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Assessing dietary change among participants in the “Diet and Health Study of Colon Adenomas”

McAuliffe, Colleen Anne, Ph.D.

The University of North Carolina at Greensboro, 1994

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ASSESSING DIETARY CHANGE AMONG PARTICIPANTS
IN THE "DIET AND HEALTH STUDY
OF COLON ADENOMAS"

by

Colleen A. McAuliffe

A Dissertation Submitted to
the Faculty of The Graduate School at
The University of North Carolina at Greensboro
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of the Requirements for the Degree
Doctor of Philosophy

Greensboro
1994

Approved by


Dissertation Adviser

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The specific aims of this research were to investigate if a computer generated nutrient analysis with personalized recommendations affected change in diet of post colonoscopy patients and to determine if people with colonic adenomas adhered to dietary recommendations and changed their diet more than people without colonic adenomas. Original dietary data collected from 120 subjects participating in a case control study were used as the baseline data. Follow-up dietary data, for the same subjects, were collected approximately three years later, and daily nutrient intakes were calculated using a quantitative food frequency. The specific dietary components of interest were fat, fiber, cholesterol, β -carotene and vitamins A, C and E. Nonparametric procedures were used to compare the subjects' initial intakes with their follow-up nutrient intakes.

As an intact group (N=120) statistically significant dietary changes were observed at follow-up for total calories, percent of calories from fat, and cholesterol intake. For each measure the change was positive. No statistically significant changes in intake for vitamins A, C and E, or fiber between baseline and follow-up were observed for the group. By case status, cases lowered caloric intake and cholesterol intake; controls lowered caloric intake, percent of calories from fat, and increased β -carotene intake. For both groups the changes observed were statistically significant. In a comparison of cases to controls, a statistically significant change was found for vitamin C intake. People identified to be at greater risk for colon carcinoma (cases) did not alter their diet and adopt

cancer risk reducing eating patterns any more than people identified to be at lower risk (controls).

Sixty-nine percent of the subjects remembered receiving the nutrient analysis with recommendations. Gender did not influence how long the recommendations were followed, but case status did. Controls adhered to the dietary recommendations for a longer time period than cases, and positive changes (lowered fat and cholesterol intake and increased β -carotene intake) were maintained over a three year period. The findings of this research suggest a brief dietary intervention, which included a personalized nutrient analysis with recommendations, positively affected dietary change among post colonoscopy patients.

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APPROVAL PAGE

This dissertation has been approved by the following committee of the
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CHAPTER I

INTRODUCTION

Colorectal cancer is the second leading cause of cancer deaths in the United States. An estimated 152,000 new cases of the disease will occur in the US in 1993 with 57,000 deaths (Boring, Squires, & Tong, 1993). Nearly one in twenty Americans will develop cancer in their lifetime, and between 1973 and 1986 this disease has increased 8.7% in whites and 22.7% in blacks (USDHHS 1989). During the past 50 years there has been only a modest decrease in the mortality from this disease.

Although early detection may offer an improved chance of survival, the benefits of colorectal screening have yet to be proven, and its actual worth remains uncertain (Knight, Fielding, & Battista, 1989; Selby & Friedman 1989; Neugut & Pita, 1989). Compounding this issue is the that belief the majority of Americans do not participate in screening programs offered. Currently only about 35% of colorectal cancers are discovered at an early stage (USDHHS 1989), and late stage surgical and chemotherapeutic treatments offer poor survival rates (Boring et al., 1993).

Marked geographic variability exists with colorectal cancer, and changes in incidence with migration suggest that this type of cancer has important environmental determinants. For many years diet has been identified as an important factor in the etiology and prevention of colorectal cancer, but the specific dietary nutrients or mechanisms responsible for the disease have not been clearly elucidated. Although the diet-cancer hypothesis is complex,

completed research has shown evidence of some nutrients acting in a protective manner and decreasing risk. Doll and Peto (1981) have estimated that 90% of large-bowel cancer deaths might be accounted for by diet. Diet is one environmental determinant which should be targeted to decrease risk. Given the magnitude of this disease and its potential preventability, it is imperative to study dietary intervention strategies. In the report Healthy People 2000, the United States Department of Health and Human Services (USDHHS) indicates that its ultimate goal is to increase the proportion of Americans who live long and healthy lives (USDHHS 1991). To reach this goal one of its objectives is a reduction of colorectal cancer deaths to no more than 13.2 per 100,000 people by year 2000 (USDHHS 1991). To meet this objective effective intervention must begin now.

Colorectal adenomatous polyps are neoplastic lesions of the large-bowel that are generally considered to be precursor lesions for the majority of colorectal carcinoma cases (Hill, Morsen, & Bussey 1978; Day 1984; Stryker, Wolff, Culp, Libbe, Ilstrup, & MacCarty 1987). Presently there are no specific dietary guidelines given to people identified at risk for colorectal cancer by diagnosis of adenomatous polyps. Use of a computer generated quantitative food frequency nutrient analysis offers a rapid and economical method to assess the diet of individuals at risk and provide personalized dietary recommendations for improvement to decrease risk. However, its use for affecting change in diet among individuals at risk for colon adenomas has never been evaluated.

Sufficient scientific research has been conducted to conclude that diet is closely related to the development of adenomatous polyps and colorectal cancer. We cannot wait for a cause and effect relationship to be established

simply because it will not happen anytime in the near future. Clinical trials are expensive, require years for completion, and are difficult to conduct with asymptomatic subjects in a free living population. If the computer generated nutrient analysis and recommendations for change has a positive affect on dietary habits, its use among people at risk for colorectal adenomas could be expanded. Ideally, such a nutrient analysis could be used as the first level of intervention for all individuals diagnosed with adenomatous polyps. Given the evidence there is no cure for colorectal cancer and survival rates are poor for both genders, identifying dietary intervention methods which are cost effective for health care providers and beneficial to recipients is essential if we are to move toward preventing colorectal carcinoma.

One purpose of the study was to determine if a computer generated nutrient analysis, with personalized recommendations for dietary improvement, affects change in diet in post colonoscopy patients. A second aim of the study was to determine if people with colonic adenomas adhered to the recommendations and changed their diet more than people without colonic adenomas.

CHAPTER II

REVIEW OF LITERATURE

Throughout the past two decades, epidemiologic studies have investigated the role of environmental factors on the incidence and mortality of colorectal carcinoma, while continuing population studies have identified lifestyle as a critical area to investigate. Dietary factors have been identified as key variables in the etiology of this chronic disease because of the direct exposure of the colon to digestive residue of food. The incidence of colorectal cancer is high in North America, Northern and Western Europe and New Zealand, while low in South America, Africa and Asia (Morsen, 1976). The disease is most prevalent in industrially developed nations. The United States has one of the highest rates of colorectal cancer in the world (USDHHS, 1989).

The study of colon cancer in migrant populations reveals international differences cannot be ascribed to variation in genetic background but must be ascribed to environmental influence (Correa & Haenszel, 1987). This is clearly illustrated by the differences in incidence seen between blacks in America and blacks in Africa (Haenszel & Dawson, 1965); between Puerto Ricans in their homeland and those who have migrated to the mainland (Martinez, Torres, & Frias, 1975); and between first and second generation Japanese immigrants to Hawaii and the mainland United States and the Japanese who have remained in Japan (Haenszel & Kurihara, 1968). In the United States the incidence of colon cancer has been greater in the north than south, with mortality rates in the

northeast exceeding the south by about 50% (Ziegler, Blot, Hower, William, & Fraumeni, 1981). The incidence of colon cancer is also greater in urban areas than in rural areas, and greater among whites than blacks (USDHHS, 1989). Based on these facts one would conclude incidence varies according to location rather than population, thus supporting the hypothesis that environment plays a critical role in the development of this disease.

The concept of diet being the most influential environmental determinant has attracted much interest throughout the nation. The possible relationship between dietary factors and colorectal cancer has been investigated in correlational, case-control, and retrospective cohort studies. Numerous studies have focused upon nutritional habits using colorectal cancer as the endpoint. Retrospective research has scrutinized alcohol consumption (Potter, McMichael & Hartshorne, 1982; Kabat, Housen, & Wynder, 1986; Klatsky, Armstrong, Freidman, & Hiatt, 1988), dietary fiber (Burkitt, 1980; Burkitt, Walker, & Painter, 1974) and fat intake (Reddy, 1986; Enstrom, 1981; Lyon & Mahoney 1988).

Of all the dietary components studied, the combined epidemiologic and experimental (animal) evidence is most suggestive for a causal relationship between high fat intake and increased occurrence of colorectal cancer. In the early seventies Wynder and Reddy (1973) proposed that colorectal carcinoma was closely associated with total dietary fat intake. Work conducted by Carroll and Khor (1975) established a worldwide correlation between colorectal cancer incidence and total fat consumption. In examining the relationship between dietary fat and colorectal cancer, two hypotheses have emerged. One hypothesis suggests that the amount of dietary fat determines both the concentration of acid and neutral sterol substrates in the large bowel along with

the composition of microflora acting on each substrate. The second hypothesis suggests that gut microflora metabolize acid and neutral sterols to active carcinogens in the large bowel (Aries, Crowther, Draser, Hill & Williams, 1969; Hill, 1975; Bansal, Rhoads & Bansal, 1978; Breuer & Goebell, 1987). There is the possibility that a high fat diet changes the composition of bile acids as well as modifies the activity of gut microflora which may in turn produce tumor promoting substances rather than act as complete independent carcinogens (Reddy, Watanabe & Weisburger, 1977). Presently, the qualitative and quantitative aspects of dietary fat remain under investigation in an attempt to determine if dietary fat acts in a uni or multifactoral manner in the pathogenesis of the colon. Today, much of the research conducted, however, has shifted from colorectal carcinoma as an endpoint to the equally logical and perhaps more attractive endpoint of colonic adenomatous polyps.

Studies based on interview data collected from cancer patients with symptoms from their cancer are inevitably prone to bias (Graham, 1980). Colorectal adenomatous polyps, which are believed to be asymptomatic, antedate the clinical diagnosis of colorectal cancer by an average of approximately 10 years (Lev, 1990). A measure of diet close to the time of polyp removal should provide useful information into the relationship of dietary factors with changes in normal colon mucosa to malignancy (Giovannucci, Stampfer, Colditz, Rimm, & Willett 1992). Case control studies designed to investigate diet and risk of adenomas are attractive because dietary information can be collected at time of endoscopy and allows for comparison of diet between asymptomatic cases and controls.

Adenoma-Carcinoma Sequence

Colorectal adenomatous polyps are proliferations of the intestinal epithelial cells. These polyps can be divided into tubular, villous, and intermediate tubulovillous types based on their microscopic appearances. All three types represent variants of the same neoplastic process and share common cytological features (Morsen, 1974; Hill et al., 1978). Both histologic and epidemiologic evidence supports the concept that these benign neoplastic lesions are regarded as precursor lesions for most cases of colorectal carcinoma. Reference to this adenoma-carcinoma sequence dates back to 1951 (Jackman & Mayo, 1951).

In a simplified presentation the adenoma-carcinoma sequence is a series of events in which genetic and environmental factors act to induce the development of a hyperproliferative state in the cells of the large bowel. These cells then develop into an adenoma, grow, and progress to carcinoma (Hill, 1991). Estimates of the prevalence of polyps come from autopsy, screening, and surveillance studies. The prevalence of adenomatous polyps ranges from 35% to 60% in the United States (Arminski & McLean 1964; Offerhaus, Giardiello, Tersmette, Mulder, Tersmette, Moore, & Hamilton, 1991). Given the concept adenomas are regarded as precursors of colorectal carcinoma, current medical recommendations are for early removal followed-up by periodic surveillance with colonoscopy at 12 and 48 months after initial diagnosis. The economic burden of this approach is substantial but usually justified given the high prevalence of colorectal cancer. The frequencies of both colonic adenomas and cancer increase with increasing age (Clark, Collan, Eide, Esteve, Ewen, Gibbs, Jensen, Koskels, MacLennan, Simpson, Stalaberg, & Zaridze, 1985), with

the highest increase in the prevalence of adenomas in the age group 50-59 (Arminski & McLean, 1964). With more and more Americans living longer, the urgency to prevent adenomas in hopes of preventing colorectal carcinoma is of national concern.

Diet and Risk of Colorectal Adenomas

The adenoma-carcinoma sequence, accepted as the natural progression for most cases of colorectal cancer (Morson, 1974), has prompted the launching of several case control studies designed specifically to investigate diet and risk of adenomas. During the past five years results from both prospective and retrospective case control studies have suggested diets high in red meat, saturated fat and alcohol and low in fruits and vegetables may increase the risk for adenomas. The protective effect of fruits and vegetables is strong, however the exact mechanisms remain unknown. Fiber intake may be protective (McKeown-Eyssen, & Bright-See, 1984) depending upon the source of the fiber.

Hoff, Moen, Trygg, Frolich, Sausar, Vatn, Gjone, and Larsen (1986) reported high consumption of fat and low consumption of fiber and cruciferous vegetables lead to increased neoplastic changes in the large bowel of study participants. Work completed by Macquart-Movlin, Riboli, Corree, Kaaks, and Berthezene (1987) revealed cases had a lower consumption of carbohydrates, potassium, magnesium and vitamin B-6 coupled with a slightly higher intake of saturated fat. In a retrospective case control study, Sandler, Lyles, Peipins, McAuliffe, Woosley, and Kupper (1993b) showed carbohydrate intake was inversely related to adenoma risk in women. Compared with women in the lowest quintile of carbohydrate consumption, those in the highest quintile were

60% less likely to develop adenomas; odds ratio (OR) = 0.39, 95% confidence interval (CI) = 0.19-0.80). Intake of fruit and intake of fiber derived from vegetables and fruits were also inversely related to adenomas in women. Total fat intake showed a positive association in women, with an OR of 2.69 for the highest versus the lowest quintile. Results were comparable for saturated fat. The risks for men were generally similar in direction and magnitude but were not statistically significant. These data (Sandler, et al., 1993b) support the hypothesis that a diet high in fat and low in carbohydrates, fruits, and fruit and vegetable fiber increases risk not only for colorectal cancer but also for precursor colorectal adenomas. In a separate analysis of the same study data, Sandler, Lyles, McAuliffe, Woosley and Kupper (1993a) found alcohol to be a significant risk factor for men but not for women. Men in the highest quartile of daily caloric intake from alcohol were more than four times likely than nondrinkers to develop adenomas, with a statistically significant trend in risk from the lowest to the highest quartile. These findings persisted after controlling for other potential risk factors for adenomas. Men in the highest tertile of beer consumption were nearly six times more likely to develop adenomas than nondrinkers. Neugut, Garbowski, Lee, Murray, Neives, Forde, Treat, Wayne, and Fenoglio-Preiser (1993), reported protective associations were observed for fish and chicken and vitamin A intake for women while elevated OR for women were found for saturated fat.

Giovannucci et al.(1992) showed that saturated fat (after adjusting for total energy intake) was positively associated with risk of colorectal adenomas in men. Relative risk (RR) for the highest versus the lowest quintile of intake was 2.0 (95% CI= 1.2-3.2). Dietary fiber was inversely associated with risk of

adenomas, and the RR for men in the highest versus lowest quintile was 0.36 (95% CI = 0.22-0.60). All sources of fiber (vegetables, fruits, grains) were associated with decreased risk of adenomas. These data provide evidence for the hypothesis that a diet high in saturated fat and low in fiber increases risk of colorectal adenomas.

Giovannucci, Stampfer, Colditz, Rimm, Tichopoulos, Rosner, Speizer, and Willett (1993), studying if dietary factors influencing methyl availability were related to colorectal adenomas, found that dietary folate was inversely associated with risk of colorectal adenomas in women. Reduced methylation of DNA may contribute to loss of normal controls on protooncogene expression. In humans hypomethylation of DNA has been observed in colorectal cancers and their adenomatous polyp precursors (Giovannucci et al., 1993). The apparent protective effect of fresh fruits and vegetables, major folate sources in the diet, on colorectal cancer incidence suggests that a methyl-deficient diet contributes to occurrence of this malignant disease.

Trock, Lanza, and Greenwald (1990) showed 31 out of 43 reviewed epidemiological studies had strong or moderate support for decreasing risk of colorectal cancer in people with high fiber low fat diets. In another combined analysis (Howe, Benito, Castelleto, Cornee, Esteve, Gallagher, Isovich, Dengao, Kaaks, Kure, L'Abbe, Lee, Lee, Miller, Peters, Potter, Riboli, Slattery, Trichopoulos, Tuyns, Tzonou, Whittemore, Wu-Williams, & Shu, 1992) of data from 13 case control studies, results show risk decreased as fiber intake increased. The inverse association was seen in 12 of the 13 studies. Inverse associations, although weak, were also seen for increased intake of vitamin C

and beta-carotene. This analysis provides substantive evidence that intake of fiber rich foods is inversely related to risk of colorectal cancer in both genders.

Nutrition and Cancer Prevention

The National Cancer Institute (NCI) has supported research in diet and chemoprevention to help attain its goal of a 50% reduction in cancer mortality by the year 2000 (USDHHS, 1986). Most of the data available on prevention is derived from animal or in vitro studies. Animal studies indicate that of all the micronutrients investigated in cancer prevention, vitamins A, C, and E, and calcium show the most promise. Data on cancer prevention in humans is limited and remains inconclusive, but both macronutrients and micronutrients are presently targeted in many clinical trials.

Micronutrients may inhibit cancer initiation or promotion. During the initiation and promotion stages, micronutrients can inhibit protooncogenes by acting as antioxidants, by inhibiting the promotion of carcinogenic agents, and by inhibiting the formation of carcinogenic substances before initiation can occur. Protooncogenes are activated by cancer initiating agents such as tannic acid, nitrosamines, and free radicals such as those generated during oxidation in the metabolism of cells, particularly during the peroxidation of polyunsaturated fatty acids (Horwitt, 1986). Activated protooncogenes become oncogenes which produce increased amounts of key proteins that control proliferation and differentiation of normal cells (Prasad, 1984). Preventive substances, like micronutrients, can also act by inhibiting the formation or the effectiveness of carcinogenic agents before the initiation stage begins. In small amounts initiating agents are not usually carcinogenic, but can become carcinogenic

when accompanied by promoter agents. Agents that promote tumors encourage transformation of cells after initiation has already occurred. Promoting agents include caffeine, saccharine, phenobarbital, and excess fat (Prasad, 1984). Colon cancer is believed to be inhibited by decreasing the activity of gut microflora which metabolizes tumorogenic compounds and increases mutagens in the feces (Reddy, 1986). Substances that reduce the amount of fecal mutagens can be deterrents to colon cancer.

