NUTRITION AND CANCER, 27(2), 150–156

General Guidelines for a Low-Fat Diet Effective in the Management and Prevention of Nonmelanoma Skin Cancer

Suzanne Jaax, Lynne W. Scott, John E. Wolf, Jr., John I. Thornby, and Homer S. Black

Abstract: A dietary intervention trial has shown a significant reduction in occurrence of actinic keratosis and nonmelanoma skin cancer in skin cancer patients who adopt diets in which the percentage of calories from fat is markedly lowered. The purpose of this study was to examine the dietary parameters of a low-fat diet found to be effective in reducing occurrence of skin cancer. Skin cancer patients were taught fat reduction strategies to complement their individual food preferences and life-styles. Diet composition was calculated using standard dietary assessment and nutrient analysis techniques. The dietary intervention was effective in reducing the percentage of calories from fat to 21% by Month 4 and maintaining that level for the remainder of the two-year study. Practical dietary advice with respect to reduction of percentage of calories from fat, along with an increase in the intake of grains, fruits, and vegetables, could make an important contribution to the management and prevention of skin cancer.

Introduction

It has been assumed for centuries that diet plays an important role in development of cancer and other major chronic diseases (1). Consequently, dietary advice, much of it anecdotal, has abounded. However, in the 1950s it became apparent that dietary habits had a major impact on public health in general, and a number of organizations and governmental bodies undertook the development of dietary guidelines as preventive measures to specific major chronic diseases, including cancer. For the most part, these recommendations have been based on the strong correlations between the incidence of certain human cancers (notably breast, colorectal, prostate, pancreatic, ovarian, and cervical) and the amount of fat in the diet (2). Whereas analytic epidemiologic studies have provided the principal evidence associating high dietary fat intake with cancer incidence (3–5), these types of studies are fraught with methodological issues that, at times, have yielded inconclusive or conflicting results. Most problems encountered have been due to the complex nature of the human diet in a free-living population; the difficulties in measuring food intake and analyzing dietary information; and the special requirements of the nutritionist, who seeks methods that accurately reflect current food intake, as opposed to the epidemiologist, who requires assessment of dietary patterns that are stable over long periods of time, usually years if cancer induction is under study (6). Generally, dietary history questionnaires and surveys, while providing epidemiologists with large sample sizes, lack validation procedures that would demonstrate that the method measures what it is intended to measure (7). This complicates observational-type studies, such as case-control studies, where potential misclassification of exposure status due to the subject's recall of dietary history impacts the association to disease status. Indeed, with regard to skin cancer, the only epidemiologic investigations, case-control and prospective studies, found no association with incidence of nonmelanoma skin cancer and dietary fat intake (8,9). Regardless of the nature of the methodological complication, it is not surprising that the public is often confused with respect to what dietary recommendations they should follow.

The rather severe limitations of observational studies of diet and cancer can be circumvented by randomized intervention designs whereby direct answers to the question of dietary impact on cancer incidence can be obtained (10). Observational studies employ instruments of limited accuracy for determination of dietary status, e.g., fat intake, whereas intervention studies create a dietary difference, then frequent dietary assessment is performed, whereby dietary fat exposure levels and disease status can be directly compared. Nonmelanoma skin cancer is particularly suited to this type of study. The high prevalence of skin cancer (11) and the identification of the relative risks of skin cancer patients developing subsequent skin cancers within two years (28% cumulative rate) (12) make it practical to obtain sufficient numbers of patients whereby significant comparisons between study groups can be made within a relatively short-term study. Furthermore, the rationale for undertaking such a study is supported experimentally. The first indication that high dietary fat intake could influence the development of ultra-

S. Jaax and L. W. Scott are affiliated with the Department of Medicine and J. E. Wolf, Jr., and H. S. Black with the Department of Dermatology, Baylor College of Medicine, Houston, TX 77030. J. I. Thornby and H. S. Black are affiliated with the Veterans Affairs Medical Center, Houston, TX 77030.

