

Chinese tea consumption is associated with longer telomere length in elderly Chinese men

Ruth Chan^{1*}, Jean Woo¹, Eddie Suen¹, Jason Leung² and Nelson Tang³

¹Department of Medicine and Therapeutics, The Chinese University of Hong Kong, Shatin, Hong Kong

²Jockey Club Centre for Osteoporosis Care and Control, The Chinese University of Hong Kong, Shatin, Hong Kong

³Department of Chemical Pathology, The Chinese University of Hong Kong, Shatin, Hong Kong

(Received 18 March 2009 – Revised 10 June 2009 – Accepted 29 June 2009 – First published online 12 August 2009)

Environmental and lifestyle factors that affect oxidative stress and inflammation may influence telomere length (TL). There are limited data to relate the effect of dietary components on TL. The present study examined the association between food groups and TL in a sample of elderly Chinese. In a sample of 2006 Chinese (976 men and 1030 women) aged 65 years and over, TL was measured by quantitative real-time PCR and daily intake of food groups was assessed by a validated FFQ. Linear regression and analysis of covariance were used to examine the association between food group intake and TL, with adjustment for demographic and lifestyle factors. In men, only Chinese tea consumption was significantly associated with TL after adjustment for demographics and lifestyle factors ($P=0.002$). Mean difference in TL for those in the highest quartile of Chinese tea consumption (>3 cups/d or >750 ml/d) as compared with those in the lowest quartile of Chinese tea consumption (≤ 0.28 cups/d or ≤ 70 ml/d) was 0.46 kb, corresponding to approximately a difference of 5 years of life. In women, intake of fats and oils was borderline and negatively associated with TL after adjustment for demographic and lifestyle factors ($P=0.037$). In conclusion, Chinese tea consumption was positively associated with TL in elderly Chinese men.

Telomeres: Tea: Diet: Ageing

Telomeres are repeats of DNA sequences located at the end of chromosomes in eukaryotic cells⁽¹⁾. They are critical in maintaining chromosome stability, preventing fusion and atypical recombination, and are essential for cell division⁽²⁾. Telomere shortening, which is sensitive to cell division rates and the level of oxidative stress, has been associated with CHD, hypertension, dementia, obesity, insulin resistance, cigarette smoking, and bone mineral density^(3–6). The underlying cellular basis for the associations has also been studied in cardiac myocyte, smooth muscle and endothelial cells^(7,8). These studies gave rise to the concept of telomere shortening as an ageing biomarker or biochronometer, in that it is a marker of cell senescence and likely represents cumulative oxidative or inflammatory stress^(6,9,10) resulting in tissue functional attenuation.

Although genetics play an important role in determining telomere length (TL)^(11,12), environmental and lifestyle factors such as smoking⁽¹³⁾, physical activity⁽⁵⁾ and life stress⁽¹⁴⁾, which affect oxidative stress and inflammation, may influence the rate of cell turnover, which, in turn, may influence telomere shortening. There are, however, limited data to relate the effect of dietary components on telomere shortening. Numerous studies have shown that diet rich in fruits, vegetables, whole grains, soya and green tea was associated with reduced mortality from chronic diseases^(15–18).

Higher consumption of whole grains, fruits, vegetables, legumes, nuts and fish was also associated with lower concentrations of biomarkers of inflammation^(19,20). It is therefore possible to speculate that dietary components associated with oxidative stress and inflammation may influence TL. A recent study has examined the associations between TL and dietary patterns and food groups in a group of white, black and Hispanic adults⁽²¹⁾. The study reported an inverse association between processed meat intake and TL, whereas no association was observed for other food groups. To our knowledge, no further studies have reported the association between diet and TL in the literature.

In view of the differences in diet pattern among Chinese and other populations^(22,23), the present study examined the association between food groups and TL in a sample of Chinese aged 65 years and over.

Subjects and methods

Study population

About 2000 men and 2000 women aged 65 years and over living in the community participated in a health survey between 2001 and 2003. They were invited to attend a health check carried out in the School of Public Health of

Abbreviations: TL, telomere length; TRF, terminal restriction fragment.

