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Ethnic Differences in Diet and Associations with Clinical Markers of Prostate Disease in New Zealand Men

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Abstract. *Objective: To examine ethnic differences in diet and dietary associations with clinical markers of prostate disease in New Zealand men. Materials and Methods: A total of 1031 males (616 New Zealand European, 230 Maori and 185 Pacific Islands) aged 40-69 years, with no history of prostate cancer, completed a questionnaire covering diet. A serum prostate specific antigen (PSA) blood analysis was also undertaken. Regression models were developed to examine the ethnic-specific levels of selected dietary components, and their relationship with PSA and urinary symptom scores. Results: The results confirmed previously found ethnic differences in the New Zealand diet. Combined tomato intake was positively-correlated with free PSA and % free PSA ($p=0.021$, $r=0.197$ and $p=0.011$, $r=0.096$ respectively). Beer intake was negatively- correlated with total PSA ($p=0.028$, $r=-0.071$) and free PSA ($p=0.004$, $r=-0.094$). Conclusion: Ethnic differences found in the consumption of foods (associated with prostate cancer) highlight the possible importance of dietary interactions for ethnic prostate cancer risk. Associations between specific foods and PSA warrant further investigation.*

Over the last three decades, prostate cancer has become a major public health problem in countries with an increasingly aged population and a Western diet; it is the most commonly diagnosed non-skin cancer of men in many Western countries (1). Overall New Zealand is ranked among the six highest countries in prostate cancer incidence (2).

Within the New Zealand population there is significant ethnic variation in cancer incidence and mortality (3). WHO age-standardised prostate cancer rates in 1998-1999 showed that, at 86.1 per 100,000, Maori males had the lowest incidence, followed by Pacific Islands males at 115.2 per 100,000 and Other (chiefly New Zealand European) males the highest incidence (118.9 per 100,000) (4). Conversely, the prostate cancer WHO age-standardised mortality rates for Pacific Islands males and Maori males in 1998-1999 (52.3 and 39.3 per 100,000, respectively) were higher than rates for Other males (22.8 per 100,000) (4).

The ethnic disparity between incidence and mortality is likely to reflect reduced health care utilisation by Maori and Pacific Islands men, as well as under-reporting in ethnic health data collection in New Zealand (5,6).Recent research has shown that prostate cancer incidence for Maori and Pacific Islands men is likely to be at least as high as that shown for New Zealand European men (7). The higher mortality rates shown for Pacific Islands and Maori men may reflect the health utilisation issues of later diagnosis and treatment, as well as more rapid disease progression.

Epidemiological research has implicated ethnic-specific diet in the genesis and progression of prostate cancer (1,8). For example, the incidence of prostate cancer is considerably lower in Asian men than in American men (1). Features of the Asian diet which may protect against prostate cancer include soy, vegetables, fish and tea (9-12). Tomato-based products have also been associated with reduced prostate cancer risk (13,14). The Western diet is high in meat and saturated fats, and low in fibre, all of which have been associated with increased risk of prostate cancer (15).

It is known that, in New Zealand, ethnic variation exists in dietary intake. The 1997 New Zealand national nutrition survey found differences in eating patterns between the main ethnic groups. Maori and Pacific Islands people eat more lamb and pork than New Zealand Europeans, and more shellfish and fried fish in batter. Ethnic variation also exists in the consumption of alcohol, with wine drinking

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more common among New Zealand Europeans and beer consumption lowest among Pacific peoples (16). Based on the higher consumption of meat products in Maori and Pacific Islands people, it could be predicted that they are at a higher diet-related prostate cancer risk.

Serum Prostate Specific Antigen (PSA) and its derivatives have been suggested as surrogate markers for both prostate cancer and benign prostatic hyperplasia (17). Presently PSA is the most useful tumour marker in clinical practice. Although an elevation in PSA is associated with prostate tissue growth, it may represent other changes, including inflammation, growth factor levels and manual manipulation of the prostate (18). The PSA level also varies with age (19), ethnicity (20) and other demographic factors (21). Studies have examined the association between dietary factors and PSA. Although two found no association with dietary factors and PSA (22,23), another found that the average PSA level increased over time with dietary intervention (24).