violet (UV) radiation-induced skin cancer in experimental animals was reported in 1939 (13). Although investigations into this relationship did not take place for another 45 years, in the 1980s a series of animal studies showed that a high level of dietary fat intake markedly shortened the time between UV exposure and tumor appearance and increased the number of tumors that developed (14,15). Furthermore, high levels of dietary fat affected skin cancer development at the promotional stage of UV carcinogenesis, i.e., after the cancer-causing dose of UV had been delivered (16). Perhaps more important, switching from a high-fat to a low-fat diet immediately after delivery of the UV-initiating dose negated the exacerbating effect of high fat intake. The latter finding suggested that dietary modification, even after a cancer-causing exposure to UV, might represent a potentially important intervention strategy in the prevention of nonmelanoma skin cancer. The potential of a low-fat intervention became apparent early in the first such clinical trial (17). Of 76 patients completing the study, those in the low-fat intervention group had reduced their percentage of calories from fat from 39% to 21% after eight weeks and maintained it at or below this level for the remainder of the two-year study. The percentage of calories from fat in the control (nonintervention) group did not drop below 36% during the study period. A clear and significant difference in numbers of actinic keratoses between groups occurred after 8–12 months, with patients in the control group diagnosed with new keratoses four times as often as those in the low-fat group. On the basis of diet alone, patients in the control group consuming high levels of fat were found to be at 4.7 times greater risk of having one or more actinic keratoses during the two-year period than similar patients in the low-fat intervention group. The large decrease in percentage of calories from fat significantly reduced the occurrence of nonmelanoma skin cancer as well, although the effect became apparent later, i.e., between 16 and 24 months (18). Although the low-fat diet was tailored for each individual in the intervention group of the clinical trial and dietary reinforcement and assessment were closely supervised by an experienced dietitian, nutrient analyses provided here suggest general dietary guidelines for a low-fat diet, the implementation of which could play an important role in the management and prevention of nonmelanoma skin cancer.

Materials and Methods

Patient Demographics

Of 133 skin cancer patients recruited for the two-year clinical trial, 115 successfully completed the study. After meeting the inclusion criteria (17,18), patients were randomly assigned to the control group (usual diet) or the intervention group (diet with 20% of calories from fat). Nineteen of the 58 patients assigned to the control group were female compared with 26 of the 57 assigned to the intervention group. The mean ages were 52.3 ± 13.2 and 50.6 ± 9.7 years for patients in the control and intervention groups, respectively. The mean body weights at time of randomization were 79.8 ± 14.9 and 79.4 ± 18.1 kg for the control and intervention groups, respectively. At baseline, the mean percentage of calories from fat was 40 ± 4% and 39 ± 3% in the control and intervention groups, respectively. There were no significant differences between groups at time of randomization due to gender, age, body weight, or percentage of calories from fat.

Dietary Intervention

Patients in the dietary intervention group attended eight weekly diet classes and monthly follow-up classes taught by a dietitian. The dietitian used an individual approach, in which patients learned how to adapt low-fat eating habits to their food preferences and life-styles. Each patient was given a "fat gram goal" (19), which defined the grams of fat that would provide 20% of calories from fat (Table 1). The caloric requirement used in determining the fat gram goal was the caloric level from the baseline food record analysis for that patient. The objective of the "grams of fat" method was to maintain the grams of fat consumed within the fat gram goal, thereby achieving the study goal of 20% of calories from fat. This method for controlling fat intake permitted considerable flexibility in food choices, since foods with little or no fat were emphasized but higher-fat foods could be included as long as the fat gram goal was not exceeded.

Patients were given educational materials that provided grams of fat in generic foods, brand-name foods, and foods in restaurants and fast-food establishments (20,21). Patients were taught fat reduction strategies, including replacing high-fat foods with foods containing little or no fat, decreasing the quantity of high-fat foods, and modifying high-fat food preparation techniques. Particular attention was paid to practical issues such as selecting low-fat foods in the supermarket and when eating out. The introduction of numerous fat-modified products into the marketplace during the study made it possible for patients to select from a much broader array of fat-free and low-fat foods.

To maintain body weight (a condition of the study to ensure that the effects observed were due to changes in fat intake but not weight loss), patients were instructed to increase their intake of carbohydrate, particularly complex carbohydrate. Patients in the intervention group were thus advised to increase their intake of grains, fruits, and vegetables to compensate for reduction in fat intake. In addition, a wide variety of foods were emphasized in order for par-

Table 1. Grams of Fat Equal to 20% of Calories From Fat at Various Calorie Levels

Calorie Level	Grams of Fat
1,200	27
1,500	33
2,000	44
2,500	56
3,000	67
3,500	78

ticipants to meet the recommended dietary vitamin and mineral allowances for their age and gender.

Nutrient Analyses

Nutrient analyses for seven sets of four-day food records were performed using the Minnesota Nutrition Data System (NDS) software (developed by the Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN), Food Database versions 3A–9A, and Nutrient Database versions 18–24 (22,23). Advantages of the NDS include the level of specificity for describing foods, the currency and completeness of the nutrient database, and the level of standardization and quality control checks incorporated into the system. Specificity in describing foods is enhanced by use of brand-name descriptions, variable ingredients in recipes, and computerized formulas for standard food preparations.