* **Corresponding author:** Dr Ruth Chan, fax +852 2606 3500, email ruthchansm@cuhk.edu.hk

the Chinese University of Hong Kong, by placing recruitment notices in community centres for the elderly and housing estates. Several talks were also given at these centres explaining the purpose, procedures and investigations to be carried out. Subjects were volunteers, and the aim was to recruit a stratified sample so that approximately 33% would be in each of these age groups: 65–69, 70–74, 75+. The present study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving human subjects were approved by the Clinical Research Ethics Committee of the Chinese University of Hong Kong. Written informed consent was obtained from all subjects. The present study includes data from 976 men and 1030 women (50.2% all subjects) for whom TL and diet data were available.

Anthropometric measurement and dietary assessment

Information was collected regarding education, history of chronic diseases including heart diseases, diabetes and hypertension, smoking habit, alcohol use, physical activity and dietary intake. Physical activity was assessed by the Physical Activity Scale for the Elderly⁽²⁴⁾. Dietary intake was assessed using a FFQ, and mean nutrient quantitation per day was calculated using food tables derived from McCance and Widdowson⁽²⁵⁾ and the Chinese Medical Sciences Institute⁽²⁶⁾. The FFQ had been validated with the BMR calculation and the 24 h sodium/creatinine and potassium/creatinine analysis⁽²⁷⁾. In the present study, we included thirteen food groups from the FFQ in the analysis. The food groups included cereals; meats and poultry; egg and egg products; fish; milk and milk products; fruits and dried fruits; vegetables; legumes/nuts/seeds; pickled vegetables; dim sum; fast food; fats and oils for cooking; and Chinese tea. Each subject was asked to complete the questionnaire – the food item, the size of each portion, the number of times of consumption each day and each week. Portion size was explained to subjects using a catalogue of pictures of individual food portions. The amount of cooking oil was estimated according to the method of preparing different foods: 0.2 tablespoon for steaming fish or stir frying half a portion of vegetables, and one tablespoon for stir frying one portion of vegetables or one portion of meat. For food items consumed less than once per week, information was obtained for consumption pattern over 1 year and the quantitation per day or week adjusted accordingly. To assess Chinese tea consumption, Chinese tea was included as one of the food items in the validated FFQ. Similar to the assessment of other food groups described above, subject was asked about the consumption frequency of Chinese tea over the past year, such as never, a few times per year, once per week, every day etc. Subject was then asked to quantify the average cup(s) of Chinese tea drank per time, using 250 ml as one cup portion.

Body weight was measured to the nearest 0.1 kg with subjects wearing a light gown, by the Physician Balance Beam Scale (Healthometer, Chicago, IL, USA). Height was measured to the nearest 0.1 cm by the Holtain Harpenden stadiometer (Holtain Ltd, Crosswell, UK). BMI was calculated as body weight in kg/(height in m²).

Telomere length measurement in leukocytes by quantitative real-time PCR

The principle of the laboratory method has been previously described⁽²⁸⁾. In short, DNA was extracted in the peripheral blood by phenol–chloroform method and stored at –80°C with concentration >100 ng/μl (whereas working samples of 20 ng/μl were prepared before analysis). TL measurement followed the method published by Cawthon⁽²⁹⁾ with modification⁽³⁰⁾. Unlike the traditional method using terminal restriction fragment (TRF) analysis, this method used the technique of quantitative real-time PCR, which has the advantage of high throughput, time-saving and reproducible estimation of TL^(29,31–33). Roche LightCycler 480 (Roche, Mannheim, Germany) was used to perform the quantitative real-time PCR with primer sequences obtained from Cawthon⁽²⁹⁾. The T/S ratio ($C_t(\text{telomere assay})/C_t(\text{single copy gene assay})$) was used to assess the relative length of telomere, while C_t is the fractional cycle number for a threshold fluorescence level to be reached during quantitative real-time PCR. The T/S ratio ($\Delta\Delta C_t$) was then plotted against a standard calibration curve using samples with pre-determined TL to obtain the TL in unit of kilobps (kb). The percentage of CV of the C_t measurements of the telomere and the single copy gene probes was 3.6 and 1.8%, respectively. Two control samples were analysed in duplicates in each batch of assays, one sample was collected from a volunteers of age of 20s representing a long telomere control (long QC, by TRF = 11.6 kb) and another one from an elderly subject representing a short telomere control (short QC, by TRF = 8.2 kb). Within-batch and between-batch analytical imprecisions were determined from over twenty batches of assays using these two samples. The within-batch and between-batch percentage of CV of $\Delta\Delta C_t$ were 11.9 and 11.2% for the long QC sample. For the short QC sample, they were 8.1 and 14.2%, respectively.

