.3	89.7±18.2+	83.2±11.9	82.1±10.3
6 minute walk m	388.0±23	429.0±44++	446.0±59	454.0±60++

* mean ± std error
** Significant group effect F=5.08 p=.05
§ Significant group effect F=6.2 p=.03
§§ Significant group x time interaction F=8.04 p=.02
\+ n = 5
++ n = 4

Figure 2:
GROUP X TIME INTERACTION FOR MAXIMAL INSPIRATORY MOUTH PRESSURES (MIP)



BLOOD GASES

Except for one chronic O_2 user (treatment group), all arterial blood gases were taken while the patients were breathing room air. As indicated in Table 5, values for blood gas determinations showed no significant difference between groups, no significant change over time and no group by time interaction. P_{O2} levels indicate mild hypoxia for both groups. P_{CO2} levels indicate mild hypercapnia for the treatment group and high normal levels of P_{CO2} for the control group. These results are consistent with the spirometric test results.

Table 5:

Comparison of pre and post-intervention blood gases for the treatment and control groups

	Treatment		Control	
Month	0	6	0	6
P_{O2}	69.8±6.4*	73.1±6.3	73.8±3.7	69.0±3.6
P_{CO2}	46.2±2.6	45.9±2.9	41.0±2.3	42.0±1.9
HCO_3	28.3±1.1	28.4±1.1	25.4±1.0	26.3±1.0
pH	7.4±0.01	7.4±0.01	7.4±0.01	7.4±.01

*mean ± std error

PERCEIVED WELL-BEING

Perceived Well-Being-Revised (PWB-R)

Table 6 displays the PWB-R total well-being scores (T) and psychological (Psy) and physical (Phy) well-being subscores at baseline and at the end of the study period. At baseline, there was no significant difference between groups for the total perceived well-being score, the psychological or physical subscores. At six months, there was no significant difference between groups in total well-being score or physical well-being subscore. However, the control group had a significantly lower psychological well-being subscore than the treatment group (Mann Whitney U = 29.0 p=.01).

Over time, there was no significant change in the total well-being score or psychological well-being subscore for the treatment group. The treatment group's lower physical well-being subscore at six months almost reached significance, p=.07. For the control group, the total well-being score was significantly lower at six months than at baseline (p=.04). There was no significant difference between baseline and six month psychological or physical well-being subscores for the control group.

When both groups were analyzed as a whole (n=11), at six months the total well-being score (p=.02) and the physical well-being subscore (p=.02) were both significantly less than the baseline scores. The psychological subscores did not change significantly over time.

Perceived Well-Being (PWB)

The total perceived well-being score for the group as a whole (n=11) at baseline was 65±3, the psychological and physical well-being subscores were 37±1 and 29±3 respectively. The PWB test scores showed similar trends as the PWB-R scores over time.

Table 6:

Mean baseline and end of study total scores (T) and psychological (Psy) and physical (Phy) subscores for the Perceived Well-Being-Revised (PWB-R)) quality of life questionnaire for malnourished patients with COPD.

	Treatment n=6		Control n=5		Total n=11	
Month	0	6	0	6	0	6
PWB-R						
Total Score	74±5*	69±3	76±7	66±5**	75±4	68±3§
Psy Subscore	49±2	49±1	47±5	42±2+	48±2	46±2
Phy Subscore	25±3	20±3	29±4	24±5	27±3	22±3§

* mean ± std error
** p=.04 control group baseline vs 6 months
§ p=.02 total group baseline vs 6 months
\+ p=.01 treatment vs control at 6 months

FACTORS CONTRIBUTING TO WEIGHT LOSS AND DECREASED NUTRIENT INTAKE

Table 7 lists factors contributing to decreased nutrient intake in COPD patients. The percentage of the 20 COPD patients interviewed who stated that these factors were a concern is indicated. Hospitalization due to exacerbation of COPD was frequently mentioned as a time of weight loss. Seventy-five percent of these malnourished patients indicated that they lost a significant amount of weight during hospitalizations prior to entering the study. They were unable to regain the lost weight after they returned home. The five patients who were hospitalized during the study all lost weight while in hospital.

Dyspnea and fatigue were the leading factors contributing to decreased intake. Sixty-five percent of the patients indicated that both of these factors were impediments to eating. Dyspnea was most frequently associated with eating too quickly, or eating large meals. Shortness of breath at meals increased in times of illness. For those on oxygen, it increased when oxygen was not used while eating. Fatigue was reported as a problem both in the morning and late in the day. Poor appetite and early satiety were problems for 60% of those interviewed. Many participants commented that eating was not pleasurable; it was an effort or they had to force themselves to eat. Fifty percent indicated that gastrointestinal discomfort in the form of bloating or burping was a problem. Only 30% of these individuals considered it to be a significant problem. The others indicated it was mild. Occasional nausea and vomiting was a concern for 30% percent of patients. These symptoms were often associated with taking antibiotics to treat or prevent further exacerbation of their disease. In one case, these symptoms were a result of theophylline toxicity. Thirty percent of patients experienced chewing difficulties. Denture discomfort and lack of energy to chew contributed to problems with chewing. Bowel problems (primarily constipation) were a concern for only a quarter of those interviewed. Four individuals (20%) indicated that intake was affected by depressed mood.

Table 7:

Factors contributing to decreased nutrient intake in malnourished patients with COPD (n=20) and the percentage of patients experiencing these problems.

Factor	Percent
Dyspnea	65%
Fatigue	65%
Poor appetite	60%
Early satiety	60%
Bloating/burping	50%
Nausea/vomiting	35%
Chewing/swallowing	30%
Bowel problems	25%
Mood/depression	20%

ACCEPTANCE OF NUTRITIONAL SUPPLEMENT

Pulmocare™ was made available to all patients in the treatment group for the duration of the study. It was also offered on a trial basis to subjects in the control group at the completion of the six month study period. Of the six patients in the treatment group, three took the supplement consistently over the study period. Generally, these three were able to consume half to one 235 ml can per day. Two others were able to take small amounts intermittently over the study period. Most of this was consumed in the first few weeks. One subject chose not to consume the supplement. Only one person reported no problems consuming this supplement. The other subjects expressed problems with feeling full or nauseated after consuming a whole can of undiluted supplement. Unpleasant taste or aftertaste of the supplement were reported as reasons for non-consumption. When taken in small amounts or mixed with other fluids such as milk or juice Pulmocare™ was better tolerated. Similar problems with tolerance were expressed from individuals in the control group who tried the product after completion of the study period.

PATIENTS' EVALUATION OF NUTRITION EDUCATION

Both the treatment and the control group completed the evaluation of nutrition education questionnaire. The treatment group completed it at the end of the study period. The control group also received individualized nutrition counselling once they had completed the study. They completed the evaluation of nutrition education after receiving counselling and at least one follow-up visit. Nine of the 11 participants returned appropriately completed evaluation questionnaires. One participant failed to return the questionnaire and another individual did not complete the form appropriately.

All patients felt that they were able to talk to the dietitian when they wanted to and that follow up contact was useful. Reasons for speaking with a dietitian included seeking advice about what to eat to help gain weight, obtaining reassurance that they were eating properly, and receiving advice on general nutrition.

Table 8 summarizes the patients evaluation of the nutrition education provided. All patients agreed that the dietitian provided useful information and that the advice that they received was suited to their special needs. All agreed that the dietitian knew what she was talking about and that after speaking with the dietitian they knew what to eat for their special needs. All either disagreed or were neutral that they could not change their diet. Fifty-five percent agreed that they felt better emotionally after speaking with the dietitian. The same percentage indicated that they felt better physically. All agreed that the dietitian provided support and encouragement. One hundred percent strongly agreed that the dietitian cared about them and that anyone with their condition should talk with a dietitian. All disagreed with the statement that there was no benefit in talking with a dietitian.

Table 8:

Frequency distribution for patients' evaluation of nutrition education (n=9). The scale used for evaluation was 1=strongly disagree, 2=disagree, 3=neither agree nor disagree, 4=agree, 5= strongly agree.

Statement	Disagree % <3	Neutral % =3	Agree % >3
1. The dietitian provided useful information	0	0	100
2. The dietitian knew what she/he was talking about	0	0	100
3. The advice I received from the dietitian was suited to my special needs	0	0	100
4. After talking with the dietitian, I knew what to eat for my special needs	0	0	100
5. After talking with the dietitian, I changed my diet	11.1	22.2	66.6
6. By talking with the dietitian, I learned I did not need to change my diet as my intake was already suited to my needs (n=8)*	37.5	25.0	37.5
7. After talking to the dietitian, I could not change my diet (n=8)*	62.5	37.5	0
8. After changing my diet, my weight improved	22.2	33.3	44.5
9. After talking with the dietitian, I felt better emotionally	11.1	33.3	55.5
10. After talking with the dietitian, I felt better physically	0	44.4	55.5
11. After talking with the dietitian, I felt in control of my condition	11.1	22.2	66.6
12. The dietitian provided support and encouragement	0	0	100
13. The dietitian cared about me	0	0	100
14. Anyone with my condition should talk with a dietitian	0	0	100
15. There was no benefit in talking with a dietitian	100	0	0

* question not completed by one participant

CHAPTER FIVE

DISCUSSION

MAJOR FINDINGS

Nutritional interventions directed toward improving nutritional status in malnourished COPD patients appear warranted because malnutrition in these patients is associated with impaired respiratory muscle function and depressed immunocompetence. These impairments are associated with increased morbidity and mortality. This study was undertaken to identify factors contributing to weight loss in COPD patients. Additionally, the effect of individualized nutrition counselling in the home environment on nutrient intake, body weight, body composition, pulmonary function and perceived well-being of COPD patients was assessed. The main findings of this study are as follows. Individualized nutrition counselling along with optional use of a nutritional supplement (Pulmocare™) was not successful in sustaining adequate nutrient intake to promote significant weight gain and improvement in nutritional status in malnourished, ambulatory COPD patients. There were no significant differences in body weight, body composition, nutrient intake, MIP, MEP or distance walked in six minutes between the treatment and the control groups or over time. The treatment group had significantly lower FEV_1% predicted and FEV_1/FVC% predicted than the control group. There was a significant decrease in general well-being and physical well-being in these patients over the study period. Hospitalization for exacerbation of COPD was indicated as a time of significant weight loss for a majority of these patients. Dyspnea, fatigue, poor appetite, early satiety, gastrointestinal discomfort and intolerance to the supplement appeared to be impediments to sustaining an adequate nutrient intake to promote nutrient repletion. Although these patients did not gain a significant amount of weight, they perceived that the nutrition education provided was beneficial.

MEASURED RESTING ENERGY EXPENDITURE AND NUTRIENT INTAKE

On average, these patients were not hypermetabolic. Mean REE of 104 ± 2% of predicted was not significantly greater than that predicted by the Harris Benedict equation.[40] This result is similar to the results of a previous study completed in the same laboratory which found REE to be 94% of predicted.[33] Although the REE was normal, it is possible that the metabolic rate of these patients is somewhat elevated. Recent reevaluations of the Harris Benedict equation to predict REE have found that this equation overpredicts REE in healthy men and women.[127,128]

Three of the patients in this study did have REEs between 110% and 115% of the predicted value. Several studies have reported hypermetabolism in malnourished patients with COPD; metabolic rates in these studies have ranged between 115% and 125% of the predicted values.[34-37,41] Differences in the measurement of REE is likely a significant factor in the variation of these results as severity of COPD and degree of malnutrition were similar. Insufficient time to allow steady state to occur,[36] lack of control in timing of measurements or incomplete fasting[35] would contribute to elevated REE measurements.[33]

Elevated REE in COPD patients has been attributed to increased work of breathing. Studies have demonstrated increased respiratory muscle O_2 consumption in COPD patients.[37,47] Increased resistive load and decreased respiratory muscle efficiency, along with high total expired ventilation contribute to the increased cost of breathing.[7,37]

Baseline energy intake was 1.6 x REE (range 1.4 - 2.2x) and baseline protein intake was 1.5 gm/kg. These results indicate that these patients were capable of consuming a diet that would normally be considered sufficient to meet energy and protein needs to at least maintain nutritional status.[129] The fact that the control group's weight was stable over the six month study period confirms that these patients consumed adequate energy for weight maintenance. These results are similar to those of other studies which reported energy intakes of 1.35 x REE[33], 1.4 x REE[8] and 1.5-1.6 x REE.[15] Based on seven-day food records, Otte reported a high habitual energy intake for malnourished patients with emphysema (2.04 x basal energy expenditure).[85] The type of food record used along with the duration and accuracy of the food record are factors that contribute to varying results.

