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INTENSIVE LIFESTYLE CHANGES MAY AFFECT THE PROGRESSION OF PROSTATE CANCER

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ABSTRACT

Purpose: Men with prostate cancer are often advised to make changes in diet and lifestyle, although the impact of these changes has not been well documented. Therefore, we evaluated the effects of comprehensive lifestyle changes on prostate specific antigen (PSA), treatment trends and serum stimulated LNCaP cell growth in men with early, biopsy proven prostate cancer after 1 year.

Materials and Methods: Patient recruitment was limited to men who had chosen not to undergo any conventional treatment, which provided an unusual opportunity to have a nonintervention randomized control group to avoid the confounding effects of interventions such as radiation, surgery or androgen deprivation therapy. A total of 93 volunteers with serum PSA 4 to 10 ng/ml and cancer Gleason scores less than 7 were randomly assigned to an experimental group that was asked to make comprehensive lifestyle changes or to a usual care control group.

Results: None of the experimental group patients but 6 control patients underwent conventional treatment due to an increase in PSA and/or progression of disease on magnetic resonance imaging. PSA decreased 4% in the experimental group but increased 6% in the control group ($p = 0.016$). The growth of LNCaP prostate cancer cells (American Type Culture Collection, Manassas, Virginia) was inhibited almost 8 times more by serum from the experimental than from the control group (70% vs 9%, $p < 0.001$). Changes in serum PSA and also in LNCaP cell growth were significantly associated with the degree of change in diet and lifestyle.

Conclusions: Intensive lifestyle changes may affect the progression of early, low grade prostate cancer in men. Further studies and longer term followup are warranted.

KEY WORDS: prostate, prostatic neoplasms, prostate-specific antigen, life style, nutrition

Increasing evidence from epidemiological and laboratory studies suggests that diet and lifestyle may have a role in the development of prostate cancer.[1–5] The intake of total and specific vegetables, tomato products (lycopene), vitamin E, selenium, vitamin C and soy products has been inversely associated with prostate cancer risk. In addition, epidemiological evidence and migrant studies indicate that the incidence of clinically significant prostate cancer is much lower in parts of the world where people eat a predominantly low fat, plant based diet.[6]

There is considerable interest in the role of diet and lifestyle changes as complementary therapy in those with prostate cancer, especially because no consensus exists regarding the relative benefits and risks of conventional treatments in many patients. Many men are making changes in diet and lifestyle in the hope of preventing or slowing the progression of prostate cancer without the benefit of data from randomized, controlled trials to help guide these decisions.

We examined if comprehensive changes in diet and lifestyle may affect the progression of prostate cancer, as measured by serial prostate specific antigen (PSA), treatment trends and serum stimulated LNCaP cell growth, in men with early, biopsy proven prostate cancer. To assess possible mechanisms mediating the relationship between changes in lifestyle and these measures we also evaluated changes in testosterone and C-reactive protein (CRP). Patient recruitment was limited to men who had chosen not to undergo any conventional treatment and who had low risk prostate cancer, as defined by baseline serum PSA and Gleason score.

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Although this decision was made for reasons unrelated to this study, the choice to perform watchful waiting was clinically reasonable in these men.[7] This subgroup of patients provided an unusual opportunity to have a nonintervention randomized control group to avoid the confounding effects of interventions such as radiation, surgery or androgen deprivation therapy.

MATERIALS AND METHODS

Patients in this study had biopsy documented prostate cancer with Gleason less than 7, serum PSA 4 to 10 ng/ml, and stages T1 and T2 disease. They had elected not to undergo conventional treatment. Patients were excluded if they had active prostatitis, had already made comprehensive lifestyle changes, had other life threatening comorbidities or major psychiatric disturbances, or were abusing alcohol, nicotine or other drugs. The University of California-San Francisco Committee on Human Research institutional review board approved this study and all patients provided proper consent. A randomized consent design was chosen to decrease the likelihood that control group patients might make diet and lifestyle changes comparable to those of the experimental group that could dilute between group differences and increase the likelihood of a type 2 error by decreasing the amount of information about the lifestyle intervention available to the control group.[8] Of the 181 patients who were eligible for the study 93 enrolled, including 44 in the experimental group and 49 in the control group. Reasons for refusal to participate were unwillingness to make or not make comprehensive lifestyle changes and/or refusal to undergo periodic testing. An additional 15 patients with Gleason scores of 7 or greater were excluded because it is a unique prognostic category with biologically distinct and more aggressive neoplasms.[9] Three experimental group patients withdrew soon after beginning the intervention because they said it was too difficult to follow and they refused further testing. No other patients were lost to followup.