A calibration curve was drawn by using $\Delta\Delta C_t$ values obtained from five different samples of pre-determined TL by TRF method. The coefficient of determination (R^2) of the linear correlation between TL by TRF method and $\Delta\Delta C_t$ was 0.63, which was similar to that reported (R^2 0.68) in the original description of the $\Delta\Delta C_t$ method by Cawthon⁽²⁹⁾. Calibration to TL in kb was carried out independently for each batch, which in a way partially corrected for between-batch variation in determination of $\Delta\Delta C_t$ and TL. After calibration to TL in kb, the within-batch and between-batch percentage of CV of TL were 8.5 and 7.5% for the long QC sample. For the short QC sample, they were 6.3 and 6.1%, respectively. A reduction in between-batch percentage of CV was noted, which reflected a partial correction of the between-batch variation in the calibration process.

Statistical analysis

Data analysis was performed using SAS version 9.1 (SAS Institute, Inc., Cary, NC, USA). Normality of the data was checked by histograms and variables were log transformed if required. Subjects' demographic and lifestyle characteristics were divided into categories to show frequency distributions whenever appropriate. ANOVA or independent *t* test was used to compare the average TL across categories of demographic and lifestyle characteristics. Linear regression was

Table 1. Characteristics of subjects and mean (SD) of telomere length (kb) across different demographic categories (Mean values and standard deviations; median values and interquartile ranges)

	Men (n 976)					Women (n 1030)				
	n	%	Mean	SD	P value*	n	%	Mean	SD	P value*
Age (years)										
65–69	291	29.8	8.97	1.65	0.116	395	38.3	9.29	2.24	0.577
70–74	348	35.7	8.80	1.63		344	33.4	9.45	2.21	
75+	337	34.5	8.70	1.58		291	28.3	9.30	2.35	
Education										
Primary or below	602	61.7	8.84	1.66	0.608	841	81.7	9.33	2.30	0.573
Secondary or above	374	38.3	8.78	1.57		189	18.3	9.43	2.10	
Current drinker										
Yes	216	22.1	8.83	1.61	0.838	32	3.1	9.11	2.19	0.541
No	760	77.9	8.81	1.63		997	96.9	9.36	2.27	
Current smoker										
Yes	121	12.4	9.04	1.68	0.096	23	2.2	9.34	2.80	0.992
No	855	87.6	8.78	1.61		1007	97.8	9.35	2.25	
Medical history of heart disease										
Yes	98	10.0	8.48	1.56	0.034	93	9.0	9.46	2.16	0.603
No	878	90.0	8.85	1.63		937	91.0	9.34	2.27	
Medical history of diabetes										
Yes	137	14.0	8.90	1.67	0.497	152	14.8	9.04	2.27	0.074
No	839	86.0	8.80	1.62		878	85.2	9.40	2.26	
Medical history of hypertension										
Yes	397	40.7	8.74	1.68	0.251	448	43.5	9.33	2.27	0.818
No	579	59.3	8.86	1.58		582	56.5	9.36	2.26	
	Mean	SD	Median	Interquartile range		Mean	SD	Median	Interquartile range	
Cigarette pack years			6.8	0.0–33.8				0.0	0.0–0.0	
BMI (kg/m ²)	23.4	3.1				23.9	3.5			
PASE	94.8	49.3				89.7	35.0			
Energy intake (kJ/d)	8658.6	2464.8				6748.7	1963.0			
Daily food group intake										
Cereals (g)	649.1	249.0				555.4	210.3			
Meat and poultry (g)	78.6	60.2				53.3	41.0			
Fish (g)	90.3	81.1				75.7	69.6			
Fruits and dried fruits (g)	264.1	188.6				258.4	219.8			
Vegetables (g)	245.5	168.6				245.8	165.4			
Dim sum (g)	69.7	66.2				41.2	43.5			
Fats and oils for cooking (g)	22.7	15.3				17.7	13.6			
Chinese tea (ml)	531.9	523.6				347.8	425.8			
Egg and egg products (g)			8.0	3.8–16.5				7.4	3.8–14.3	
Milk and milk products (g)			10.0	1.0–35.9				6.4	0.0–25.4	
Legumes, seeds and nuts (g)			30.0	15.0–56.1				25.6	12.2–49.4	
Pickled vegetables (g)			0.0	0.0–0.8				0.0	0.0–0.7	
Fast food (g)			0.0	0.0–7.8				0.0	0.0–4.7	