Self reported intakes are only estimates of true habitual intakes. There are several sources of error for estimated food records. These include omission of foods due to memory lapse, inability of the respondent to adequately estimate portion sizes and difficulties in converting portion sizes recorded in household measures to gram weights for computer analysis.[97] In the present study, completed food records were reviewed with the participants to minimize these sources of error. Also, participants may alter their intake during the recording period to simplify the recording process or to please the investigator.[130]

The food records obtained for this study represent intake during periods of relative disease stability. Measurement of energy intake during an interval of disease stability does not necessarily reflect intake during intercurrent illness. Exacerbation of disease and increased dyspnea, can lead to a significant reduction in nutrient intake.

There were no significant differences between groups for protein and energy intakes. There was a significant time effect for energy but not for protein. Due to the small sample size and therefore, limited power, a significant difference in energy intake was not found between any two time points.

In view of the limitations of the small sample size to find significance, the trends in intake should be noted. For the treatment group the highest recorded intake occurred at .5 months. At three and six months the intake decreased toward baseline levels. All subjects in the treatment group had increased energy and protein intakes at .5 months while those in the control group did not. The mean difference in energy intake at .5 months for the treatment group was approximately 500 kcal. It was 145 kcal for the control group. For protein intake, the mean difference, between baseline and .5 months, was 15 gm for the treatment group and -3 gm for the control group. Protein intake for the treatment group tended to be higher at all time points. If the sample size was larger, these differences may have become significant. These trends suggest that individualized nutrition counselling had a positive effect on protein intake. Additionally, it appeared to encourage increased energy intake for at least the first two weeks after an initial nutrition counselling session.

ANTHROPOMETRIC MEASURES

The finding of no significant differences over time or between groups in anthropometric measures is consistent with no significant differences over time or between groups in energy or protein intake. These patients encountered many obstacles to sustaining an increased nutrient intake over the study period.

IMPEDIMENTS TO INCREASING ORAL INTAKE

In this study, several factors appeared to contribute to difficulties in sustaining an adequate nutrient intake to promote nutrient repletion and weight gain. Exacerbation of COPD and hospitalization resulted in weight loss for all five patients who were admitted to hospital over the study period. Typical patterns of weight change observed for the COPD patients in this study are shown in Appendix 12. Additionally, at the time of baseline interviews, the majority of patients indicated that they had lost significant amounts of weight during previous hospitalizations. Dyspnea, poor appetite and early satiety also contributed to difficulties in increasing intake. Fatigue made shopping and meal preparation difficult and contributed to decreased intake at a meal. COPD patients with the greatest degree of dyspnea have been reported to have the lowest energy intakes.[27] Nausea resulting from antibiotics taken to treat or prevent exacerbations of COPD was also a problem for some participants. One subject in the treatment group lost two kg over 10 days due to nausea and vomiting resulting from theophylline toxicity. These impediments to eating are consistent with findings of other investigators.[8]

Although Pulmocare™ was provided to the treatment group as a convenient way to increase nutrient intake, only half of the patients in the treatment group were able to consistently take 120 to 235 ml of this supplement daily (180-350 kcal/day). Dislike of the taste of Pulmocare™ and intolerance to this supplement were reasons why the supplement was not consumed to a greater extent. The high fat content of Pulmocare™ (55%) may have contributed to intolerance. It was better tolerated if taken in small amounts or if it was diluted with other fluids such as milk or juice. Some food records indicated that the supplement had replaced energy provided in the patients' usual diet.

These findings are similar to those of other studies which were not able to achieve adequate intake to promote weight gain with nutrition counselling and/or provision of supplement. Impediments to sustaining adequate intake in these studies included a decline of supplement usage over time and reduction of usual intake during the period of supplementation.[15,16,93] In one uncontrolled study, 10 subjects were encouraged to consume an additional 750-1000 kcal/day in the form of high calorie liquid supplements (Sustacal®, Ensure®). No home dietary records were kept but compliance with supplement usage was surveyed at follow-up which ranged between 12-22 months. All patients reported that they took the supplements in the prescribed amount early in the study period but that usage declined over time due to taste fatigue.[93] Knowles and associates found considerable variation in the ability of COPD outpatients to increase nutrient intake when they were provided with an oral supplement (Susacal®) for eight weeks. Supplement calories were often substituted for normal diet calories.[16] In another study, patients in the refed group were not able to tolerate more than 240 ml/day of Isocal HCN® in addition to their normal diet. Bloating and fullness limited the amount of supplement that could be consumed. Additionally, there was a tendency for patients to decrease their usual food intake while receiving supplement.[15]

In contrast, six malnourished emphysema patients admitted to a clinical research unit gained 3.0 kg and increased TSF and MAMC when given an ad libitum diet. This diet was supplemented if energy intake was less than 150% of the patients' basal metabolic rate. The period of nutrition intervention was two weeks.[14] Efthimiou and associates[12] were also successful in promoting adequate energy and protein intake to promote weight gain. Over a three month period, ambulatory malnourished COPD patients took a nutritional supplement (Build-Up®, Carnation) in addition to their regular diet. All patients tolerated this supplement without complication. After the period of supplementation, the patients reduced their energy and protein intakes to levels very similar to baseline levels and all patients lost weight. Yoshikawa and associates were also successful in achieving improvement in nutritional status, increased body weight and skinfolds, with provision of a branch chain enriched enteral formula in addition to the usual diet of their ambulatory COPD patients.[86]

Diet plus injections of recombinant methionyl human growth hormone (GH)[88] and enteral tube feeding[13] have also been successful in promoting weight gain in malnourished COPD patients. A pilot study in which seven malnourished COPD patients received a nutritionally adequate diet plus daily subcutaneous injections of GH documented weight gain and improved nitrogen balance over a three week period.[88] In a short-term (16 days) inpatient tube-feeding study, patients gained an average of 2.4 kg after receiving enteral formula that provided an additional 1000 calories/day to their usual intake.[13] It appears that refeeding studies in which patients are not responsible for their own meal preparation or in which the supplement provided is well tolerated, weight gain and improvement in nutritional status can be achieved over the short-term.

PULMONARY FUNCTION AND RESPIRATORY MUSCLE STRENGTH

This study found no significant pre or post-intervention differences in pulmonary function, respiratory muscle strength tests or distance walked in six minutes. The treatment group had significantly lower FEV_1% predicted and FEV/FVC% predicted than the control group. These results suggest that clinically, the treatment group was more severely obstructed than the control group. The trend for greater hypercapnia in the treatment group is consistent with their lower FEV_1. The more severe airflow obstruction in the treatment group may have made it more difficult to demonstrate the effectiveness of nutrition intervention in this group.

The mean MIP and MEP values for the all subjects were greater than one standard deviation below the mean reported for normal elderly.[122] This indicates respiratory muscle weakness in this group of COPD patients.

There was a significant group by time interaction for MIP. The treatment group had a mean increase in MIP of 24% at retest while the control group had a mean decrease of 24%. Although not significant, the trends for MEP and distance walked in six minutes were similar to that of MIP. In the treatment group, MEP increased by 17% at retest while there was no increase for the control group. The distance walked in six minutes at retest increased by 10% for the treatment group and 2% for the control group. A larger sample size would more conclusively determine whether these differences would be significant or not.

MIP is determined in COPD patients both by mechanical disadvantage to respiratory muscles consequent to hyperinflation of the lung and generalized muscle weakness. In this study, lung volumes were not measured; therefore, it was not possible to correct MIP and MEP results for altered lung volumes. Increased residual volume will place the inspiratory muscles at increased mechanical disadvantage. The resulting MIP would be lower than that which would result if the residual volume was not increased. The fact that there was no significant difference in flow rates over time, however, indicates that it was unlikely that the difference in the change in MIP between the control and the treatment group was a result of altered lung volumes.

Several other factors need to be considered in the interpretation of pulmonary function, respiratory muscle strength and six-minute walking tests. To minimize a learning effect, the patients practised the manoeuvres for the MIP and MEP tests prior to performing the test and completed a practice six-minute walk. There is much intersubject variability in MIP and MEP measures. Repeat tests on the same individual, however, show little intrasubject variation.[121] A training effect was found to be significant between an initial and a second 12-minute walk but further increases were not significant on subsequent walks.[122] Additionally, it would be expected that any training effects would be equally distributed between the treatment and the control group.

Walking test performance also depends on various factors including motivation, endurance, respiratory function, cardiovascular fitness, and neuromuscular function. It is difficult to quantify the effect that these other factors may have on test results. However, it would be expected that these factors would affect the treatment and control group equally.

The mechanism behind the significant group by time interaction for MIP and the trends for MEP and the six-minute walk is not clear. Nutritional status is one of several factors contributing to respiratory muscle strength. Malnutrition impairs respiratory muscle function by decreasing the availability of energy substrates and altering the structure of muscle fibres.[36] Animal studies have confirmed that restricted energy intake results in a reduction of diaphragmatic muscle fibre size primarily in the fast-twitch muscle fibres.[64] If the adverse effects of malnutrition on respiratory muscle function could be attributed to reductions in muscle size, then successful nutrition intervention would be expected to show increases in lean body tissue for measureable physiologic change. One clinical study supports this. When a group of ventilator dependent COPD patients were given TPN nutritional support there was a significant positive correlation between body cell mass and inspiratory muscle strength.[62] No significant difference in FFM was documented in this study. Therefore, increased respiratory muscle mass in the treatment group is an unlikely explanation for this interaction.

Studies in populations without COPD have suggested that muscle function in malnutrition may be independent of muscle size.[131] In vivo studies using ulnar nerve stimulation and measurement of the contraction force of the extensor pollicis longus have suggested that muscle function is abnormal with energy deprivation and returns to normal with refeeding. These changes occur independent of changes in FFM. This suggests a possible biochemical abnormality in the respiratory muscles of COPD patients that may be reversible with enhanced nutrient intake.

This study's finding of no significant differences in pulmonary function and respiratory muscle strength tests over time along with no significant body weight change or body composition change are consistent with those of other studies. Refeeding studies in malnourished COPD patients where little or no weight gain occurred also did not document significant changes in respiratory muscle function.[15,16] Only studies which reported significant weight gain and improvement in nutritional status were able to document significant improvement in respiratory muscle performance. Along with significant weight gain, Wilson and associates[14] reported a 41% increase in respiratory muscle strength. These results need to be interpreted with caution; however, as the number of subjects was small (n=6). Also, there was no control group. The effects of nutritional repletion on muscle function during the short refeeding period of two weeks may be related more to repletion of muscle water and potassium rather than to muscle protein anabolism and reconstruction.[15] In the study conducted by Efthimiou and associates,[12] the mean weight gain was 4.4 kg over the 3 month period of dietary supplementation. TSF and mid-arm-muscle circumference also increased significantly during this period. This group reported significant increases in respiratory muscle strength, hand grip strength and distances walked in six minutes. At the same time, sternomastoid fatigability was decreased. Significant increases in maximal expiratory pressure and mean sustained inspiratory pressure were shown in Whittaker and colleagues' successful short-term tube feeding study.[13]

PERCEIVED WELL-BEING

PWB-R Analyzed by Group

At baseline both groups had similar perceptions of general well-being, psychological and physical well-being. The treatment group's perceived physical well-being, however, tended to be somewhat lower than that of the control group. These results may be explained by the fact that the treatment group had a greater degree of airway obstruction than the control group. Therefore, they may have encountered greater physical limitations. Over the study period, the treatment group had no significant change in any of the perceived well-being scores. However, there was a trend for a decrease in physical well-being while psychological well-being remained constant. The control group experienced a significant decrease in general well-being over the six month period. No significant differences were found between baseline and six month psychological and physical well-being subscores. The tendency for both perceived physical and psychological well-being to decrease was observed.