A possible role of vitamin C in prevention or reduction of fecal mutagens in stool has been observed in two studies (Dion, Bright-See, Smith, & Bruce, 1982; Schiffman, 1987). In a study of diet and fecapentaene levels, Shiffiman (1987) found a strong protective effect for both dietary and supplemental vitamin C intake. Consumptions of citrus fruit and supplemental vitamin E were also significantly negatively associated with fecapentaene levels. Thus, if fecal mutagens are truly associated with colon carcinogenesis, vitamin C may play an independent role or may act synergistically with vitamin E in blocking their effects. Vitamins C and E have powerful antioxidant properties (Machlin & Bendich, 1987), and vitamin C indirectly participates in a number of key oxidative and reductive enzyme systems. An adequate supply of vitamin C, therefore, ensures the proper functioning of many life supporting biochemical systems. Vitamins C and E have both been identified as having a major role in counteracting and controlling oxygen toxicity, and vitamin E, especially together with vitamin C is an excellent nitrate trapping agent (Mirvish, 1986; Chen, Biossonneault, & Glavert, 1988). Some animal experiments and human studies suggest vitamin E may protect against cancer, however, most human studies do not generally support the hypothesis of its protective effect (Knekt, 1991). Few

epidemiologic studies have investigated the association of dietary vitamin E and cancer. The studies that have investigated the association of dietary vitamin E with cancer have been of case control or cohort design and have been mostly negative. A greater number of studies have investigated the association of serum alpha-tocopherol with cancer. Results of investigations examining prediagnostic serum vitamin E and cancer have been inconsistent. No association of serum or plasma alpha-tocopherol and the development of cancer at all sites was found in three cohort studies (Gey, Brubacher, & Stahelin, 1982; Willett, Polk, Underwood, Stampfer, Pressel, Rosner, Taylor, Schneider, & Hames, 1984; Wald, Thompson, Densem, Boreham, & Bailey, 1988). Low levels of serum alpha-tocopherol were associated with increased risk of cancer at all sites in three other cohort studies (Kok, Van Duijn, Hifmann, Vemeeren, Bruijn, & Valkenburg, 1987; Kenkt, 1988; Kenkt, Aromaa, Maatela, Aaran, Nikkari, Hakama, Hakulinen, Petro, Saxen, & Teppo, 1988). Presently, sufficient scientific evidence does not exist to support the hypothesis that vitamin E may be protective against cancer.

Vitamin A occurs in the diet in both precursor and preformed forms. The provitamins, derived from plant sources, are primarily carotenes, with the most important being β -carotene. The preformed vitamins, retinoids or retinol, are derived from animal sources. Carotenoids and retinoids have separate functions. Carotenoids block initiators of cancer, and β -carotene is a significant antioxidant which deactivates carcinogenic free radicals and prevents them from transforming cells (Prasad & Rama, 1985). Retinoids act as antipromoters of cancer by decreasing the overproduction of m-RNA's specific to certain oncogenes, thereby discouraging rapid cell growth (Watson & Leonard, 1986). A

report by the National Research Council (1982) suggested an inverse relationship between serum levels of vitamin A and subsequent risk of cancer in general. Vitamin A plays a major role in the maintenance of epithelial tissues by inhibiting the transition of cells to metaplasia and inducing malignant cells to differentiate back to normal (Watson & Leonard, 1986). Of all the evidence for an inverse relationship between cancer and the antioxidant micronutrients, vitamins C and E and carotenoids (Block, 1991; Zeigler, 1989), the strongest for anticarcinogen effect exists for beta-carotene (Pryor, 1991).

β -carotene is unique because it is the most abundant of the carotenoids that humans can metabolize to vitamin A and it appears to function through a mechanism that does not require conversion to vitamin A. Low levels of β -carotene are consistently associated with the subsequent development of lung cancer (Zeigler, 1991). In direct contrast, results of case control studies on the relation of carotenoids and colorectal cancer risk are inconsistent. Three cohort studies (Shekelle, Lepper, Lius, Maliza, Ragnor, & Rossof, 1981; Pahanini-Hill, Chao, Ross, & Henderson, 1987; Heilbrun, Momura, Hankin, & Stemmermann, 1989) failed to detect an association between dietary carotenoids and cancer of the colon or rectum. Kune, Kune, and Watson (1987) present data that suggest an inverse relationship between dietary carotenoids and cancer of the colon. The role of serum carotenoids in the prevention of colorectal cancer is weakly supported by prospective serum studies. Lower levels of β -carotene in individuals who subsequently developed colon cancer were found in four out of five studies of prediagnostic serum, but, the association was not statistically significant (Nomura, Stemmermann, Heilbrun, Salkeld, & Vuillemier, 1985; Schober, Comstock, Helsing, Salkeld, Morris, Rider, & Brookmeyer, 1987; Wald,

Thompson, Densem, Boreham, & Bailey, 1987; Connett, Kuller, Kjelsberg, Polk, Collins, Rider, & Hulley, 1989; Comstock, Helzlsouer, & Bush, 1991). For now it is unclear whether β -carotene or another component of fruits and vegetables is protective against cancer at any site. Since carotenes are the major precursor forms of vitamin A, occurring naturally in fruits and vegetables, this provitamin must be considered in the context of accounting to some degree, for the protective effect of fruits and vegetables. Further investigations of β -carotene are needed, particularly to examine strength of association and range of effective exposure at different cancer sites (Zeigler, 1991). The importance of other carotenoids and other constituents of fruits and vegetables must also be examined.

Evidence suggests dietary calcium may prevent colorectal cancer by forming calcium soaps to neutralize the effect of bowel irritating bile acids and fatty acids (Lipkin & Newmark, 1985; Vogel & McPherson, 1989). In a study designed to investigate calcium as a mitigating factor in colon cancer, Slattery, Sorrenson, and Ford (1988) observed a protective effect for both genders. Presently, NCI is funding a randomized, double blind, placebo-controlled multicenter clinical trial testing the efficacy of dietary calcium supplementation in preventing recurrence of adenomatous polyps of the large bowel in persons at high risk for this condition.

Research has also suggested dietary fiber may help prevent colon carcinoma, and there are several mechanisms by which fiber can exert a beneficial effect. Fiber may act by diluting concentrations of potentially carcinogenic agents or promoters in the bowel (Greenwald, Lanza, & Eddy, 1987); by decreasing colonic transit time thereby reducing the opportunity for

bacterial production of carcinogenesis (Burkitt et al., 1974; Reddy, Hedges, Laasko, & Wynder, 1978; Zaridze, 1983), or by lowering bowel pH which lowers colonic enzyme activity (Kritchevsky, 1986). The fiber hypothesis is attractive and has great heuristic value, yet scientific evidence remains elusive. A major problem exists with accurately measuring and comparing fiber types in foods consumed (Marlett, 1990). International studies support an inverse relationship between total fiber intake and colon cancer in slightly over one-half of the published studies (Vogel & McPherson, 1989). Nutritional and biochemical variability of the fiber sources consumed by populations limit the usefulness of these data. Results of case control studies are inconsistent. When fiber has been found to be protective, it has generally been of fruit and vegetable origin rather than from cereals (Vogel & McPherson, 1989; Potter, 1990). Since other factors in fruits and vegetables have been implicated, most notably, β -carotene and vitamin C, the current evidence for a protective effect of fiber in colon cancer is inconclusive (Ausman, 1993).

Research designed to examine the relationship between cancer and diet is a challenge. The difficulty in demonstrating a stronger association between colorectal cancer and specific nutrients may be attributed to several factors (Rogers & Longnecker, 1988; Willett, 1990). Dietary exposures are unique in that they cannot be classified as absent or present. A diet is also a complex mixture of interrelated exposures, and diets are relatively homogeneous among individuals within a population. Measures of diet are also inherently error prone. Determining a mechanistic association between nutrients or foods and cancer will remain difficult because of the complex chemical composition of foods. Nutrients that both increase and decrease risk may coexist in the same food.

Evidence for this intra-food interaction is illustrated by the differences in risk estimates found for dairy products, which vary in fat, protein, and calcium content (Phillips, 1975).

Macronutrients, numerous micronutrients and fiber continue to be investigated as potential colon carcinogenesis chemopreventive agents. To date no study supports the use of any single dietary component or synthetic agents for the prevention of colon cancer. This is not surprising given that the etiology of colon cancer is considered multifactorial. It is highly probable that an individual's dietary pattern or the mix of nutrients and foods consumed is what affects cancer risk, rather than a single nutrient or food.

Despite the lack of concrete scientific evidence linking dietary factors to cancer prevention, there is the need to consider evidence which will allow sound advice on reducing risk at the earliest possible time (Greenwald et al., 1987). Based on current knowledge, the NCI believes prudent dietary guidelines that will promote good health and reduce the risk of cancer are warranted.

Dietary Guidance

Dietary guidance is not a new activity, since nutritionists have been advising Americans about their dietary practices for over a century (Abel, 1890; Atwater, 1902). The USDA published its first dietary plan in 1916 (Hunt, 1916). Between 1917 and the mid 1940's, food guides were published by both public and private organizations (National Dairy Council, 1941; American Meat Institute; 1942; USDA, 1943; 1946). Although differing in presentation all food guides stressed a common theme that Americans should strive to eat sensibly and balance the proportion of nutrient dense foods with foods of less nutrient

density. Since the 1940's, the evolution of dietary guidance has corresponded to the growth and depth in our ability to understand the relationship between health and disease. The role of diet in the prevention of chronic diseases such as diabetes, cardiovascular disease and cancer has escalated with cancer prevention receiving the most attention during the last decade.

The National Cancer Act Amendments (1974) commissioned the National Cancer Program to explore the role of nutrition in the treatment, rehabilitation, and causation of cancer. The amendments required NCI to provide for a program to disseminate and interpret information relevant to the cause, prevention and treatment of cancer. To meet this mandate, in 1975, the NCI established the Diet, Nutrition and Cancer Program. The first dietary guidelines designed specifically for cancer prevention were issued by the NCI in 1979 (Upton, 1979). The guidelines were general and had little impact on the American people.

In the early eighties the National Research Council (NRC) conducted a comprehensive review of existing evidence linking dietary factors to cancer risk. Based on the results of this review NRC published interim dietary guidelines (NRC, 1982). Thereafter the NCI (1984; 1987) American Cancer Society (ACS) (1984) and the American Institute for Cancer Research (1990) published similar dietary recommendations to lower the risk of cancer. These guidelines are consistent with general dietary recommendations proposed by US government agencies (USDA & USDHHS, 1990). All agencies repeated the same basic message advising Americans to consume more dietary constituents associated with protection against cancer (grains, fruits, vegetables) and less foods associated with increased risk (calories, fat, alcohol). Increased consumption of

recommended foods might help decrease consumption of undesirable foods and lower risk. In 1988 NCI issued its current dietary guidelines for decreasing cancer risk. The NCI guidelines specifically recommend that Americans reduce fat intake to less than 30% of total calories, increase fiber intake to 20-30 g/d with an upper limit of 35 grams, include a variety of fruits and vegetables in the daily diet, avoid obesity, consume alcoholic beverages in moderation if at all, and minimize consumption of salt-cured, salt-pickled or smoked foods (Butrum, Clifford, & Lazza, 1988). The guidelines published by NCI, NRC and ACS all provide Americans with sound nutritional advice based on scientific evidence worthy of recognition. Health officials agree the main challenge today is no longer to determine what eating patterns to recommend to Americans but rather how to implement these recommendations (Institute of Medicine, 1991). Results from national food consumption surveys indicate the shift in attention is long overdue.

The Second National Health and Examination Survey (NHANES II) examined a representative sample of approximately 12,000 US adults (National Center for Health Statistics, 1981) using a detailed 24-hour recall. Although NHANESII data were collected prior to the issuance of the NCI, ASC and NCR guidelines and do not address the impact of these guidelines, analyses did provide evidence that very few Americans even approach levels of recommended intakes. Data indicated 17% of the population ate no vegetables at all on the day of the survey, not even potatoes; 41% of the population had no fruit on the survey day and only one fourth had a fruit or vegetable rich in vitamin A or C. Only 10% consumed the recommended five servings daily (Block, 1991). In a another national survey (USDA, 1988) completed six years after the

NCI dietary guidelines were issued, only about half of the participants had even one serving of a citrus fruit or juice in 4 days, and only one third had a dark green or yellow vegetable once in 4 days. Only 9% of Americans consumed five or more servings of fruit and vegetables per day. Results from another national survey (Patterson, Block, Rosenberger, Pee, & Kahle, 1990) showed that on any given day, only 60% of Americans had even one serving of fruit or juice.

Randall, Marshall, Brasure, and Graham (1991) designed a case control study specifically to examine compliance with NCI dietary guidelines, using multidimensional measures of diet and identifying seven gender specific patterns. Results of this study showed that no one pattern signified compliance with all guidelines. High fat patterns in both genders and the low-cost pattern in women were the best indicators of poor compliance with the guidelines. At all levels of each pattern, subjects failed to achieve the goal of obtaining no more than 30% of calories from fat. The data suggest that few subjects complied with the recommendations in the NCI guidelines to consume no more than 30% of calories from fat. Among males, subjects in the uppermost tertile of the high fat patterns had mean fat intakes of 40% of calories. For women subjects, a high of 36.5% of calories from fat was observed.

The task of designing effective strategies for implementing dietary guidelines has proven to be a more difficult challenge than their establishment. Health officials have agreed the task will require a shared responsibility among all Americans (USDHHS, 1991). The Surgeon General's Report on Nutrition and Health (USDHHS, 1988) was developed as an authoritative source of information on which to base nutrition policy decisions. This report presents a comprehensive review of the evidence linking diet to chronic disease, and its

consensus on the scientific basis of diet-disease relationships established a foundation from which to develop policies and programs to implement dietary recommendations. The report concludes that the federal role in implementation is necessary but not sufficient. Commitment from state, local, private and voluntary sections are essential for promoting improved food choices by Americans.

The report identifies implications for public health policy regarding nutrition practices. These include 1) dietary guidance; 2) improved education of the public about dietary choices most conducive to good health, especially for groups at greatest risk; 3) incorporation of nutrition services into health care programs for all Americans; 4) improved monitoring of nutritional status, especially among high risk groups, and 5) identification of effective educational methods to help the public translate dietary recommendations into appropriate food choices (USDHHS, 1988). Objectives were developed with the assumption that there is no longer any doubt about the importance of diet to the health of Americans. It is true chronic disease results from multiple causes, only some of which are dietary. However, because the magnitude of chronic disease is so great, even a small decrease in risk as a result of dietary change should be expected to produce substantial improvements in the overall health of Americans (McGinnis & Nestle, 1989).

Implementation should begin with getting accurate practical information about dietary recommendations to people in formats that are relevant and comprehensible to them (Institute of Medicine, 1991). The information distributed must identify the components of a healthy diet and link such a diet with a decreased risk for chronic disease. Helping people alter their food

choices in a more healthful direction will require both individual and public responsibility. The individual, family, community, private industry, government and health care professionals must become involved. The individual is both starting point and the ultimate target of a movement towards improving dietary practices (USDHHS, 1991). The Institute of Medicine (1991) believes successful implementation of dietary recommendation can be achieved by 1) enhancing awareness, understanding and acceptance of dietary recommendations; 2) creating legislative, commercial and educational environments supportive of the recommendations, and 3) improving the availability of foods and meals that facilitate the implementation of recommendations. One tactic for increasing the prevalence of healthful eating patterns in Americans will require altering nutrition education. This can be accomplished by presenting consistent messages in education programs and by broadening exposure to formal and informal nutrition education (Institute of Medicine, 1991).

The responsibility of helping Americans adhere to dietary recommendations falls heavily on health care professionals whose training provide them with the skills to translate science into practice (USDHHS, 1991). Today more than ever people are looking to nutritionists, dietitians, nurses, physicians, and health educators to provide clear useful information on the relationship of risk and diseases as well as practical guidelines for making food selections in a manner that adheres to dietary recommendations (Institute of Medicine, 1991). Health care professionals need to integrate nutrition education for all clients and patients at the time of initial patient contact as well as at follow-up medical visits (Institute of Medicine, 1991). Nutritional

assessment and education activities should be an integral part of primary care prevention services (Block, 1993). As specialists in human nutrition and food science, the challenge of changing dietary practices to reduce risk of chronic disease lies primarily on nutritionists and dietitians. The need to create avenues for providing effective practical nutrition education for individuals identified to be at risk should be a priority. Nutritionists will have to take their places as leaders in the discipline of nutrition if this major health care effort is to be successful (Bronner, 1991).

There have been few studies of nutrition education interventions designed to produce qualitative dietary changes, that being changes in type of foods consumed daily. Face to face communication combined with supplementary printed materials has been studied in adults who have been identified to be at risk for the development of chronic disease because of lifestyle or the presence of other risk factors (Meyers & Henderson, 1974; MRFIT Research Group, 1982). Studies (Arntzenius, Kromhant, Barth, Reiber, Brusckke, Buis, van Gent, Kempen-Voogd, Strikwerda, & van der Velde, 1985; Bruno, Arnold, Jacobson, Winick, & Wynder, 1983; Carmody, Istvan, Matarazzo, Connor, & Connor, 1986) evaluating programs providing information needed to change knowledge, instruction in methods of monitoring change and guidance on how to achieve gradual incremental change in dietary habits show positive results among people at risk. Perhaps people who are aware of their risk for chronic disease are more motivated to adhere to dietary recommendations to decrease risk.

Nutrition education consists of three phases; namely, assessment, intervention, and evaluation or monitoring (Glanz, 1985). In the past formal nutrition education was seldom provided to patients except during treatment

requiring hospitalization and then it was often limited to brief bedside instruction prior to discharge. Today nutrition education can occur in a wide variety of settings which allows nutrition education to be targeted at individuals, identified to be at risk, who want to voluntarily alter their dietary practices in hopes of decreasing risk. To meet this need the development, implementation and evaluation of rapid, easy, economical and effective methods is warranted.

The ultimate goal of nutrition education is a permanent change in eating patterns to reduce risk for disease, and an evaluation of the success of the goal should be measured in terms of patient change in dietary intake over time (Edwards, Mullins, & Clarke, 1986; Talmage, Hughes, & Eash, 1978). Quick methods of intervention are needed because long term interventions are not feasible when attempting to change the dietary habits of large populations. Public involvement in such programs dwindle over time and the expense and human resources needed to implement such programs are substantial. Considering the current national health care crisis conducting such programs is prohibitive. Yet having nutrition services as part of preventive health care cannot be ignored. Estimates by the US Senate (Select Committee on Nutrition and Human Needs, 1977) indicate that an average 20 per cent reduction in incidence, prevalence and costs in most chronic disease categories could be seen by improving American dietary practices.

The translation of the NCI guidelines into food recommendations is attractive and should be the message content of any nutrition intervention strategy. Eating pattern messages should emphasize eating less high fat foods, choosing low fat dairy products, and consuming more whole grains, starches, legumes, fruits, and vegetables (Sorenson, Hunt, Morris, Donnelly, Freeman,

Ratcliff, Hsiesh, Larson, & Ockere, 1990; Potter, Graves, Finnegan, Mullis, Baxter, Crockett, Elmer, Gloeb, Hall, Hertog, Pirie, Richardson, Rooney, Slavin, Synder, Splett, & Vinsnawath, 1990).

Computer Assisted Instruction

Increased interest in health promotion and disease prevention has created new opportunities to explore creative approaches for the assessment of American dietary habits. Advances in computer technology have led to an expanded use of computerized nutrient analysis programs in health care settings. Due to time constraints and major budget cuts in health care, nutritionists and health educators are making use of programs that analyze dietary intake (Sorensen, Seltzer, & Wyse, 1983). Laptop and personal computers offer an easy, practical and rapid method for assessing the dietary practices of individuals at risk (Smith & Lloyd-Still, 1983; Johnson, Selzer, Blankenhorn, Nessim, Harlow, Caldarara, & Azen, 1983; Dwyer & Sutor, 1984).

Printouts of nutritional analyses are impressive in appearance. Most list calculations for total calories, protein, carbohydrate, fat, including a breakdown for saturated, polyunsaturated and monounsaturated, vitamin, mineral and fiber intake. Printouts often provide the patient with the Recommended Daily Allowances (RDA) based on the individual's gender and age and compare their estimated nutrient intake with the RDA. Computer Assisted Instruction (CAI) provided by the printouts allow for personalized education messages tailored to the food habits of the individual seeking dietary guidance (Wise, Liddell, & Lolke, 1987).

Smiciklas-Wright, Peclian, Byrd-Bredenner, and Shannon (1984) found computer generated nutrition information was helpful to patients when it was simple, direct, readable and understandable. Byrd-Bredbenner, Lewis, Dacis, and Antanitis (1988) reported that individuals had little difficulty in reading and interpreting and comprehending information from personalized printouts correctly. Subjects whose printouts included only nutrient data generally had more difficulty using the information to draw appropriate conclusions and identify practical ways to improve dietary habits than did individuals whose printouts included short eating pattern messages or advice statements listing foods to include in their daily diet to reduce risk. Dennison, Dennison, Ward, and Wu, (1992) assessed the receptiveness of older adults to the use of computers in a nutrition education program and found that such subjects were very receptive to CAI.