Based on this background and previous findings of demographic influences on levels of PSA by the Wellington Region Community Prostate Study group (21), we undertook additional research on this group, in order to examine ethnic differences in diet and dietary associations with clinical markers of prostate disease in New Zealand men. Namely total, complexed and free PSA, and urinary tract symptoms.

Materials and Methods

From January 2000 to February 2002, a total of 1617 males were recruited into the Wellington Regional Community Prostate Study co-ordinated through the Wellington School of Medicine and Health Sciences of the University of Otago, New Zealand. In order to ensure an adequate representation of Maori and Pacific Islands men, participants were enrolled by two separate means. Initially subjects were identified in census area units containing at least 5% Maori and 5% Pacific Islands populations. Males aged between 40 and 69 years were invited to attend a local clinic where a blood sample was taken and a detailed questionnaire was completed (Phase 1). The total number recruited into Phase 1 was 698.

The second mode of recruitment was through the identification of individuals who had been screened as part of the Wellington hepatitis and diabetes-screening programme for Maori and Pacific Islands populations. After ethics approval, blood samples were retrieved from the Hepatitis Foundation of New Zealand and subjects were contacted and asked to complete the study questionnaire (Phase 2). The total number recruited into Phase 2 was 919.

Ethnicity was determined on a self-identification basis (25). Subjects also completed an International Prostate Symptom Score Sheet (IPSS) (26) and declared if they have ever had any evidence of prostate disease. Subjects with elevated total PSA levels of >4.0 ng/ml were referred back to their general practitioner for further evaluation and exclusion of prostatic malignancy.

In order to ensure subjects were affiliated to the three ethnic groups in question and aged between 40-69, age and ethnicity selection criteria were applied to the combined Phase 1 and Phase 2 group (1617). Subjects were also excluded on the basis of suspected prostate cancer (through PSA testing, GP diagnosis or prostate biopsy). Of the remaining 1405 eligible subjects, 1031 completed the food-frequency questionnaires (616 New Zealand European, 230 Maori and 185 Pacific Islands subjects).

Questionnaires were self-administered and covered occupation, education, medical history and diet. Most dietary information was obtained *via* a subset of questions from a food frequency questionnaire (FFQ) previously validated in another New Zealand study (27). Only the questions regarding tomatoes had not been validated previously. These questions were included because tomatoes have been found protective for prostate cancer in some studies (13,14). The FFQ consisted of 36 questions on food items, some of which have been associated with prostate cancer, including: fruit, vegetables, lamb, pork, fish, shellfish and alcohol.

A 10ml tube of blood was collected, and centrifuged within 4 hours of sampling. Serum was stored at -70˚C until assayed. Sera from each subject were tested for total PSA, complexed PSA (cPSA) and free PSA (fPSA). Laboratory blood analysis for PSA and free PSA was carried out using an Elecsys 2010 (Roche Diagnostics, Mannheim, Germany) assay. Blood analysis for cPSA was carried out on a Bayer ACS-180 analyser (Tarrytown, New York, USA). Assays were performed according to manufacturers' specifications (28).

Daily intake of food and drink (g/day or ml/day) was calculated by multiplying the frequency of consumption (seven options, from never to every day) by the portion size (four options, from half to three or more).

Statistical analyses. Statistical analyses were performed with SPSS version 10.1 for the PC. Two separate sets of analyses were developed, first to examine the ethnic-specific levels of our selected dietary components, and second to investigate the relationship between these dietary components, PSA and urinary symptom scores.

Logistic regression models were developed to test for ethnic differences in the cooking technique employed. Potential confounding factors, such as age, were controlled for within the model.

We used linear models to estimate the effect of all the dietary components (including total meat and vegetable intake) on total PSA, cPSA, fPSA, ratio of free to total PSA (%fPSA) and urinary symptom score. Because PSA and its various derivatives were found to be log-normally distributed, the logarithm of all PSA derivatives was used in analyses.

Backwards Stepwise Regression models were developed to examine if the association between those dietary components initially found to correlate to PSA and its various derivatives on a bivariate level, was due to confounding alone (29). Ethnicity, age, body mass index and smoking status were controlled for within the models.

For dietary components found to be associated with PSA and its various derivatives, quartiles of intake and the average PSA per quartile were calculated.