PWB-R and PWB Analyzed as a Whole Group (n=11)

Small sample size limits the interpretation of the above results. Consequently, scores for PWB-R were reanalyzed for the group as a whole. The baseline general well-being score of 75 ± 4 is lower than that of 88 ± 1 reported for a sample of 121 elderly people (60-98 yr) living independently (GT Reker personal communication). Mean psychological well-being score for this group of elderly was 49 ± .5, which is similar to that of 48 ± 2 for the COPD patients in this study. The physical well-being score of 27 ± 3 for the COPD patients was lower than 39 ± 1 that was documented in the elderly group (GT Reker personal communication). It appears, therefore, that the COPD patients in this group perceived their general well-being to be poorer than the elderly population in general. Physical well-being was perceived to be the component of well-being that is compromised. This is a realistic finding for patients with severe COPD.[91]

Over the study period, there was a significant decrease in the general PWB-R well-being score and the physical well-being subscore. These results are consistent with the contention that perceived physical well-being is more closely linked to one's actual physical condition than perceived psychological well-being.[92]

The results for the PWB showed the same trends as the results for the PWB-R. The mean baseline PWB general well-being score of 65 ± 3 observed in the present study is similar to the baseline mean score of 64 ± 2 observed in a study which investigated the stability of perceived well-being in COPD patients.[91] Thirty-eight ambulatory patient with severe COPD completed the PWB scale on two occasions, at baseline and one year later. There was no specific intervention given to these patients over the year. Although the exact subscores for physical and psychological well-being were not reported, the investigators reported that physical well-being scores tended to fall toward the lower end of the scale. The psychological subscore tended to be closer to normal. The results of the present study are consistent with these findings. At follow-up testing, the general well-being score, physical and psychological well-being subscores for the 38 COPD patients were slightly but not significantly higher.[91] These findings are in contrast to those of the present study. At the six month retest, there was a significant decrease in general well-being score and physical well-being subscore. There were insufficient data provided to allow for comparison of the actual severity of COPD and nutritional status of the 38 COPD patients with those in the present study. Greater clinical severity of illness for the patients in the present study may explain the differing retest trends for the perceived well-being scores.

A study which used a psychiatric test (SCL-90R) to examine general well-being, indices of depression, and anxiety in outpatients with COPD concluded that psychiatric factors appeared to only play a minor role in nutritional depletion.[4] Another study found that both well nourished and malnourished COPD patients showed a moderate degree of depression.[93] The definition of psychological well-being for the PWB scale is "the presence of positive emotion such as happiness, contentment, joy and peace of mind and the absence of negative emotions such as fear, anxiety, and depression".[92] Considering this definition, it appears that the psychological scores obtained in this study are in agreement with these other studies.

In COPD patients, there is a common triad of dyspnea, especially on exertion, intermittent cough and fatigue. This triad is a major contributor to disability.[91] A study which evaluated quality of life in 985 COPD patients showed that quality of life was severely compromised. These patients' scores on the Sickness Impact Profile (SIP) indicated that the patients' illness had a very substantial effect on psychological and physical function. Their scores for the Profile of Mood States showed relatively high levels of anxiety, depression, hostility, fatigue and confusion.[94]

The perceived well-being scores from this study and those of other investigators, along with results from six-minute walk tests suggest that factors such as limited physical abilities and endurance impede COPD patients' ability to increase nutrient intake.

EVALUATION OF NUTRITION EDUCATION

The aim of nutrition counselling, the process of individualizing instruction to enable clients to become self-sufficient in managing personal nutritional care, is to encourage voluntary behaviour changes. Therefore, it is essential to consider behavioral issues as well as principles of nutritional management. Challenges to successful nutrition education include long-standing eating habits which have become ingrained and occur at a subconscious level. Changes to these behaviours are difficult because patients are not aware of their behaviour and habits. Poor health limits the patient's ability to shop and cook. Additionally, physical changes reflect body status rather than behaviour and are subject to influences apart from nutrition education. Other challenges to successful nutrition education include client factors such as limited finances, poor living conditions and inadequate food storage and cooking facilities.[95,132] From personal observation of the subjects' homes and discussions with the study participants, it was evident that all the participants had adequate, although limited for some, finances, living conditions, food storage and cooking facilities. Consequently, nutrition knowledge, physical limitations and long-standing eating habits were the primary challenges to the success of nutrition intervention.

Results of the evaluation of the nutrition education questionnaire indicated that all patients felt that they could speak with the dietitian when they wanted to and that follow-up contact was useful. There was agreement that the dietitian provided useful information that was suited to their needs. They also indicated that they knew what to eat for their special needs. These results indicate that these patients did not perceive that lack of nutrition knowledge was a problem.

Only two thirds of the patients agreed that they changed their diet after speaking with the dietitian. Reasons for not changing their diet could include that they felt that they did not require changes, or that the nutrition counselling provided was so tailored to their needs that they did not recognize that changes had been made. Those who were neutral or disagreed when asked if they could not change their diet after speaking with the dietitian may have been unwilling to accept the idea that they were unable to make changes due to physical or other situational limitations. The patients' appreciation of the psychosocial value of contact with the dietitian may explain the discrepancy in the patients' perception of physical benefit (ie weight gain) and the benefit of talking with a dietitian. More than half of the participants felt that they felt better emotionally and were in control of their condition after talking to the dietitian while less than half felt that their weight improved. Other important components of the relationship that developed between the participants and the dietitian also could influence the participants' perception of benefit of the intervention. All participants perceived the dietitian to be a credible source of information. They also indicated that the dietitian provided support, encouragement and cared about them. The results of evaluation of nutrition education obtained in the present study are similar to those obtained in a group of oncology patients.[95]

The stability of the treatment groups' psychological well-being may be a reflection of the psychosocial benefit that they obtained from receiving individualized nutrition counselling. One goal of nutrition counselling was to emphasize the patient's ability to problem-solve and to focus on strengths rather than weaknesses. It has been documented that psychological well-being is positively correlated with problem-focused coping strategies.[91]

RELATED OBSERVATIONS

The role of a dietitian is to translate the science of nutrition into the art of furnishing optimal nourishment to people. Dietetics is not an exact science. Each recipient of a dietitian's services provides a unique challenge. The following quotations and observations, which were gathered over the six month study period, illustrate the diverse concerns of these patients. Each of these can influence nutrient intake and nutritional status.

> "I know you are trying to help me Janet but when you can't breathe you can't eat."
>
> "That was the day that I was at my niece's third birthday. My breathing got so bad that I had to go sit in the car with the air conditioner on. Finally, I just had to go home to bed." (intake 1300 kcal vs usual 2700-3000 kcal)

These statements illustrate the limitations that shortness of breath associated with COPD place on nutrient intake. Often it is a real struggle for these patients to perform the activities of daily living such as shopping, meal preparation and eating.

> "I think that I would eat better if my dishes were done."
>
> "I would eat if someone cooked for me."

These quotes illustrate the concerns of a widower who lived alone. Like many elderly people, he found preparing meals for himself and eating by himself difficult. Generally, older men find it more difficult to prepare meals due to lack of experience in food preparation. Elderly individuals are more likely to have a balanced diet, both in nutrient intake and variety, if they live with a spouse.[131]

"I like to eat what I like to eat."

Habit and custom play a large role in the elderly's food choices. Cultural factors, ethnicity, religious and traditional beliefs all influence food preferences. Introducing new food items or making modifications to usual food patterns can be difficult to achieve when eating habits have been lifelong.

> "I haven't been eating well lately. I've been feeling down. Sometimes, I just break into tears."

Living with a chronic disease can be depressing. Throughout the study period the woman who made the above statement struggled to deal with the limitations that her disease placed on her life. For this woman, depression made increasing nutrient intake a difficult task.

> "I would die before I drank that stuff. It's a step down isn't it?"

One patient in the treatment group made this statement in reference to an untouched case of Pulmocare™. It illustrates how important it is to question why clients are not willing to make recommended changes to their diet. Rather than perceiving a supplement as something positive, he perceived that the need to take a supplement (formula feeding) was an indication of a deterioration of his condition.

CHAPTER SIX

SUMMARY AND CONCLUSIONS

SUMMARY

The purpose of this study was to identify dietary and personal factors which contribute to failure to gain weight and failure to increase nutrient intake in malnourished, ambulatory patients with COPD. As well, the effect of individualized nutrition counselling, in the home environment, on nutrient intake, body weight, body composition, pulmonary function and perceived well-being in these patients was assessed. Additionally, the patients' perceptions of the nutrition education provided were evaluated. Acceptance and usage of Pulmocare™ was also monitored.

Baseline measurements of REE indicated that these patients were not hypermetabolic. Individualized nutrition counselling did not result in significant increases in protein and energy intake. There was no significant group effect for protein or energy intake. The treatment group tended to have the highest level of intake over the first two weeks of the study. After this, energy intake tended to decline toward baseline levels.

There was no significant group or time effect for body weight, TSF, SSF, MAMC or AMA. There was a significant time effect for FFMBIA but no two points were identified as being significantly different.

Consistent with other refeeding studies which were not able to achieve adequate intake to achieve weight gain, the present study found no significant time or group effects for any of the pulmonary function tests, MIP, MEP or distance walked in six minutes.

Many factors contributed to difficulties in promoting increased nutrient intake to promote weight gain. Hospitalization for exacerbations of COPD frequently resulted in weight loss. Dyspnea, fatigue, poor appetite, early satiety, and gastrointestinal discomfort were impediments to increasing nutrient intake. Additionally, Pulmocare™ was not well tolerated.

When the study participants were analyzed as one group, perceived well-being scores were similar to those previously reported for COPD patients. These patients perceived their general well-being to be poorer than that of the elderly population in general. Physical well-being was the component of well-being that was compromised. The present study found a significant decrease in general well-being and physical well-being over the study period.

All patients perceived that the dietitian provided credible, useful information that was suited to their needs. Nutrition counselling was considered beneficial. All subjects indicated that COPD patients should see a dietitian.

LIMITATIONS TO THE STUDY

Small sample size, which compromised the power of the study, was a major limitation. Additionally, the participants were not a random sample of malnourished COPD patients. Physician referral to the study and voluntary participation in the study bias the sample toward cooperative, motivated and mentally alert individuals. This group may have been more interested in diet or motivated to modify their diet than others.

CONCLUSIONS

COPD patients are at nutritional risk. Weight loss and malnutrition are a significant problem for many COPD patients. Associations have been made between malnutrition and impaired respiratory function, decreased immunocompetence and increased morbidity and mortality in COPD patients. Therefore, it is important to investigate the reversibility of malnutrition in this group.

In the present study, individualized counselling along with provision of a nutritional supplement was not successful in promoting adequate nutrient intake for weight gain and improvement in nutritional status. Inability to promote weight gain can be attributed to limitations to augmenting nutrient intake. Increased energy needs especially during exacerbation of disease may also contribute to difficulty in attaining weight gain. The physical limitations placed on these patients due to exacerbation of COPD, dyspnea and fatigue rather than lack of nutrition knowledge or motivation to modify their diets made it difficult to intervene successfully. Weight loss appears to be episodic during periods of disease exacerbation rather than continual. In constrast to the present study, studies which were able to achieve adequate intake for nutrient repletion were able to document improvements in respiratory function and sense of well-being in COPD patients.