Experimental group patients were prescribed an intensive lifestyle program that included a vegan diet supplemented with soy (1 daily serving of tofu plus 58 gm of a fortified soy protein powdered beverage), fish oil (3 gm daily), vitamin E (400 IU daily), selenium (200 mcg daily) and vitamin C (2 gm daily), moderate aerobic exercise (walking 30 minutes 6 days weekly), stress management techniques (gentle yoga based stretching, breathing, meditation, imagery and progressive relaxation for a total of 60 minutes daily) and participation in a 1-hour support group once weekly to enhance adherence to the intervention.[10] The diet was predominantly fruits, vegetables, whole grains (complex carbohydrates), legumes and soy products, low in simple carbohydrates and with approximately 10% of calories from fat.[11] The diet is intensive but palatable and practical. In earlier studies most patients were able to adhere to this diet for at least 5 years.[10–13]

A registered dietitian was available for nutrition education and counseling. A nurse case manager contacted patients by telephone once weekly for the first 3 months and once monthly thereafter. Control group patients were asked to follow the advice of their physicians regarding lifestyle changes. All therapeutic decisions, including whether to undergo conventional treatment during the study course, were deferred to the personal physician of each patient.

Serum PSA was measured twice at baseline and at 1 year. Patients were counseled to avoid activities that might affect PSA for 3 days prior to testing, including sexual activity, exercise and digital rectal examination. Serum PSA was measured at Memorial Sloan-Kettering Cancer Center prospectively by a heterogeneous sandwich magnetic separation assay with the Immuno 1™ System. Testosterone was measured by a competitive immunoassay with an Immulite® automated analyzer.

LNCaP cells were grown in 75 cm^2 flasks in RPMI-1640 medium without phenol red, as previously described in detail.[12] Cells were collected using 0.25% Trypsin-ethylenediaminetetraacetic acid (Sigma Chemical Co., St. Louis, Missouri) and then experiments were performed in duplicate (5×103 cells per well in 96-well plates). After 24 hours fresh medium composed of 10% fetal bovine serum (FBS) or 10% human serum was replaced and the cells were incubated (37·C, 5% CO_2) for 48 hours. FBS served as a control for each assay and results are expressed as percent FBS. Cell growth was assessed by MTS Assay (Promega, Madison, Wisconsin). For apoptosis cells were plated at a density of 1×104 cells per well in 96-well culture plates and incubated as described for the growth assay. After 48 hours apoptosis was detected by Cell Death Detection ELISA^{PLUS} (Roche Applied Science, Indianapolis, Indiana). CRP determinations were done in duplicate by ultrasensitive enzyme-linked immunosorbent assay with 1.6 ng/ml sensitivity, and with intra-assay and interassay coefficients of variation of 3.9% and 5.1%, respectively.

Dietary intake assessing the percent of calories from fat and mg cholesterol was measured with a semiquantitative food frequency questionnaire. Nutrient assessment was calculated elsewhere using United States Department of Agriculture food composition tables and other sources. The frequency and duration of exercise and of stress management techniques were assessed by self-reporting questionnaires. Attendance at group support sessions was recorded. The level of adherence to the recommended lifestyle change was based on a formula validated in previous studies.[13] A total score of 1 indicated 100% adherence to the program and 0 indicated no adherence.

Eligible patients were randomly assigned to the control or the intervention group. Assessment of outcome measures was done while blinded to group assignment.