Adoption of computers in nutrition education allows for more efficient use of time for both the nutritionist and client. Nutritionists can become more effective educators using computers for dietary assessment and individualized recommendations based on the analysis generated. Computerized nutrition education programs can also provide meaningful and productive use of time spent by patients in ambulatory care clinic waiting rooms. Individuals identified at risk are scheduled for regular medical check-ups which would permit follow-up dietary assessment to be coordinated at time of visit. The decreasing cost of computers coupled with continuing budget cuts for nutritional services in health care facilities makes CAI a practical and sound economic venture.

CHAPTER III

METHODS

The purposes of this study were to determine if computer generated nutrient analyses, with personalized recommendations for improvement, affects change in diet over time in post colonoscopy patients and if people with colonic adenomas (cases) adhered to the recommendations and changed their diet more than people without colonic adenomas (controls). The study consisted of a random sample of 120 subjects from the 645 participants of the "Diet and Health Study of Colon Adenomas" conducted between July 1988 and March 1990 at the University of North Carolina at Chapel Hill, School of Medicine, Department of Medicine, Division of Digestive Diseases and Nutrition. The original dietary data collected from the 120 subjects in the "Diet and Health Study" were used as the baseline data. The follow-up dietary data for the same subjects were collected approximately 3 years later, between January and July 1993. To answer the two research questions, the baseline and follow-up dietary data were compared.

Target Population

The purpose of the "Diet and Health Study of Colon Adenomas" was to learn more about the etiology of colorectal adenomas. It was designed as a case-control study to examine a number of potential risk factors for adenomas including diet. Study participants in the "Diet and Health Study of Colon Adenomas" were drawn from consecutive patients who underwent colonoscopy at University of North Carolina Hospitals between July 1, 1988 and March 1.

1990. There were 2094 colonoscopies performed during these dates. Patients were excluded for any of the following reasons: 1) age less than 30 years, 2) polyposis (defined as >100 polyps), 3) colitis of any type, 4) previous colon resection, 5) previous colon cancer, 6) incomplete examination, or 8) previous adenoma. Cases were defined as patients with one or more adenomatous polyps. Controls were defined as individuals with no adenomatous polyps.

There were 973 patients who met eligibility requirements. Eligible patients were interviewed over the telephone about past health, diet, exercise, and occupation. Telephone interviews were conducted by the investigator, who held the position of Research Nutritionist at the University of North Carolina at Chapel Hill, School of Medicine, between July 25, 1988 and March 1, 1990. At time of each interview the interviewer was blinded to the case status of the patient. Of the 973 eligible subjects, interviews were declined by 161. The interviews of 130 subjects were incomplete. An additional 37 subjects who completed the interviews were subsequently found to be ineligible and were eliminated. A total of 645 subjects remained in the study (236 case subjects and 409 control subjects). Case and control subjects were similar with respect to gender, body mass index (BMI), race, marital status, and education. The case subjects were slightly older than the control subjects. A complete detailed target population profile of the "Diet and Health Study of Colon Adenomas" is included in this document (Appendix A).

Nutrient Analysis

Within two weeks of being interviewed, all participants of the "Diet and Health Study of Colon Adenomas" were mailed a computer generated nutrient

analysis based on their interview responses (Appendix B). Attached to the analysis were recommendations for improving diet. The recommendations specifically addressed how to limit fat and cholesterol and increase Vitamins A and C intake (Appendix C). Participants also received a flyer developed specifically for the study explaining the terms found in the computer printed nutrient analysis and recommendations (Appendix D). No other nutrition education material was provided to study participants.

Sample Population

Using a table of random numbers, (Remington & Schork, 1985; Table A-1) subjects from the "Diet and Health Study" were randomly selected and recruited to participate in the follow-up study. To locate, recruit and enroll 120 alive and willing participants, 315 subjects files were needed. All subjects were contacted by telephone by the investigator. Of the 315 potential participants, 81 had moved and left no forwarding number, 14 were too ill to participate, 18 had died, 82 had no current phone listing in the University of North Carolina Hospitals patient database, and 120 agreed to participate.

Informed Consent

Verbal consent was obtained over the telephone prior to conducting the dietary interview. Each subject was given a complete explanation of the study purpose. Subjects were informed that all information would be kept confidential and they could decline to be interviewed or withdraw at any time without penalty. This study was approved by the Committee on the Protection of Human Subjects and by the School of Medicine at the University of North Carolina at Chapel Hill

and by the Institutional Review Board at the University of North Carolina at Greensboro.

Instrument

The dietary instrument used to collect data for this study, as well as for the "Diet and Health Study of Colon Adenomas", consisted of a food frequency questionnaire developed at the National Cancer Institute. Use of the same instrument to collect data at baseline and follow-up allowed for comparison of dietary data. This food frequency questionnaire is known as the Health Habits and History Questionnaire (Appendix E). Subjects were asked about their intake of more than 100 foods that have been shown to contribute to the intake of calories and 17 macronutrients and micronutrients in the national diet. The instrument has been validated against data obtained from four 4-day diet records and recalls collected for 1 year prior to administering the questionnaire (Block, 1991b). Participants were asked to estimate how often, on average, they consumed each of the food items during the previous year. A 1-year period was chosen to provide a full cycle of seasons in hopes that responses would be independent of time of year. Subjects were also asked to estimate whether their usual portion size was small, medium, large, or extra large for each food item. Additional questions address consumption of foods eaten in restaurants, consumption of foods not mentioned in the questionnaire and frequency and type of fat used in cooking. The questionnaire is low cost, easy to administer, in wide use, and represents state-of-the-art assessment of usual dietary intake.

The nutrient database used for analysis was developed from the Second National Health and Nutrition Examination Survey II database (Block, Hartman,

Dresser, Carroll, Gannin, & Gardner, 1986). The food list and database, when used to calculate nutrients from a diet record, has yielded correlations of $r > 0.70$ with a more detailed method (Block, et al., 1986). An important feature of the Health Habits and History Questionnaire analysis is the use of age-sex specific portion sizes in conjunction with the respondents reported portion sizes.

Data Collection

The printed version of the Health Habits and History Questionnaire provided by the National Cancer Institute was used to collect all dietary data. Upon completion of the Health Habits and History Questionnaire, participants were asked five additional questions (Appendix F) addressing the usefulness of the personalized nutrient analysis each received. Participant responses were hand recorded and the paper copy of the interview was kept under lock and key. All data were entered into a password protected database. All subjects were identified by subject codes, and no person identifiers were kept with such materials. All data entries were verified by a second person for accuracy.

Telephone Survey

Subjects were contacted by telephone because a well designed and well administered telephone survey is comparable to, and may be better than, other methods for obtaining dietary information (Fox, Heimendinger, & Block, 1992). The average time needed to complete the dietary interview was 30 minutes. To keep an accurate record of calls a Patient Information Interview Contact Sheet (Appendix G) was developed for this project and maintained on each participant. The form provides documentation for each call to a potential respondent. The

day and time each call was placed and the result of each call was recorded. The following codes were used; Day: M=Monday; T=Tuesday; W=Wednesday; R=Thursday; F=Friday; or S=Saturday; Times: Mn=called between 8:00am-Noon; A=called between 12:01 -5:00pm; or E=called between 5:01-9:00pm.

To contact potential participants three call attempts were made during morning, afternoon, and evening hours on different days of the week. For each call the investigator allowed the telephone to ring seven times before hanging up. No messages were left on answering machines. The result of each contact was recorded on the Summary Contact Sheet with one of the following codes: C=contact; B=busy line; D=disconnected number; or NA=no answer. When the telephone call resulted in contact success the Summary Contact Sheet was coded with one of five categories 1) interview completed; 2) appointment for interview set up; 3) refused to participate; 4) unable to participate (too ill or died); or 5) moved away. Of the 120 subjects who participated, about 25% agreed to be interviewed at time of initial telephone contact with the remaining participants requesting an appointment to be interviewed at a later date. An average of five call attempts were needed to make initial contact with potential subjects. Interviews were conducted between January and July 1993.

Recruitment

Special significance was attached to the introduction of a survey interview because this is the point at which most refusals occur (Dillman, Gorton & Frey, 1976). Once the actual interview begins, very few respondents terminate before the last question is asked. Results of telephone surveys show that most people who refused will listen to the entire recruitment statement before attempting to

terminate (Dillman et al., 1976). Consequently, the recruitment statement provides an important opportunity to persuade the potential participant the survey is important and worth the time. To promote an optimal response rate and keep refusals to a minimum, a recruitment statement was created for this study and read to all potential participants at the time of the initial telephone contact.

Recruitment Statement

Hello. May I please speak to _____.

If not at home or unable to come to the phone at this time, I identified myself and asked when was the best time to call back. If the potential participant was home:

This is Colleen McAuliffe from the University of North Carolina at Chapel Hill. You may recall I contacted you once before a few years back. At that time I asked you questions about your diet. I am conducting a follow-up study about dietary change over time among patients who have undergone colonoscopy. Participation requires a single telephone interview which lasts about 30 minutes. Once again I will be asking you questions about your diet. The information gained from this study may help us to better understand what factors influence dietary change over time. May I set up an appointment with you for an interview?

Statistical Methods

Prior to onset of the study, a sample size calculation was constructed to simultaneously detect reductions in percentage of calories from fat and milligrams of cholesterol between baseline and follow-up measures. The hypotheses of interest were 1) the mean percentage of calories from fat in the sample population is lower at follow-up than baseline and 2) the mean cholesterol in the sample population is lower at follow-up than baseline. To

control for an overall type I error rate of 0.05, each test used an alpha = 0.025. The sample of 120 guaranteed a 95% power of detecting a 30 milligram decrease in cholesterol and a 2.5% decrease in percentage of calories from fat.

Analysis of Food Frequency Data

Each subjects' nutrient intakes were calculated by an analysis program provided by the Division of Cancer Prevention and Control, National Cancer Institute. The program was identified as Personal Computer Dietary Analysis System (PCDAS), and the analysis procedure incorporated the nutrient content of each food item, the frequency, and the age-sex specific portion size to calculate each daily nutrient intake.

Analysis of Dietary Change

Once the daily nutrient intakes were calculated, all baseline and follow-up data were pooled together for the analysis of dietary change. The change in a daily nutrient intake, measured as the absolute change from the baseline value, was calculated for each nutrient. The specific dietary components of interest in this study were fat, cholesterol, vitamin A, and vitamin C because they were individually addressed in the recommendations given to subjects participating in the "Diet and Health Study of Colon Adenomas". The additional questions on the questionnaire addressed the usefulness of the nutrient analysis. These questions were used to calculate how many subjects thought the nutrient analysis was helpful, how many followed the recommendations and for how long they followed the recommendations.

Nonparametric procedures were used to compare the subjects' initial nutrient intakes with their follow-up nutrient intakes. Nonparametric procedures were used because there was concern that certain distributional assumptions on the data, required under the parametric procedures, did not hold. The nonparametric procedures are appropriate regardless of the distribution of the data, as long as the observations are independent.

In answering the first study question, the Wilcoxon signed rank test was used to determine if there was a change in diet, specifically fat, cholesterol, vitamin A and vitamin C, between baseline and follow-up measurements. The comparison between baseline and follow-up was done for the overall group, for each gender, and for each case status.

The second study question was to determine if the dietary change was different for cases than for controls, and the Wilcoxon rank sum test was used in this determination. The comparison of change between cases and controls was done for the overall group and for each gender. Included in the second study question was whether the cases were more likely than the controls to alter their diets and adhere to the recommendations for change given with the nutrient analysis. This was assessed by using the Chi-Square test for association between case status and the responses to the additional questions. All nonparametric procedures were performed using SAS software (SAS Institute Inc., 1989).

Although the primary purpose of the study was to compare baseline and follow-up measures of fat, cholesterol, vitamin A and vitamin C, comparisons with other dietary components which may be linked to certain forms of cancers were possible. These included fiber, vitamin E and β -carotene. Dietary

modifications pertaining to these components may be appropriate in future recommendations to post colonoscopy patients. Baseline, follow-up and amount of change, by case status, for these dietary components were examined.

CHAPTER IV

RESULTS

Results of the statistical analysis are presented in Tables 1 through 10 of this chapter. A total of 120 subjects participated in the study, and demographics of the study population are presented in Table 1.

Table 1
Demographics of the Study Population (N=120)

	Case n(%)	Control n(%)	Total n(%)
Gender	40 (33.3)	80 (66.7)	120
Male	18 (45.0)	28 (35.0)	46 (38.3)
Female	22 (55.0)	52 (65.0)	74 (61.7)
Race			
White	29 (72.5)	56 (70.0)	85 (70.8)
Black	11 (27.5)	24 (30.0)	35 (29.2)

Data given as number and percent.

Baseline and follow-up measures of interest included age, weight, body mass index (BMI), total calories, percent of calories from fat, vitamins A & C, and cholesterol. Baseline and follow-up measures, along with amount of change, for the intact study population are presented in Table 2. On average, the entire study population lost 11.7 pounds, lowered their caloric intake by 191 calories and lowered their percent calories from fat by almost 2 percent. They also

increased their vitamin A intake by an average of 179 IUs and lowered their cholesterol intake by an average of 24 mgs.

Table 2

Baseline and Follow-up Measures* for the Study Population (N=120)

Measures	Baseline	Follow-up	Change
Age	59.8 (9.8)	63.1 (10.0)	3.3 (1.1)
Weight (lbs.)	183.9 (114.7)	172.1 (40.0)	-11.7 (107.2)
BMI (kg/m ²)	27.0 (5.8)	27.6 (6.3)	0.4 (2.1)
Calories	1743 (741)	1551 (547)	-191 (591)
% Calories from Fat	38.1 (6.7)	36.2 (6.8)	-1.9 (6.9)
Vitamin A (IUs)	7688 (4306)	7867 (4042)	179 (4049)
Vitamin C (mgs)	129.4 (74.1)	132.1 (62.9)	2.8 (67.9)
Cholesterol (mgs)	253.2 (149.4)	229.4 (133.2)	-23.8 (139.8)

Data given as mean and standard deviation for each measure.

*Mean (sd)

The data were also stratified by gender and case status. Baseline and follow-up measures, with amount of change, by gender are presented in Table 3. The female study population lost an average of 19 pounds. Males and females lowered their caloric intakes by averages of 296 calories and 126 respectively. Baseline and follow-up measures, with amount of change, by case status are presented in Table 4. Cases and controls lowered their caloric intake by averages of 196 calories and 189 calories, respectively. The average cholesterol intake was lowered by 34 mgs for cases and 19 mgs for controls.

Table 3

Baseline and Follow-up Measures* by Gender (N=120)

Measures	Males (n=46)			Females (n=74)		
	Baseline	Follow-up	Change	Baseline	Follow-up	Change
Age	61.2 (9.8)	64.5 (9.8)	3.4 (0.9)	58.9 (9.8)	62.2 (10.1)	3.3 (1.2)
Weight (lbs.)	176.1 (37.1)	176.3 (38.4)	0.2 (9.1)	188.7 (143.3)	169.5 (41.0)	-19.2 (136.1)
BMI (kg/m ²)	25.6 (4.5)	25.5 (4.7)	-0.1 (1.5)	27.8 (6.3)	28.9 (6.9)	0.7 (2.4)
Calories	2164 (803)	1867 (610)	-296 (717)	1481 (562)	1355 (39.5)	-126 (490)
% Calories from Fat	38.1 (6.6)	37.6 (6.4)	-0.4 (6.5)	38.0 (6.9)	35.2 (6.9)	-2.8 (6.9)
Vitamin A (IUs)	8715 (4211)	9489 (4790)	774 (4395)	7049 (4268)	6858 (3130)	-191 (3802)
Vitamin C (mgs)	145.9 (88.2)	137.8 (71.2)	-8.0 (70.4)	119.1 (62.3)	128.6 (57.4)	9.5 (65.5)
Cholesterol (mgs)	323 (189.4)	296.3 (158.9)	-26.7 (193.0)	209.8 (96.4)	187.7 (93.6)	-22.1 (94.3)

Data given as mean and standard deviation for each measure.

*mean (sd)

Table 4

Baseline and Follow-up Measures* by Case Status (N=120)

Measures	Cases (n=40)			Controls (n=80)		
	Baseline	Follow-up	Change	Baseline	Follow-up	Change
Age	60.4 (10.2)	63.7 (10.1)	3.3 (0.8)	59.5 (9.7)	62.8 (10.0)	3.3 (1.3)
Weight (lbs.)	218.4 (186.6)	176.5 (37.7)	-41.9 (182.7)	166.6 (41.5)	169.9 (41.2)	3.3 (11.3)
BMI (kg/m ²)	27.9 (7.2)	27.8 (6.5)	-0.2 (1.9)	26.5 (5.0)	27.5 (6.3)	0.7 (2.1)
Calories	1637 (602)	1441 (430)	-196 (547)	1796 (800)	1607 (591)	-189 (615)
% Calories from Fat	38.7 (6.7)	36.5 (7.4)	-2.2 (8.0)	37.7 (6.8)	36.0 (6.5)	-1.8 (6.3)
Vitamin A (IUs)	7447 (4304)	7403 (3557)	-43.8 (4344)	7808 (4329)	8099 (4266)	290.3 (3717)
Vitamin C (mgs)	118.2 (69.7)	114.9 (52.7)	-3.3 (56.3)	134.9 (76.0)	140.7 (66.1)	5.8 (72.8)
Cholesterol (mgs)	231.5 (137.7)	197.9 (91.1)	-33.6 (134.2)	264.1 (154.6)	245.1 (147.9)	-19.0 (143.1)

Data given as mean and standard deviation for each measure. *mean (sd)

The Wilcoxon signed rank test was conducted to determine if the computer generated nutrient analysis, with personalized recommendations for dietary improvement, affected change in diet in post colonoscopy patients. As an intact group of 120 post colonoscopy patients, statistically significant changes from baseline to follow-up were found for total calories (lowered 191 calories; $p=0.0006$), percent calories from fat (lowered 1.9%; $p=0.0017$), and cholesterol intake (lowered 23.8 mgs; $p=0.0440$) The Wilcoxon signed rank statistics and their associated p-values are presented in Table 5.

Table 5

Wilcoxon Signed Rank Test Results[†] for the Study Group and by Gender (N=120)

Measures	Study Group	Males	Females
	N=120	n=46	n=74
Age	3513.5 (0.0001)**	540.5 (0.0001)**	1315.5 (0.0001)**
Weight (lbs.)	484.0 (0.1167)	9.0 (0.8981)	283.5 (0.0696)*
BMI (kg/m ²)	569.5 (0.0645)*	-23.0 (0.7528)	378.0 (0.0123)**
Calories	-1279.0 (0.0006)**	-227.5 (0.0113)**	-420.5 (0.0224)**
% Calories from Fat	-1182.0 (0.0017)**	-65.5 (0.4803)	-620.0 (0.0006)**
Vitamin A (IUs)	234.0 (0.5422)	97.5 (0.2917)	-6.5 (6.9724)
Vitamin C (mgs)	157.0 (0.6827)	-29.5 (0.7511)	138.5 (0.4593)
Cholesterol (mgs)	-767.5 (0.0440)**	-80.0 (0.3880)	-359.5 (0.0521)*

[†]Data given as Wilcoxon signed rank statistic(S) and associated p-value.
(p-value)

* p-value < 0.1

** p-value < 0.05

Further analysis was conducted with the data stratified by gender. Results show both males and females had changed (lowered) their daily caloric intake at time of follow-up. For both genders, the change was statistically significant. As a group, males showed no other statistically significant changes in diet between baseline and follow-up. Female study participants showed a statistically significant change from baseline to follow-up for weight (lost 19.2lbs.; $p=0.0696$), BMI (increased 0.7 kg/m^2 ; $p=0.0123$), calories (lowered 126 calories; $p=0.0224$), percent of calories from fat (lowered 2.8 %; $p=0.0006$) and cholesterol intake (lowered 22.1 mgs; $p=0.0521$). Table 5 presents the results for each gender.