Despite not gaining weight, the patients perceived that the nutrition counselling was beneficial. Psychosocial benefits of contact with a dietitian are important elements of nutrition counselling.

Nutrition intervention is an important component of the treatment of COPD but facilitating improvement to nutrient intake for ambulatory COPD patients is a challenge. Dietitians have an important role to play in providing nutritional care for these patients. It is important to monitor nutritional status closely because it can change quickly. Earlier intervention and more aggressive nutrition support may be required for these patients.

FUTURE RESEARCH

-Documentation of long-term impact of nutrition intervention on morbidity, quality of life, number of hospital admissions, and mortality

-Establishment of selection criteria to identify those most likely to benefit from nutritional support

-Evaluation of the effect of alternate approaches to nutrition support in the home such as home tube feeding

-Evaluation of innovative ways to provide community support to promote improved nutritional intake of chronically ill individuals who have limited abilities and resources for meal preparation

CHAPTER SEVEN

RECOMMENDATIONS FOR PRACTICE

COPD patients are at nutritional risk. The following are recommendations for assessing and monitoring the nutritional status of these patients. Strategies for nutritional intervention are also provided.

NUTRITIONAL ASSESSMENT

Baseline Assessment

Minimum Nutritional Assessment should include:

-weight, %IBW, %UBW, weight history

-complete diet history including assessment of:

dyspnea, fatigue, anorexia, early satiety, bloating, nausea, vomiting, chewing and swallowing, bowel problems, use of supplements

living conditions, cooking and food storage facilities, shopping arrangements, use of community services (meals on wheels, homemakers)

3 or 4 day food records

Additional Nutritional Assessment Measures:

-Anthropometry: skinfolds, mid-arm muscle circumference, BIA

-Biochemical parameters: electrolytes, complete blood count, total protein, albumin, phosphorus, magnesium, amd calcium. (Note hemoglobin is not a good indicator of iron status in COPD because it tends to be elevated in response to hypoxia)

-Respiratory function tests: spirometry, respiratory muscle pressures, distance walked in 6 minutes

Estimating Nutrient Needs:

-Ideally, indirect calorimetry should be used to assess energy expenditure.

-Optimal energy level remains controversial.
1.4 to 1.6 x REE seems appropriate for weight maintenance

-1.7 x REE for nutritional repletion for stable COPD patients.

-Protein recommendations for repletion 1.5 - 2.0 gm/kg seem reasonable.

-There is little data to support use of diets with altered fat to CHO ratios in stable COPD patients. Diets containing approximately 50% CHO have been well tolerated by patients with severe airflow obstruction.

-A daily multivitamin and mineral supplement is recommended for patients who are not consistently consuming a variety of foods from all food groups.

-Patients on regular doses of steroids may require calcium supplementation and sodium dietary modifications.

RECOMMENDATIONS FOR COUNSELLING AMBULATORY COPD PATIENTS

Nutrition counselling needs to be individualized to accommodate the unique dietary concerns of each COPD patient. However, the following general suggestions and strategies for nutritional therapy of COPD patients outlined on table 9 may be beneficial.

General Suggestions

-Focus on making meal preparation and eating as easy and enjoyable as possible.

-Encourage patients to take advantage of days that they feel well to maximize food intake and to prepare extra portions of food to freeze and use on days when they do not feel as well.

-Advise patients to organize their kitchens so that minimal effort is expended reaching for utensils, dishes and food during meal preparation.

-Suggest smaller more frequent easy to prepare meals and snacks.

-Encourage patients to rest for a few minutes before eating, to eat slowly, relax at meal times and use oxygen, if necessary, while eating.

-Ensure adequate fluid intake 1.5-2.0 L/day. Nutrient containing fluids should be recommended for weight losing patients, water for those who do not require extra energy intake.

-As required, recommend that patients utilize community support services such as homemakers, volunteer shopping programs and meals on wheels.

Table 9: Impediments to adequate oral intake and counselling strategies for nutritional therapy of COPD patients

Complaint	Recommendations
Dyspnea	-Simplify meal preparation, convenience foods or homemade, previously prepared and frozen meals may be helpful -Rest for 5-10 minutes before eating -Relax and eat slowly at meal times -Use bronchodilators before meals and/or oxygen while eating -Try soft moist easy to chew foods -Drink nutrient dense fluids if unable to eat solids
Fatigue	-Choose easy to prepare foods -Rest before meals -Eat larger meals at times when least fatigued -Obtain assistance with shopping and meal preparation -Try foods which are soft and moist that require little chewing or drink nutrient dense fluids
Anorexia	-Make nutrient dense foods a priority -Make favourite foods readily available -Eat small frequent meals and snacks over the day -Push intake as much as possible
Early Satiety	-Eat nutrient dense foods first -Limit fluids with meals, take fluids between meals -Eat small meals and snacks frequently
Bloating/Burping	-Eat smaller more frequent meals -Eat slowly to avoid swallowing air -Avoid gas forming foods, individual to patient
Chewing/Swallowing	-Choose soft, moist, easy-to-chew nutrient dense foods
Bowel Problems (Constipation)	-Encourage increased fibre intake and adequate fluids -Try prune juice or fruit lax -Encourage regular activity as tolerated
Mood/Depression	-Provide encouragement and support, -Push intake on "better days" -Refer to appropriate counselling services

USE OF SUPPLEMENTS

Liquid oral nutritional supplements are a convenient way to increase nutrient intake. Encourage patients to try a number of different supplements and to consume those that they prefer. Altered fat to CHO ratio supplements do not seem to be necessary for this group of patients. Finding a supplement that they tolerate well and like is critical to continued supplement usage.

MONITORING NUTRITIONAL STATUS

Nutritional status needs to be monitored on a regular basis. Patients should be weighed on a properly calibrated scale regularly. Nutritional status can change quickly. During exacerbations of disease, intake tends to decrease at a time when nutrient needs increase. Intervention should be initiated early. If a patient is not responding to oral supplementation, more aggressive nutritional support, such as a tube feed, should be considered, especially when patients are hospitalized.

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GLOSSARY

Acinus:

the terminal respiratory unit or gas exchange unit; it consists of the respiratory bronchiole, alveolar duct and alveolar sacs

Alveoli:

the air cells of the lungs

Airway Obstruction:

increased resistance to airflow in the conducting passages of the respiratory tract

Asthma:

sudden, periodic dyspnea accompanied by wheezing caused by a spasm of the bronchial tubes or by swelling of the mucous membranes

Atelectasis:

collapse of distal lung air spaces after obstruction of the proximal airway and consequent absorption of the trapped gas

Bronchiectasis:

chronic dilatation of a bronchus or bronchi, with a secondary infection that usually involves the lower portion of the lung

Chronic obstructive bronchitis:

pulmonary disease characterized by 1) excessive tracheobronchial mucous secretions causing cough with expectoration for at least 3 months of the year for more than 2 consecutive years and 2) structural changes in the small airways causing narrowing and obstruction

Chronic obstructive pulmonary disease (COPD):

clinically significant, irreversible airway obstruction due to chronic obstructive bronchitis, emphysema, asthma or a combination of these conditions

Compliance:

change in volume per unit change in pressure; measure of elastic resistance of the total respiratory structure, chest wall or lungs

Cyanosis:

slightly bluish, grayish, or dark purple discoloration of the skin due to presence of abnormal amounts of reduced hemoglobin in the blood

Diffusion capacity:

the rate of gas transfer through a unit area of a permeable membrane per unit of gas pressure difference across it

Diffusion:

transfer of oxygen and carbon dioxide between the acini and the pulmonary capillary bed

Dyspnea:

difficult or laboured breathing

Emphysema:

pulmonary disease characterized by destruction of alveolar septa and enlarged airspaces distal to the terminal bronchiole

Forced Expiratory Flow (FEF_{25-75}):

mean forced expiratory flow during the middle half of the FVC

Forced Expiratory Volume (FEV):

volume of air forcefully and rapidly expired following a maximum inspiration during a specified time period

Forced Expiratory Volume in 1 second (FEV_1):

the FEV measured during the first second of expiration; expressed as a percentage of the forced vital capacity (FEV_1/FVC%) it indicates the severity of airways obstruction in COPD

Forced Vital Capacity (FVC):

volume of air forcefully and rapidly expired after a maximal inspiratory effort

Functional Residual Capacity (FRC):

volume of air in the lungs at the end of a normal expiration when all the respiratory muscles are relaxed

Hypercapnia: increased partial pressure of carbon dioxide in the arterial blood; occurs during hypoventilation, CO_2 inhalation and CO_2 retention.

Hypoxemia:

deficient blood oxygenation

Maximal Expiratory Pressure (MEP):

measurement of the maximal pressure generated at the mouth when an individual makes maximal efforts against a closed airway during expiration; an indication of expiratory respiratory muscle strength

Maximal Inspiratory Pressure (MIP):

measurement of the maximal pressure generated at the mouth when an individual makes maximal efforts against a closed airway during inspiration; an indication of inspiratory muscle strength

Maximum Voluntary Ventilation (MVV):

maximum volume of air that an individual can ventilate by breathing as quickly and deeply as possible during a specific time period; it is an index of respiratory muscle endurance

Perfusion:

blood flow through the pulmonary capillary bed

Pulmonary surfactant:

dipalmitoyl phosphatidylcholine, a liquid that lines the alveoli and decreases the surface tension of the alveolar walls

Residual Volume (RV):

the volume of air remaining in the lungs at the end of a maximum expiration

Respiratory failure:

severe hypercapnia or hypoxemia or both

Respiratory quotient:

ratio of carbon dioxide production to oxygen consumption resulting from metabolism of energy substrates

Total lung capacity (TLC):

volume of air contained in the lungs at the end of a maximum inspiration

Tachypnea:

abnormally rapid respiration

Ventilation/perfusion ratio:

ratio of pulmonary alveolar ventilation to pulmonary capillary perfusion It indicates the degree of matching between gas distribution (ventilation) and blood distribution in the lungs

Ventilation:

the rhythmic movement of air into and out of the lungs

Vital Capacity:

the maximum volume of air that can be expired after a maximum inspiration

Sources for glossary: references 17,18,50,117.

APPENDICES

APPENDIX 1

ASSESSMENT OF PULMONARY FUNCTION IN COPD

ASSESSMENT OF PULMONARY FUNCTION

Pulmonary function tests provide a means of quantification of COPD and evaluation of its response to therapy. Patients with COPD can be followed with objective tests including spirometry, blood gases, maximal inspiratory and expiratory mouth pressures and distance walked in six or twelve minutes. Pulmonary function tests and arterial blood gas analysis are used in combination with history and physical examination to identify the various disease syndromes (asthma, chronic bronchitis or emphysema) and their severity.[17,18]

Spirometry

Routine spirometry provides basic pulmonary function assessment needed for screening, diagnosis, and evaluation of prognosis and therapy in patients with obstructive pulmonary disease. The most important physiologic measurements derived from spiromety are the forced vital capacity (FVC) and the forced expiratory volume in one second (FEV_1). FVC is the total volume exhaled when a subject inspires maximally and then exhales as hard and as completely as he/she can. FEV_1 is the total volume exhaled in the first second of this manoeuvre. Measurements are compared to predicted normal values based on the patient's age, sex and height. Normally FEV_1 is about 80% of the FVC. In obstructive diseases FEV_1 is reduced more than the FVC resulting in a low FEV_1/FVC%. Expressing FEV_1 as a percent of FVC avoids the effects of superimposed restrictive disease and allows comparison between individuals with different body sizes.[17,18,118]

There is variation in the interpretation of what is considered mild, moderate, and severe obstruction. Generally, a decrease in FEV_1/FVC to less than 75-80% of the predicted value indicates airway obstruction. Mild obstruction can be interpreted for a ratio between 60% and 80%. Ratios between 45% and 60% indicate moderate obstruction and less than 45% indicate severe obstruction.[17,18,118]

Arterial Blood Gases and pH

Arterial oxygen tension (Pa_{O2}), arterial carbon dioxide tension (Pa_{CO2}), and pH are easily measured in blood samples with blood gas electrodes. Arterial blood gases are essential to the assessment of severity of lung function impairment in patients with moderate to severe COPD. Normal Pa_{O2} in young adults at sea level is 80-100 mmHg. This value normally decreases with age.