Baseline equivalence of the 2 groups was analyzed using the independent sample t test in the case of continuous variables and the chi-square test of association in the case of categorical variables. Between group differences in baseline to 12-month changes in clinical and behavioral outcomes were compared using ANCOVA with baseline values as covariates. Although control patients were not asked to make changes in diet and lifestyle, some did so in varying degrees, that is 18% to 137% (experimental group 58% to 316%). As a secondary analysis, we correlated the degree of lifestyle change with changes in serum PSA, LNCaP cell growth, LNCaP apoptosis, serum testosterone and CRP across the 2 groups regardless of group assignment with baseline values as a covariate. Natural log transformation achieved normality (ln-CRP). All reported significance levels are 2-sided and $p < 0.05$ was considered the required value for concluding that there were significant differences between the groups.

RESULTS

At baseline there were no significant differences between the groups in demographic or clinical measures (table 1). Subject age, PSA and Gleason scores in those who were randomized into the study but refused to participate were not significantly different from values in those who participated.

After 1 year adherence to the intervention was 95% in the experimental group and 45% in the control group. There were no adverse events attributable to the lifestyle intervention. Diet, exercise, stress management techniques and group support improved significantly more in the experimental group than in the control group (table 2).

Six control group patients withdrew before 12 months and underwent conventional treatment, including radical prostatectomy in 3, and androgen deprivation, external beam radiation and brachytherapy in 1 each. Four of these patients underwent conventional treatment due to an increase in PSA

TABLE 1. *Participant demographic and baseline characteristics*

	Intervention	Control	p Value
No. subjects	44	49	
Mean age ± SD	65 ± 7	67 ± 8	0.25
% Married/cohabitating	66	76	0.31
% Employment:			0.64
Full/part time	54	49	
Retired	46	51	
Mean PSA ± SD (ng/ml)	6.32 ± 1.72	6.28 ± 1.66	0.92
Mean cholesterol ± SD (mg/dl)	204 ± 42	203 ± 39	0.90
Mean low density protein ± SD (mg/dl)	129 ± 36	127 ± 33	0.75
Mean high density protein ± SD (mg/dl)	48 ± 11	50 ± 13	0.57
Mean triglycerides ± SD (mg/dl)	133 ± 77	135 ± 88	0.94
Mean Ln-CRP ± SD	−0.0310 ± 1.1	0.2767 ± 0.8	0.16
Mean wt ± SD (kg)	80 ± 13.6	80 ± 11.3	0.75
Mean LNCaP apoptosis ± SD (% FBS)	48.16 ± 22.1	44.33 ± 33.0	0.55
Mean testosterone ± SD (ng/dl)	414 ± 860	387 ± 100	0.20
Mean Gleason ± SD (Sum)	5.7 ± 0.5	5.7 ± 0.7	0.80

To convert cholesterol, LDL and HDL to mmol multiply by 0.0259, to convert triglycerides to mmol multiply by 0.0113 and to convert testosterone to nmol multiply by 0.0347.