A logistic regression model was developed to test whether the prevalence of elevated PSA varied significantly by a number of measured demographic and clinical characteristics, namely age, ethnicity, other dietary intakes, smoking status, body mass index, and socio-economic position as measured by the NZDep96 index (30). Due to the non-random sampling method in our study, the

Table I. *Age-adjusted mean intake (g/day or ml/day).*

Food /Beverage Item	Total	European	Maori	Pacific Islands
Raw tomatoes	17.9	17.6	14.9	22.9
Cooked tomatoes	31.0	25.6	29.6	51.9
Tomato juice	2.6	2.1	2.0	4.9
Tomato soup	12.3	8.4	11.3	26.4
Tomato sauce*	88.3	97.5	78.3	69.4
Table tomato sauce	3.8	3.2	4.0	5.5
Combined tomato	143.0	144.0	126.9	159.0
Oranges	40.3	36.0	33.6	62.7
Combined green vegetables	48.5	40.2	59.3	63.1
Combined root vegetables	167.5	180.6	197.4	117.3
Lamb	15.6	11.2	15.1	31.1
Pork	10.5	7.0	13.6	19.0
Combined meat	25.5	17.8	27.9	49.2
Baked fish	19.2	12.4	18.4	43.8
Fried fish	17.4	14.7	17.2	27.2
Tinned fish	7.2	5.6	6.2	14.2
Combined fish	43.5	32.5	41.5	84.3
Shellfish	9.40	4.10	16.70	18.70
Beer	152.42	176.74	153.43	70.03
Wine	25.23	35.94	10.94	9.52
Sherry/Port	1.15	1.21	1.55	0.445
Whiskey	4.14	4.93	3.54	2.11

* Includes canned tomatoes, tomato puree and tomato paste included in pasta-based dishes. The quantity of pasta-based food was multiplied by 0.2 to estimate tomato sauce intake.

Table II. *Difference in intake between the three ethnic groups (p-value).*

Food Item	European *cf*. Maori	European *cf*. Pacific Islands	Maori *cf*. Pacific Islands
Raw tomatoes	0.240	0.034	<0.001
Cooked tomatoes	0.403	<0.001	<0.001
Tomato juice	0.920	0.020	0.040
Tomato soup	0.276	<0.001	<0.001
Tomato sauce	0.035	0.004	0.436
Table tomato sauce	0.148	<0.001	0.043
Combined tomato intake	0.141	0.428	0.067
Oranges	0.690	<0.001	<0.001
Combined green vegetables	<0.001	<0.001	0.534
Combined root vegetables	0.253	<0.001	<0.001
Lamb	0.058	<0.001	<0.001
Pork	<0.001	<0.001	0.012
Combined meat intake	0.002	<0.001	<0.001
Baked fish	0.026	<0.001	<0.001
Fried fish	0.5619	<0.001	<0.001
Tinned fish	0.724	<0.001	<0.001
Combined fish	0.236	<0.001	<0.001
Shellfish	<0.001	<0.001	0.409
Beer	0.244	<0.001	<0.001
Wine	<0.001	<0.001	0.814
Sherry/Port	0.177	0.597	0.269
Whiskey	0.197	0.015	0.297

All least significant differences are reported and significant differences (*p*-values) determined using the Holm multiple comparison procedure [p1=0.05/(n-i+1)][43]

potential for selection bias was investigated and partially controlled for by including factors within the model likely to be associated with participation.

Results

There were significant dietary differences between the three New Zealand ethnic groups (see Tables I and II).

Combined tomato intake was significantly positively associated with fPSA and %fPSA ($p=0.021$, r=0.107 and $p=0.011$, r=0.096, respectively). Intake of tomato sauce was positively correlated with %fPSA ($p=0.015$, r=0.098).

There was a weak negative association between daily beer and total PSA ($p=0.028$, r=-0.071) and fPSA ($p=0.004$, r=-0.094). All these associations remained after controlling for likely confounding factors within the model, including ethnicity, intake of root vegetables (carrots, sweet potato and taro), broccoli, silverbeet, cabbage and watercress.

Other associations were found between fried fish and total PSA, cooked tomatoes and total and fPSA. However, these associations became non-significant when examined within the multivariate model. No association was found between PSA and cooking methods ($p=0.734$).

The association between dietary factors and PSA was not affected by ethnicity.