The Pa_{CO2} is the best indicator of alveolar ventilation. Hypoventilation is always associated with elevated Pa_{CO2}. Normal values range from 32-46 mmHg.[116]

Maximal Static Inspiratory and Expiratory Respirtory Pressures

Maximal inspiratory (MIP) and expiratory (MEP) pressures produced at the mouth during static efforts reflect respiratory muscle strength. The simplest method to measure inspiratory and expiratory pressures is to have a subject make maximal efforts against a closed airway. The preferred technique is that of Black and Hyatt.[119] To generate MEP the subject inhales to total lung capacity and makes an expiratory effort for several seconds. To generate MIP, the subject exhales to residual volume and makes a powerful inspiratory effort. Normal values have been reported for the relationship of MIP and MEP to age and gender.[119,121] The coefficients of variation for repeated measurements of MIP and MEP are small. This indicates little intrasubject variation. Interindividual variation, however, can be large.[121] Lung volume can also influence MIP and MEP. When a low MIP is found in COPD, it may simply reflect the fact that the patient made the effort at a higher than normal lung volume.

Six- and Twelve-Minute Walking Tests

A 12-minute walking test is a useful and reproducible exercise tolerance measure which provides a practical guide to everyday disability in COPD.[122,123] A high correlation between a six and 12-minute walking test indicate that they are similar measures of exercise tolerance.[124] There is a potential training effect for this test; therefore, the test should be carried out twice to achieve reproducible results.[122]

APPENDIX 2

ETHICS APPROVAL CERTIFICATES

The University of British Columbia
Office of Research Services

C91-122

CLINICAL SCREENING COMMITTEE FOR RESEARCH
AND OTHER STUDIES INVOLVING HUMAN SUBJECTS

C E R T I F I C A T E of A P P R O V A L

INVESTIGATOR:	McCargar, L.
UBC DEPT:	Family & Nutr Sci
INSTITUTION:	Univ-UBC
TITLE:	The effect of individualized nutrition intervention in malnourished, ambulatory patients with chronic obstructive pulmonary disease (COPD)
NUMBER:	C91-122
CO-INVEST:	Le Patourel, J.A. Road, J.
APPROVED:	MAY 27 1991

The protocol describing the above-named project has been reviewed by the Committee and the experimental procedures were found to be acceptable on ethical grounds for research involving human subjects.

Dr. S. Segal, Chairman
Clinical Screening Committee

Shirley A. Thompson
for Dr. R.D. Spratley
Director, Research Services

THIS CERTIFICATE OF APPROVAL IS VALID FOR THREE YEARS
FROM THE ABOVE APPROVAL DATE PROVIDED THERE IS NO
CHANGE IN THE EXPERIMENTAL PROCEDURES

RESEARCH COMMITTEE

NOTICE OF RESULT OF EVALUATION

EVALUATION DATE:	August 8, 1991
PRINCIPAL INVESTIGATOR:	Dr. J. Road
RESEARCH TITLE:	"The Effect of Individualized Nutrition Intervention in Malnourished Ambulatory Patients with Chronic Obstructive Pulmonary Disease"
PROPOSAL NO:	91.37

Your Research Proposal has been evaluated and the result is as follows:

Final approval granted.

JOEL OGER, M.D., F.R.C.P. (C)
Chair, Research Committee
University Hospital

JO/cjs

APPENDIX 3

CONSENT FORM

CONSENT FORM

The University of British Columbia

Investigators: J. Le Patourel RDN, Dr. J. Road, Dr. L. McCargar
Dr. S. Whittaker, S. Ross MSc, RDN

Title of the Project:

The Effect of Individualized Nutrition Intervention in Malnourished, Ambulatory Patients with Chronic Obstructive Pulmonary Disease (COPD)

Malnutrition is common among patients with COPD. Poor nutritional status has been associated with a decline in lung function and a decreased resistance to infection. The purpose of this project is to determine relationships between nutrient intake at home and nutrition status in patients with COPD. Whether changes in nutrition status will result in changes in lung function, the strength of muscles responsible for drawing air into the lungs, and an individual's sense of well-being will also be assessed.

I understand that as a participant in this study, I will be required to:

1. Keep four 4-day food records, answer questions regarding my diet, food preferences and weight changes. (Diet history interviews will take approximately 2 hours).

2. Come to UBC hospital for tests at the beginning of the study and at the end of the study (6 months later). These tests will involve giving a blood sample, performing lung function tests that involve blowing into a tube through a mouth piece, and going for a 6-minute walk. A respiratory technician will take an arterial blood sample that will be analyzed for oxygen and carbon dioxide concentrations. Arterial puncture for blood gases can lead to a small hematoma at the site of the puncture. When done properly, there is no danger of arterial damage. Also, my metabolic rate will be measured by having me breathe normally while resting under a plexiglass hood. (These tests will take approximately 3 hours on each visit).

3. Have one of the investigators (Janet Le Patourel) come to my home 3 or 4 times during the study to provide instruction on how to complete food records and diet.

4. Have body weight and height taken and body composition measured by calipers at the beginning, middle and end of the study. At these times, my body composition will be calculated by measuring the resistance between two pairs of electrodes placed on one of my wrists and one of my ankles as well.

5. Complete a short questionnaire to assess my perception of well-being at the beginning and end of the study.

6. Be willing to follow a diet which encourages an increased take of calories and protein. This diet will include regular foods and may include commercial nutritional supplements such as Pulmocare^{TM}.

I realize that upon giving consent to participate in this study, I will be randomly assigned to one of two groups. Dietary intake and nutrition status will be monitored equally for both groups. Individuals in one group will be encouraged to consume their usual diet while those in the other will be encouraged to increase caloric intake.

It is unlikely that any side effects will occur as a result of participating in this study. My participation in this study is voluntary. I am aware that I may refuse to participate or that I may withdraw from the study at any time without jeopardy to continued medical care.

If I have further questions concerning this study, I can contact Janet Le Patourel at 733-1078 or Dr. Linda McCargar at 228-6869 or Dr. Jeremy Road at 822-7128.

Thus, I ________________ hereby voluntarily consent to participate in this study and acknowledge that I have received a copy of this consent form. I have read and understand the contents of this form.

Date ______________

Signature of Participant ________________________

Signature of Witness __________________________

APPENDIX 4

DIET HISTORY QUESTIONNAIRES

NUTRITION ASSESSMENT
DIET HISTORY

Date: ____________________

Name: ____________________

Address: ____________________

Phone: ____________________ Sig Other: ____________________

Birth Date: ____________________ Age: __________ Sex: __________

Ht: ________ Current Wt: ________ IBW: __________ Usual Wt: ________

Wt Young Adult: ________ Max Wt: __________ Min Wt: ________

Recent Wt Changes: ________________ Time at recent Wt: ________

Appetite: ____________________ N/V: ____________________

Bloating/Burping: ______________ Dyspnea: ____________________

Fatigue: ____________________ Bowel Problems: ________________

Chewing/Swallowing: ____________________

Food Allergies: ____________________

Food Intolerances: ____________________

Previous Diets: ____________________

Supplements: ____________________

Medications: ____________________

____

Alcohol: ____________________ Smoking: ____________________

Living Conditions: ____________________

Cooking: ____________________ Shopping: ____________________

Restaurant Dining: ____________________

Activity Level: ____________________

Hospitalizations past year: ____________________

DIET RECALL

Breakfast: ______________________________

____

____

____

____

Snack: ______________________________

____

Lunch: ______________________________

____

____

____

____

Snack: ______________________________

____

Dinner: ______________________________

____

____

____

____

Snack: ______________________________

____

DIET HISTORY
6 MONTHS

Weight:__________ Weight Changes /6 mo. ______________

Appetite:________________

Bloating/Burping:_____________________

Fatigue:______________________________

Chewing/Swallowing:______________________

N/V:_______________________________________

Early Satienty:___________________________

Dyspnea:____________________________________

Bowel Problems:_____________________________

Supplements:_________________________________

Medications:____

____

____

Alcohol:______________________________

Smoking:______________________________

Living Conditions:____

Restaurant Dining:____

Activity Level:____

Hospitalizations over 6 mo.____________________________________

APPENDIX 5

FOOD FREQUENCY QUESTIONNAIRE

FOOD	Per Day	Per Week
MILK/MILK PRODUCTS:		
Milk		
Cheese		
Yogurt		
Cottage Cheese		
Ice Cream		
Custards/Puddings		
BREADS/CEREALS:		
Bread: Whole Grain		
Bread: Refined		
Cereal		
Rice		
Pasta		
Muffins		
FATS/OILS:		
Butter		
Margarine		
Oil		
Gravy		
Fried Food		
Salad Dressing		
Cream		
Cream Cheese		
Cream Sauce		
ALCOHOL:		
SODIUM:		
Salt at Table		
Salt in Cooking		
Processed Meat, Cheese		
Canned, Pkg. Soup		
Canned, Frozen Entrees		
Salty Snack Foods		
Pickles, Olives		
Fast Foods		
Condiments/Sauces		
SUPPLEMENTS:		

	Per Day	Per Week
MEATS/ALTERNATES:		
Beef/Pork		
Poultry (skin)		
Fish		
Liver		
Eggs		
Peanut Butter		
Nuts, Seeds		
Legumes		
Casseroles		
FRUIT:		
Vitamin-C Rich		
Juice		
VEGETABLES:		
Vitamin-A Rich		
Vitamin-C Rich		
SUGARS/SWEETS:		
Added Sugar		
Jam, Jelly, Honey		
Cakes, Pies		
Cookies		
Candy, Chocolate		
Soft Drinks		
SPECIAL DIET FOODS:		
OTHER:		

KDD 73-2-87

APPENDIX 6

FOUR-DAY FOOD RECORD FORM

GUIDELINES FOR KEEPING A FOOD RECORD

A FOOD RECORD IS A DETAILED DESCRIPTION OF EACH FOOD OR BEVERAGE ITEM TAKEN OVER 24 HRS OF A DAY. AN ACCURATELY COMPLETED FOOD RECORD CAN PROVIDE VALUABLE INFORMATION ABOUT THE NUTRITIONAL CONTENT OF AN INDIVIDUAL'S USUAL DIET.

TO ASSESS YOUR DIET RECORD CORRECTLY, WE MUST BE ABLE TO CLEARLY PICTURE THE FOODS AND BEVERAGES THAT YOU HAVE RECORDED. THE GUIDELINES BELOW DESCRIBE THE INFORMATION THAT IS IMPORTANT FOR YOU TO RECORD. PLEASE READ THESE GUIDELINES BEFORE YOU START.

PLEASE KEEP A RECORD OF EVERYTHING THAT YOU EAT OR DRINK ON THE ATTACHED FORMS FOR 4 DAYS IN A ROW. INCLUDE 1 WEEKEND DAY IN THE RECORDING PERIOD.

1. THE PORTION SIZE (QUANTITY) NEEDS TO BE ACCURATELY RECORDED
   -Please don't guess if you can measure!

It may be helpful to measure how much your regular glasses, cups and bowls contain before you start. You can describe portion sizes in as many ways as you like. The attached food pictures are provided to help you with portion sizes.