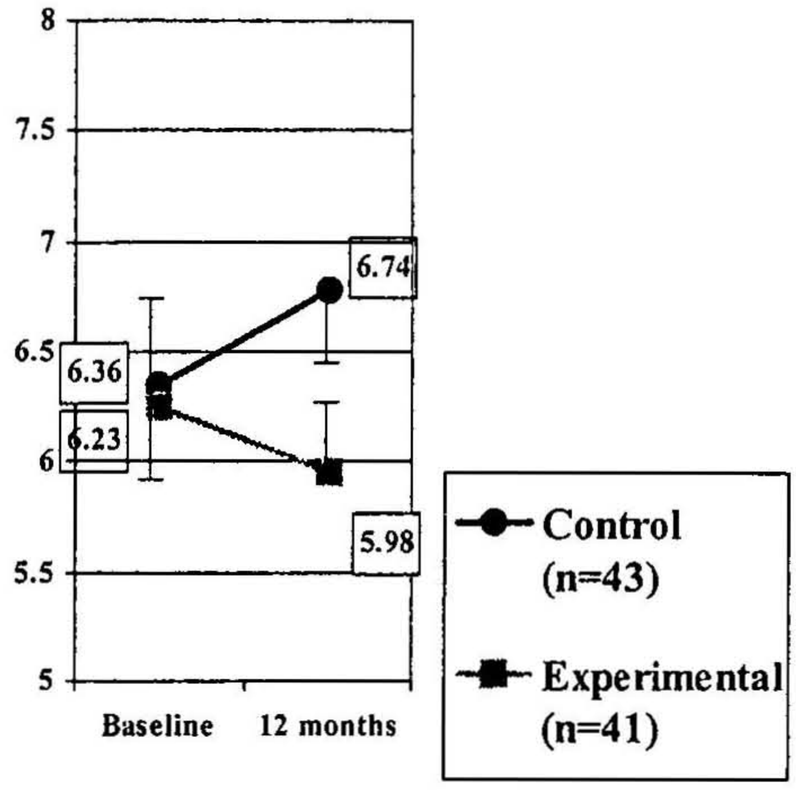

FIG. 1. Mean changes ± SEM in PSA in ng/ml between experimental and control groups after 1 year.

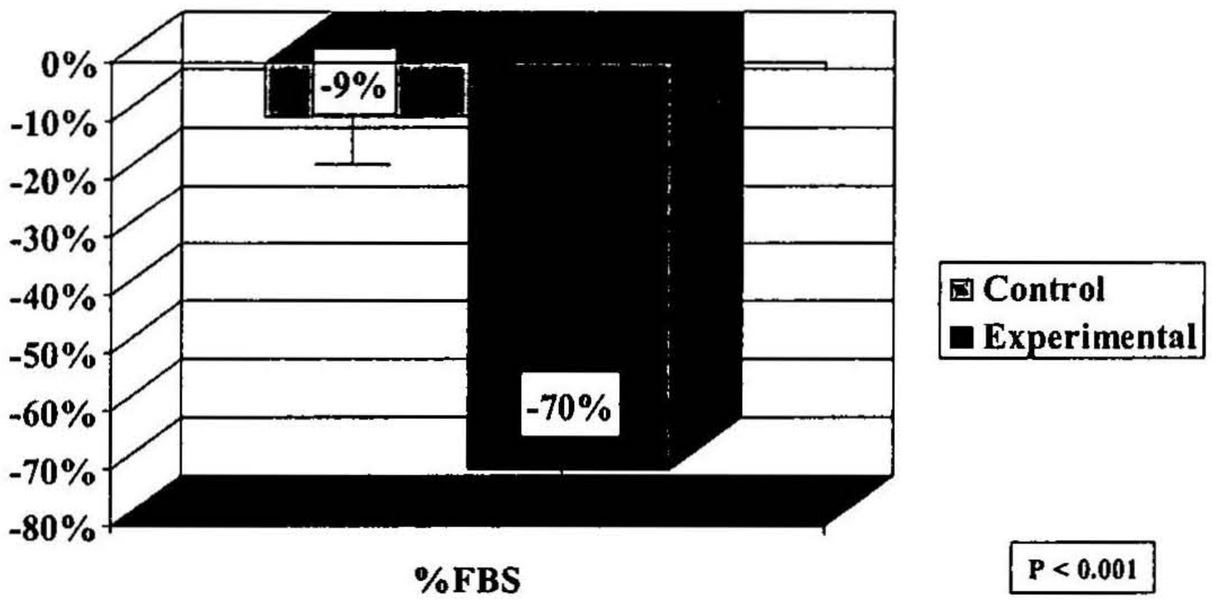

FIG. 2. Mean changes ± SEM in percent serum stimulated LNCaP cell growth from baseline to 1 year in experimental and control groups.

during the study and 2 underwent it due to progression of prostate cancer, as assessed by magnetic resonance imaging compared with earlier studies. In contrast, no experimental group patients underwent conventional treatment during the study.

Changes in serum PSA and LNCaP cell growth from baseline to 12 months were significantly different between the groups, showing more favorable changes in the experimental group. Specifically serum PSA decreased an average of 0.25 ng/ml or 4% of the baseline average in the experimental group but it showed an average increase of 0.38 ng/ml or 6% of the baseline average in the control group ($F = 5.6$, $p = 0.016$, fig. 1). Serum from experimental group patients inhibited LNCaP cell growth by 70%, whereas serum from control group patients inhibited growth by only 9% ($p < 0.001$, fig. 2). CRP decreased more in the experimental group ($p = 0.07$). There were no significant differences between the groups in serum testosterone or in apoptosis (table 3).