Dietary Assessment

Baseline and follow-up dietary data were compiled from seven-day food records, from which four days (Monday, Wednesday, Saturday, and Sunday) were selected for analyses. Food records were verified for types of foods, amounts, and methods of preparation by the dietitian (23). All patients in the control and intervention groups were examined at four-month intervals after baseline (Months 4, 8, 12, 16, 20, and 24) by dermatologists unaware of their dietary treatment assignments. At that time, patients were also scheduled to see the dietitian to verify their food records used for nutrient analyses. Comparisons of dietary data between the two groups at baseline (at time of randomization) and between the combined responses from Months 4–24 were made using an unpaired t-test (24) (Tables 1–5). Patient body weight, total calories, and percentage of calories from fat within the control and the intervention groups were compared by analysis of variance across each of the four-month evaluation intervals (Figures 1–3). Significant changes from baseline in these parameters were assessed using Dunnett's multiple comparison criteria. Cross-sectional comparisons between groups were performed using nonpaired t-tests. In all comparisons, significance was based on two-tailed tests with $p \leq 0.05$.

Numbers of new confirmed skin cancers per patient were totaled in eight-month intervals of the two-year study period. Each of the last two eight-month periods within each dietary group was compared with the respective initial eight-month period using the Wilcoxon signed rank test. Corresponding eight-month periods of the two dietary groups were compared using the Wilcoxon rank-sum test.

Results

The effectiveness of the dietary intervention is reflected in Figures 1–3. There were no significant differences in caloric intake (Figure 1) or weight loss (Figure 2) between control and intervention groups at any time during the two-year study. However, by Month 4, patients in the intervention group had reduced their percentage of calories from fat from 39% to 21% ($p < 0.001$), a level maintained during the remainder of the study (Figure 3).

The influence of reduction in the percentage of calories from fat on skin cancer occurrence is shown in Figure 4 (25). Skin cancer occurrence in the control group did not change significantly from the baseline period, i.e., at either of the two later eight-month periods. However, cancer occurrence in the intervention group was significantly ($p < 0.02$) lower in the last eight-month period than at baseline, and the two dietary groups differed significantly ($p < 0.01$) during the last eight-month evaluation period. The cumula-

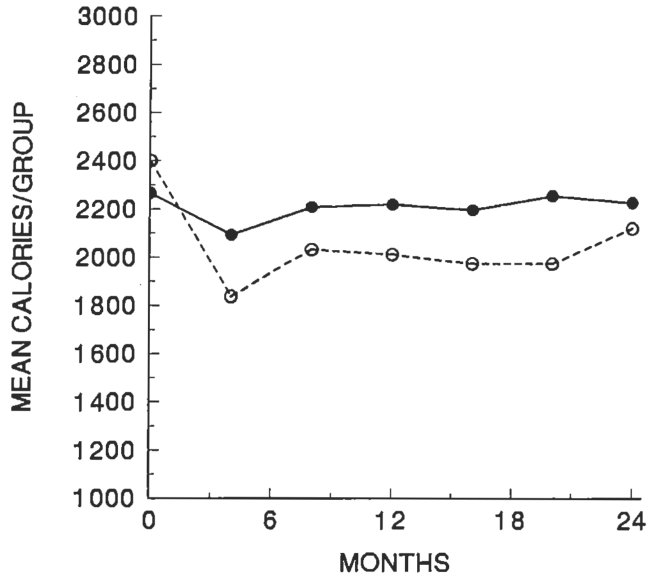

Figure 1. Mean caloric intake of control (*filled circles*) and low-fat dietary intervention (*open circles*) groups.

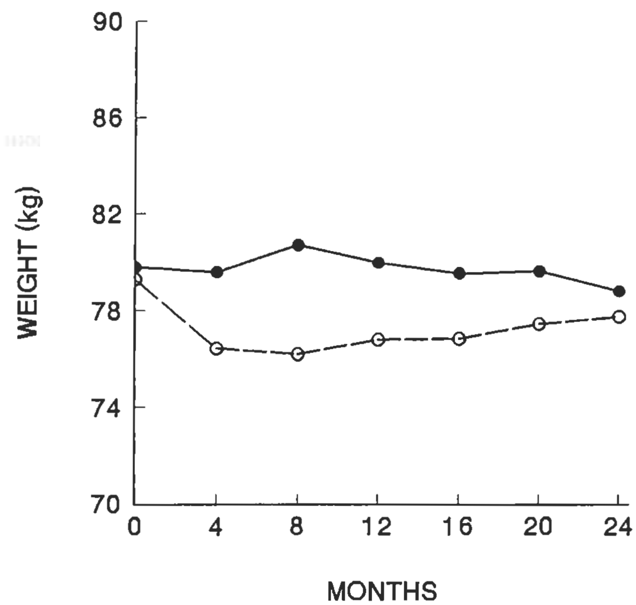

Figure 2. Mean body weights of control (*filled circles*) and low-fat dietary intervention (*open circles*) groups.