No association was found between urinary symptom scores and any of the dietary components.

A significant negative association between elevated PSA and beer intake was found. The likelihood of having elevated PSA decreased slightly with increasing beer intake ($p=0.023$).

The average PSA and fPSA decreased by quartile of beer intake. There was a 0.75 ng/ml and 0.14 ng/ml difference between the 1st and 4th quartile of beer intake for PSA and fPSA respectively (Table III).

The average fPSA and %fPSA increased by quartile of combined tomato intake. There was a 0.04 ng/ml and 3% difference between the 1st and 4th quartile of combined tomato intake for PSA and %fPSA, respectively (Table IV).

The average %fPSA increased by quartile of tomato sauce intake. There was a 5% difference between the 1st and 4th quartile of tomato sauce intake (Table V).

Discussion

The progression of prostate cancer is determined by endocrine factors, which can be influenced by environmental

Table III. *Average PSA and fPSA by quartile of beer intake.*

	1st quartile beer intake (ml/day) [a*]	2nd quartile beer intake (ml/day) [b*]	3rd quartile beer intake (ml/day) [c*]	4th quartile beer intake (ml/day) [d*]
PSA	1.78 ng/ml	1.14 ng/ml	1.08 ng/ml	1.03 ng/ml
fPSA	0.46 ng/ml	0.35 ng/ml	0.32 ng/ml	0.32 ng/ml

[a*] up to 1.40 ml/day beer intake.
[b*] between 1.40 and 46.69 ml/day beer intake.
[c*] between 46.69 and 210.00 ml/day beer intake.
[d*] over 210.00 ml/day beer intake.

Table V. *Average %fPSA by quartile of tomato sauce intake.*

	1st quartile tomato sauce intake (ml/day) [a*]	2nd quartile tomato sauce intake (ml/day) [b*]	3rd quartile tomato sauce intake (ml/day) [c*]	4th quartile tomato sauce intake (ml/day) [d*]
%fPSA	32%	36%	36%	37%

[a*] up to 0.12 ml/day tomato sauce intake.
[b*] between 0.12 and 1.00 ml/day tomato sauce intake.
[c*] between 1.00 and 3.00 ml/day tomato sauce intake.
[d*] over 3.00 ml/day tomato sauce intake.

Table IV. *Average fPSA and %fPSA by quartile of combined tomato intake.*

	1st quartile combined tomato intake (g/day) [a*]	2nd quartile combined tomato intake (g/day) [b*]	3rd quartile combined tomato intake (g/day) [c*]	4th quartile combined tomato intake (g/day) [d*]
fPSA	0.33 ng/ml	0.35 ng/ml	0.36 ng/ml	0.37 ng/ml
%fPSA	34%	34%	36%	37%

[a*] up to 22.34 g/day combined tomato intake.
[b*] between 22.34 and 101.13 g/day combined tomato intake.
[c*] between 101.13 and 202.95 g/day combined tomato intake.
[d*] over 202.95 g/day combined tomato intake.

factors, such as diet (31). A study has suggested that dietary intake, especially fats, may increase the risk of aggressive prostate tumours in older males (32).

This current study aimed to explore whether ethnic differences in diet and associations between diet and clinical markers of prostate disease might explain ethnic differences in the rates of prostate cancer.

In New Zealand ethnic prostate cancer incidence rates are inaccurate because of under-reporting in health data for Maori and Pacific Islands men. There are ethnic differences in mortality rates, with Pacific Islands and Maori men shown to have higher rates than New Zealand Europeans (33). Under-utilisation of the health system is a probable influence on the increased mortality rates for Pacific Islands and Maori males (6), however ethnic differences in disease progression may also contribute. Given the probable inaccuracy of the incidence rates, it appears important to examine mortality as an indication of increased disease burden for Pacific Islands and Maori men.

In the first part of this study, we investigated ethnic differences in consumption of foods that have been suggested to influence prostate cancer. There were significant dietary differences between the three New Zealand ethnic groups. The most significant difference in dietary practice was between New Zealand European and Pacific Islands subjects. The age group studied (40-69 years) probably influenced the magnitude of this difference. Results from this current study confirm the results of other New Zealand studies (16,34) and show that this generation of Pacific Islands people have traditional diets, eating more taro, shellfish and fresh vegetables and drinking less alcohol than New Zealand Europeans (16). The dietary pattern of Maori found in this study more closely resembled that of New Zealand European subjects, which may be because Maori now live a more westernised rather than traditional lifestyle (35).