When stratified by case status, cases had a statistically significant change in caloric intake (lowered 196 calories; $p=0.0500$) and cholesterol intake (lowered 33.6 mgs; $p=0.0627$). Controls had a statistically significant change in total calories (lowered 189 calories; $p=0.0059$) and percent of calories from fat (lowered 1.8%; $p=0.0062$). Table 6 presents the results of the Wilcoxon signed rank test by case status.

Table 6

Wilcoxon Signed Rank Test Results[†] by Case Status (N=120)

Measures	Cases n=40	Controls n=80
Age	410.0 (0.0001)**	1542.5 (0.0001)**
Weight (lbs.)	-32.5 (0.5690)	413.5 (0.0167)**
BMI (kg/m ²)	-20.0 (0.7147)	426.0 (0.0157)**
Calories	-145.0 (0.0500)*	-566.0 (0.0059)**
% Calories from Fat	-104.0 (0.1649)	-562.5 (0.0062)**
Vitamin A (IUs)	15.0 (0.8432)	126.0 (0.5490)
Vitamin C (mgs)	-59.0 (0.4347)	211.0 (0.3145)
Cholesterol (mgs)	-138.0 (0.0627)*	-254.5 (0.2244)

[†]Data given as Wilcoxon signed rank statistic(S) and associated p-value.
(p-value)

* p-value < 0.1

** p-value < 0.05

Further analysis stratified subjects by gender and case status. Table 7 presents the results of the Wilcoxon signed rank test for subjects once stratified by gender and case status. Male cases showed no statistically significant changes in measures. Male controls showed a statistically significant change for total calories (lowered 337 calories; p=0.0254). Female cases showed statistically significant changes in measure for total calories (lowered 167 calories; p=0.0883), percent of calories from fat (lowered 4.2%; p=0.0164), and cholesterol (lowered 37.0 mgs; p=0.0201). Female controls showed statistically

significant changes for body weight (gained 5.0 pounds; $p=0.0027$), BMI (increased 1.0 kg/m^2 ; $p=0.0033$), percent of calories from fat (lowered 2.2%; $p=0.0139$) and vitamin C (increased 17.0 mgs; $p=0.0835$).

Table 7

Wilcoxon Signed Rank Test Results[†] Stratified by Gender and Case Status (N=120)

Measures	Males		Females	
	Cases (n=18)	Controls (n=28)	Cases (n=22)	Controls (n=52)
Age	85.5 (0.0001)**	20.3 (0.0001)**	126.5 (0.001)**	638.5 (0.0001)**
Weight (lbs.)	2.5 (0.8752)	1.5 (0.9688)	-21.5 (0.4357)	265.0 (0.0027)**
BMI (kg/m^2)	-12.5 (0.4536)	2.5 (0.9480)	0.5 (1.000)	269.0 (0.0033)**
Calories	-26.5 (0.2645)	-96.0 (0.0254)**	-52.5 (0.0883)*	-178.0 (0.1056)
% Calories from Fat	13.0 (0.5876)	-58.5 (0.1877)	-71.5 (0.0164)**	-266.0 (0.0139)**
Vitamin A (IUs)	23.5 (0.3247)	23.0 (0.6095)	-19.5 (0.5392)	40.0 (0.7194)
Vitamin C (mgs)	7.5 (0.7660)	-24.0 (0.5939)	-39.5 (0.2068)	190.0 (0.0835)*
Cholesterol (mgs)	-3.5 (0.8986)	-35.5 (0.4288)	69.5 (0.0201)**	-91.0 (0.4125)

[†]Data given as Wilcoxon signed rank statistic(S) and associated p-value.
(p-value)

* p-value < 0.1

** p-value < 0.05

The second specific aim of this study was to investigate whether people with colonic polyps (cases) were more motivated to adhere to the personalized recommendations and, as a result, changed their diets more than the people without colonic polyps (controls). The Wilcoxon rank sum test was used to

assess this for the overall group and for each gender. The Wilcoxon rank sum statistics and their associated p-values are presented in Table 8. Regarding vitamin C intake, female cases decreased intake by 8.3 mgs and female controls increased their intake by 17.0 mgs. This difference in change was statistically significant ($p=0.0431$). No other statistically significant differences were found between cases and control in regards to change in nutrient intake.

Table 8

Wilcoxon Rank Sum Test Results[†] for the Study Group and by Gender (N=120)

Measures	Study Group	Males	Females
	N=120	n=46	n=74
Age	2302.0 (0.4836)	412.0 (0.8014)	769.5 (0.4842)
Weight (lbs.)	2124.0 (0.0994)*	427.0 (0.9370)	669.5 (0.0664)*
BMI (kg/m ²)	1881.5 (0.1138)	393.0 (0.6344)	584.5 (0.2371)
Calories	2440.0 (0.9136)	448.0 (0.5813)	795.0 (0.7272)
% Calories from Fat	2452.0 (0.8608)	459.5 (0.4177)	769.0 (0.5116)
Vitamin A (IUs)	2403.0 (0.9268)	438.0 (0.7442)	773.0 (0.5425)
Vitamin C (mgs)	2206.5 (0.2357)	445.0 (0.6284)	653.5 (0.0431)**
Cholesterol (mgs)	2340.0 (0.6581)	453.0 (0.5067)	708.0 (0.1683)

[†]Data given as Wilcoxon rank sum statistic(S) and associated p-value.
(p-value)

* p-value < 0.1

** p-value < 0.05

Results to the questions addressing the usefulness of receiving the personalized nutrient analysis are summarized in Table 9. As an intact group of

120 subjects, 83 subjects (69.2%) remembered receiving the nutrient analysis and 37 subjects (30.8%) did not. When stratified by gender, 27 males (58.7%) and 56 females (75.7%) remembered receiving the nutrient analysis. When stratified by case status, 28 cases (70.0%) and 55 controls (68.7%) remembered receiving the analysis. Of the 83 subjects who remembered receiving the analysis, 82 subjects (98.8%) found the information helpful. When stratified by gender, fifty-six females (100%) and 26 males (96.3%) found the information helpful. When stratified by case status, 27 cases (96.4%) and 55 controls (100%) found the information helpful. Out of those subjects who found the nutrient analysis useful, 75 subjects (91.5%) followed the recommendations and 7 (8.5%) did not. Out of the 75 subjects, 24 were males and 51 were females; by case status, 25 were cases and 50 were controls. Of the 75 subjects who followed the recommendations, the mean length of time spent adhering to the recommendations was 14.9 weeks (sd=33.9). The mean length of time spent adhering to the recommendations was similar for males and females, but was much longer for controls (18.5 weeks) than cases (8.2 weeks). Sixteen subjects reported they presently still follow some of the recommendations and 104 subjects (86.7%) reported they do not currently follow any of the recommendations. Of the 16 subjects who presently follow the recommendations, 4 are males and 12 are females; by case status, 5 are cases and 11 are controls. Limiting fat intake, in an attempt to keep total percent of calories from fat under 30%, is the one recommendation all 16 subjects reported they presently follow.

Table 9

Summary of Usefulness of Nutrient Analysis with Recommendations

		Overall	Male	Female	Case	Control
		N=120	n=46	n=74	n=40	n=80
Remember receiving analysis†	Yes	83 (69.2)	27 (58.7)	56 (75.7)	28 (70.0)	55 (68.7)
	No	37 (30.8)	19 (41.3)	18 (24.3)	12 (30.0)	25 (31.2)
Was information helpful†	Yes	82 (98.8)	26 (96.3)	56 (100)	27 (96.4)	55 (100)
	No	1 (1.2)	1 (3.7)	0 ----	1 (3.6)	0 ----
Followed recommendations†	Yes	75 (91.5)	24 (92.3)	51 (91.1)	25 (92.6)	50 (90.9)
	No	7 (8.5)	2 (7.69)	5 (8.9)	2 (7.4)	5 (9.1)
If followed, how long (weeks)*	----	14.9 (33.0)	16.5 (42.3)	14.1 (27.7)	8.2 (8.1)	18.5 (40.1)
Do you presently follow recommendations†	Yes	16 (13.3)	4 (8.7)	12 (16.2)	5 (12.5)	11 (13.7)
	No	104 (86.7)	42 (91.3)	62 (83.8)	35 (87.5)	69 (86.2)

† Data given as number and percentage. n(%)

* Data given as number and standard deviation. mean (sd)

Thirty-nine subjects (32.5%) reported there were other health factors which may have motivated them to modify their diets, while 81 subjects (67.5%) reported no additional health factors exist which may have lead to dietary change. Health factors reported include hypercholesterolemia, cardiovascular disease, diabetes, obesity or overweight, major surgery, renal disease, and diagnosis of cancer in a body organ other than the large bowel.

When questioned about where they receive advice about diet and nutrition, many subjects reported two primary sources. Seventy-five subjects (62.5%) named a doctor, 54 subjects (45%) reported magazines, 35 subjects (29.2%) stated a dietitian or nutritionists, 19 subjects (15.8%) stated television, 6

subjects (5.0%) subjects stated a friend and 2 subjects (1.7%) reported other.

Table 10 summarizes the subjects responses by gender and case status.

Table 10

Summary for Sources of Nutrition Advice (N=120)

	<u>Overall</u>	<u>Male</u> n=46	<u>Female</u> n=74	<u>Case</u> n=40	<u>Control</u> n=80
Where did you get advice?*					
Dietitian	35 (29.2%)	8 (17.4%)	27 (36.5%)	16 (40.0%)	10 (23.7%)
Doctor	75 (62.5%)	32 (69.6%)	43 (58.1%)	28 (70.0%)	47 (58.7%)
TV	19 (15.8%)	6 (13.0%)	13 (17.6%)	4 (10.0%)	15 (18.7%)
Magazines	54 (45.0%)	17 (37.0%)	37 (50.0%)	13 (32.5%)	41 (51.2%)
Friends	6 (5.0%)	3 (6.5%)	3 (4.1%)	1 (2.5%)	5 (6.2%)
Other	2 (1.7%)	0 ----	2 (2.7%)	0 ----	2 (2.5%)

Data given as number and percent. n (%)

* Most people gave two responses.

When asked if they would have liked to receive more detailed information on how to improve their diet, 76 (63.3%) subjects responded yes and 43 subjects (35.8%) responded no. Of the 76 subjects who would have liked to receive more detailed nutritional information, 34 subjects (44.7%) would have liked menus, 20 subjects (26.3%) requested a weekly meal plan and 22 subjects (29%) wanted low fat recipes. Thirty-four percent of the subjects requesting more detailed information were male and 66% were female.

To determine if cases and controls were equally interested in and motivated to follow the recommendations and alter diet the Chi-Square test for

association was used. Results show cases were not significantly different from controls. The Chi-Square test, adjusting for small samples, produced a test statistic of 0.000 with a p value of 1.000. The results indicate case subjects were not significantly different from control subjects in regards to interest in the analysis or motivation to follow the recommendations.

The dietary data collected allowed for comparison of other dietary components which may be linked to certain types of cancer. These include fiber, vitamin E, and β -carotene. Baseline, follow-up and amount of change, by case status, for these three dietary components were also analyzed. Baseline and follow-up measures for fiber, vitamin E, and β -carotene, along with amount of change by case status are presented in Table 11. Cases and controls decreased their fiber intake by 0.5 grams and 0.6 grams respectively. The average vitamin E intake was lowered by 0.6 TEs for cases and 0.3 TEs for controls. Both cases and controls increased β -carotene intake by averages of 278.8 mgs for cases and 603.6 mgs for controls.

Table 11

Baseline and Follow-up Measures* for Fiber, Vitamin E and β -Carotene by Case Status (N=120)

Measures	Cases (n=40)			Controls (n=80)		
	Baseline	Follow-up	Change	Baseline	Follow-up	Change
Fiber (g)	12.6 (5.6)	12.0 (4.4)	-0.5 (6.2)	13.4 (6.7)	12.7 (5.0)	-0.6 (5.9)
Vitamin E (TE)	7.1 (2.7)	6.4 (2.4)	-0.6 (3.6)	7.8 (3.8)	7.4 (3.1)	-0.3 (3.6)
β -Carotene (mgs)	3332.9 (2636.6)	3611.7 (2519.6)	278.8 (2870.1)	3182.4 (2461.1)	3786.0 (2666.6)	603.3 (2790.0)

Data given as mean and standard deviation for each measure. *mean (sd)

Table 12 presents results of the Wilcoxon signed rank test by case status for fiber, vitamin E and β -Carotene. The change in β -carotene intake by controls was statistically significant (increased 603.0 TEs; p-value=0.0141), whereas, the change in intake by cases was not statistically significant. No statistically significant changes were found for fiber or vitamin E by either cases or controls.

Table 12

Wilcoxon Signed Rank Test Results[†] for Fiber, Vitamin E and β -Carotene by Case Status (N=120)

Measures	Cases n=40	Controls n=80
Fiber (g)	-10.5 (0.8813)	-187.0 (0.3731)
Vitamin E (TE)	-80.5 (0.2848)	-174.5 (0.4060)
β -Carotene	66.0 (0.3817)	507.0 (0.0141)**

[†] Data given as Wilcoxon signed rank statistic (S) and associated p-value.
(p-value)

*p-value<0.1

**p-value<0.05

CHAPTER V

DISCUSSION

The specific aims of this dissertation research were to investigate if a computer generated nutrient analysis, with personalized recommendations for dietary improvement, affected change in diet in post colonoscopy patients and to determine if people with colonic adenomas adhered to the recommendations and changed their diet more than people without colonic adenomas. It was also of interest to investigate whether the study subjects found the nutrient analysis useful and to determine how long they followed the recommendations.

One-hundred-twenty subjects were randomly selected from the "Diet and Health Study" population. Of the 120 subjects in the follow-up study, 38.3% were males and 61.7% were females; 70.8% were Caucasian and 29.2% were black. Thirty-three percent of the subjects were classified as cases, (people diagnosed with one or more adenomatous polyps), and 67% were classified as controls (adenoma free). All study subjects completed two telephone dietary interviews. The first, at time of entry into the "Diet and Health Study", the second, approximately three years later, at time of follow-up. Baseline and follow-up measures were compared to identify changes in diet. Measures of primary interest were percent of calories from fat and intakes of cholesterol, vitamin A, and vitamin C. The data were initially analyzed for the intact group. Further analysis was conducted with the data stratified by gender and case status. Dietary data collected allowed for comparison of other dietary

components. Baseline, follow-up and amount of change by case status, for fiber, vitamin E and β -carotene were also examined.

As a group (N=120), statistically significant changes in diet were found for total calories, percent of calories from fat, and cholesterol. At time of follow-up, the group had lowered daily caloric intake, lowered daily percent of calories from fat, and lowered cholesterol intake. The nutrient analysis mailed to each subject was derived from individual responses given during the dietary interview. The recommendations provided subjects with specific eating pattern messages targeting how to limit fat and cholesterol intake and consume adequate vitamins A and C. Specifically, subjects were advised to limit fat intake to 30% of total calories, keep daily cholesterol intake under 300 mgs, and to consume sufficient amounts of vitamin A rich foods to meet the RDA (1,000 ug RE for males; 800 ug RE for females), and sufficient amounts of vitamin C rich foods to meet the RDA (60 mgs males and females). The eating pattern messages emphasized eating less high fat foods; choosing low fat dairy products, and consuming more whole grain, starches, fruits, and vegetables. The messages provided subjects with information needed to adopt cancer risk reducing eating patterns. If followed these recommendations would be expected to lower an individuals total fat and cholesterol intake.

As a group study subjects had a baseline percent of fat intake at 38.1% per day, which exceeded the current upper limit set by the National Cancer Institute, the American Heart Association, and the American Dietetic Association recommendations for healthy eating patterns. All three organizations recommend that the percent of calories from fat not exceed 30%. At follow-up the study group had a percent of fat intake at 36.2%. Although 36.2% of

calories fat still exceeds the upper limit, the change of -1.9% from baseline to follow-up was statistically significant. As a group, baseline cholesterol intake was 253.2 mgs per day, which is below the recommended limit of 300 mgs per day. At follow-up the group had lowered their cholesterol intake to 229.4 mgs per day, and the change of -23.8 mgs per day was statistically significant. No statistically significant changes in intake for vitamin A or vitamin C between baseline and follow-up measures were observed for the group. These findings suggest the specific eating pattern messages addressing fat and cholesterol intake, set forth in the recommendations, may have prompted subjects to alter their food choices which resulted in the positive changes in dietary measures found at time of follow-up assessment. Since a high intake of dietary fat is strongly associated with adenomatous polyps (Sandler, et al., 1993; Giovannucci et al., 1992), the dietary changes of lowered fat and cholesterol found with this sample of post colonoscopy patients are important. It is possible that being enrolled as a participant in the case control "Diet and Health Study" may have motivated subjects to take action and modify their diet based on the findings of their colonoscopy. People who participate in research projects, as a group, may be different from those in the general population. Perhaps as a group they are more concerned about their health and are highly motivated to adhere to dietary recommendations to reap the benefits of decreased risk. It is well established that behavior is more likely to be changed when it is associated with an identifiable benefit (Guthrie, 1978). The benefits of avoiding colorectal cancer are innumerable. Research (Bruno et al., 1983; Arntzenius et al., 1985; Carmody et al., 1986) evaluating programs needed to change knowledge and

guidance on how to achieve gradual incremental change in dietary habits have shown positive results among people at risk for disease.

The dietary intervention given to these subjects was brief and did not include individual or group counseling. Previous research has shown brief dietary interventions can be effective in producing incremental dietary changes with subjects at risk for cancer (Schapira, Kumar, Lyman & Baile, 1991) and cardiovascular disease (Baron, Gleason, Crowe & Mann, 1990). The subjects in this study modified their diets in a positive manner (lowered fat and cholesterol intake) and maintained these changes for 3 years. Reid and Mulcahy (1987) reported high risk cardiac subjects maintained positive dietary changes six years after initial dietary intervention.

Further analysis was conducted with data stratified by gender and case status. For gender, analysis revealed both males and females lowered their daily caloric intake at the time of follow-up. For both genders, the change was statistically significant. The lowered caloric intake can be attributed to the decrease in total fat consumed. As a group, males did not show any other statistically significant changes in diet between baseline and follow-up. Females showed four other statistically significant changes in measure from baseline to follow-up. These included weight status, body mass index, percent of calories from fat, and cholesterol intake. As a group, the mean weight change for females was a loss of 19.2 pounds. The standard deviation of 136.1 pounds must be carefully considered when interpreting the drastic weight reduction. A review of weight status of female subjects revealed many were morbidly overweight at time of entering the "Diet and Health Study". With a high percentage of females being overweight, the probability of a such a high mean

increases. Females lowered their percent of calories from fat by 2.8%, and lowered cholesterol intake by 22.1 mgs. These results suggest the dietary recommendations given to female subjects were followed and contributed to the positive changes found at follow-up. Females may have been more eager to follow the recommendations in an attempt to promote weight loss rather than decrease their risk of adenomatous polyps or colon cancer. The nutrient analysis along with the recommendations was given to all study subjects free of charge. Female subjects may have recognized the benefit of receiving such accurate personalized information without cost, but male subjects did not.