Food groups and telomere length

PASE, Physical Activity Scale for the Elderly.

* Comparison of telomere length across groups or between groups by ANOVA or independent *t* test, respectively.

used to examine the association of TL and food group intake. Unadjusted and multivariable models were used to examine the associations between TL and per 1 SD unit increase in food group intake. Multivariable model was adjusted for age (in years, continuous), BMI (in kg/m², continuous), energy intake (in kJ/d, continuous), education (below secondary, secondary or above), current drinker (yes or no), current smoker (yes or no), pack years of smoking (continuous), Physical Activity Scale for the Elderly (continuous), medical history of heart diseases (yes or no), diabetes (yes or no) and hypertension (yes or no). Significant associations were further characterised by calculating adjusted mean TL across categories of food group intake using analysis of covariance. Interactions between sex and food group intake were tested with cross product terms. Statistical tests were considered significant if $P < 0.05$ (two sided).

Results

Measurement of TL was carried out in 2006 subjects (976 men and 1030 women). There were no significant differences in demographic and lifestyle characteristics between those included in the present study (n 2006) and those not included in the present study (n 1994) (data not shown). For those included in the present study, their characteristics are shown in Table 1. Mean age was 72.8 (SD 5.0) years for men and 72.0 (SD 5.2) years for women. In men, older age tended to show shorter TL, but the difference in TL across age groups did not reach statistical significance ($P=0.116$). Those had history of heart disease showed shorter TL than those did not ($P=0.034$). In women, average TL did not differ significantly across different categories of demographic and lifestyle characteristics.

In men, only Chinese tea consumption was significantly associated with TL after multivariable adjustment ($P=0.002$; Table 2). No association was detected between other food groups and TL in the multivariable models. In women,

only intake of fats and oils was negatively associated with TL after adjustment for demographic and lifestyle factors ($P=0.033$).

When consumption of Chinese tea was divided into quartiles in men, mean TL was highest in those subjects in the highest quartiles of Chinese tea consumption (Table 3). Mean difference in TL for those in the highest quartile of Chinese tea consumption (>3 cups/d or >750 ml/d) as compared with those in the lowest quartile of Chinese tea consumption (≤ 0.28 cups/d or ≤ 70 ml/d) was 0.46 kb, corresponding to approximately a difference of 5 years of life⁽³⁴⁾. The association remained significant after full adjustment for demographic characteristics, lifestyle factors and medical history ($P < 0.001$). In women, mean TL was lowest in those subjects in the highest quartiles of cooking fats and oils intake (Table 4). The association was only statistically significant after adjustment for demographic characteristics, lifestyle factors and medical history ($P=0.037$).

Discussion

Diet rich in fruits, vegetables, whole grains, soya and green tea was associated with reduced mortality from chronic diseases^(15–18) and lower concentrations of biomarkers of inflammation^(19,20). It is thus speculated that dietary components associated with oxidative stress and inflammation may influence TL. A recent study has reported an inverse association between processed meat intake and TL, whereas no association was observed for other diet features with TL⁽²¹⁾. In the present study, of all food groups examined, only Chinese tea consumption was positively associated with TL and the association was only observed in men, whereas in women, intake of fats and oils was borderline and negatively associated with TL.