Pearson correlations between changes in serum PSA, LNCaP, apoptosis, testosterone and CRP, and following recommended lifestyle changes in the entire sample indicated that the extent to which participants made changes in diet and lifestyle was significantly related to decreases in PSA ($r = -0.23$, $p = 0.035$, fig. 3) and to LNCaP cell growth ($r = -0.37$, $p < 0.001$, fig. 4). There were no significant associations between the degree of lifestyle changes and changes in CRP, testosterone or apoptosis. Comparisons of baseline values in the 6 control group patients who received

TABLE 2. *Differences in lifestyle change scores between groups ($p < 0.001$)*

Group	Mean Baseline ± SE	Mean 12 Mos ± SE	Mean Baseline-12-Mo Change ± SE	F (df)
Dietary fat (% calories from fat):				
Experimental	28.9 ± 1.8	11.2 ± 0.4	−17.7 ± 1.4	130.7 (1.81)
Control	26.2 ± 1.2	25.3 ± 8.8	−0.9 ± 1.1	
Dietary cholesterol (mg/day):				
Experimental	230.4 ± 21.6	7.5 ± 1.9	−222.9 ± 21.8	98.3 (1.81)
Control	218.0 ± 19.2	182.1 ± 19.3	−35.9 ± 16.0	
Exercise (days/wk):				
Experimental	3.1 ± 0.4	4.8 ± 0.3	1.7 ± 0.4	14.7 (1.80)
Control	3.3 ± 0.4	3.3 ± 0.4	0.0 ± 0.4	
Exercise (mins/wk):				
Experimental	120.8 ± 18.8	262.9 ± 38.8	142.1 ± 32.7	11.4 (1.80)
Control	186.1 ± 27.6	160.6 ± 21.3	−25.5 ± 26.8	
Stress management (days/wk):				
Experimental	2.1 ± 0.4	5.7 ± 0.3	3.6 ± 0.4	46.2 (1.80)
Control	2.0 ± 0.4	2.3 ± 0.5	0.3 ± 0.4	
Stress management (mins/wk):				
Experimental	39.6 ± 11.0	315.7 ± 20.9	276.0 ± 20.9	102.5 (1.80)
Control	71.3 ± 22.1	75.7 ± 19.1	4.4 ± 18.0	
% Overall lifestyle index:				
Experimental	41.4 ± 3.8	94.8 ± 3.8	53.4 ± 4.2	115.2 (1.80)
Control	45.4 ± 2.9	45.1 ± 3.5	−0.3 ± 3.0	

TABLE 3. *Baseline to 12-month change in clinical outcomes by group*

Group	Mean Baseline ± SD	Mean 12 Mos ± SD	Mean Baseline-12-Mo Change ± SD	p Value
PSA (ng/ml):				
Experimental	6.23 ± 1.7	5.98 ± 1.7	−0.25 ± 1.2	0.016
Control	6.36 ± 1.7	6.74 ± 2.1	0.38 ± 1.3	
Total cholesterol (mg/dl):				
Experimental	205.0 ± 42	172.6 ± 34	−32 ± 39.4	<0.001
Control	200.6 ± 39	202.8 ± 37	2 ± 25.7	
Low density protein (mg/dl):				
Experimental	130.9 ± 35	101.2 ± 25	−30 ± 31.3	<0.001
Control	125.2 ± 33	124.1 ± 30	−1 ± 25.2	
High density protein (mg/dl):				
Experimental	47.3 ± 10	41.9 ± 12	−5 ± 8.3	<0.001
Control	48.3 ± 12	49.3 ± 12	1 ± 6.8	
Triglycerides (mg/dl):				
Experimental	133.0 ± 78	138.0 ± 96	5 ± 65.4	0.52
Control	137.1 ± 91	150.9 ± 93	14 ± 77.5	
LNCaP growth (% FBS):				
Experimental	105.50 ± 19.0	35.56 ± 9.2	−69.94 ± 19.5	<0.001
Control	91.40 ± 19.2	82.34 ± 36.8	−9.06 ± 42.8	
LNCaP apoptosis (% FBS):				
Experimental	48.16 ± 22.1	125.38 ± 127.0	77.23 ± 120.6	0.27
Control	45.16 ± 33.7	90.18 ± 128.0	45.02 ± 112.7	
Ln-CRP (mg/l):				
Experimental	−0.0310 ± 1.1	−0.2782 ± 1.0	−0.2472 ± 0.8	0.07
Control	0.2767 ± 0.8	0.2121 ± 0.9	−0.0646 ± 0.9	
Testosterone (ng/dl):				
Experimental	414.2 ± 86	443.3 ± 117	29 ± 96	0.53
Control	387.0 ± 100	435.0 ± 155	48.0 ± 123	
Wt (kg):				
Experimental	80 ± 13.8	76 ± 10.0	−4.5 ± 6.2	<0.001
Control	80 ± 11.4	80 ± 11.4	0 ± 3.9	