When data were stratified by case status, cases (n=40) showed a statistically significant change between baseline and follow-up for caloric intake and cholesterol intake. For both measures, cases lowered their intake. Controls (n=80) showed statistically significant changes for total calories, percent of calories from fat and β -carotene intake. For all measures the change was positive. These findings suggest both cases and controls adhered to the recommendations given. It is not surprising subjects were able to modify percent of calories from fat. The mass media campaign we are all exposed to, promoting low fat and fat free food items, has made most Americans conscious about fat intake. Implementing the recommendations to lower fat intake may have been less difficult than would be expected. The recommendations given may have acted as a prompt to get subjects started on a lower fat dietary regimen while mass media messages acted as continual reinforcement.

During the past few years β -carotene has received a fair amount of exposure in both scientific journals and the popular press. The recommendations given to study subjects did not specifically address β -

carotene. Vitamin A, without any mention about its precursor or preformed status, was addressed. Eating pattern advice statements for vitamin A encouraged increased consumption of dark green, deep yellow and richly colored vegetables and fruits, which are excellent sources of β -carotene. Although no statistically significant changes were observed for vitamin A intake, the results suggest, that controls did increase their consumption of foods rich in β -carotene. The recommendations along with the combined media messages delivered by private industry, government agencies, and professional associations may have jointly lead to the positive change observed by control subjects. Interestingly, fiber has also been widely addressed by commercial organizations and government agencies, but was not addressed in the recommendations. No positive change in fiber intake was observed at time of follow-up by either case status. Perhaps if fiber had been addressed in the recommendations a positive change in fiber intake would have been observed. To date vitamin E has received only little exposure in the popular press, and for good reason. Scientific evidence supporting its protective effect against cancer at any site remains controversial. It is not surprising that vitamin E intake remained relatively constant between baseline and follow-up measurement.

In summary, females showed a greater number of positive dietary changes at time of follow-up than did males. Positive changes for two measures were observed for cases. Controls showed a positive change for three measures. Regardless of how the data were stratified, the dietary recommendations positively altered the diets of each group. When examined simultaneously by gender and case status, female cases lowered total calories, percent of calories from fat, and cholesterol; female controls lowered body

weight, and percent of calories from fat. Male cases showed no statistically significant changes in dietary measures. Male controls showed a statistically significant change for only one measure, total calories. These findings suggest female (case or control) post colonoscopy subjects in the "Diet and Health Study" followed the dietary recommendations to improve diet and adopt cancer risk reducing eating patterns to a greater degree than male (case or control) post colonoscopy subjects. Again it is possible, female study participants were motivated to follow the recommendations and altered their diet to promote weight reduction rather than lower the risk for colorectal carcinoma.

When comparing cases to controls, at baseline and follow-up, results show the only statistically significant dietary change between the two groups occurred for vitamin C intake. For vitamin C, female cases decreased intake by 8.3 mgs and female controls increased their intake by 17.0 mgs. This difference in change between the two groups was statistically significant. Even with this difference, daily vitamin C intake for all groups exceeded the recommended dietary allowance (RDA) at baseline and follow-up. No other statistically significant changes were found for dietary measures between cases and controls in regards to nutrient intake. This finding suggests that people identified to be at higher risk for colon cancer (cases) did not modify their eating patterns to decrease risk any more than people who were identified to be at lower risk (controls).

To determine the usefulness of the nutrient analysis and recommendations, subjects were asked to respond to five additional questions. Overall, 69% of the study subjects (N=120) remembered receiving the nutrient analysis. This is impressive when one considers a mean of 3.3 years elapsed

between baseline and follow-up interviews, and no specific instructions were given to the subjects about the nutrient analysis or recommendations. Subjects received a cover letter, the nutrient analysis with recommendations, and an explanation sheet to help them interpret the terms found in the nutrient analysis. Formal follow-up was never mentioned. Of the 83 subjects who remembered the analysis with recommendations, 100 percent of both females and controls remembered receiving the information, while 96.3% of the males and 96.4% of the cases remembered receiving the information. This finding is important. The information sent to the subjects was basic and inexpensive to produce. The nutrient analysis with recommendations is one section of a routine computer printout investigators can request when dietary data is analyzed from the Health Habits and History Questionnaire (HHHQ). The fact that so many subjects remembered receiving the information and altered their diet is encouraging and lends strong support to the proposal that all post colonoscopy subjects should complete the HHHQ, have their diets analyzed, and receive the accompanying computer generated recommendations for dietary improvement. Implementing such a proposal would allow all post colonoscopy patients to receive basic personalized nutrition education as part of routine medical care. This does not occur, however, in our present health care system. Nutritional assessment and intervention should be an integral component of primary medical care screening and prevention services (Block, 1993). Patients in need of nutritional assessment and guidance are usually referred to an out-patient dietitian or a nutritionist in private practice, both of which operate on a fee for service basis. The cost of such services make them prohibitive for many individuals. Consequently, many people never receive any nutritional guidance until they are

already suffering from a chronic yet potentially preventable disease. Oncology patients, receiving radiation or chemotherapy treatments, are given dietary guidance as part of standard medical care with out-patient or inpatient status. Numerous publications devote their entire content to nourishing the cancer patient (Schwalb & Crosson, 1988). Yet, few people receive dietary guidance, based on sound scientific data, enabling them to adopt cancer risk reducing eating patterns to prevent disease. If follow-up dietary assessment and recommendations were to be integrated with present follow-up medical care for high risk post colonoscopy patients, measurable reductions in colorectal cancer might be observed a decade from now. Dietary intervention initiated now could propel the nation's goal of a reduction of colorectal cancer deaths to no more than 13.2 per 100,000 people by year 2000 (USDHHS, 1991) into reality. Colonic adenomas are logical endpoints where nutrition intervention may have a positive impact.

Of the subjects who remembered receiving the nutrient analysis and recommendations, 98.8% of them found the information to be helpful for improving their diet. Out of the 98.8% subjects who found the information helpful, 91.5% followed the recommendations given. When stratified by gender and case status, adherence to the recommendations was over ninety percent for all groups; males, females, cases, and controls. Thus, gender or case status did not seem to influence whether or not a subject followed the recommendations. The adult public receives nutrition information from a wide variety of sources, some of which are more reliable and accurate than others. It is possible that subjects in this research study were highly motivated to adhere to recommendations for two reasons; first, the source of the recommendations, the

National Cancer Institute, is reputable and well exposed; second, the recommendations were given at the time of screening for a fatal disease. These two factors may have had a powerful synergistic impact on the subjects willingness to modify diet to adopt cancer risk reducing eating patterns.

As an intact group, mean time for adhering to the recommendations was just under 4 months at 14.9 weeks. Gender did not influence how long the recommendations were followed, case status did. Controls adhered (18.5 weeks) to the recommendations for a longer time period than cases (8.2 weeks). This is impressive and indicates that the simple straight forward nutrient analysis with the eating pattern message recommendations is an inexpensive and effective method to reach a large group of people at the time of routine medical screening. Even if dietary recommendations only bring about marginal or small dietary changes, such changes have a higher chance of being maintained over time than more drastic changes (Angove, 1979). A small decrease in risk as a result of dietary change can be expected to produce substantial improvements in the overall health of Americans (McGinnis & Nestle, 1989). The results of this study indicate small but significant positive dietary changes were maintained over a three year period. These small significant positive dietary changes deserve the attention of professionals working with this population. Results suggest high risk post colonoscopy patients are willing and able to modify their diet, to decrease risk, if recommendations are provided. The simplicity and practicability of eating pattern messages without individual counseling appears to have had a positive impact on the eating habits of the subjects in this study. Of great significance is the finding that thirteen percent of the study subjects are still following the recommendations. Of those currently following the

recommendations, by gender, 16.2% are female and 8.7% are males; by case status, 12.5% are cases and 13.7% are controls. These findings indicate, for this sample of people, controls adhered to the recommendations longer than cases. The finding that a greater percentage of controls have followed the recommendations longer than cases is of interest because it is in contrast to results for dietary intervention with females at risk for breast cancer. Schapira et al. (1991) reported females identified to be at greater risk for developing breast cancer, were more motivated to follow dietary recommendations targeted at reducing breast cancer risk than those at lesser risk.

The results showed 41.9% of the females and 40.0% of the cases mentioned other health factors which may have influenced them to improve their diet. However, no subject reported receiving dietary guidance or nutritional counselling for a medical condition during the time period between baseline and follow-up data collection. It is possible subjects who adhered to the recommendations and improved their diet did so to help prevent or control a chronic disease other than colorectal cancer, such as heart disease or diabetes. Therefore, the desire to adopt cancer risk reducing eating patterns and target or prevent one chronic disease may actually help lower an individuals risk for another chronic disease. This is understandable, since many Americans present a profile of co-morbidity. The recommendations to lower risk for the major chronic diseases are based on similar principles and often overlap (USDHHS, 1988). Whether a person targets improving their diet for the prevention of one disease over another should not be central. The important issue is, dietary recommendations are implemented which promote positive dietary changes to lower risk. The key is to get accurate practical information to the public,

especially those identified to be at high risk. This is not occurring. Currently, post colonoscopy patients do not receive any dietary guidelines as a component of their health care. Many Americans are unaware of the current dietary recommendations established to decrease risk and potentially prevent cardiovascular disease and cancer (Schapira, Kumar, Lyman & McMillan, 1990) simply because dietary guidance is seldom incorporated into routine medical care.

The findings that 62.5% of the study subjects identified a physician and 45% named magazines as their primary sources for nutrition information are discouraging. Only 29.2% of the subjects identified dietitians or nutritionists as their primary source for nutrition information. Most physicians are not trained to be experts in nutrition. The minimum number of classroom hours of nutrition education recommended for medical students by the National Academy of Sciences is 25-30 hours. Sixty percent of American medical schools, however, provide fewer than 20 hours of nutrition education, and only 24% of American medical schools require their students to complete a single course in human nutrition (National Research Council, 1985). Compound the lack of training physicians receive with the time constraints they face when seeing patients during clinic appointments, and it is evident physicians cannot be providing accurate practical dietary recommendations to patients.

For a wide variety of reasons, Americans want nutrition information that can be applied to their lives, more so if they are at risk for a potentially preventable chronic disease. To be worthwhile dietary recommendations disseminated to people at risk must lead to dietary changes which will lower a population's risk for disease. The information given must be accurate,

understandable, and practical. The findings of this research suggest the computer generated nutrient analysis with recommendations did affect change in diet in post colonoscopy patients. The dietary changes were all positive and maintained at the time of follow-up, three years later. These findings suggest that the nutrient analysis with recommendations was an effective method of nutrition education for producing and maintaining small significant positive dietary changes among post colonoscopy patients.

The Health Habits and History food frequency questionnaire developed at the National Cancer Institute is a comprehensive instrument, available at no cost, and easy to administer in both epidemiologic and clinical research. Currently, it is widely used in investigations targeting dietary habits and chronic disease, especially cancer, in population groups. Foods listed on the questionnaire and their associated nutrient values were developed using dietary data from the Second National Health and Nutrition Examination Survey nutrient database. The questionnaire has been previously determined to be a reliable and valid instrument. It can be self-administered or interviewer-administered using paper and pencil or computer assisted, dependent upon where, when, and what group of subjects are being studied. Use of the food frequency questionnaire by investigators will enhance comparability between studies examining diet and chronic disease, specifically cancer. This is a strength of the instrument and becomes increasingly important as diet continues to be scrutinized in the prevention of cancer at any site. The data management and analytic tools needed to utilize the food frequency questionnaire are provided free of charge by the Division of Cancer Prevention and Control.

The food frequency questionnaire can be administered in about 30 minutes, ensuring that respondent burden is kept to a minimum. The format of the instrument is logical, well organized, and comprehensive. Another strength of the instrument is the use of age-sex specific portion sizes in conjunction with the respondents reported portion size. When administered properly the questionnaire will be representative of usual diet, allowing for associations of dietary factors correlated with adverse health outcomes. The instrument is in wide use and is well respected among investigators conducting research targeting the role of diet in the prevention of cancer.

One weakness of this food frequency questionnaire is its lack of representation of ethnic foods. In its current form it would not be an acceptable instrument for collection of dietary data among minorities, such as the Hispanic, Asian-American, Mexican-American, Chinese-American and American-Indian populations. The questionnaire would need to be expanded to include foods normally consumed by these groups. To accommodate the additional foods the nutrient database would need to be expanded, however, this is not any easy task and is a limitation of the questionnaire and analysis package. As the American population continues to diversify, the food habits of these groups will need to be addressed. The current version of the questionnaire became available in March 1989 and does not reflect newly developed food products currently consumed by many Americans, especially those of the low-fat, reduced-fat or no-fat, and reduced calorie category. Expanding questions 17 and 18, which address fat intake, would be beneficial. Expanding the dairy products section to include a wider variety of cheeses and cheese products would also be attractive and strengthen the questionnaire. The performance of

this food frequency is dependent upon the subjects ability to accurately quantify food consumption and portion size. This is a limitation if the questionnaire is to be self-administered. It is imperative that the study subjects' ability to quantify their dietary habits be evaluated prior to the onset of administering the questionnaire.

The recommendations which were provided to post colonoscopy study patients are still appropriate and do address dietary components which have been linked with chronic diseases such as cancer. The recommendations have many strengths. The one page format of the recommendations is ideal, and its brevity does not overwhelm the patient. The eating pattern messages are clear, concise, and accurate, and they are personalized in that they identify a subject's intake for fat, cholesterol, vitamins A and C and evaluate these against the current recommended intake for each dietary component. Subjects are very receptive to receiving these recommendations. As a first level dietary intervention strategy, designed to target at risk individuals encouraging voluntary dietary modification, the single page is of value. Factor in the ease at which it can be produced for a large number of patients, at no cost, and it becomes priceless.

Based on scientific evidence presented in the literature, timely evaluations of the recommendations produced by the analysis package, with appropriate revisions would be prudent. Information exists which suggest adequate consumption of fiber and β -carotene may be protective against cancer at certain sites. Inclusion of these two dietary components in future dietary recommendations generated by the analysis package would be beneficial. The absence of these two dietary components could be considered a limitation of the

recommendations evaluated in this research. At this time the addition of vitamin E to the recommendations would not be advocated. Maintaining the recommendations to a single page is strongly supported; a strength lies with its conciseness.

Finally, this dissertation research combined nutrition research with nutrition education. Using the intact study population, an inexpensive, readily available dietary intervention method was evaluated. The primary purpose of the "Diet and Health Study of Colon Adenomas" was to learn more about the role of diet in the etiology of colorectal adenomas. The specific aim of this study was to evaluate change in diet over time among post colonoscopy patients who had received brief dietary intervention. Providing the nutrient analysis with recommendations to participants and evaluating how it affected dietary intake over time brought about the marriage of nutrition research with nutrition education. A logical union which does not occur often, but should. The National Cancer Institute advocates public education on dietary modification while the research on diet and cancer and chemoprevention continues (Greenwald, 1993). As new directions in dietary studies in cancer emerge, nutrition education methods with an emphasis on cancer risk reducing eating patterns should be evaluated. Ideally, nutrition education strategies could be included in many study designs as an integral component of a scientific proposal addressing dietary habits and cancer.

CHAPTER VI

SUMMARY CONCLUSIONS AND RECOMMENDATIONS

The specific aims of this dissertation research were to investigate if a computer generated nutrient analysis with personalized recommendations for dietary improvement, affected change in diets of post colonoscopy patients, and to determine if people with colonic adenomas adhered to the recommendations and changed their diets more than people without colonic adenomas. The study consisted of a random sample of 120 subjects from the 645 participants of the "Diet and Health Study of Colon Adenomas". The original dietary data collected from the 120 subjects was used as baseline data. After the initial interview all subjects were mailed a nutrient analysis with personalized recommendations for improving diet. The follow-up dietary data, for these same subjects, were collected approximately three years later. To answer the two study questions baseline and follow-up dietary data were compared. The hypotheses of interest were 1) the mean percentage of calories from fat in the sample population is lower at follow-up than baseline and 2) the mean cholesterol in the sample population is lower at follow-up than baseline. The sample of 120 guaranteed a 95% power of detecting a 30 milligram decrease in cholesterol and a 25% decrease in percentage of calories from fat. Nonparametric procedures were used to compare the subjects' initial nutrient intakes with their follow-up nutrient intakes. The comparison between baseline and follow-up was completed for the overall group, for each gender and for each case status. Dietary data collected

allowed for comparison of other dietary components. Baseline, follow-up and amount of change, by case status, for fiber, vitamin E and β -carotene were also examined.

As an intact group (N=120) statistically significant dietary changes were found for total calories, percent of calories from fat and cholesterol. For each measure the change was positive. Subjects lowered total caloric intake, percent of calories from fat and cholesterol intake. When stratified by gender, results showed both genders lowered daily caloric intake. The change was statistically significant. As a group males showed no other statistically significant changes in diet. Females showed statistically significant changes for weight status, percent of calories from fat and cholesterol intake. For each dietary measure the change was positive. By case status, both cases and controls lowered daily caloric intake. The change was statistically significant for both groups. Cases also lowered cholesterol intake, while controls lowered total percent of calories from fat. For both groups the changes were statistically significant. No statistically significant changes in vitamins A, C and E or fiber intake between baseline and follow-up measures were observed for any group. However, controls increased β -carotene intake between baseline and follow-up and the change was statistically significant.

Females showed a greater number of positive dietary changes at time of follow-up than did males. Positive changes for two measures were observed for cases. Controls showed a positive change for three measures. When comparing cases to controls a statistically significant dietary change was found for vitamin C intake. Even with the change, vitamin C intake for cases and controls exceeded the RDA at both baseline and follow-up. Subjects in this

study identified to be at higher risk for colon carcinoma (cases) did not alter their diets and adopt cancer risk reducing eating patterns any more than subjects identified to be at lower risk.(controls).

Sixty-nine percent of the subjects remembered receiving the nutrient analysis with recommendations of which 98.8% found the information useful for improving diet. The mean time spent adhering to the recommendations was just under four months. Gender did not influence how long the recommendations were followed, case status did. Controls adhered to the dietary recommendations for a longer time period than cases.

Conclusions

The findings of this research suggest that a brief dietary intervention, which included a personalized nutrient analysis with recommendations, positively affected dietary changes among post colonoscopy patients. However, the dietary recommendations may have not been solely responsible for the positive dietary changes observed. Media coverage of dietary habits and the prevention of cancer may have also influenced subjects throughout the three year period. The brief dietary intervention was inexpensive, and results suggest it is an effective method for producing and maintaining small significant positive dietary changes in this sample of study subjects. The fact such a large percentage of the study participants adhered to the dietary recommendations is encouraging. The results lend strong support to the proposal that all post colonoscopy patients, identified to be at risk for adenomatous polyps, receive brief dietary intervention at routine medical screening. Coordinating nutritional assessment with routine medical follow-up care would be worthwhile, especially

when diet has been identified as a critical factor in the disease process. Results suggest the brief dietary intervention evaluated in this study affected the eating habits of post colonoscopy patients in a positive manner, and the positive dietary changes of lowered fat and cholesterol intake and increased β -carotene intake were maintained in this sample three years after intervention.

Recommendations

Based on the results of this research it would be of interest and appropriate to further evaluate the nutrient analysis with expanded recommendations among post colonoscopy patients in relation to polyp recurrence. Additional dietary components for which recommendations would be appropriate include fiber and β -carotene. Eating pattern messages modeling those evaluated in this study could be developed for fiber and β -carotene. Ideally, high risk patients would have their diets assessed at each scheduled follow-up colonoscopy. Findings from the colonoscopy and pathology reports, that being adenoma presence or absence, could be correlated with dietary intake changes. Further dietary intervention could be initiated based on the findings at follow-up visits. Patients identified to be at highest risk could receive more advanced dietary intervention. Subjects who participated in this follow-up study expressed a desire to receive more detailed nutrition information on ways to improve diet. Specifically, menus, meal plans, and low fat recipes were requested. Future studies could include these materials as one component of the dietary intervention and evaluate their impact on dietary habits among high risk post colonoscopy patients.