Beneficial effects of tea on health are extensively reviewed^(35,36). There are three major types of tea, depending on the level of fermentation. They are green (unfermented),

Table 2. Linear regression between telomere length (kb) and per 1 SD increase in the intake of different food groups

Food group	Men				Women			
	Unadjusted		Multivariable adjusted†		Unadjusted		Multivariable adjusted	
	<i>B</i>	<i>P</i> value	<i>B</i>	<i>P</i> value	<i>B</i>	<i>P</i> value	<i>B</i>	<i>P</i> value
Cereals (g/d)	−0.025	0.596	−0.012	0.858	0.021	0.659	−0.015	0.866
Meat and poultry (g/d)	0.030	0.595	0.060	0.284	0.008	0.912	−0.016	0.763
Egg and egg products (g/d)*	0.008	0.888	0.014	0.803	−0.003	0.968	−0.034	0.646
Fish (g/d)	−0.006	0.914	0.016	0.814	0.070	0.263	0.070	0.343
Milk and milk products (ml/d)*	0.079	0.292	0.079	0.157	0.061	0.428	0.061	0.549
Fruits and dried fruits (g/d)	−0.094	0.061	−0.094	0.103	0.066	0.389	0.022	0.701
Vegetables (g/d)	−0.034	0.593	−0.017	0.701	0.017	0.770	−0.015	0.854
Legumes, seeds and nuts (g/d)*	−0.010	0.851	−0.002	0.977	0.072	0.311	0.051	0.505
Pickled vegetables (g/d)*	0.035	0.503	0.034	0.527	−0.070	0.320	−0.088	0.229
Dim sum (g/d)	0.066	0.080	0.132	0.066	0.044	0.666	0.003	0.970
Fast food (g/d)*	−0.011	0.834	−0.017	0.757	0.048	0.502	0.018	0.817
Fats and oils for cooking (ml/d)	0.031	0.615	0.015	0.818	−0.136	0.047	−0.150	0.033
Chinese tea (ml/d)	0.157	0.001	0.157	0.002	−0.043	0.419	−0.043	0.500

PASE, Physical Activity Scale for the Elderly.

* Log transformed for linear regression.

† Adjusted for age (in years, continuous), BMI (in kg/m², continuous), energy intake (in kJ/d, continuous), education (below secondary, secondary or above), current drinker (yes or no), current smoker (yes or no), cigarette pack years (log transformed, continuous), PASE (continuous), medical history of heart disease (yes or no), diabetes (yes or no) and hypertension (yes or no).

Table 3. Adjusted telomere length by quartiles (Q) of Chinese tea consumption in men (Mean values with their standard errors)*

Chinese tea consumption	Intake range (cups/d)†	Telomere length (kb)			
		Unadjusted		Multivariable adjusted‡	
		Mean	SE	Mean	SE
Q1 (n 236)	≤ 0.28	8.72	0.11	8.73	0.15
Q2 (n 230)	> 0.28–1.99	8.84	0.11	8.84	0.16
Q3 (n 275)	> 1.99–3.00	8.56	0.10	8.54	0.16
Q4 (n 233)	> 3.00	9.20	0.11	9.19	0.16
P value		< 0.001		< 0.001	

* By analysis of covariance.

† 1 cup = 250 ml.

‡ Adjusted for age (in years, continuous), BMI (in kg/m², continuous), energy intake (in kJ/d, continuous), education (below secondary, secondary or above), current drinker (yes or no), current smoker (yes or no), cigarette pack years (log transformed, continuous), Physical Activity Scale for the Elderly (continuous), medical history of heart disease (yes or no), diabetes (yes or no) and hypertension (yes or no).