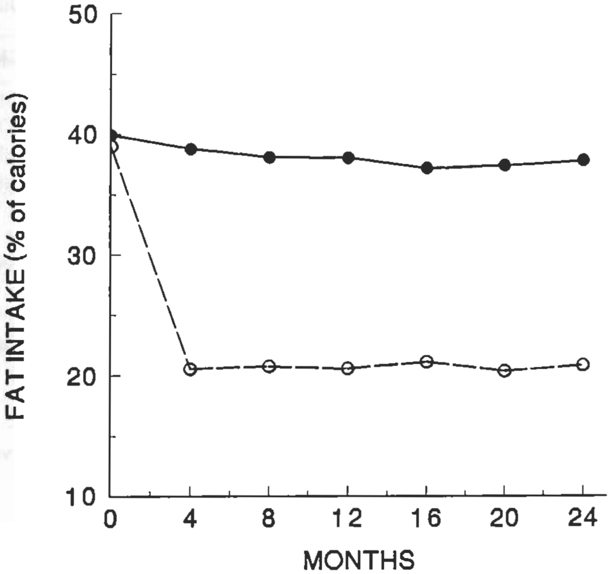

Figure 3. Dietary fat intake of control (*filled circles*) and low-fat dietary intervention (*open circles*) groups.

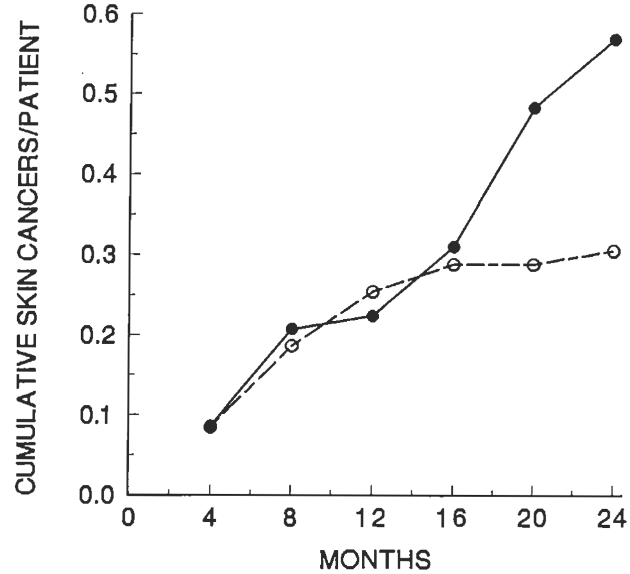

Figure 4. Influence of dietary fat intake on nonmelanoma skin cancer occurrence in control (*filled circles*) and low-fat dietary intervention (*open circles*) groups. Data points reflect cumulative numbers of nonmelanoma skin cancers per patient at each follow-up period.

tive rate of occurrence of nonmelanoma skin cancer, i.e., cumulative numbers of skin cancers per patient per time period, was 0.21 and 0.19 during the first eight-month period of the study and 0.26 and 0.02 during the last eight-month period for control and intervention groups, respectively.

Dietary composition at baseline and during the study is shown in Table 2. There were no statistically significant differences in total caloric intake between the two groups. However, there were significant changes introduced in the intervention group in all three major dietary variables. The percentage of calories from fat was reduced in the intervention group by 47%, i.e., between baseline and the mean of Months 4–24. The percentage of calories from carbohydrates increased in the intervention group by 36% and was

Table 2. Major Dietary Variables of Control and Low-Fat Dietary Intervention Groups[a]