Several promising chemopreventive agents have been identified from epidemiologic, in vitro, and animal studies. Currently, ongoing chemopreventive trials sponsored by the National Cancer Institute targeting colon carcinoma include the dietary components vitamins C and E, fiber, calcium, and β -carotene. While awaiting the results of these clinical trials, cost effective dietary intervention strategies designed to target high risk individuals and encourage voluntary change in diet must be developed and applied.

BIBLIOGRAPHY

- Abel, M. M. (1890). Practical sanitary and economic cooking adapted to persons of moderate and small means. New York: American Public Health Association; 188.
- American Cancer Society. (1984). Special report: nutrition and cancer cause and prevention. A CA-Cancer Journal for Clinicians, 34, 121-126.
- American Institute for Cancer Research. (1990). Dietary guidelines to lower cancer risk. American Institute for Cancer Research; Washington, DC., 1-88.
- American Meat Institute. (1942). A guide to good eating. Poster. Chicago: National Dairy Council.
- Angove, R. (1979). Nutrition education for change. Journal of Human Nutrition, 33, 65-69.
- Aries, V., Crowther, J. S., Drasar, B. S., Hill, M.J., & Willaims, R. (1969). Bacteria and the etiology of cancer of the large bowel. Gut, 10, 334-345.
- Arminski, T. C., & McLean, D. W. (1964). Incidence and distrubition of adenomatous polyps of the colon and rectum based on 1,000 autopsy examinations. Diseases of the Colon and Rectum, 7, 249-261.
- Arntzenius, A. C., Kromhout, J. D., Barth, J. H. C., Reiber, A. V. G., Brusckke, B., Buis, C. M., van Gent, P., Kempen-Voogd, S., Strikwerda, S., & van der Velde, K. (1985). Diet, lipoproteins and the progression of coronary atherosclerosis: the Leiden Intervention Trial. New England Journal of Medicine, 312, 805-811.
- Atwater, W. O. (1902). US department of Agriculture. Priciples of nutrition and nutritive values of foods. Farmers Bulletin, 142, 48.
- Ausmen, L. M. (1993). Fiber and colon cancer: does the current evidence justify a preventive policy. Nutrition Reviews, 5, 57-63.
- Bansal, B. R., Rhoads, J. E., & Bansal, S. C. (1978). Effects of diet on colon carcinogenesis and the immune system in rats treated with 1,2-Dimethylhydrazine. Cancer Research, 38, 3293-3303.
- Baron, J., Gleason, R., Crowe, B., & Mann, J. (1990). Preliminary trial of the effect of general practice based nutritional advice. British Journal of General Practice, 40, 137-141.

- Block, G. (1991a). Vitamin C and cancer prevention: the epidemiologic evidence. American Journal of Clinical Nutrition, 53, 270S-282S.
- Block, G. (1991b). Dietary guidelines and the results of food consumption surveys. American Journal of Clinical Nutrition, 53, 356-357.
- Block, G. (1993). Micronutrients and cancer: time for action? Journal of the American Cancer Institute, 85, 846-848.
- Block, G., Hartman, A. M., Dresser, C.M., Carroll, M. D., Gannin, J., & Gardner, L. (1986). A data-based approach to diet questionnaire design and testing. American Journal of Epidemiology, 124, 453-469.
- Boring, C. C., Squires, T. S., & Tong, T. (1993). Cancer statistics, 1993. A CA-Cancer Journal for Clinicians, 43, 7-26.
- Breuer, N. F., & Goebell, H. (1987). Bile acids and cancer of the large bowel. Digestive Diseases, 5, 65-77.
- Bronner, Y. (1991). Healthy people 2000: a call to action for ADA members. Journal of the American Dietetic Association, 91, 1520-1521.
- Bruno, R. C., Arnold, L., Jacobson, M., Winick, M., & Wynder, E. (1983). Randomized controlled trial of a nonpharmacologic cholesterol reduction program at the worksite. Preventive Medicine, 12, 523-532.
- Bryd-Bredbenner, C., Lewis, M., Davis, B., & Antanitis, R. (1988). Computer analyzed dietary intake printouts: guidelines for their design and student comprehension. Journal of the American Dietetic Association, 83, 311-316.
- Burkitt, D. P. (1980). Fiber in the etiology of colorectal cancer. In Winawer, S., Schottenfeld, D., & Sherlock, P. Colorectal Cancer: Prevention, Epidemiology and Screening, New York: Raven Press 13-19.
- Burkitt, D. P., Walker, A. P., & Painter, N. S. (1974). Dietary fiber and disease. Journal of the American Medical Association, 229, 1068-1071.
- Butrum, R. R., Clifford, C. K., & Lanza, E. (1988). NCI dietary guidelines: rational. American Journal of Clinical Nutrition, 48, 888-895.
- Carmody, T. P., Istvan, J. D., Matarazzo, S. L., Connor, S. L., & Connor, W. E. (1986). Applications of social learning theory in the promotion of heart healthy diets: the Family Heart Study intervention model. Health Education Research, 1, 13-27.
- Carroll, K. K., & Khor, H. T. (1975). Dietary fat its relation in tumorigenesis. Progress in Biochemical Pharmacology, 10, 308.
- Chen, L. H., Biossonneault, G. A., & Glavert, H. P. (1988). Vitamin C, vitamin E and cancer (review). Anticancer Research, 8, 739-748.

- Clark, J. C., Collan, Y., Eide, T. J., Esteve, J., Ewen, S., Gibbs, N. M., Jensen, O. M., Koskela, E., MacLennan, R., Simpson, J.G., Stalsberg, H., & Zaridze, D. G. (1985). Prevalence of polyps in an autopsy series from areas with varying incidence of large-bowel cancer. International Journal of Cancer, 36, 179-186.
- Comstock, G. W., Helzlsouer, K. J., & Bush, T. L. (1991). Prediagnostic serum levels of carotenoids and vitamin E as related to subsequent cancer in Washington County, Maryland. American Journal of Clinical Nutrition, 53, 296S-264S.
- Connett, J., Kuller, L. H., Kjelsberg, M. O., Polk, B. F., Collins, G., Rider, A., & Hulley, S., B. (1989). Relationship between carotenoids and cancer: The multiple risk factor intervention trial (MRFIT) study. Cancer, 64, 126-134.
- Correa, P., & Haenszel, W. (1987). The epidemiology of large bowel cancer. Advances in Cancer Research, 26, 2-141.
- Day, D. W. (1984). The adenoma-carcinoma sequence. Scandinavian Journal of Gastroenterology, 104, 99-107.
- Dennison, D., Dennison, K. F., Ward, J. Y., & Wu, Y. W. (1992). Satisfaction of senior citizens in a nutrition education program with and without computer assisted instruction. Journal of Nutrition for the Elderly, 12, 15-31.
- Dillman, D. A., Gorton, G., & Frey, J. H. (1976). Reducing refusal rates for telephone interviews. Public Opinion Quarterly, 40, 66-70.
- Dion, P. W., Bright-See, E. B., Smith, C. C. & Bruce, W., R. (1982). The effects of dietary ascorbic acid and alpha-tocopherol on fecal mutagenicity. Mutation Research, 102, 27-37.
- Doll, R., & Peto, R. (1981). The causes of cancer: quantitative estimates of avoidable risks of cancer. Journal of the National Cancer Institute, 66, 1191-1308.
- Dwyer, J., & Sutor, C. W. (1984). Assessing needs, evaluating computer options. Journal of the American Dietetic Association, 84, 302-312.
- Edwards, P. K., Mullins, R. M., & Clarke, B. (1986). A comprehensive model for evaluating innovative nutrition programs. Journal of Nutrition Education, 18, 10-15.
- Enstrom, J. E. (1981). Reassessment of the role of dietary fat in cancer etiology. Cancer Research, 41, 3722-3723.
- Fox, T. A., Heimendinger, J., & Block, G. (1992). Telephone surveys as a method for obtaining dietary information: a review. Journal of the American Dietetic Association, 92, 729-732.

- Gey, K. F., Brubacher, G. B., & Stahelin, H. B. (1982). Plasma level of antioxidant vitamins in relation to ischemic heart disease and cancer. American Journal of Clinical Nutrition, 45, 1368-1377.
- Giovannucci, E., Stampfer, M. J., Colditz, G., Rimm, E. B., & Willet, W. C. (1992). Relationship of diet to risk of colorectal adenomas in men. Journal of the National Cancer Institute, 84, 91-98.
- Giovannucci, E., Stampfer, M. J., Colditz, G. A., Rimm, E. B., Trichopoulos, D., Rosner, B. A., Speizer, F. E., & Willet W. C. (1993). Folate, methionine and alcohol intake and risk of colorectal adenoma. Journal of the National Cancer Institute, 85, 875-883.
- Glanz, K. (1985). Nutrition education for risk factor reduction and patient education: a review. Preventive Medicine, 14, 721-752.
- Graham, S. (1980). Diet and cancer. American Journal of Epidemiology, 112, 247-251.
- Greenwald, P. (1993). New directions in dietary studies in cancer of the National Cancer Institute. Abstract presented at the Conference on Nutrition and Biotechnology in Heart Disease and Cancer. Research Triangle Park, NC, December 5-7, 1993.
- Greenwald, P., Lanza, E., & Eddy, O. A. (1987). Dietary fiber in the reduction of colon cancer risk. Journal of the American Dietetic Association, 87, 1178-1188.
- Guthrie, H. A. (1978). Is education not enough? Journal of Nutrition Education, 10, 57-58.
- Haenszel, W., & Dawson, E. A. (1965). A note on mortality from cancer of the colon and rectum in the United States. Cancer, 18, 265-272.
- Haenszel, W. M., & Kurihara, M. (1968). Studies of Japanese migrants: mortality from cancer and other diseases among Japanese in the United States. Journal of the National Cancer Institute, 40, 43-51.
- Heilbrun, L., Nomura, A., & Stemmermann, G., N. (1989). Diet and colorectal cancer with special reference to fiber intake. International Journal of Cancer, 44, 1-6.
- Hill, M. J. (1975). Metabolic epidemiology of dietary factors in large bowel cancer. Cancer Research, 35, 3398-3402.
- Hill, M.J. (1991). European organization for cooperation in cancer prevention: research and policy for primary prevention of colorectal cancer background of intervention trial. In Large-bowel Cancer: policy prevention, research and treatment, ed. by Rozen, P., Reich, C. B., & Winawar, S. J. Basel: Karger, 25-37.

- Hill, M. J., Morson, B. G., & Bussey, H. J. (1978). Aetiology of adenoma-carcinoma sequence in the large bowel. Lancet, 1, 245-247.
- Hoff, G., Moen, I. E., Trygg, W., Frolich, J., Sausar, M. V., Vatn, M., Gjone, E., & Larsen, S. (1986). Epidemiology of polyps in the rectum and sigmoid colon: evaluation of nutritional factors. Scandinavian Journal of Gastroenterology, 21, 199-204.
- Horwitt, M. K. (1986). The promotion of Vitamin E. Journal of Nutrition, 116, 1371-1377.
- Howe, G. R., Benito, E., Castelleto, R., Corree, J., Esteve, J., Gallagher, R. P., Isovich, J. M., Dengao, J., Kaaks, R., Kure, G. A., Kure, S., L'Abbe, K. A., Lee, H. P., Lee, M., Miller, A. B., Peters, R. K., Potter, J. D., Riboli, E., Slattery, M. L., Trichopoulos, D., Tuyns, A., Tzonou, A., Whittemore, A. S., Wu-Willaims, A. H., & Shu, Z. (1992). Dietary intake of fiber and decreased risk of cancer of the colon and rectum; evidence from combined analysis of 13 case-control studies. Journal of the National Cancer Institute, 24, 1887-1896.
- Hunt, C. L. (1916). United States Department of Agriculture. Food for young children. Farmers Bulletin, 717, 1-22.
- Institute of Medicine. (1991). Improving America's Diet and Health. ed. Thomas, P. R. National Academy Press, Washington, DC., 1-226.
- Jackman, R. J., & Mayo, C. W. (1951). The adenoma-carcinoma sequence of the colon. Surgical Gynecology and Obstetrics, 93, 327-330.
- Johnson, R. L., Selzer, R., Blankenhorn, D. H., Nessims, S., Harlow, C., Caldarara, L., & Azen, S. P. (1983). Nutrient analysis-a computerized seven-day food record system. Journal of the American Dietetic Association, 83, 667-671.
- Kabat, G. C., Howson, C. P., & Wynder, E. L. (1986). Beer consumption and rectal cancer. International Journal of Epidemiology, 15, 494-501.
- Klatsky, A. L., Armstrong, M.A., Friedman, G. D., & Hiatt, R. A. (1988). The relations of alcoholic beverage use to colon and rectal cancer. American Journal of Epidemiology, 128, 1007-1015.
- Knekt, P. (1988). Serum vitamin E level and risk of female cancer. International Journal of Cancer, 17, 281-286.
- Knekt, P. (1991). Role of vitamin E in the prophylaxis of cancer. Annals of Medicine, 23, 3-12.
- Knekt, P., Aromaa, A., Maatela, J., Aaran, R. K., Nikkari, T., Hakama, M., Hakulinen, T., Petro, R., Saxen, E., & Teppo, L. (1988). Serum vitamin E and risk of cancer among Finnish men during a 10-year follow-up. American Journal of Epidemiology, 127, 28-41.

- Knight, K. K., Fielding, J. E., & Battista, R. N. (1989). Occult blood screening for colorectal cancer. Journal of the American Medical Association, 261, 587-594.
- Kok, F. J., Van Duijn, C. M., Hifmann, A., Vermeeren, R., Bruijn, A. M., & Valkenburg, H. A. (1987). Micronutrients and the risk of lung cancer. (letter). New England Journal of Medicine, 316 1416.
- Kritchevsky, D. (1986). Diet, nutrition and cancer: the role of fiber in cancer. Cancer, 58, 1830-1836.
- Kure, S., Kure, G. A., & Watson, L. F. (1987). Case-control study of dietary etiology factors: the Melbourne colorectal cancer study. Nutrition and Cancer, 9, 21-42.
- Lev, R. (1990). Adenomatous polyps of the colon. New York: Springer-Verlag, 1-136.
- Lipkin A. T., & Newark, T. P. (1985). Effect of dietary calcium on colonic epithelial-cell proliferation in subjects at high risk for familial colonic cancer. New England Journal of Medicine, 313, 1381-1384.
- Lyon, J. L., & Mahoney, A. W. (1988). Fried foods and the risk of colon cancer. American Journal of Epidemiology, 128, 1000-1006.
- Machlin, L. J., & Bendich, A. (1987). Free radical tissue damage: protective role of antioxidant nutrients. Federation of American Societies for Experimental Biology Monographs, 1, 441-445.
- Macquart-Movlin, G., Riboli, E., Cornee, J., Kaaks, R., & Berthezene, P. (1987). Colorectal polyps and diet: a case-control study in Marseilles. International Journal of Cancer, 40, 179-188.
- Marlett, J. A. (1990). Analysis of dietary fiber in human foods. In: Kritchevsky, D., Bonfield, C., & Anderson, J. W. eds. Dietary Fiber Chemistry, Physiology, and Health Effects. New York: Plenum Press, 31-48..
- Martinez, I., Torres, R., & Frias, Z. (1975). Cancer incidence in the United States and Puerto Rico. Cancer Research, 35, 3265-3271.
- McGinnis, M. J., & Nestle, M. (1989). The surgeon general's report on nutrition and health: policy implications and implementation strategies. American Journal of Clinical Nutrition, 49, 23-28.
- McKeown-Eyssen, G. E., & Bright-See, E. (1984). Dietary factors in colon cancer: international relationships. Nutrition and Cancer, 6, 160-170.
- Meyers, A. J., & Henderson, J. B. (1974). Multiple risk factor reduction in the prevention of cardiovascular disease. Preventive Medicine, 3, 225-236.

- Mirvish, S. S. (1986). Effects of vitamin C and E on N-nitroso compound formation, carcinogenesis and cancer. Cancer, 58, 1842-1850.
- Morsen, B. C. (1976). Evolution of Cancer of the colon and rectum. Cancer, 34, 845-849.
- Morsen, B. C. (1974). The polyp cancer sequence in the large bowel. Proceedings of the Royal Society of Medicine, 67, 451-51.
- MRFIT Research Group. (1982). Multiple Risk Factor Intervention Trial: risk factor changes and mortality results. Journal of the American Medical Association, 248, 1465-1477.
- National Cancer Act Amendemts of 1974. (1974). Public Law 93-352.
- National Center for Health Statistics. (1981). Plan and operation of the second national health and nutrition examination survey, 1976-1980. Vital and health statistics series 1. Programs and collection series, no. 5. Washington DC, US Government Printing Office, PHS, 81-1317.
- National Cancer Institute. (1984). Cancer prevention: good news, better news, best news. Washington, DC, US Government Printing Office. NIH publication no. 84-2671, 1-15.
- National Cancer Institute (1987). Diet, nutrition and cancer prevention. A guide to food choices. Washinton, DC. Government Printing Office. NIH publication no. 87-2878, 1-18.
- National Dairy Council. (1941). A guide to good eating. Leaflet. Chicago, National Dairy Council.
- National Research Council. (1982). Diet Nutrition and Cancer. Committee on diet, nutrition and cancer. Assembly of Life Sciences, National Academy Press, Washington, DC., I-1-XVIII-7.
- National Research Council. (1985). Nutrition Education in U.S. Medical Schools. National Academy Press, Washington, D.C., 5-11.
- Neugut, A. L., & Pita, S. (1989). Role of sigmoidoscopy in screening for colorectal cancer: a critical review. Gastroenterology, 95, 492-499.
- Neugut, A. I., Garbowski, G. C., Lee, W. C., Murray, B. A., Nieves, J. W., Forde, K. A., Treat, M. D., Waye, J. D., & Fenoglio-Preiser, C. (1993). Dietary risk factors for the incidence and recurrence of colorectal adenomatous polyps. Annals of Internal Medicine, 118, 91-95.
- Nomura, A. M., Stemmermann, G. N., Heilbrun, L., Salkeld, R. M., & Vuilleumier, J. P. (1985). Serum vitamin levels and the risk of cancer of specific sites in men of Japanese ancestry in Hawaii. Cancer Research, 45, 2369-2372.