oolong (partially fermented) and black (fermented) tea⁽³⁵⁾. Both green and oolong teas are popular for Chinese population. In the present study, a positive association was observed between TL and Chinese tea consumption. The result was consistent with studies showing reduced risk of cancer⁽³⁶⁾, diabetes^(38,39), CVD⁽³⁷⁾ and mortality⁽³⁸⁾, and reduced level of inflammatory markers⁽³⁹⁾. The antioxidative properties of tea and its constituent nutrients may protect telomeres from oxidative damage in the normal ageing process. Tea is rich in polyphenols and contains carotenoids, tocopherols, ascorbic acid, minerals and certain phytochemical compounds^(35,40). These constituents have been suggested to work against oxidative damage in several ways, including scavenging harmful reactive nitrogen and oxygen species, acting as metal chelators and inhibiting lipoxygenase, cyclooxygenase and xanthine oxidase enzymes^(35,41,42). Although some studies suggested that subjects with high tea consumption were prone to have healthier lifestyle^(43,44), we tried to control for all possible confounding factors in the analyses. The positive association between TL and Chinese tea

consumption in men remained significant in the multivariable model, and it was unlikely that the significant association observed in men was due to chance. In contrast, there were some possibilities to explain the lack of association between tea consumption and TL in women; for example, the differences in demographic and lifestyle characteristics between men and women, such as education, smoking status and alcohol use. There was also no adjustment for psychosocial stress or socioeconomic factors such as income or occupation level before retirement in the present study. These factors have been associated with TL in previous studies^(14,45) and may be associated with dietary factors and therefore could be confounders. Moreover, the hormonal differences between men and women, and the somatic cell selection primarily be apparent in older women, may provide a survival advantage and a greater resistance to oxidative stress by telomeres in women⁽⁴⁶⁾. Therefore, the diet impact on TL may be less pronounced in women than in men.

In the present study, a significant and inverse association between intake of fats and oils and TL was observed in

Table 4. Adjusted telomere length by quartiles (Q) of intake of fats and oils for cooking in women (Mean values with their standard errors)*

Fats and oils consumption	Intake range (serving/d)†	Telomere length (kb)			
		Unadjusted		Multivariable adjusted‡	
		Mean	SE	Mean	SE
Q1 (n 223)	≤ 0.62	9.49	0.15	9.45	0.40
Q2 (n 273)	> 0.62–1.24	9.57	0.14	9.58	0.40
Q3 (n 240)	> 1.24–1.55	9.28	0.15	9.25	0.40
Q4 (n 291)	> 1.55	9.09	0.13	9.04	0.40
P value		0.059		0.037	

* By analysis of covariance.

† 1 serving = 15 g.

‡ Adjusted for age (in years, continuous), BMI (in kg/m², continuous), energy intake (in kJ/d, continuous), education (below secondary, secondary or above), current drinker (yes or no), current smoker (yes or no), cigarette pack years (log transformed, continuous), Physical Activity Scale for the Elderly (continuous), medical history of heart disease (yes or no), diabetes (yes or no) and hypertension (yes or no).

women when adjusting for demographic characteristics, lifestyle factors and medical history. Cooking method may be one possible reason to explain for the inverse association between TL and fats and oils intake. For example, eating raw vegetable is not common for Chinese and vegetable cooking methods in Chinese differ from those of the Western countries. For Chinese population, the majority of oil is used for cooking vegetables and the usual method is to stir-fry them. Our sample, similar to the general Hong Kong population, used mainly maize and peanut oil for cooking⁽⁴⁷⁾. Previous studies have suggested that stir-fry not only destroys nutrient values of vegetables⁽⁴⁸⁾, but also gives rise to a wide variety of mutagenic substances that are carcinogenic^(49,50). Epidemiological studies also reported that high temperature cooking was associated with elevated risk of cancer^(51,52).

No association between TL and other well-known factors such as age and smoking was observed in the present study. The result was different from previous studies^(13,21). However, many factors should be considered in the comparison between the present and previous studies. Firstly, the mean age of this sample was approximately 72 years, compared with the age range of 18–76 years for women in one study⁽¹³⁾ and 45–84 years for men and women in the other⁽²¹⁾. In examining association between TL and age, large number of subjects over a wide age range would be preferable. Our sample did not include young or middle-age subjects. Secondly, an overall of 7.2% of this sample (12.4% in men and 2.2% in women) was current smoker, compared with 18.4% for women in one study⁽¹³⁾ and a range of 9.9–13.3% for men and women in the other study⁽²¹⁾. The small proportion of current smoker in our sample may also explain the lack of association between TL and smoking.