Dietary Variable	Baseline	*P* Value	Within Study	*P* Value
Total calories, kcal				
Control	2,265 ± 659		2,196 ± 615	
Intervention	2,400 ± 629	NS	1,995 ± 564	NS
Percentage of calories from:				
Fat				
Control	39.9 ± 4.4		37.8 ± 4.1	
Intervention	38.9 ± 3.4	NS	20.7 ± 5.5	0.0001
Carbohydrate				
Control	42.7 ± 8.1		44.6 ± 6.9	
Intervention	44.2 ± 5.4	NS	60.3 ± 6.3	0.0001
Protein				
Control	15.5 ± 2.9		15.7 ± 2.4	
Intervention	15.6 ± 2.3	NS	17.7 ± 2.2	0.0001
Alcohol				
Control	3.2 ± 4.5		3.2 ± 3.9	
Intervention	2.8 ± 3.8	NS	3.2 ± 3.4	NS
Grams from:				
Fat				
Control	100.9 ± 34.3		92.9 ± 29.9	
Intervention	104.4 ± 31.4	NS	45.5 ± 16.3	0.0001
Carbohydrate				
Control	240.9 ± 79.1		244.9 ± 78.7	
Intervention	264.5 ± 74.1	NS	303.5 ± 101.4	0.0009
Protein				
Control	86.1 ± 24.9		83.9 ± 21.1	
Intervention	93.6 ± 29.9	NS	86.5 ± 23.3	NS

a: Values are means ± SD of diets at time of randomization into study (baseline) and from 4 to 24 mos (within study).

Table 3. Dietary Fat and Cholesterol[a]

Dietary Variable	Baseline	*P* Value	Within Study	*P* Value
Percentage of calories from:				
Saturated fat				
Control	13.5 ± 2.6		12.8 ± 2.0	
Intervention	13.4 ± 2.1	NS	6.6 ± 1.8	0.0001
Polyunsaturated fat				
Control	8.3 ± 2.0		7.8 ± 1.4	
Intervention	7.9 ± 1.9	NS	4.5 ± 1.3	0.0001
Monounsaturated fat				
Control	15.2 ± 2.2		14.4 ± 1.7	
Intervention	14.8 ± 1.6	NS	7.6 ± 2.2	0.0001
Quantity from:				
Saturated fat, g				
Control	34.2 ± 12.8		31.6 ± 11.4	
Intervention	35.8 ± 11.2	NS	14.6 ± 5.5	0.0001
Polyunsaturated fat, g				
Control	20.8 ± 8.5		19.1 ± 6.1	
Intervention	21.2 ± 8.1	NS	9.9 ± 3.7	0.0001
Monounsaturated fat, g				
Control	38.4 ± 14.1		35.3 ± 11.7	
Intervention	39.7 ± 12.3	NS	16.7 ± 6.4	0.0001
Cholesterol, mg				
Control	345.6 ± 155.5		322.2 ± 120.7	
Intervention	321.3 ± 149.1	NS	184.5 ± 61.3	0.0001
Linoleic acid, g				
Control	18.5 ± 7.7		16.9 ± 5.6	
Intervention	18.8 ± 7.4	NS	8.5 ± 3.3	0.0001
P/S Ratio[b]				
Control	0.64 ± 0.22		0.64 ± 0.14	
Intervention	0.61 ± 0.20	NS	0.72 ± 0.16	0.0065

a: Values are means ± SD at time of randomization into study (baseline) and from 4 to 24 mos (within study).
b: Polyunsaturated-to-saturated fat ratio.

significantly greater than that of the control group ($p < 0.001$). The percentage of calories from protein increased in the intervention group by 13% and also was significantly greater than that of the control group ($p < 0.001$). There were no significant changes in percentage of calories from alcohol within the intervention group or between groups.

With respect to fat, the grams of fat consumed by patients in the intervention group were reduced 56% and were 49% of the level consumed by patients in the control group (Table 2). In the intervention group the reduction in percentage of calories from saturated, polyunsaturated, and monounsaturated fat was 43–51% (Table 3). Cholesterol intake was reduced in the intervention group by about 43%. The polyunsaturated-to-saturated fat (P/S) ratio increased in the intervention group from 0.61 to 0.72, resulting in a significant within-study difference ($p < 0.01$) between control and intervention groups. By cross-sectional analysis, these differences were found in the last three follow-up periods, i.e., the last 12 months. Although patients in the intervention group were instructed to decrease their intake of all types of fat to achieve the goal of 20% of calories from fat, the 59% reduction in grams of saturated fat surpassed the 53% reduction in grams of polyunsaturated fat, resulting in an increase in the P/S ratio. A slight, but not statistically significant, reduction in animal protein intake was accompanied by a slight, but statistically significant ($p < 0.001$), increase in plant protein intake in the intervention group (Table 4).