For example, you might record:

Volume

1 cup or 8 oz or 250 mL of 2% milk
1 tablespoon or 15 mL or peanut butter or cream cheese
1 teaspoon or 5 mL of sugar or honey

Size

1 "2 inch by 3/4 inch by 3/4 inch" piece of chedda: cheese
1 medium egg, poached
1 small apple
1 2" diameter digestive biscuit
1 medium bran muffin

Weight

2 ounces or 60 grams of lean hamburger meat or chicken or fish (use labels on packages to help you)

2. DETAILED DESCRIPTION OF FOOD ITEMS IS ESSENTIAL
-tell us as much as you can about the foods that you eat
BE SPECIFIC ABOUT THE <u>TYPE</u> OF FOOD, <u>BRAND NAME</u> IF APPLICABLE AND THE <u>CONTENT OF MIXED DISHES</u>

for example:

if you have cookies please tell us what type (chocolate chip) what brand name (Dare 's or homemade) as well as the size (2")

if you have milk, tell us if its canned or fresh and whether it is skim, 2% or whole as well as the amount eg 4oz

DESCRIBE MIXED FOODS AS IF YOU WERE WRITING A RECIPE
Everyone has their own way of making everyday foods- please tell us how you do it!

for example:

If you make a cheese sandwich, do you use margarine or butter? Do you add mayonnaise or miracle whip, or lettuce or tomato slices. What type of cheese and bread did you use? How much of each item did you use?

Attaching recipes for items such as casserole dishes or labels from prepackaged foods such as frozen dinners is helpful.

If you didn't make the food yourself, describe the contents as best you can. eg. If you had 1 cup of tuna casserole, let us know that it was about 1/2 macaroni, and 1/4 tuna and 1/4 peas and celery and cream sauce.

3. RECORD IMMEDIATELY AFTER EACH MEAL AND SNACK
-take your food record with you if you go out to eat. Please keep track throughout the day-its easy to forget exactly what you have eaten!

. HERE IS AN EXAMPLE FOR YOU TO USE AS A GUIDE:

	ID Number	DAY OF WEEK:	DATE:
	JL 4	Monday	10/05/87 (D)(M)(Y)

TIME & PLACE Hour & Min. AM/PM	FOOD & BEVERAGE ITEMS Use a separate line for each item. Describe carefully as if writing a recipe.	QUANTITY Specify each measure: g, ", oz,tsp,cup,ml.	CODES
12:30 p.m.	Macaroni and Cheese		
	- cooked macaroni noodles	1 cup	
	- homemade cheese sauce	1/2 cup	
	(made with butter, flour)		
	cheddar cheese and 2% milk)		
	Tomato Juice,	4 oz. glass	
	Whole Wheat Dinner Roll	1 - 2" Diam.	
	Margarine (Soft Tub)	2 tsp.	

WHEN YOU HAVE COMPLETED YOUR FOUR DAY FOOD RECORD, PLEASE TELL US
(A) WHETHER THIS WAS A USUAL WEEK'S DIET FOR YOU

YES, this record describes my usual diet __________
NO, this was not my usual diet because ____________________

I would usually eat ______________________________

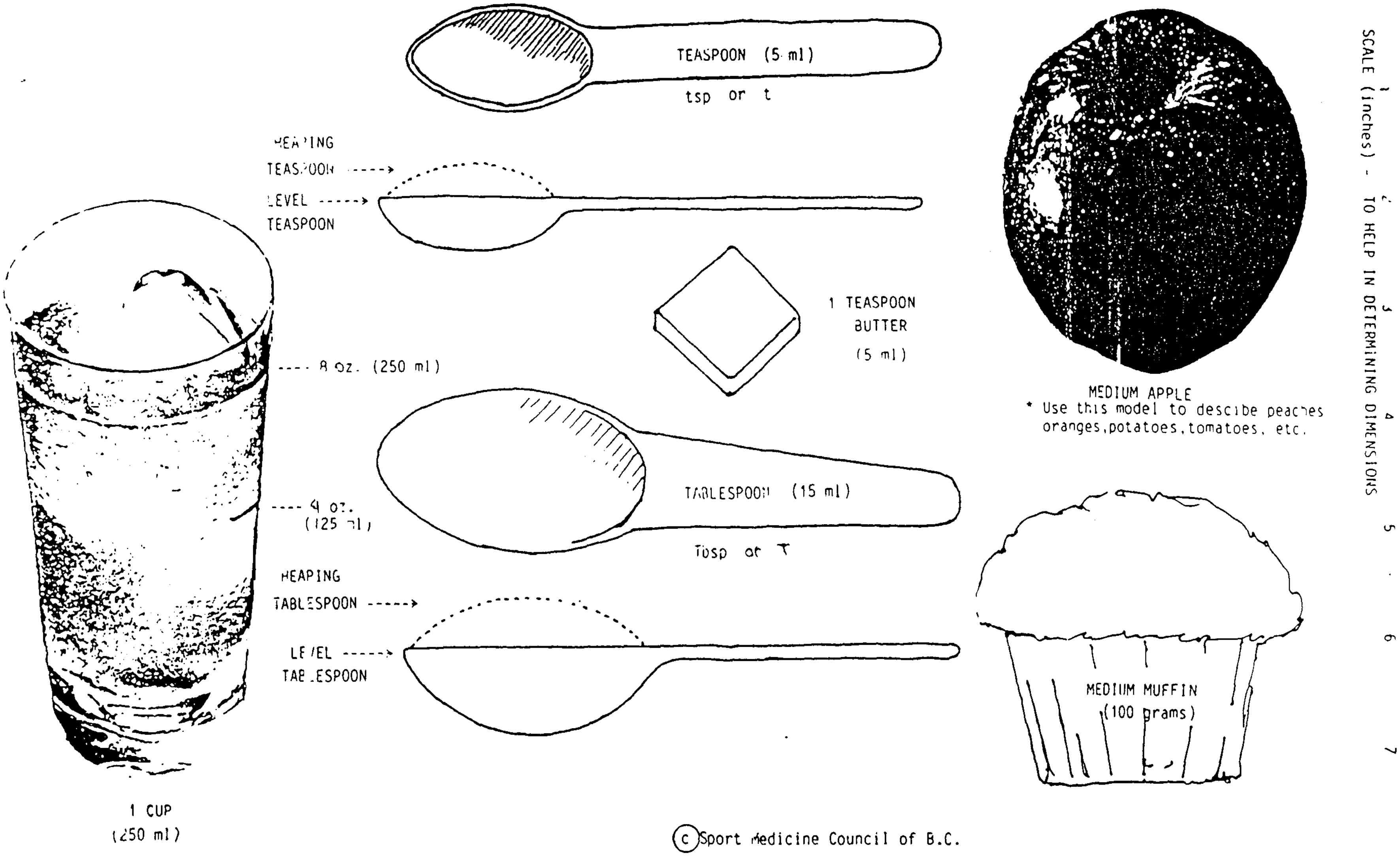
(B) WHETHER YOU TOOK ANY VITAMIN OR MINERAL SUPPLEMENTS THIS WEEK

NO, I did not take any vitamin or mineral pills __________
YES, I took the following vitamin/mineral pills: (please tell us the Brand name, the number of pills you took, and what the label says each pill contains)

ID NUMBER | DAY OF WEEK | DATE

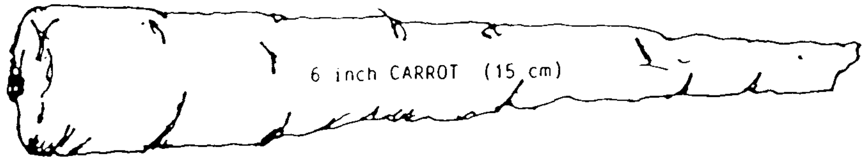
TIME & PLACE			FOOD & BEVERAGE ITEMS	QUANTITY	COD
Hour & Min. AM/PM	HOME	AWAY	Use a separate line for each items. Describe carefully as if writing a recipe.	Specify each measure: g." oz, tsp. cup	For Off Use Onl

oder ______ ID ______ Number ______ Entered

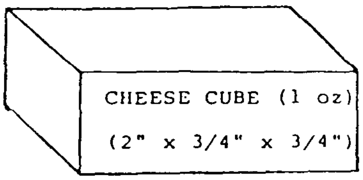

TEASPOON (5 ml)
tsp or t
HEAPING TEASPOON
LEVEL TEASPOON
1 TEASPOON BUTTER (5 ml)
MEDIUM APPLE
* Use this model to descibe peaches oranges, potatoes, tomatoes, etc.
8 oz. (250 ml)
4 oz. (125 ml)
TABLESPOON (15 ml)
Tbsp or T
HEAPING TABLESPOON
LEVEL TABLESPOON
MEDIUM MUFFIN (100 grams)
1 CUP (250 ml)
SCALE (inches) - TO HELP IN DETERMINING DIMENSIONS
1
2
3
4
5
6
7

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19
SCALE (cm) - TO HELP IN DETERMINING DIMENSIONS

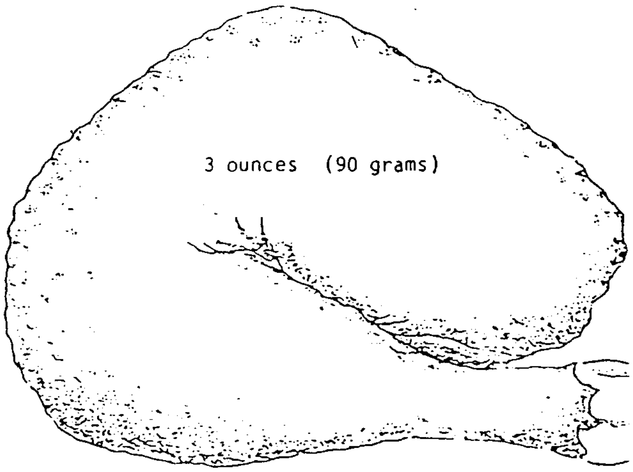
II. SIZE



III. WEIGHTS



4 ounces of raw meat = 3 ounces of cooked meat

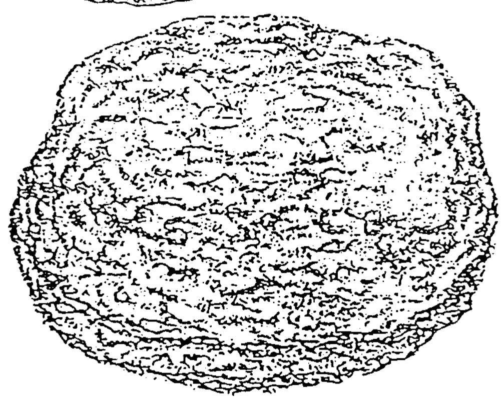


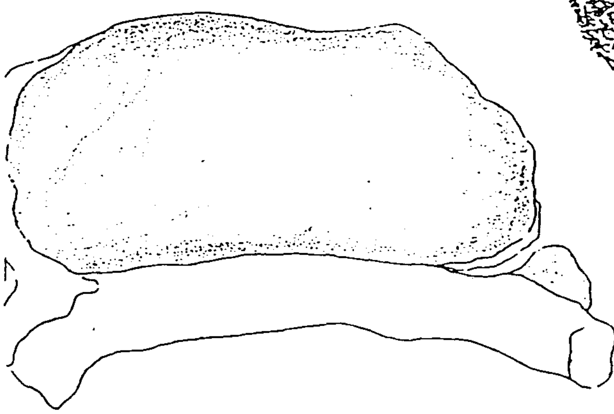
alf a chicken breast and wing or one leg and thigh cooked is equal to about 3 ounces of meat.

1 ounce= 30 grams

One hamburger pattie this thick is equal to 3 ounces.

One hamburger pattie this thick is equal to 2 ounces.





A pork chop this thick is equal to 3 ounces.

A pork chop this thick is equal to 2 ounces.