There are limitations in the present study. The data are cross-sectional in nature and TL at a single point in time may not reflect telomere attrition, which is the construct of great interest. Inter-individual differences in TL are present at birth; thus, changes in TL over time may not mirror cross-sectional differences in absolute TL, even if age adjusted⁽⁵³⁾. In addition, we agree with the presence of heterogeneity in TL among different white cell populations. However, blood samples of the present study cohorts were collected from healthy elderly, and at the time of collection they did not suffer from any acute illness. Therefore, it could be assumed that they had steady cell counts of the leucocyte populations. The measurement only reflects the mean TL rather than specific length for particular leucocyte population. Furthermore, the same method of measurement of TL in the mixed white cells population had been used in many other studies and found to be correlated with biological parameters^(54–56). The reliance on self-report of health conditions may be another limitation in the present study. The cross-sectional design of the present study may also lead to survival bias. Those with diseases (and likely shorter TL) may have died earlier, so that the survivors may have relatively longer TL⁽⁵⁷⁾. Therefore, fewer associations with diseases or lifestyle factors may be observed. Furthermore, data obtained at one time point regarding environmental factors that are related to chronic inflammatory or oxidative stress may not be an accurate reflection of life course factors or cumulative stress.

In conclusion, the present cross-sectional study showed that Chinese tea consumption was positively associated with TL in elderly Chinese men. Intake of fats and oils may be associated with TL in elderly Chinese women. However, the underlying mechanisms by which tea and its constituent nutrients affect TL, and the interplay of diet constituents, choice of cooking methods and other lifestyle factors on TL remain to be determined in future prospective studies and clinical trials.

Acknowledgements

We wish to thank all subjects for their participation. The present study was supported by grants from the National Institute of Health, USA, with grant number 1R01, the Research Grants Council of Hong Kong, CUHK 4101/02M, and the Centre for Nutritional Studies, The Chinese University of Hong Kong. R. C. conceptualised the study, designed the analytic approach, interpreted the data and drafted the manuscript. J. W. conceptualised the study and contributed to revision of the manuscript. E. S. was involved in laboratory analysis and contributed to revision of the manuscript. J. L. contributed to data management and statistical analysis. N. T. provided expertise for laboratory analysis and contributed to revision of the manuscript. The authors declared no conflict of interests.

References

- Goronzy JJ, Fujii H & Weyand CM (2006) Telomeres, immune aging and autoimmunity. *Exp Gerontol* **41**, 246–251.
- Blasco MA (2007) The epigenetic regulation of mammalian telomeres. *Nat Rev Genet* **8**, 299–309.
- Wong JM & Collins K (2003) Telomere maintenance and disease. *Lancet* **362**, 983–988.
- Baird DM (2006) Telomeres. *Exp Gerontol* **41**, 1223–1227.
- Aviv A (2006) Telomeres and human somatic fitness. *J Gerontol A Biol Sci Med Sci* **61**, 871–873.
- Bekaert S, De Meyer T & Van Oostveldt P (2005) Telomere attrition as ageing biomarker. *Anticancer Research* **25**, 3011–3021.
- Minamino T & Komuro I (2007) Vascular cell senescence: contribution to atherosclerosis. *Circ Res* **100**, 15–26.
- Serrano AL & Andres V (2004) Telomeres and cardiovascular disease: does size matter? *Circ Res* **94**, 575–584.
- Monaghan P & Haussmann MF (2006) Do telomere dynamics link lifestyle and lifespan? *Trends Ecol Evol* **21**, 47–53.
- von Zglinicki T & Martin-Ruiz CM (2005) Telomeres as biomarkers for ageing and age-related diseases. *Current Molecular Medicine* **5**, 197–203.
- Slagboom PE, Droog S & Boomsma DI (1994) Genetic determination of telomere size in humans: a twin study of three age groups. [see comment]. *Am J Hum Genet* **55**, 876–882.
- Bischoff C, Graakjaer J, Petersen HC, *et al.* (2005) The heritability of telomere length among the elderly and oldest-old. *Twin Research Hum Genet Official J Int Soc Twin Studies* **8**, 433–439.
- Valdes AM, Andrew T, Gardner JP, *et al.* (2005) Obesity, cigarette smoking, and telomere length in women. *Lancet* **366**, 662–664.
- Epel ES, Blackburn EH, Lin J, *et al.* (2004) Accelerated telomere shortening in response to life stress. *Proc Natl Acad Sci U S A* **101**, 17312–17315.