To convert cholesterol, LDL and HDL to mmol multiply by 0.0259, to convert triglycerides to mmol multiply by 0.0113 and to convert testosterone to nmol multiply by 0.0347.

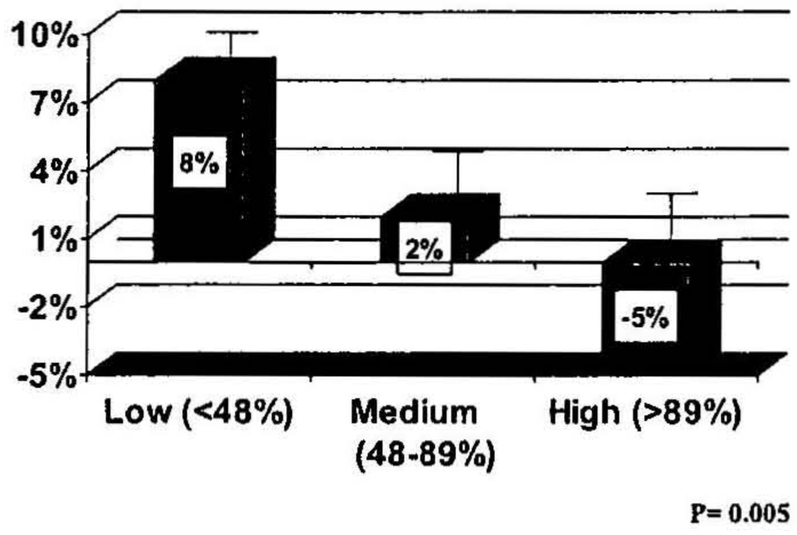

FIG. 3. Mean relationship ± SEM of degree of lifestyle change and changes in PSA across 2 groups by tertiles.

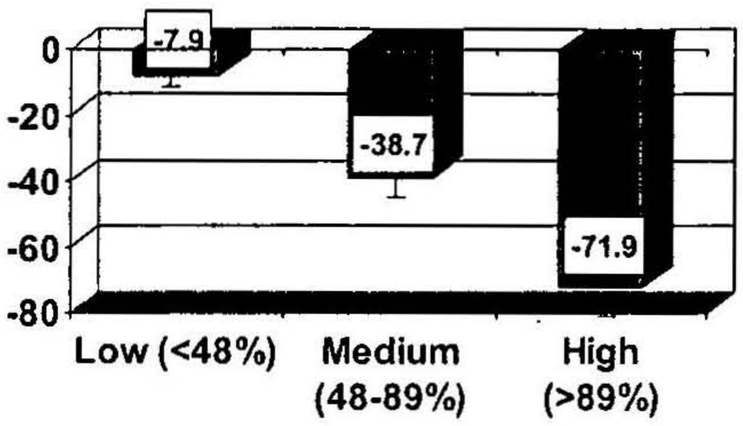

FIG. 4. Mean relationship ± SEM of degree of lifestyle change and changes in LNCaP cell growth across 2 groups by tertiles.

treatment with values in controls who did not require treatment by 12 months did not reveal any significant differences in any of these measures.

DISCUSSION

The primary end point of this study was serum PSA because PSA is the most widely used surrogate or intermediate measure for assessing the outcomes of virtually any treatment for prostate cancer. Mean serum PSA decreased in the experimental group but increased in the control group. Although these differences were statistically significant, the magnitude of these changes was relatively modest. However, the direction of change may be clinically significant since an increase in PSA predicts clinical progression, ie regional or systemic metastasis, in the majority of men with prostate cancer.[14–16] In addition, recent trials of surveillance alone in those with clinically localized prostate cancer have shown that a change in serum PSA kinetics is 1 of the strongest determinants of eventual treatment.[7,14] These differences in PSA after 1 year may have been greater if 6 control group patients had not undergone conventional treatment during the study due to increasing PSA before 1-year PSA values could be determined.