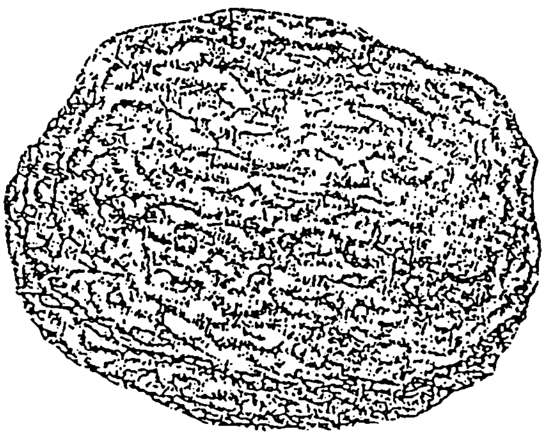
One slice of meat this thick is equal to 3 ounces.

Two slices of meat this thick are equal to 3 ounces

MEAT (cooked)

One slice of meat this thick ↴
is equal to 3 ounces.

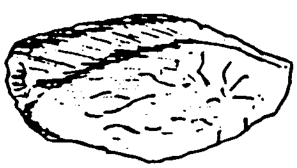
One slice of meat this thick ↴
is equal to 1½ ounces.



HAMBURGER PATTY (cooked)

One hamburger patty this thick ↴
is equal to 3 ounces.

One hamburger patty this thick ↴
is equal to 2 ounces.



STEWING MEAT (cooked)

One piece of cooked stewing meat
this size is equal to ½ ounce.

APPENDIX 7

PERCEIVED WELL-BEING QUESTIONNAIRE

PERCEIVED WELL-BEING SCALE - REVISED

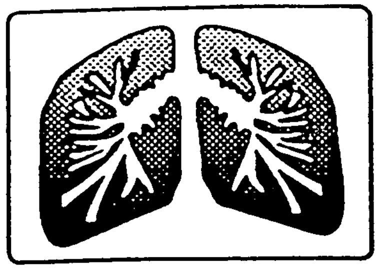
This questionnaire contains a number of statements related to your mental and physical well-being. Read each statement carefully, then indicate the extent to which you agree or disagree by circling one of the alternatives provided. For example, if you STRONGLY AGREE, circle SA following the statement. If you MODERATELY DISAGREE, circle MD. If you are UNDECIDED, circle U. However, try to use the UNDECIDED category sparingly.

SA	A	MA	U	MD	D	SD
STRONGLY AGREE	AGREE	MODERATELY AGREE	UNDECIDED	MODERATELY DISAGREE	DISAGREE	STRONGLY DISAGREE

1.	I have many physical complaints.	SA	A	MA	U	MD	D	SD
2.	No one really cares whether I am dead or alive.	SA	A	MA	U	MD	D	SD
3.	I think that I have a lung condition.	SA	A	MA	U	MD	D	SD
4.	I have plenty of physical energy.	SA	A	MA	U	MD	D	SD
5.	I am often bored.	SA	A	MA	U	MD	D	SD
6.	I have aches and pains.	SA	A	MA	U	MD	D	SD
7.	It is exciting to be alive.	SA	A	MA	U	MD	D	SD
8.	Sometimes I wish that I never wake up.	SA	A	MA	U	MD	D	SD
9.	I am in good shape physically.	SA	A	MA	U	MD	D	SD
10.	I feel that life is worth living.	SA	A	MA	U	MD	D	SD
11.	I think my health is deteriorating.	SA	A	MA	U	MD	D	SD
12.	I don't seem to care about what happens to me.	SA	A	MA	U	MD	D	SD
13.	I don't get tired very easily.	SA	A	MA	U	MD	D	SD
14.	I can stand a fair amount of physical strain.	SA	A	MA	U	MD	D	SD
15.	I have peace of mind.	SA	A	MA	U	MD	D	SD
16.	I am afraid of many things.	SA	A	MA	U	MD	D	SD
17.	I have a good appetite for food.	SA	A	MA	U	MD	D	SD

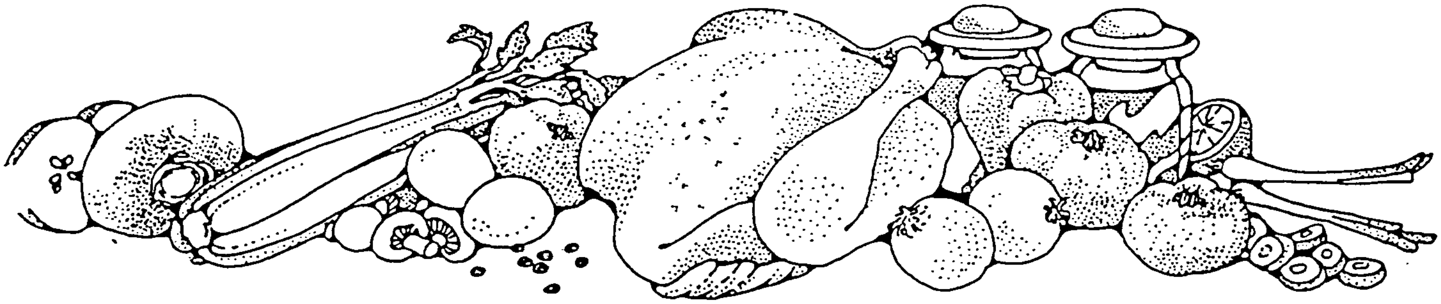
APPENDIX 8

NUTRITIONAL GUIDELINES FOR COPD PATIENTS



A BITE AT A TIME

NUTRITION AND COPD

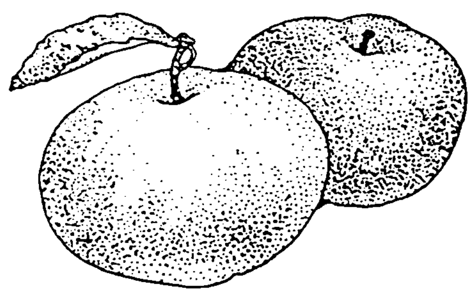


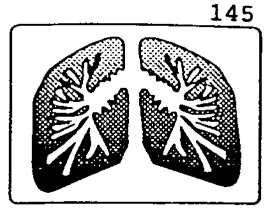
For:________________________________

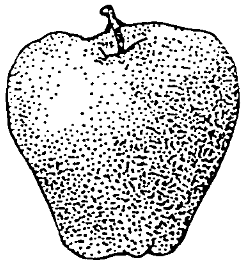
Date:________________________________

If you have any questions or concerns, please call:

Janet Le Patourel, nutritionist, at 733-1078.







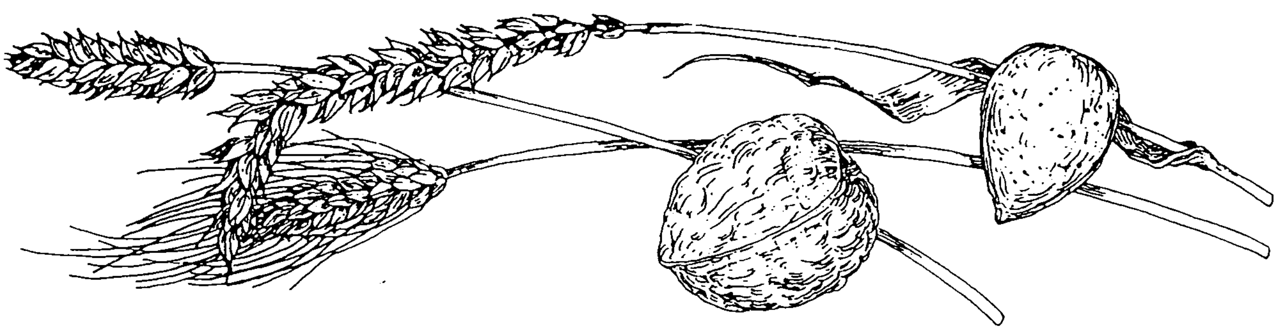
A BITE AT A TIME

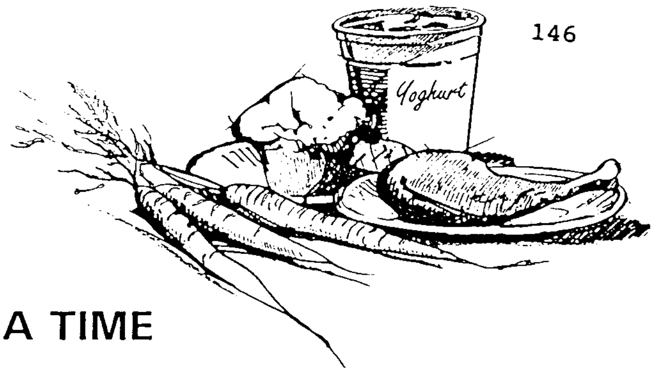
NUTRITION AND COPD

People with COPD use a lot of energy just breathing. That is why it is important to choose foods that will provide you with enough energy.

What you eat may help you breathe more easily, feel less tired, fight infections more readily and shorten time spent in the hospital.

This information along with advice from your dietitian may help to make eating and breathing easier.





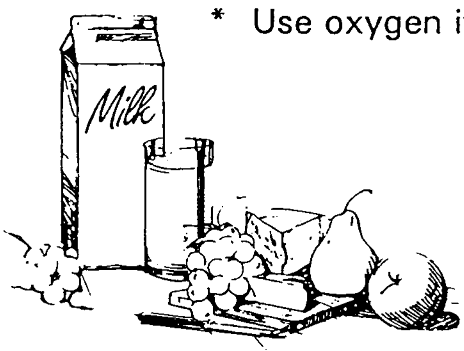
A BITE AT A TIME

NUTRITION AND COPD

EASY DOES IT!

-Make meal preparation and eating as easy as possible

* Choose foods that are easy to make (or have someone cook for you)
* Eat many small meals throughout the day (See **"Easy to Prepare Meal and Snack Ideas"**)
* Make double of what you need and freeze some
* Keep utensils and dishes where you can get them easily
* Rest before eating
* Relax at meal times -don't rush!
* Use oxygen if necessary, while eating



TO INCREASE THE NUTRITIONAL VALUE OF FOODS

* Swirl beaten eggs into soups while heating, or sprinkle grated cheese over soups before serving.

* Cook hot cereals in milk instead of water.

* Use 'fortified milk" instead of milk for making pudding, custard, cream soup , hot chocolate, etc.

  To make 'fortified milk' mix 1 cup of powdered milk with 1 litre of whole milk.

* Add butter or margarine to soups, hot cereals, or other hot entrées.

* Use cream instead of milk on cereals or in cream soups.

* Use spreads such as margarine, mayonnaise, cream cheese, and salad dressings and sauces and gravies liberally.

VITAMIN AND MINERAL SUPPLEMENTS

* A multiple vitamin and mineral supplement is recommended if your diet is more limited than usual.

* Large doses of individual vitamins and mineral are not recommended.

* Nutritional supplement drinks such as Ensure™, Ensure Plus™, are available in most drugstores and may be convenient alternatives if you find food preparation difficult.

Pulmocare™

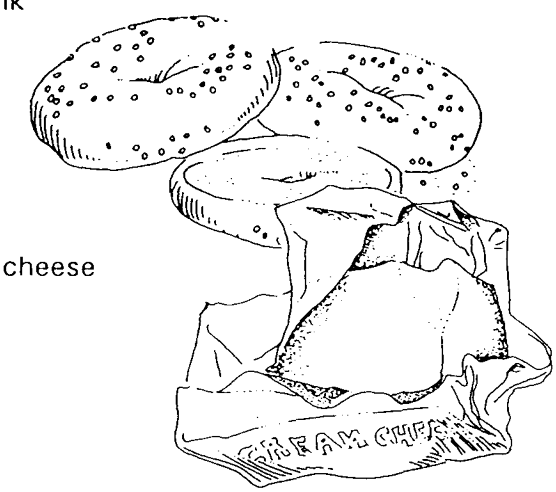
Pulmocare™ is a high-energy, low-carbohydrate drink that contains all of the essential nutrients. It has been specifically designed for people who have difficulty breathing. It is a good between meal snack to supplement your usual diet.