Other aspects of the intervention diet are found in Table 5. Vitamin E intake decreased in patients in the intervention group and was significantly lower than in the control group

Table 4. Comparison of Animal and Plant Protein Intake[a]

Dietary Variable	Baseline	*P* Value	Within Study	*P* Value
Animal protein, g				
Control	60.1 ± 21.2		59.1 ± 16.8	
Intervention	64.4 ± 23.2	NS	55.6 ± 15.3	NS
Plant protein, g				
Control	24.9 ± 8.5		24.2 ± 7.2	
Intervention	28.2 ± 10.9	NS	30.1 ± 10.3	0.0007

a: Values are means ± SD at time of randomization into study (baseline) and from 4 to 24 mos (within study).

Table 5. Major Dietary Vitamin and Fiber Variables[a]

Dietary Variable	Baseline	*P* Value	Within Study	*P* Value
Vitamin E, mg α-TE				
Control	11.6 ± 4.9		10.8 ± 3.5	
Intervention	12.4 ± 6.2	NS	7.1 ± 2.8	0.0001
Vitamin D, μg				
Control	5.4 ± 4.0		5.5 ± 3.0	
Intervention	7.9 ± 10.2	NS	5.7 ± 3.1	NS
Vitamin C, mg				
Control	99.1 ± 69.9		94.0 ± 41.6	
Intervention	110.5 ± 62.9	NS	123.6 ± 71.5	0.0087
Vitamin A, μg RE				
Control	1,106.8 ± 903.4		1,097.7 ± 470.7	
Intervention	1,262.3 ± 1,178.7	NS	1,231.1 ± 571.1	NS
β-Carotene, μg				
Control	3,367.5 ± 3,558.4		3,277.3 ± 2,129.6	
Intervention	4,231.2 ± 5,406.1	NS	4,264.8 ± 2,456.9	0.0252
Fiber, g				
Control	17.6 ± 6.9		17.2 ± 6.4	
Intervention	19.9 ± 8.7	NS	22.5 ± 8.4	0.0002

a: Values are means ± SD at time of randomization into study (baseline) and from 4 to 24 mos (within study). α-TE, α-tocopherol equivalents; RE, retinol equivalents.

($p < 0.001$) as a result of the decreased intake of vegetable oils, which are rich in this antioxidant vitamin. Vitamins D and A did not differ significantly between groups, whereas vitamin C, β-carotene, and fiber intake in patients in the intervention group differed significantly ($p < 0.05$) from that in the control group, reflecting an increase in fruit and vegetable consumption.

Discussion

This study demonstrates that substantial dietary changes, which are effective in reducing the occurrence of actinic keratosis and nonmelanoma skin cancer (17,18), can be initiated and maintained in a free-living population of skin cancer patients. These patients were successful in reducing their fat intake from 39% of calories at baseline to approximately 21% of calories at the four-month evaluation and maintaining their percentage of calories from fat for the duration of the study. When the study goal of 20% of calories from fat is placed in perspective, data from the Third National Health and Nutrition Examination Survey (NHANES III, collected from 1988 to 1991) indicated that, in the United States, the percentage of calories from fat was 34.7% for persons between 50 and 59 years of age (26).

Inasmuch as the current study was specifically designed to examine the influence of dietary fat on nonmelanoma skin cancer, stability of body weight and caloric intake were required to prevent any possible confounding effect due to these variables. Compensation for the reduction in calories, due to the decrease in fat, was achieved by a 36% increase in percentage of calories from carbohydrates and is reflected in equivalent body weights and caloric intakes between patients in both groups. The primary strategy recommended for increasing carbohydrate intake was to increase consumption of grains, fruits, and vegetables. This strategy introduces an additional potential variable; i.e., a statistically significant inverse relationship has been reported between nonmelanoma skin cancer and high vegetable intake (27). Overall, epidemiologic evidence strongly suggests that increased vegetable and fruit intake lowers the risk for a number of site-specific cancers (28).