In addition to PSA as the primary outcome, we included changes in serum stimulated LNCaP cell growth for monitoring disease progression. The LNCaP cell line has been used extensively for studying the mechanisms and benefits of various therapeutic interventions. This cell line was initially derived from a patient with androgen dependent prostate cancer and it has been used in numerous studies to investigate factors that may stimulate or decrease prostate cancer cell growth. The current results indicate that serum from experimental group patients decreased the growth of LNCaP prostate cancer cells almost 8 times more than serum from control group patients (9% vs 70%, p <0.001), suggesting that comprehensive lifestyle changes may have affected tumor growth as well as PSA. Although such an in vitro system has its limitations, the results are provocative. The observation that changes in PSA and in LNCaP cell growth were significantly related to the extent to which participants had changed their lifestyle supports the hypothesis that intensive changes in diet and lifestyle may affect the progression of prostate cancer. Investigations done by others support this hypothesis.[4,17,18]

Also, we considered the possibility that changes in diet and lifestyle may have affected PSA production without affecting tumor growth and the underlying prostate cancer disease process. However, 2 recent articles failed to show any effect of

a diet low in fat and high in fiber, fruits and vegetables on PSA after 4 years in men who did not have prostate cancer, perhaps because the diet was not as low in fat and did not include exercise or stress management.[19,20] In addition, it did not appear that changes in serum testosterone were responsible for the changes in serum PSA because changes in this end point were unrelated to serum PSA.

Consistent with findings in earlier studies in patients with ischemic heart disease who followed a similar program of diet and lifestyle changes, experimental group participants had significant decreases in body weight and improvements in the lipid profile compared with those in the control group. It is unlikely that changes in weight alone were responsible for the changes in PSA observed in the current study since we did not observe a statistically significant correlation between changes in weight and changes in PSA ($r = 0.169$, $p = 0.14$). Cardiovascular disease is the leading cause of death in men and women in the United States, and it is the primary or secondary cause of death in most men with prostate cancer.

Therefore, this lifestyle intervention may have benefits beyond any possible favorable effects on the progression of prostate cancer. In addition, since there is a significant rate of recurrence following any conventional treatment for prostate cancer, our findings may encourage some patients to make changes in diet and lifestyle as an adjunct to conventional treatment in the hope of decreasing the risk of recurrence.

A limitation of the current study is that it cannot provide definitive conclusions concerning the effect of our intervention on disease specific survival. Any intervention, including diet and lifestyle, may affect the progression of prostate cancer without necessarily affecting survival. Because patients in this study had early, less aggressive tumors, they would be unlikely to show changes in clinical progression in only 1 year. We will continue to follow these patients for a longer period to determine the number undergoing conventional treatment, and the rates of recurrence, metastasis and death.

CONCLUSIONS

Patients with low grade prostate cancer were able to make and maintain comprehensive lifestyle changes for at least 1 year, resulting in significant decreases in serum PSA and a lower likelihood of standard treatment. In addition, substantially decreased growth of LNCaP prostate cancer cells was seen when such cells were incubated in the presence of serum from those who made lifestyle changes. These findings suggest that intensive changes in diet and lifestyle may beneficially affect the progression of early prostate cancer. Additional trials of such therapy appear warranted.

Representatives Nancy Pelosi and John Murtha, and Senators Arlen Specter and Ted Stevens provided support, Rusty Nicar performed CRP analyses, and Jennifer Daubenmier, Billy Gao, Dennis Malone and the referring physicians from University of California, San Francisco, California Pacific Medical Center, Kaiser Permanente and Marin General Hospital contributed to the study. Nutrient assessment was done at Harvard School of Public Health.

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EDITORIAL COMMENT

This article is an example of the increasing efforts to apply clinical trials science to the claims of complementary medicine. Several criticisms seem appropriate.