EASY TO PREPARE

MEAL AND SNACK IDEAS

MINI MEALS

* Instant hot cereal, topped with cream or milk
* Cold cereal with cream or milk
* French toast (frozen) with butter and syrup
* Poached, scrambled, boiled eggs
* Toast with peanut butter, cheese or cream cheese
* Melted cheese on a bagel or English muffin
* Hot chocolate or Ovaltine
* Carnation Instant Breakfast
* Croissant or muffin with cheese, cream cheese or peanut butter



FAST MEALS

* **Soups:**

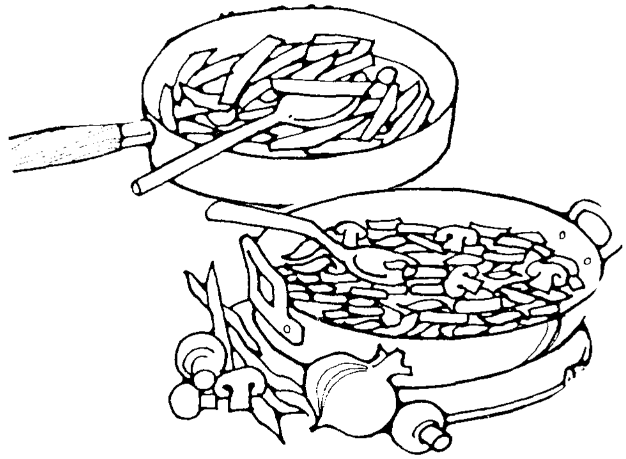
    Cream soup made with milk
    Hearty soups such as:

        pea or lentil
        chunky beef or chicken
        chowders

for extra flavour and calories sprinkle grated cheese (cheddar, parmesan) on top of hot soup

* **One-Dish Meals:**

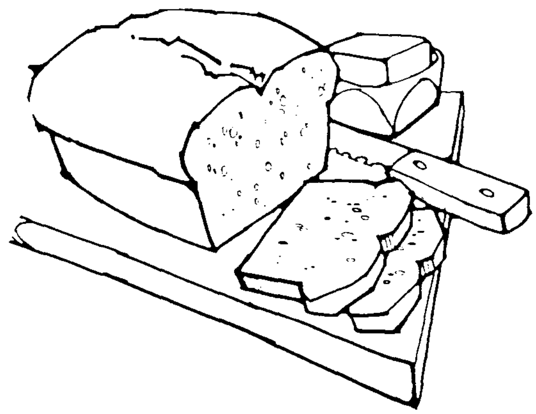
Frozen or homemade:



macaroni and cheese
shepherd's pie
stew
meat pies
lasagna
tuna noodle casserole
pyrogies/ sour cream
ravioli
chili

* **Sandwiches:**

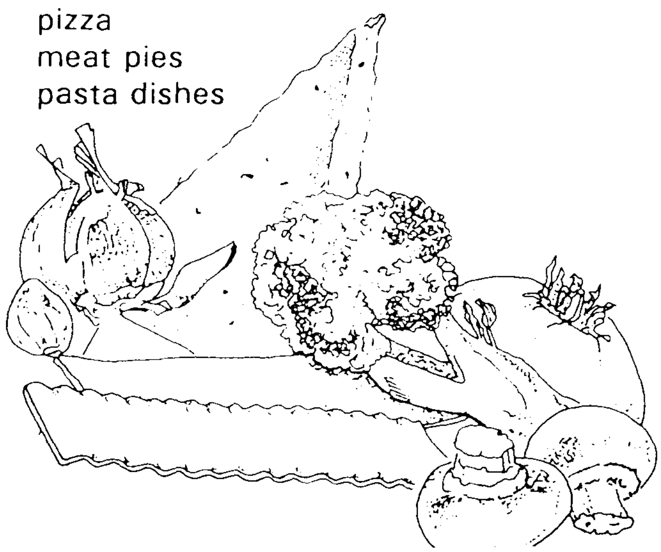
try these fillings on bread, toast or crackers:



tuna or salmon
sardines
hard boiled egg
sliced chicken, beef, turkey
peanut butter
cheese
liverwurst, pate

* **Delicatessen and take-out foods:**

BBQ chicken
quiche
cornish pasties
pizza
meat pies
pasta dishes



APPENDIX 9

MEAL PLAN AND SHOPPING LIST

UNIVERSITY HOSPITAL

EATING PLAN FOR ____________________

DIET ____________________

BREAKFAST	
LUNCH	
DINNER	

IETITIAN ______________________ PHONE NUMBER ____________

ATE ______________________

6/91

UNIVERSITY HOSPITAL

SHOPPING LIST FOR ____________________

DIET ____________________

MEAT/FISH/POULTRY	DAIRY	DELI
BAKESHOP	STAPLES	FRESH PRODUCE
FROZEN FOOD	CONVENIENCE FOODS	CANNED FOODS
BEVERAGES	OTHER	

DATE ____________________

APPENDIX 10

DAILY RECORD FORM

DAILY RECORD

ID:____

MONTH:________ DATE:________________

1 MEALS: Snacks: MEDS: ILL:	2	3	4	5	6	7
8	9	10	11	12	13	14
15	16	17	18	19	20	21
22	23	24	25	26	27	28

COMMENTS:

WEEK 1: WEIGHT________
ILLNESS________________________________
MEDICATION CHANGES________________________________

WEEK 2: WEIGHT________
ILLNESS________________________________
MEDICATION CHANGES________________________________

WEEK 3: WEIGHT________
ILLNESS________________________________
MEDICATION CHANGES________________________________

WEEK 4: WEIGHT________
ILLNESS________________________________
MEDICATION CHANGES________________________________

DAILY RECORD

ID:____

MONTH:________ DATE:________________

1 MEALS: SNACKS: PLMCRE: MEDS: ILL:	2	3	4	5	6	7
8	9	10	11	12	13	14
15	16	17	18	19	20	21
22	23	24	25	26	27	28

COMMENTS:

WEEK 1: WEIGHT________
ILLNESS____
MEDICATION CHANGES________________________________

WEEK 2: WEIGHT________
ILLNESS____
MEDICATION CHANGES________________________________

WEEK 3: WEIGHT________
ILLNESS____
MEDICATION CHANGES________________________________

WEEK 4: WEIGHT________
ILLNESS____
MEDICATION CHANGES________________________________

APPENDIX 11

EVALUATION OF NUTRITION EDUCATION

QUESTIONNAIRE

THE UNIVERSITY OF BRITISH COLUMBIA

School of Family and
Nutritional Sciences
Division of Human Nutrition
2205 East Mall
Vancouver, B.C. Canada V6T 1W5

EVALUATION OF NUTRITION EDUCATION

INSTRUCTIONS: Please read each question or statement carefully before responding.

1. Were you able to talk with a dietitian when you wanted to? (circle choice)

   Yes No

   If no, please explain:

2. Did you talk to a dietitian more than once? (circle choice)

   Yes No

   If yes, was the follow up contact useful? (circle choice)

   Yes No

   Please explain:

3. Why did you talk to a dietitian from UBC? (check any that apply)

   ______for advice about what to eat to help gain weight

   ______for reassurance that I was eating properly

   ______for general nutrition advice

   ______I am not sure

   ______someone recommended I see the dietitian

   ______other (comments):

4. Circle how much you agree or disagree with each statement:
   1 disagree strongly
   2 disagree
   3 neither agree nor disagree
   4 agree
   5 agree strongly

	disagree strongly				agree strongly
i. the dietitian provided useful information	1	2	3	4	5
ii. the dietitian knew what she was talking about	1	2	3	4	5
iii. the advice I received from the dietitian was suited to my special needs	1	2	3	4	5
iv. after talking to the dietitian I knew what to eat for my special needs	1	2	3	4	5
v. after speaking with the dietitian I changed my diet	1	2	3	4	5
vi. by talking with the dietitian I learned I did not need to change my diet as my intake was already suited to my needs	1	2	3	4	5
vii. After talking with the dietitian I could not change my diet	1	2	3	4*	5*

* If you circled 4 or 5, were any of these a problem?

_____knowing what to eat
_____getting to the store
_____finding the foods I needed in the store
_____the foods that I needed cost too much
_____preparing meals and snacks
_____eating?
_____other?

viii. after changing my diet my weight improved	1	2	3	4	5

		disagree strongly				agree strongly
ix.	after talking with the dietitian I felt better emotionally?	1	2	3	4	5
x.	after talking with the dietitian I felt better physically	1	2	3	4	5
xi.	after talking with the dietitian I felt in control of my condition	1	2	3	4	5 NA
xii.	the dietitian provided support and encouragement	1	2	3	4	5
xiii.	the dietitian cared about me	1	2	3	4	5
xiv.	anyone with my condition should talk with a dietitian	1	2	3	4	5
xv.	there was no benefit in talking with the dietitian	1	2	3	4	5

5. Have you any other comments about the contact you had with the dietitian?

Thank you.
Your answers will help us to provide better nutrition education services.

Janet Le Patourel
School of Family and Nutritional Sciences, UBC
822-2502

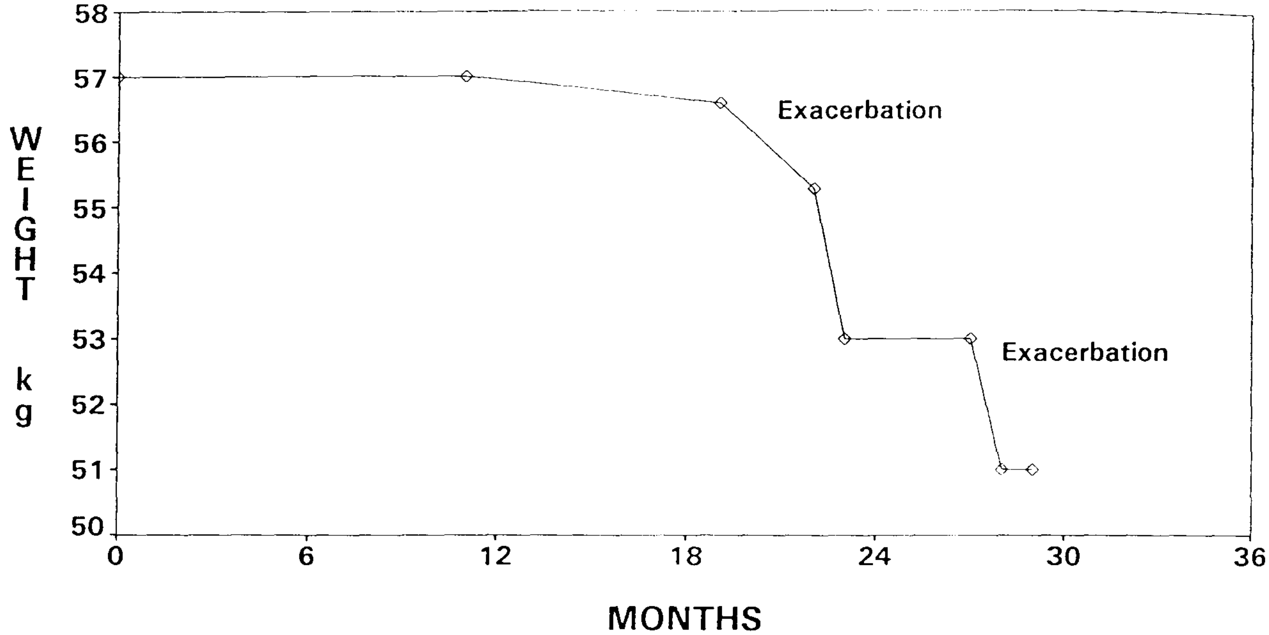
Please return your completed questionnaire in the enclosed stamped return envelope.

APPENDIX 12

TYPICAL PATTERNS OF WEIGHT CHANGE IN COPD PATIENTS

Typical Patterns of Weight Change In COPD Patients

Patient A



Patient B