Pacific Islands men have the highest mortality rate for prostate cancer, followed by Maori men and lowest in New Zealand Europeans. These differences in mortality could be contributed to by differences in rates of disease progression, influenced by dietary factors. However, the results of dietary components that have been associated with prostate cancer risk were variable and did not reflect an ethnic trend that could explain the differences in mortality rate. For example, Pacific Islands subjects were potentially most at risk from higher meat intake, but had possible protection from the highest fish intake. While New Zealand Europeans were potentially most at risk by more alcohol, and lower fruit and vegetable consumption, they had possible protection from eating more tomato sauce-based dishes. Perhaps the interaction of these dietary components is the important factor in determining risk and disease progression. For example, the protective effect of a high intake of bioavailable lycopene may modify the effects of other potentially harmful substances, such as alcohol. Results highlight the complexity of the risk/protective mechanisms conferred by dietary factors. Further research is needed to investigate the associations observed in this study and to help clarify the mechanisms by which certain foods might promote or protect against prostate cancer.

In the second part of this study we investigated clinical markers of prostate disease– PSA levels and urinary symptoms, in relation to diet. Studies in the early 1990s

confirmed that elevated total PSA could be used to identify patients with prostate cancer (36). The majority of PSA (70 to 90%) circulates as a complex (cPSA), while a smaller proportion (10-30%) circulates as fPSA (37). %fPSA is lower in many patients with prostate cancer. Studies in men with a PSA level between 4 and 10 ng/ml and a %fPSA greater than 25% indicate a prostate cancer risk of less than 8%, while a %fPSA less than 10% indicates a greater than 56% risk (37).

The effect of diet on PSA levels could be direct or *via* hormonal pathways involving androgens. PSA expression is differentially regulated by androgens acting on prostate cells (38). Androgenic activity can be influenced by diet; vegetarian men have been found to have less testosterone available for androgenic action (39). Diet can also affect PSA expression independently of hormonal pathways. For example, soy has been found to decrease PSA secretion and expression in androgen-independent prostate cell lines (40).

Several dietary factors were found to be significantly associated with markers of prostate disease. A negative association was found between beer consumption and both total serum PSA and free PSA; this could be due to a hormonal effect of the female flowers of the hop plant, which are used as a preservative and flavouring in beer. Hop phyto-oestrogens have been studied recently for their oestrogenic activity (41,42). It is possible that, with higher beer consumption, increased levels of phyto-oestrogens counteract the stimulatory effect of testosterone on the prostate, inhibit growth and interrupt PSA expression and secretion. The positive correlation found between tomato intake, specifically tomato paste, and %fPSA, supports the protective role of lycopene suggested in the literature.

The average PSA and fPSA was found to decrease moderately by quartile of beer intake. For example, there was a 0.75 ng/ml PSA difference between the 1st and 4th quartile of beer intake. fPSA and %fPSA were found to increase moderately by quartile of combined tomato and tomato sauce intake. There was a 5% increase in %fPSA between the 1st and 4th quartile of tomato sauce intake. These differences in PSA and %fPSA may be important on two levels: first, a standard diagnostic cut-off for these components is used to determine possible prostate cancer; second, it is possible that this degree of change in PSA could indicate tendencies in prostate cancer risk.

The strength of this study is the recruitment of a large number of Maori and Pacific Islands subjects, allowing some insights to be gained into differing dietary patterns, prostate cancer risk and PSA levels. However, limitations include the potential inaccuracies involved with the brief FFQ which did not cover total dietary intake, meaning it was not possible to control for other dietary factors including energy intake. However, it is reassuring that the dietary patterns found in this study are consistent with those found in other New Zealand studies (16, 34).

Conclusion

Ethnic differences in mortality could be contributed to by differences in rates of disease progression, influenced by dietary factors. However, the results did not reflect a consistent ethnic trend and highlight the complexity of the risk/protective mechanisms conferred by dietary factors. Further research is needed to ascertain whether the associations found between tomato and beer intake and PSA levels are biologically important, or are merely factors to be considered when interpreting PSA results clinically.

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