- Offerhaus, G. J., Giardiello, F. M., Tersmette, K. W. F., Mulder, J. W. R., Tersmette, A. C., Moore, G. W., & Hamilton, S. R. (1991). Ethnic differences in the anatomical location of colorectal adenomatous polyps. International Journal of Cancer, 49, 641-644.
- Paganini-Hill, A., Chao, A., Ross, R. K., & Henderson, B. E. (1987). Vitamin A, beta-carotene and the risk of cancer: a prospective study. Journal of the National Cancer Institute, 79, 443-448.
- Patterson, B. H., Block, G., Rosenberger, W. F., Pee, D., & Kahle, L. L. (1990). Fruit and vegetables in the american diet: data from the NHANESII survey. American Journal of Public Health, 80, 1443-1449.
- Phillips, R. L. (1975). Role of life-style and dietary habits in risk of cancer among seventh-day adventists. Cancer Research, 35, 3513-3522.
- Potter, J. D. (1990). The epidemiology of fiber and colon cancer. In: Kritchevsky, D., Bonfield, C., & Anderson, J. W. eds. Dietary Fiber Chemistry, Physiology, and Health Effects. New York: Plenum Press, 431-445.
- Potter, J. D., Graves, K. L., Finnegan, J. R., Mullis, R. M., Baxter, J. S., Crockett, S., Elmer, P. J., Gloeb, B. D., Hall, N. J., Hertog, J., Pirie, P., Richardson, S. L., Rooney, B., Slavin, J., Synder, M. P., Splett, P., & Visnawath, K. (1990). The cancer and dietary intervention project: a community based intervention to reduce nutrition related risk of cancer. Health Education Research, 5, 489-503.
- Potter, J. D., McMichael, A. J., & Hartshorne, J. M. (1982). Alcohol and beer consumption in relation to cancer of the bowel and long colon: an extended correlation analysis. Journal of Chronic Disease, 35, 833-842.
- Prasad, K. N., & Rama, B. N. (1985). Nutrition and Cancer. In: Bland, J. ed: Yearbook of Nutritional Medicine. New Canaan, Ct: Keats Publishing Inc., 179-211.
- Prasad, K. N. (1984). Vitamins against cancer. Arvada, Co: Nutrition Publishing House, Inc., 36-37.
- Pryor, W. A. (1991). The antioxidant nutrients and disease prevention, what do we know and what do we need to find out. American Journal of Clinical Nutrition, 53, 391s-3s.
- Randall, E. D., Marshall, J. R., Brasure, J., & Graham, S. (1991). Patterns in food use and compliance with NCI dietary guidelines. Nutrition and Cancer, 15, 141-158.
- Reddy, B. S. (1986). Diet and colon cancer: evidence from human and animal model studies. In, Reddy, B. S., Cohen, L. A. Diet, nutrition and cancer: a critical evaluation. Volume I macronutrients and cancer. Boca Raton, FL: CRDC Press, Inc., 47-67.

- Reddy, B. S. (1986). Amount and type of dietary fat and colon cancer; animal model studies. Progress in Clinical Biological Research, 222, 295-309.
- Reddy, B. S., Watanabe, K., & Weisburger, J. H. (1977). Effects of high fat diet on colon carcinogenesis in F-344 rats treated with 1,2-Dimethylhydrazine, methylazoxymethanol acetate and methylnitrosources. Cancer Research, 37, 4156-59.
- Reddy, B. S., Hedges, A. R., Laasko, K., & Wynder, E. L. (1978). Fecal constituents of a high risk North American and a low risk Finnish population for the development of large bowel cancer. Cancer Letters, 4, 217-222.
- Reid, V., & Mulcahy, R. (1987). Nutrient intakes and dietary compliance in cardiac patients: 6-year follow-up. Human Nutrition: Applied Nutrition, 41A, 311-318.
- Remington, R. D., & Schork, M. A. (1985). Statistics and Applications to the Biological and Health Sciences. New Jersey: Prentice-Hall Publishing, 619.
- Rogers, A. E., & Longnecker, M. D. (1988). Dietary and nutritional influence on cancer: a review of epidemiologic and experimental data. Laboratory Investigation, 59, 729-756.
- Sandler, R. S., Lyles, C. M., McAuliffe, C. A., Woosley, J. T., & Kupper, L. L. (1993a). Cigarette smoking, alcohol and risk of colorectal adenomas. Gastroenterology, 104, 1445-1451.
- Sandler, R. S., Lyles, C. M., Peipins, L. A., McAuliffe, C.A., Woosley, J. T., & Kupper, L. L. (1993b). Diet and risk of colorectal adenomas: macronutrients, cholesterol and fiber. Journal of the American Cancer Institute, 85, 884-891.
- SAS Institute (1989). Cary, NC: SAS Institute, Inc.
- Schapira, D. V., Kumar, N. B., Lyman, G. H., & Baile, W. F. (1991). The effect of duration of intervention and locus of control on dietary change. American Journal of Preventive Medicine, 7(6), 341-347.
- Schapira, D. V., Kumar, N. B., Lyman, G. H., & McMillan, S. C. (1990). The value of current nutrition information. Preventive Medicine, 19, 45-53.
- Schiffman, M. H. (1987). Diet and faecal genotoxicity. Cancer Surveys, 6, 653-672.
- Schober, S. E., Comstock, G. W., Helsing, K. J., Salkeld, R. M., Morris, J. S., Rider, A. A., & Brookmeyer, R. (1987). Serologic precursors of cancer: I. Prediagnostic serum nutrients and colon cancer risk. American Journal of Epidemiology, 126, 1033-1041.

- Schwalb, E., & Crosson, K. (1988). Helping you help your patients: the patient education program of the National Cancer Institute. Patient Education, 15(5), 651-655.
- Selby, J. V., & Friedman, D. G. (1989). Sigmoidoscopy in the periodic health examination of asymptomatic adults. Journal of the American Medical Association, 261, 555-601.
- Shekelle, R., Lepper, M., Lius, S., Maliza, C., Ragnor, R. J., & Rossof, A. H. (1981). Dietary vitamin A and risk of cancer in the western electric study. Lancet, 2 1185-1189.
- Slattery, M. L., Sorrenson, A. W., & Ford, M. H. (1988). Dietary calcium intake as a mitigating factor in colon cancer. American Journal of Epidemiology, 128, 504-514.
- Smiciklas-Wright, H., Peclian, S., Byrd-Bredbenner, C., & Shannon B. (1984). Clients' comprehension of a computer analyzed dietary intake printout. Journal of Nutrition Education, 16, 67-69.
- Smith, A. E., & Lloyd-Still, J. D. (1983). Value of computerized dietary analysis in pediatric nutrition: an analysis of 147 patients. Journal of Pediatrics, 103, 820-823.
- Sorensen, G., Hunt, M. K., Morris, D. H., Donnelly, G., Freeman, L., Ratcliffe, B. J., Hsieh, J., Larson, K., & Ockene, J. K. (1990). Promoting healthy eating patterns in the worksite: the Treatwell intervention model. Health Education Research, 5, 505-515.
- Sorensen, A., Seltzer, R., & Wyse, B. (1983). Personal computers for health. The Professional Nutritionist, 15, 1-3.
- Stryker, S. J., Wolff, B. G., Culp, C. E., Libbe, S. D., Ilstrup, D. M., & MacCarty, R. L. (1987). Natural history of untreated colonic polyps. Gastroenterology, 93, 1009-1013.
- Talmage, H., Hughes, M., & Eash, M. J. (1978). The role of evaluation research in nutrition education. Journal of Nutrition Education, 10, 169-172.
- Trock, B., Lanza, E., & Greenwald, P. (1990). Dietary fiber, vegetables and colon cancer: critical review and meta-analysis of the epidemiologic evidence. Journal of the National Cancer Institute, 82, 650-661.
- Upton, A. C. (1979). Statement on diet, nutrition and cancer. Hearings of the Subcommittee on Nutrition, Senate Committee on Agriculture, Nutrition and Forestry. Washington, DC: US Government Printing Office, 56-1510.
- US Senate. (1977). Select Committee on Nutrition and Human Needs: dietary goals for the United States. Washington, DC., Government Printing Office, 1-83.

- USDA United States Department of Agriculture.(1943). War Food Administration. Nutrition and Food Conservation Branch. National wartime nutrition guide. Folder. Washington, DC, US Government Printing Office.
- USDA United States Department of Agriculture. (1946). Agriculture Research Services. Bureau of Human Nutrition and Home Economics. National Food Guide rev. ed Leaflet no. 288. Washington, DC, Government Printing Office.
- USDA United States Department of Agriculture. (1988). Human Nutrition Information Service. Nationwide food consumption survey, continuing survey of food intakes by individuals, women 19-50 and their children 1-5 years, 4 days. Washington, DC., 1-220.
- USDA-United States Department of Agriculture and USDHHS-United States Department of Health and Human Services. (1990). Nutrition and your health: dietary guidelines Americans, 3rd ed. Home and Garden Bulletin, 232, 5-14.
- USDHHS United States Department of Health and Human Services. (1986). NCI Monographs: cancer control objectives for the nation: 1985-2000. Washington, DC: NIH publication no. 86-2880, 3-93.
- USDHHS-United States Department of Health and Human Services. (1989). National Institutes of Health, National Cancer Institute. Cancer statistics review, 1973-1988. Bethesda MD:NIH publication no. 89-2789, I.12-VI.16.
- USDHHS United States Department of Health and Human Services. (1988). The Surgeon General's Report on Nutrition and Health. Washington, DC, PHS no. 88-50210, 177-247.
- USDHHS-United States Department of Health and Human Services. (1991). Healthy People 2000: National Health Promotion and Disease Prevention Objectives. Public Health Service; Washington DC: PHS no. 91-50213; 1-129.
- Vogel, V. G., & McPherson, R. S. (1989). Dietary epidemiology of colon cancer. Hematology/Oncology Clinics of North America, 3, 35-62.
- Wald, N. J., Thompson, S. G., Densem, J. W., Boreham, J., & Bailey, A. (1987). Serum vitamin E and subsequent risk of cancer. British Journal of Cancer, 56, 69-72.
- Wald, N. J., Thompson, S. G., Densem, J. W., Boreham, J., & Bailey, A. (1988). Serum beta-carotene and subsequent risk of cancer: results from the BUPA study. British Journal of Cancer, 57, 428-433.
- Watson, R. R., & Leonard, T. K. (1986). Selenium and vitamins A, E and C: nutrients with cancer prevention properties. Journal of the American Dietetic Association, 86, 505-510.

- Wise, A., Liddell, J. A., & Lolke, G., M. (1987). Food habits and nutrition education-computer aided analysis of data. Human Nutrition Applied Nutrition, 41A, 118-134.
- Willett, W. C. (1990). Nutritional Epidemiology. New York: Oxford Publishing, 1-396.
- Willett, W. C., Polk, B. F., Underwood, B. A., Stampfer, M. J., Pressel, S., Rosner, B., Taylor, J. O., Schneider, K. & Hames, C. G. (1984). Relation of serum vitamin A and E and carotenoids to the risk of cancer. New England Journal of Medicine, 310, 430-434.
- Wynder, E. L., & Reddy, B. S. (1973). Studies of large bowel cancer: human leads to experimental application. Journal of the National Cancer Institute, 50, 1099.
- Zaridze, D. G. (1983). Environmental etiology of large bowel cancer. Journal of the National Cancer Institute, 70, 389-400.
- Zeigler, R. G. (1989). A review of epidemiologic evidence that carotenoids reduce the risk of cancer. Journal of Nutrition, 119, 116-122.
- Zeigler, R. G. (1991). Vegetables, fruits and carotenoids and the risk of cancer. American Journal of Clinical Nutrition, 53, 251S-259S.
- Zeigler, R. G., Blot, W. J., Hower, R., William, B. A., & Fraumeni, J. F. (1981). Protocol for a study of nutrition factors and low risk of colon cancer in southern retirement areas. Cancer Research, 41, 3724-3726.

APPENDIX A
TARGET POPULATION PROFILE

Table A-1

Target Population Profile

	Cases n (%)	Controls n(%)	Total
	n=236 (36.6)	n=409 (63.4)	N=120
Gender			
Male	152(44.5)	165(40.3)	270(41.9)
Females	131(55.5)	244(59.7)	375(58.1)
Mean Age (min,max)	62.3(31,89)	58.7(30,88)	60.2(30,89)
Mean Height (min,max)	66.8(59,76)	66.3(53,78)	66.5(53,78)
Mean Weight (min,max)	168.7(95,330)	162.8(72,375)	164.9(72,375)
Mean BMI (min,max)	26.5(13.4, 50.1)	26.0(14.2,52.3)	26.2(13.4,52.3)
Male	25.3(13.4,44.8)	25.3(14.6,52.3)	25.3(13.4,52.3)
Female	25.3(17.6,50.1)	26.4(14.2,51.1)	26.8(14.2,51.1)
Race			
White	146(61.9)	264(64.6)	410(63.6)
Black	88(37.3)	136(33.3)	224(34.7)
Hispanic	1(0.4)	4(1.0)	5(0.8)
American Indian	1(0.4)	2(0.5)	3(0.5)
Asian American	0	2(0.5)	2(0.3)
Pacific Islander	0	1(0.2)	1(0.2)
Marital Status			
Single	17(7.2)	43(10.5)	60(9.3)
Married	134(57.0)	244(59.8)	378(58.8)
Widowed	52(22.1)	82(20.1)	134(20.8)
Divorced	32(13.6)	39(9.6)	71(11.0)
Education			
Highest Grade Level			
Mean (min,max)	10.7(0,17)	11.2(0,17)	11.0(0,17)
0	1(0.4)	3(0.7)	4(0.6)
1-5	25(0.8)	40(10.0)	65(10.3)
6-8	49(21.2)	67(16.7)	116(18.3)
9-11	41(17.1)	77(19.2)	118(18.6)
12	46(19.9)	72(17.9)	118(18.6)
13-16	37(16.0)	67(16.7)	104(16.4)
17	32(13.9)	76(18.9)	108(17.1)

APPENDIX B
REPRESENTATIVE NUTRIENT ANALYSIS OF INTERVIEWED SUBJECT

Table B-1

Representative Nutrient Analysis of Interviewed Subject

Please note that the following nutrient values should be considered as estimates rather than as precisely accurate values. They are based on the frequency of consumption and portion sizes estimated on the diet questionnaire.

AVERAGE DAILY NUTRIENTS		RECOMMENDED RANGES
TOTAL CALORIES	3947.9 CALORIES	DEPENDS ON AGE, SEX, ACTIVITY
PROTEIN	156.2 GRAMS	.39GRAMS PER POUND BODY WT
TOTAL FAT	156.4 GRAMS	BASED ON CALORIES
CARBOHYDRATE	479.0 GRAMS	BASED ON CALORIES
CALCIUM	2710.7 MILLIGRAMS	800-1200 MILLIGRAMS
PHOSPHORUS	2948.8 MILLIGRAMS	800-1200 MILLIGRAMS
IRON	24.4 MILLIGRAMS	10-18 MILLIGRAMS
SODIUM	5193.9 MILLIGRAMS	1100-3300 MILLIGRAMS
POTASSIUM	5506.7 MILLIGRAMS	1875-5625 MILLIGRAMS
VITAMIN A	10677.3 IUs	4000-5000 IUs
THIAMIN (B-1)	2.5 MILLIGRAMS	1-1.5 MILLIGRAMS
RIBOFLAVIN (B-2)	4.7 MILLIGRAMS	1.2-1.7 MILLIGRAMS
NIACIN	32.8 MILLIGRAMS	13-19 MILLIGRAMS
VITAMIN C	299.5 MILLIGRAMS	60-100 MILLIGRAMS
SATURATED FATE	66.7 GRAMS	APPROX 1/3 OF FAT
OLEIC ACID	58.5 GRAMS	APPROX 1/3 FAT
LINOLEIC ACID	14.9 GRAMS	APPROX 1/3 FAT
CHOLESTEROL	631.0 MILLIGRAMS	150-300 MILLIGRAMS
DIETARY FIBER	17.2 GRAMS	20-30 GRAMS
FOLATE	492.4 MICROGRAMS	400 MICROGRAMS
VITAMIN E	14.3 ALPHA TE	8-10 ALPHA TE

Table B-2

Representative Nutrient Analysis of Interviewed Subject

AVERAGE DAILY NUTRIENTS		RECOMMENDED RANGES
RETINOL EQUIV	1979.0 RE'	1000RE
CAROTENE ESTIMATE	2433.1 MICROGRAMS	NOT ESTABLISHED
RETINOL ESTIMATE	1573.0 MICROGRAMS	NOT ESTABLISHED
POLYUNSAT/SAT FAT RATIO	0.22	0.6-1.0
SODIUM/POTASSIUM RATIO	0.94	NOT ESTABLISHED
PERCENT OF CALORIES		
FROM FAT	35.6 PERCENT	LESS THAN 30 PERCENT
FROM PROTEIN	15.8 PERCENT	12%OR MORE, IF OVER 60
FROM CARBO	48.5 PERCENT	50-68 PERCENT
FROM ALCO/BEV	0.1	

REPORTED WEEKLY FREQUENCY OF CONSUMING CERTAIN FOODS:

ANY FRUIT:	24.5	BEEF:	4.0
CITRUS FRUITS AND JUICES:	4.3	PORK:	0.2
ANY VEGETABLE:	11.5	HOT DOGS/LUNCH MEATS:	0.8
VEGETABLE EXCEL POTATO/RICE:	6.0	BUTTER OR MARGARINE:	7.2
SALAD:	1.0	CHEESES EXCL COTTAGE	7.5
CARROTS:	0.6	WHOLE MILK:	21.0
TOMATOES:	0.2	ICE CREAM:	4.0
DEEP YELLOW OR DARK GREEN VEG:	1.0	PASTRIES, SWEETS, SUGAR:	24.8
WHOLE GRAINS OR BRAN CEREAL:	8.0	FRIED FISH OR CHICKEN:	0.0
EGGS (# OF EGGS)	4.0		
ALCOHOLIC BEVERAGES	0.3		

Your reported weight is 160 pounds. The desirable weight for your height is approximately 141-155 pounds.

APPENDIX C
REPRESENTATIVE RECOMMENDATIONS FOR CHANGE
PROVIDED TO AN INDIVIDUAL SUBJECT

Representative Recommendations For Change Provided To An Interviewed Subject

Your total fat intake is 35.60% of your total calories. Experts in heart disease and cancer recommend that your fat intake should be limited to 30% of your total calories. To improve your diet and your health, consider eating fewer fatty foods and more vegetables, fruits and grains. Choose more fish, poultry, beans and grains as protein sources, limit your fat intake of sweets, butter and margarine; switch to lowfat milk products. Good Health!

Your vitamin C Intake from your diet is approximately 299 mg per day. Congratulations. This is excellent. This vitamin aids in healing and promotes healthy bones and gums, and it may be important in helping to prevent a number of major diseases. Among the stresses that can increase your need for this vitamin are infections, burns, cigarette smoking, and the prolonged use of medicines, including aspirin. Help yourself to health by eating 1-2 servings of vitamin C sources everyday, such as citrus fruits and juices, tomatoes, strawberries, broccoli and other dark green vegetables.

Your diet is providing approximately 10677 IU of vitamin A per day. This meets or exceeds the recommended daily allowance. This vitamin is essential to the health of eyes, bones, skin and many other tissues. You can consume more vitamin A by eating more dark green, deep orange and other richly colored vegetables and fruits, such as cantaloupe, peaches, apricots, carrots, winter squash, sweet potatoes, spinach, broccoli and southern greens. Liver and liverwurst are excellent sources. Some mixed dishes can be good sources if they contain carrots or cheese, such as beef stew, pizza, etc.

Your intake of cholesterol is 631 mg per day. The recommended level is less than 30 mg per day. Your intake exceeds the recommended limit. Cholesterol is a type of fat which is found in animal products such as meat, whole milk and cheese, butter, cream and shrimp. Eating foods high in cholesterol can increase cholesterol levels in your blood, which in turn may increase your risk of heart attack. It is recommended that you eat more cereals, fruits and vegetables; reduce your intake of eggs; and choose lean meats such as fish and poultry, and lowfat milk and cheese products. You should consider reducing the number of eggs you eat. Each egg contains 250 mg cholesterol.

APPENDIX D
EXPLANATION OF TERMS FOUND IN NUTRIENT ANALYSIS

EXPLANATION OF TERMS FOUND IN NUTRIENT ANALYSIS

NUTRITION: Combination of processes by which the body receives and uses the materials necessary for maintenance, for energy, and for growth and renewal.

CALORIE: A unit used to express heat or energy value of food. Calories come from carbohydrate, protein, fat, and alcohol.

GRAM: Nutrient substance in food necessary for life. Carbohydrates, proteins, fats, minerals, vitamins, and water are nutrients.

CARBOHYDRATE: One of the three major energy sources in foods. The most common carbohydrates are sugars and starches. Carbohydrates are found in foods from the milk, vegetable, fruit and starch/bread groups.

PROTEIN: One of the three major nutrients in food. Protein provides about 4 calories per gram. Protein is found in foods from the milk and meat groups. Smaller amounts of protein are found in foods from the vegetable and starch/bread groups.