Although the ameliorating effect of caloric restriction on tumorigenesis has long been recognized, recent interest has focused on the relative importance of calorie vs. fat restriction, with some evidence suggesting that the major effects of dietary fats relate primarily to their contribution to caloric intake (29,30). In a prospective study, no significant associations were observed between risk of basal cell carcinoma and energy-adjusted intake of dietary fat (9). In contrast, others have reported that associations from international data between total fat consumption and the incidence of breast, colon, and prostate cancer were independent of total caloric intake or the P/S ratio (31). Regardless, a diet in which the level of fat intake has been reduced, as in this study, but in which no effort is made to maintain caloric intake, might be expected to convey even greater protection to nonmelanoma skin cancer occurrence and certainly to provide collateral health benefits (32). Thus a singular strategy of reducing fat intake, within the fat gram goal of 20% of calories from fat, should be an effective aid for the management and prophylaxis of nonmelanoma skin cancer. Practically, this would involve substitution of low-fat foods (lean meat, skinless poultry, fish, fat-free and low-fat dairy products) for their higher-fat counterparts, food preparation techniques requiring little or no fat (broiling or grilling) as opposed to high-fat techniques (frying), and substitution of fat-modified foods (e.g., fat-free and low-fat salad dressings, mayonnaise, margarine) for their high-fat counterparts. Consumption of a low-fat diet without specific efforts to maintain a constant caloric intake has been shown to result in weight loss (33,34), a result often sought by patients and one that can convey additional health benefits. Recipes, menus, techniques for food preparation, brand-name low-fat products, tips for selecting low-fat foods in restaurants, and educational materials are useful in advising patients with respect to low-fat diets (20,21,35).

In conclusion, practical dietary advice with respect to reduction of percentage of calories from fat could make an important contribution to the management and prevention of nonmelanoma skin cancer.

Acknowledgments and Notes

This study was supported by National Cancer Institute Grant CA-44383 from the National Institutes of Health (Bethesda, MD). Address reprint requests to Dr. Homer S. Black, Photobiology Laboratory, Veterans Affairs Medical Center, 2002 Holcombe Bl., Houston, TX 77030. Phone: (713) 794-7637. FAX: (713) 794-7638. E-mail: hblack@bcm.tmc.edu.

Submitted 19 July 1996; accepted in final form 18 September 1996.