The fact that no one switched to active therapy in the experimental group is no surprise Relative PSA decreases in the experimental group, while "significant," were meager, especially when one considers that the coefficient of variation for most PSA assays is 15% and the number of patients in the groups is relatively small. Also, PSA decrease differences do not necessarily translate into differences in progression or survival. Experimental serum seemed to contain

something that differentially inhibited cell line growth but so what. Just because these serums were different does not mean that they were good. They might have also killed normal cells.

This report undoubtedly will excite the aficionados and devotees of lifestyle changes for cancer but it should also give pause to the skeptics. Appropriately it will encourage other and more vigorous scientific scrutinies of complementary medicine strategies. For those of us taking care of patients with prostate cancer it will reinforce the use of lifestyle changes in management. Even if scientific evidence is still meager, complementary medicine approaches have strong appeal in practicing the medical art since they give the patient an active role in his care and promote an attitude of optimism and hope.

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REPLY BY AUTHORS

Changes in diet and lifestyle are of profound interest to many patients with prostate cancer. A large number of patients make such changes, often independent of doctor advice or knowledge. Quantitative information about their effects are lacking and more trials need to address such issues.

While many people believe that changing diet and lifestyle decrease the quality of life—"am I going to live longer or is just going to seem longer?"—patients in the experimental group reported marked improvements in quality of life.[1,2] In contrast, many patients report a decrease in quality of life, including impotence and incontinence, following conventional treatments. Six patients in the control group received conventional treatments because progression of prostate cancer was evident.

All of the PSA tests were performed in the same laboratory at Memorial Sloan-Kettering Cancer Center using a precise procedure. These results are accurate and precise with day-to-day coefficients of variation of less than 4.2%. A mean difference in PSA of 10% is different than the individual variation in a given patient. Regarding the sample size, Maseri et al stated, "The larger the number of patients that have to be included in a trial in order to prove a statistically significant benefit, the greater the uncertainty about the reason why the beneficial effects of the treatment cannot be detected in a smaller trial."[3] In other words, a treatment needs to be potent for its effects to be statistically significant in a smaller sample. While there is not a direct correlation between change in PSA and differences in progression or survival, PSA is used as a primary end point meausre of virtually all standard treatments of prostate cancer. Also, it is unusual for prostate cancer to metastasize if PSA levels are decreasing.

Change in LNCaP cell growth is a standard test used for evaluating the effects of conventional treatments on prostate cancer in the laboratory, and so it should also be useful in evaluating the effects of diet and lifestyle changes. Although it is true that chemotherapy and radiation may kill normal as well cancerous cells, we are not aware of any evidence that fruits vegetables, whole grains, legumes and soy products kill normal cells. Indeed, evidence suggests that substances present in these foods, such as lycopene, flavonoids, sulphoraphanes, omega-3 fatty acids, isoflavones, polyphenols, lignans and other substances, are protective of normal cells. The significant correlation between degree of changes in diet and lifestyle and degree of change in PSA and LNCaP cell growth adds to the strength of evidence.

While the evidence linking the effects of diet and lifestyle on the development and progression of prostate cancer is not conclusive, it is hardly meager. A wide body of evidence from animal studies, epidemiological studies of large groups of humans, case reports and now evidence from a carefully conducted randomized controlled trial indicates that changes in diet and lifestyle may reduce the risk of prostate cancer and may affect its rate of progression. Since there is a significant rate of recurrence following any conventional treatment for prostate cancer, our findings may encourage some patients to make changes in diet and lifestyle as an adjunct to conventional treatment in hopes of reducing the risk of recurrence.

Also, these same changes in diet and lifestyle have beneficial effects that go beyond those that may favorably affect the progression of prostate cancer, including significant reductions in low density protein cholesterol and weight. Cardiovascular disease is the leading cause of death of men and women in the United States and either the primary or secondary cause of death of most men with prostate cancer, and obesity is of widespread concern. In earlier randomized controlled trials we found that these changes in diet and lifestyle may reverse the progression of even severe coronary heart disease.[4,5] The recent INTERHEART study of 30,000 patients from 52 countries found that almost 95% of coronary heart disease could be prevented by changing diet and lifestyle.[6] And the only side effects are beneficial ones.

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