FAT: One of the three major energy sources in food. A concentrated source of calories; about 9 calories per gram. Fat is found in foods from the fat and meat groups. Some kinds of milk also have fat; some foods from the starch and bread group also contain fat.

SATURATED FAT: Tends to raise blood cholesterol levels. It comes primarily from animals and is often hard at room temperature. Examples of saturated fat are butter, lard, meat fat, solid shortening, palm oil and coconut.

UNSATURATED FAT: Tends to lower blood cholesterol levels. It comes from plants and is usually liquid at room temperature. Examples of unsaturated fats are vegetable oils such as corn, cottonseed, sunflower, safflower, soybean, olive and peanut oil.

FIBER: An indigestible part of certain foods. Fiber is important in the diet as roughage or bulk. Fiber is found in foods from the starch and bread, vegetable and fruit groups. There are two types of fiber: soluble and insoluble.

EXPLANATION OF TERMS FOUND IN NUTRIENT ANALYSIS (CONTINUED)

SOLUBLE FIBER: Has high water-holding capability and turns to gel during digestion. This slows digestion and the rate of nutrient absorption from the stomach and intestine. This type of fiber is found in oat bran, pectin (from fruits and vegetables) and various gums which are found in nuts, seeds and legumes such as beans, lentils and peas.

INSOLUBLE FIBER: Is found in foods such as wheat bran and other whole grains, and has poor water-holding capability. It appears to speed the passage of foods through the stomach and intestines and increases bulk.

MINERAL: Substance essential in small amounts to build and repair body tissue or control functions of the body. Calcium, iron, magnesium, phosphorous, potassium, sodium, and zinc are minerals.

VITAMINS: Substances found in food, needed in small amounts to assist in body processes and functions. Some vitamins are A, B-Complex, C, D, E, and K.

CHOLESTEROL: A fat like substance normally found in blood. A high level of cholesterol in the blood has been shown to be a major risk factor for developing heart disease. Dietary cholesterol is found in all animal products, but is especially high in egg yolks and organ meats. Eating foods high in dietary cholesterol and saturated fat tends to raise the level of blood cholesterol. Foods of plant origin such as fruits, vegetable, grains, and legumes contain no cholesterol. Cholesterol is found in foods from the milk, meat and fat food group.

SODIUM: A mineral needed by the body to maintain life.

ALCOHOL: An ingredient in a variety of beverages including beer, wine, liqueur and cordials. Pure alcohol yields about 7 calories per gram, of which more than 75% is available to the body.

APPENDIX E
HEALTH HABITS AND HISTORY QUESTIONNAIRE



Diet and Health Study

Program in Digestive Diseases and Nutrition
University of North Carolina
Chapel Hill, North Carolina

Health Habits and History Questionnaire

TODAY'S DATE

MONTH	DAY	YEAR
<input type="text"/>	<input type="text"/>	<input type="text"/>

LAST NAME	FIRST NAME	MI
<input type="text"/>	<input type="text"/>	<input type="text"/>

STREET ADDRESS:

CITY:	STATE	ZIP
<input type="text"/>	<input type="text"/>	<input type="text"/>

SOCIAL SECURITY NUMBER:

PERSONAL INFORMATION, HABITS

1. When were you born? / /
month day year
2. How old are you now? years
3. Sex: 1. Male 2. Female
4. Race or ethnic background:

1. <u> </u> White, not of Hispanic origin	4. <u> </u> American Indian/Alaskan native
2. <u> </u> Black, not of Hispanic origin	5. <u> </u> Asian
3. <u> </u> Hispanic	6. <u> </u> Pacific Islander
5. What is the highest grade in school that you have completed:
 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17+
6. What is your marital status? 1. Single 3. Widowed
 2. Married 4. Divorced/Separated
8. Have you smoked at least 100 cigarettes in your entire life? 1. *NO 2. YES

IF YES: About how old were you when you first started smoking cigarettes fairly regularly? years old
 On average, how many cigarettes did you smoke per day during the entire time you smoked? cigarettes per day
 Do you smoke cigarettes now? 1. NO 2. YES
 IF NO: How old were you when you stopped smoking? years old
 IF YES: On the average about how many cigarettes a day do you smoke now? cigarettes

9. Have you ever smoked a pipe or cigars regularly? 1. NO 2. YES

IF YES: For how many years? years
 About how much? pipes or cigars per
(day or week)
 1 2

10. During the past six months, have you taken any vitamins or minerals?
 1. NO 2. Yes, but not regularly 3. Yes, fairly regularly

IF YES, what do you take fairly regularly? Indicate # or PILLS per DAY, WEEK or MONTH

<i>Multiple Vitamins</i>		<u>Day</u>	<u>Week</u>	<u>Month</u>	
1. One-a-day type	___ pills per	1 ___ 2 ___	3 ___	3 ___	
2. Stress-tabs type	___ pills per	1 ___ 2 ___	3 ___	3 ___	
3. Therapeutic, Theragran type	___ pills per	1 ___ 2 ___	3 ___	3 ___	
What brand of multiple vitamin/mineral do you usually take _____					
<i>Other Vitamins</i>		<u>Day</u>	<u>Week</u>	<u>Month</u>	<u>How many milligrams or IUs per pill?</u>
4. Vitamin A	___ pills per	1 ___ 2 ___	3 ___	3 ___	___ IU per pill
5. Vitamin C	___ pills per	1 ___ 2 ___	3 ___	3 ___	___ mg per pill
6. Vitamin E	___ pills per	1 ___ 2 ___	3 ___	3 ___	___ IU per pill
7. Calcium or dolomite	___ pills per	1 ___ 2 ___	3 ___	3 ___	___ mg per pill
8. Beta carotene	___ pills per	1 ___ 2 ___	3 ___	3 ___	___ mg per pill
9. Vitamin D	___ pills per	1 ___ 2 ___	3 ___	3 ___	___ IU per pill
<i>Other:</i>					
1. <u> </u> Yeast		2. <u> </u> Selenium		3. <u> </u> Zinc	4. <u> </u> Iron
5. <u> </u> Beta carotene		6. <u> </u> Cod liver oil		7. <u> </u> Other	

11. Are you on a special diet?
 1. NO 2. Weight loss 3. For medical condition 4. Vegetarian
 5. Low salt 6. Low cholesterol 7. Weight gain
12. How often do you eat the following foods from *restaurants or fast food places*?

RESTAURANT FOOD	1 Almost every day	2 2-4 times a week	3 Once a week	4 1-3 times a month	5 5-10 times a year	6 1-4 times a year	7 Never or less than once a year
Fried chicken							
Burgers							
Pizza							
Chinese foods							
Mexican foods							
Fried fish							
Other foods							

FOODS: INTRODUCTION

The next section is about your *usual* eating habits. I will be asking you a number of questions about the foods you normally eat. For each type of food, I would like you to tell me how many times you eat it per day, week, month or year. I would also like to know, for each food, whether your normal serving size is small, medium, or large (or even extra large). If you're not sure, I can tell you what we mean by a "medium" serving for any food in the list.

Thinking back over the past year, how often do you eat the following foods.

	Medium Serving	How often?					Your Serving Size			Comments
		d a y	w e e k	m o n t h	y e a r	n e v e r	S	M	L	
FRUITS & JUICES										
EXAMPLE - Apples, applesauce, pears	(1) or 1/2 cup			4					✓	
Not counting juices, how many fruits do you usually eat?										
Apples, applesauce, pears	(1) or 1/2 cup									
Bananas	1 medium									
Peaches, apricots (canned, frozen or dried, whole year)	(1) or 1/2 cup									
Peaches, apricots, nectarines (fresh, in season)	1 medium									
Cantaloupe (in season)	1/4 medium									
Watermelon (in season)	1 slice									
Strawberries (fresh, in season)	1/2 cup									
Oranges	1 medium									
Orange juice or grapefruit juice	6 oz. glass									
Grapefruit	(1/2)									
Tang or other breakfast drinks	6 oz glass									
Other fruit juices, fortified fruit drinks, Hi-C	6 oz. glass									
Any other fruit, including berries, fruit cocktail	1/2 cup									
VEGETABLES										
Not counting salad or potatoes, about how many vegetables do you eat?										
How often do you eat raw vegetables (such as carrots, celery, cauliflower, etc.) as a snack										
String beans, green beans	1/2 cup									
Peas	1/2 cup									
Chili with beans	3/4 cup									
Other beans such as baked beans, pintos, kidney beans, limas	3/4 cup									

		D	W	M	Y	N	S	M	L	Comments
		a	k	e	r	v				
Corn	1/2 cup									
Winter squash, baked squash	1/2 cup									
Tomatoes, tomato juice	(1) of 6 oz.									
Red chili sauce, taco sauce, salsa picante	2 Tblsp. sauce									
Broccoli	1/2 cup									
Cauliflower or Brussels sprouts	1/2 cup									
Spinach (raw)	3/4 cup									
Spinach (cooked)	1/2 cup									
Mustard greens, turnip greens, collards	1/2 cup									
Cole slaw, cabbage, sauerkraut	1/2 cup									
Carrots, or mixed vegetables containing carrots	1/2 cup									
Green salad	1 med bowl									
Salad dressing, mayonnaise (including on sandwiches)	2 Tblsp									
French fries and fried potatoes	3/4 cup									
Sweet potatoes, yams	1/2 cup									
Other potatoes, including boiled, baked, potato salad	(1) or 1/2 cup									
Rice	3/4 cup									
Any other vegetable, incl. cooked onions, summer squash	1/2 cup									
Butter, margarine, other fat on vegetables, potatoes etc	1 tsp.									
MEAT, FISH, POULTRY & MIXED DISHES		D	W	M	Y	N	S	M	L	
		a	k	e	r	v				
Hamburgers, cheeseburgers, meat loaf, beef burritos/taco	1 medium									
Beef - steaks, roasts	4 oz									
Beef stew or pot pie with carrots, other vegetables	1 cup									
Liver, including chicken livers	4 oz									
Pork, including chops, roasts	4 oz.									
Fried chicken	2 sm or 1 lg pc									
Chicken or turkey, roasted, stewed or broiled	2 sm or 1 lg pc									
Fried fish or fish sandwich	4 oz. or 1 sand									
Tuna fish, tuna salad, tuna casserole	1/2 cup									
Shell fish (shrimp, lobster, crab, oyster, etc.)	(5) 1/4 cup, 3 oz									
Other fish, broiled or baked	4 oz									
Spaghetti, lasagna, other pasta with tomato sauce	1 cup									
Pizza	2 slices									
Mixed dishes with cheese (such as macaroni and cheese)	1 cup									
Liverwurst	2 slices									
Hot dogs	2 dogs									
Ham, bologna, salami, lunch meats	2 slices									
Vegetable soup, vegetable beef, minestrone, tomato soup	1 med. bowl									
Other soups	1 med. bowl									
BREADS/SALTY SNACKS/SPREADS		D	W	M	Y	N	S	M	L	
		a	k	e	r	v				
Biscuits, muffins, burger rolls (incl. fast foods)	1 med piece									
White bread (including sandwiches), bagels, etc. crackers	2 slices, 3 crackers									
Dark bread, including whole wheat, rye, pumpernickel	2 slices									
Corn bread, corn muffins, corn tortillas	1 med piece									
Salty snacks (such as chips, popcorn)	2 handfuls									
Peanuts, peanut butter	2 Tblsp									

		D	W	M	Y	N	S	M	L	Comments
		a	k	e	r	v				
Butter on bread or rolls	2 pats									
Margarine on bread or rolls	2 pats									
Gravies made with meat drippings, or white sauce	2 Tbsp									
BREAKFAST FOODS		D	W	M	Y	N	S	M	L	
		a	k	e	r	v				
High fiber, bran or granola cereals, shredded wheat	1 med bowl									
Highly fortified cereals - Product 19, Total, Just Right	1 med bowl									
Other cold cereals, such as Corn Flakes, Rice Krispies	1 med bowl									
If you eat cold cereal, what kind do you eat most often										
Cooked cereals	1 med. bowl									
Grits	1 med bowl/									
Sugar added to cereal	2 tsp									
Eggs	1 egg = small, 2 eggs = medium									
Bacon	2 slices									
Sausage	2 patties or links									
SWEETS		D	W	M	Y	N	S	M	L	
		a	k	e	r	v				
Ice cream	1 scoop									
Ice milk	1 scoop									
Doughnuts, cookies, cakes, pastry	1 pc - 3 cookies									
Pumpkin pie, sweet potato pie	1 med slice									
Other pies	1 med slice									
Chocolate candy	small bar, 1 oz									
Other candy, jelly, honey, brown sugar	3 pc, 1 Tbsp									
DAIRY PRODUCTS		D	W	M	Y	N	S	M	L	
		a	k	e	r	v				
Cottage cheese	1/2 cup									
Other cheese and cheese spreads	2 slices or 2 oz									
Flavored yogurt, frozen yogurt	1 cup									
Plain yogurt	1 cup									
Whole milk and bevs. with whole milk (not on cereal)	8 oz. glass									
2% milk and bevs with 2% milk (not on cereal)	8 oz. glass									
Skim milk, 1% milk or buttermilk (not on cereal)	8 oz glass									
BEVERAGES		D	W	M	Y	N	S	M	L	
		a	k	e	r	v				
Regular soft drinks	12 oz can-bottle									
Diet soft drinks	12 oz can-bottle									
Beer	12 oz can-bottle									
Wine	1 med glass									
Liquor	1 shot									
Decaffeinated coffee	1 med cup									
Coffee, not decaffeinated	1 med cup									
Tea (hot or iced)	1 med cup									
Lemon in tea	1 tsp									
Non-dairy creamer in coffee or tea	1 Tbsp									
Milk in coffee or tea	1 Tbsp									
Cream (real) or Half and Half in coffee or tea	1 Tbsp									
Sugar in coffee or tea	2 tsp									
Artificial sweetener in coffee or tea	1 packet									
Glasses of water, not counting in coffee or tea	8 oz glass									

Think about your diet over the last year and the responses you have just made on this questionnaire. Are there any foods not mentioned which you ate at least once a week, even in small quantities, or ate frequently in a particular season? Consider other meats, breakfast foods, catsup, green chilies or jalapenos, avocado (guacamole), Mexican dishes, Chinese or other ethnic foods, other fruits or vegetables, as well as nutritional supplements (bran, etc.).

	How Often?		Your Serving Size		
	Day	Week	S	M	L
1.					
2.					
3.					
4.					
5.					
6.					

- | | | | | | |
|----|---------------------------|----|-----------------------------|----|--------------------------------|
| 43 | asparagus | 22 | instant breakfast, metropol | 21 | pancakes, waffles |
| 44 | avocado | 69 | lemons or lemon juice | 67 | papayas |
| 46 | bean sprouts | 66 | mangoes | 61 | pineapple or pineapple juice |
| 48 | beets | 7 | Mexican dishes | 10 | Polish or Italian sausage |
| 71 | bran | 24 | milkshake | 62 | prunes or prune juice |
| 33 | catsup | 5 | mixed dish with chicken | 23 | pudding |
| 6 | Chinese dishes | 4 | mixed dish with meat | 9 | refried beans or bean burritos |
| 64 | cranberry juice cocktail | 12 | noodles | 8 | seafood Creole |
| 11 | cream soups | 70 | nuts and seeds | 31 | sour cream, dips |
| 32 | diet salad dressing | 41 | onions | 42 | summer squash |
| 65 | grapes | 25 | other dairy products | 44 | sweet green peppers |
| 34 | green chilies, jalapenos | 26 | other dessert, sweet | 45 | sweet red peppers |
| 63 | Hi-C | 79 | other vegetable/fruit | 3 | tofu |
| 68 | honey dew or casaba melon | 88 | other not mentioned here | 1 | veal, lamb |

	1 Seldom/Never	2 Sometimes	3 Often/Always
15 How often do you eat the skin on chicken?			
How often do you eat the fat on meat?			
How often do you add salt to your food?			
How often do you add pepper to your food?			
How often do you eat food seasoned with garlic?			

16. How often do you use fat or oil in cooking? For example, in frying eggs, meat or vegetables?
 _____ time(s) per. 1 day 2 week 3 month

17. What do you usually cook with? (one or two answers OK)
- | | |
|---|---|
| 1 <input type="checkbox"/> Don't know or don't cook | 5 <input type="checkbox"/> Oil |
| 2 <input type="checkbox"/> Soft margarine | 6 <input type="checkbox"/> Lard, fatback, bacon fat |
| 3 <input type="checkbox"/> Stick margarine | 7 <input type="checkbox"/> Pam or no oil |
| 4 <input type="checkbox"/> Butter | |
18. What kind of fat do you usually add to cooked vegetables, potatoes etc.? (one or two answers OK)
- | | |
|--|--|
| 1 <input type="checkbox"/> Do not add fat | 4 <input type="checkbox"/> Butter |
| 2 <input type="checkbox"/> Soft margarine | 5 <input type="checkbox"/> Half butter, half margarine |
| 3 <input type="checkbox"/> Stick margarine | 6 <input type="checkbox"/> Lard, fatback, bacon fat |
19. How tall are you? ____ feet ____ inches.
20. How much do you weigh? ____ pounds

INTERVIEWER REMARKS

Interviewer initials

Respondent's cooperation was:

- | |
|---------------|
| 1 = very good |
| 2 = good |
| 3 = fair |
| 4 = poor |
| 9 = unknown |

The overall quality of this interview is:

- | |
|------------------------|
| 1 = high quality |
| 2 = generally reliable |
| 3 = questionable |
| 4 = unsatisfactory |
| 9 = unknown |

The MAIN REASON for unsatisfactory or questionable quality of information was because the respondent:

- | |
|--|
| 1 = did not know enough information regarding the topic |
| 2 = did not want to be more specific |
| 3 = did not understand or speak English well |
| 4 = was bored or uninterested |
| 5 = was upset, angry, or emotionally unstable |
| 6 = had poor hearing or speech |
| 7 = was confused or distracted by frequent interruptions |
| 8 = was physically ill |
| 9 = unknown |

Total time for interview _____ minutes

APPENDIX F

**ADDITIONAL QUESTIONS ASKED TO DETERMINE THE USEFULNESS
OF PERSONALIZED NUTRIENT ANALYSIS**

ADDITIONAL QUESTIONS ASKED TO DETERMINE USEFULNESS OF NUTRIENT ANALYSIS

1. Do you remember receiving a nutrient analysis of your diet?

No Yes IF YES

Did you find the information helpful?

No Yes IF YES

Did you follow the recommendations?

No Yes IF YES

For how long?

2. What do you remember most about the nutrient analysis?

3. Do you presently follow any of the recommendations?

No Yes IF YES

What do you follow?

4. Are there any other factors that might have influenced you to alter your diet?

No Yes IF YES

What are these factors?

Sources of nutritional advice?

5. Would you have found it helpful to receive more detailed information on how to alter your diet?

No Yes IF YES

What type of information?

APPENDIX G
PATIENT INFORMATION INTERVIEW CONTACT SHEET

MEDICAL PATIENT INFORMATION INTERVIEW CONTACT SHEET

STUDY SUBJECT: _____

STUDY ID#: _____

RECORD #: _____

ADDRESS: _____

1ST PHONE: _____ 2ND PHONE: _____

SIGNIFICANT OTHER: _____

RELATIONSHIP TO SUBJECT: _____

RESULT OF CONTACT: _____

WILLING TO PARTICIPE: YES NO

(IF NO, RECORD REASON IN COMMENTS SECTION)

DATE OF INTERVIEW: _____

TIME OF INTERVIEW: _____

DATE DATE ENTERED INTO COMPUTER: _____ INITIALS _____

DATE DATA VERIFIED: _____ INITIALS: _____

COMMENTS: _____