References

1. O'Connor, TP, and Campbell, TC: "Dietary Guidelines." *Prog Clin Biol Res* **222,** 731–771, 1986.
2. Mettlin, C: "Methodological Issues in Epidemiologic Studies of Dietary Fat and Cancer." *Prog Clin Biol Res* **222,** 3–15, 1986.
3. Armstrong, B, and Doll, R: "Environmental Factors and Cancer Incidence and Mortality in Different Countries, With Special Reference to Dietary Practices." *Int J Cancer* **15,** 617–631, 1975.
4. Enig, MG, Munn, RJ, and Kenney, M: "Dietary Fats and Cancer Trends—A Critique." *Fed Proc* **37,** 2215–2220, 1978.
5. Hegsted, DM: "Exercise, Calories, and Fat: Future Challenges." *Adv Exp Med Biol* **322,** 1–6, 1992.
6. Lyon, JL, Gardner, JW, West, DW, and Mahoney, AM: "Methodological Issues in Epidemiological Studies of Diet and Cancer." *Cancer Res* **52** Suppl, 2040s–2048s, 1992.
7. Block, G: "A Review of Validations of Dietary Assessment Methods." *Am J Epidemiol* **115,** 494–505, 1982.
8. Graham, S: "Results of Case-Controlled Studies of Diet and Cancer in Buffalo, New York." *Cancer Res* **43** Suppl, 2409–2413, 1983.
9. Hunter, DJ, Colditz, GA, Stampfer, MJ, Rosner, B, Willett, WC, et al.: "Diet and Risk of Basal Cell Carcinoma of the Skin in a Prospective Cohort of Women." *Ann Epidemiol* **2,** 231–239, 1992.
10. Henderson, MM: "Role of Intervention Trials in Research on Nutrition and Cancer." *Cancer Res* **52** Suppl, 2030s–2034s, 1992.
11. Miller, DL, and Weinstock, MA: "Nonmelanoma Skin Cancer in the United States: Incidence." *J Am Acad Dermatol* **30,** 774–778, 1994.
12. Karagas, MR: "Occurrence of Cutaneous Basal Cell and Squamous Cell Malignancies Among Those With a Prior History of Skin Cancer." *J Invest Dermatol* **102** Suppl, 10s–13s, 1994.
13. Baumann, CA, and Rusch, HP: "Effect of Diet on Tumors Induced by Ultraviolet Light." *Am J Cancer* **35,** 213–221, 1939.
14. Black, HS, Lenger, W, Gerguis, J, and Thornby, JI: "Relation of Anti-Oxidants and Level of Dietary Lipid to Epidermal Lipid Peroxidation and Ultraviolet Carcinogenesis." *Cancer Res* **45,** 6254–6259, 1985.
15. Black, HS: "Role of Dietary Factors in UV-Carcinogenesis." *Cancer Bull* **45,** 232–237, 1993.
16. Black, HS, Thornby, JI, Gerguis, J, and Lenger, W: "Influence of Dietary Omega-6, -3 Fatty Acid Sources on the Initiation and Promotion Stages of Photocarcinogenesis." *Photochem Photobiol* **56,** 195–199, 1992.
17. Black, HS, Herd, JA, Goldberg, LH, Wolf, JE, Thornby, JI, et al.: "Effect of a Low-Fat Diet on the Incidence of Actinic Keratosis." *N Engl J Med* **330,** 1272–1275, 1994.
18. Black, HS, Thornby, JI, Wolf, JE, Jr, Goldberg, LH, Herd, JA, et al.: "Evidence That a Low-Fat Diet Reduces the Occurrence of Non-Melanoma Skin Cancer." *Int J Cancer* **62,** 165–169, 1995.
19. Clifford, C, and Stables, G: "Dietary Guidelines." In *Cancer Prevention and Control,* P Greenwald, BS Kramer, and DL Weed (eds). New York: Dekker, 1995, pp 749–764.
20. DeBakey, ME, Gotto, AM, Scott, LW, and Foreyt, JP: *The Living Heart Brand Name Shopper's Guide—Revised and Updated.* New York: MasterMedia, 1993.
21. DeBakey, ME, Gotto, AM, and Scott, LW: *The Living Heart Guide to Eating Out.* New York: MasterMedia, 1993.
22. Schekel, BF, Sievert, YA, and Buzzard, IM: "Source of Data for Developing and Maintaining a Nutrient Database." *J Am Diet Assoc* **88,** 1268–1271, 1988.
23. Dennis, B, Ernst, N, Hjortland, M, Tillotson, J, and Grambach, V: "The NHLBI Nutrition Data System." *J Am Diet Assoc* **77,** 641–647, 1980.
24. SAS Institute: *SAS/STAT User's Guide,* 4th ed. Cary, NC: SAS Institute, 1989, version 6, vol 2.
25. Black, HS, Thornby, JI, Wolf, JE, Goldberg, JH, Herd, JA, et al.: "Influence of a Low-Fat Diet on Incidence of Actinic Keratosis and Non-Melanoma Skin Cancer" (abstr). *Congr Eur Soc Photobiol, 6th, Cambridge, UK, September 1995*, p 7.
26. Centers for Disease Control: "Daily Dietary Fat and Total Food-Energy Intakes. Third National Health and Nutrition Examination Survey, Phase I, 1988–91." *Morbidity Mortality Weekly Rep* **43,** 116–125, 1994.
27. Kune, GA, Bannerman, S, Field, B, Watson, LF, Cleland, H, et al.: "Diet, Alcohol, Smoking, Serum β-Carotene, and Vitamin A in Male Nonmelanocytic Skin Cancer Patients and Controls." *Nutr Cancer* **18,** 237–244, 1992.
28. Steinmetz, KA, and Potter, JD: "Vegetables, Fruit, and Cancer. I. Epidemiology." *Cancer Causes Control* **2,** 325–357, 1991.
29. Tannenbaum, A: "The Genesis and Growth of Tumors. II. Effects of Caloric Restriction per se." *Cancer Res* **2,** 460–467, 1942.
30. Weindruch, R, Albanes, D, and Kritchevsky, D: "The Role of Calories and Caloric Restriction in Carcinogenesis." *Hematol Oncol Clin North Am* **5,** 79–89, 1991.
31. Hursting, SD, Thornquist, M, and Henderson, MM: "Types of Dietary Fat and Incidence of Cancer at Five Sites." *Prev Med* **19,** 242–253, 1990.
32. National Research Council. *Diet and Health: Implications for Reducing Chronic Disease Risk.* Washington, DC: National Academy Press, 1989.
33. Lichtenstein, AH, Ausman, LM, Carrasco, W, Jenner, JL, Ordovas, JM, et al.: "Short-Term Consumption of a Low-Fat Diet Beneficially Affects Plasma Lipid Concentrations Only When Accompanied by Weight Loss." *Arterioscler Thromb* **14,** 1751–1760, 1994.
34. Schaefer, EJ, Lichtenstein, AH, Lamon-Fava, S, McNamara, JR, Schaefer, MM, et al.: "Body Weight and Low-Density Lipoprotein Cholesterol Changes After Consumption of a Low-Fat Ad Libitum Diet." *J Am Med Assoc* **274,** 1450–1455, 1995.
35. DeBakey, ME, Gotto, AM, Scott, LW, and Foreyt, JP: *The New Living Heart Diet.* New York: Simon & Schuster, 1996.