15. Lockheart MS, Steffen LM, Rebnord HM, *et al.* (2007) Dietary patterns, food groups and myocardial infarction: a case-control study. *Br J Nutr* **98**, 380–387.
16. Brunner EJ, Mosdol A, Witte DR, *et al.* (2008) Dietary patterns and 15-y risks of major coronary events, diabetes, and mortality. *Am J Clin Nutr* **87**, 1414–1421.
17. Osler M, Heitmann BL, Gerdes LU, *et al.* (2001) Dietary patterns and mortality in Danish men and women: a prospective observational study. *Br J Nutr* **85**, 219–225.
18. Shimazu T, Kuriyama S, Hozawa A, *et al.* (2007) Dietary patterns and cardiovascular disease mortality in Japan: a prospective cohort study. *Int J Epidemiol* **36**, 600–609.
19. Lopez-Garcia E, Schulze MB, Fung TT, *et al.* (2004) Major dietary patterns are related to plasma concentrations of markers of inflammation and endothelial dysfunction. *Am J Clin Nutr* **80**, 1029–1035.
20. Chrysoshoou C, Panagiotakos DB, Pitsavos C, *et al.* (2004) Adherence to the Mediterranean diet attenuates inflammation and coagulation process in healthy adults: The ATTICA Study. *J Am Coll Cardiol* **44**, 152–158.
21. Nettleton JA, Diez-Roux A, Jenny NS, *et al.* (2008) Dietary patterns, food groups, and telomere length in the Multi-Ethnic Study of Atherosclerosis (MESA). *Am J Clin Nutr* **88**, 1405–1412.
22. Maskarinec G & Meng L (2001) An investigation of soy intake and mammographic characteristics in Hawaii. *Breast Cancer Res* **3**, 134–141.
23. Sowers MR, Crawford S, McConnell DS, *et al.* (2006) Selected diet and lifestyle factors are associated with estrogen metabolites in a multiracial/ethnic population of women. *J Nutr* **136**, 1588–1595.
24. Washburn RA, Smith KW, Jette AM, *et al.* (1993) The Physical Activity Scale for the Elderly (PASE): development and evaluation. *J Clin Epidemiol* **46**, 153–162.
25. Paul AA & Southgate DAT (1978) *McCance & Widdowson's: The Composition of Foods*, 4th ed. London: HMSO.
26. Yang Y, Wang G & Pan X (2002) *China Food Composition 2002*. Peking: University Medical Press.
27. Woo J, Leung SSF, Ho SC, *et al.* (1997) A food frequency questionnaire for use in the Chinese population in Hong Kong: Description and examination of validity. *Nutr Res* **17**, 1633–1641.
28. Woo J, Tang N, Suen E, *et al.* (2008) Telomeres and frailty. *Mech Ageing Dev* **129**, 642–648.
29. Cawthon RM (2002) Telomere measurement by quantitative PCR. *Nucleic Acids Res* **30**, e47.
30. Gil ME & Coetzer TL (2004) Real-time quantitative PCR of telomere length. *Mol Biotechnol* **27**, 169–172.
31. Norwood D & Dimitrov DS (1998) Sensitive method for measuring telomere lengths by quantifying telomeric DNA content of whole cells. *Biotechniques* **25**, 1040–1045.
32. Baird DM (2005) New developments in telomere length analysis. *Exp Gerontol* **40**, 363–368.
33. Gardner JP, Kimura M, Chai W, *et al.* (2007) Telomere dynamics in macaques and humans. *J Gerontol A Biol Sci Med Sci* **62**, 367–374.
34. Frenck RW Jr, Blackburn EH & Shannon KM (1998) The rate of telomere sequence loss in human leukocytes varies with age. *Proc Natl Acad Sci U S A* **95**, 5607–5610.
35. Cabrera C, Artacho R & Gimenez R (2006) Beneficial effects of green tea—a review. *J Am Coll Nutr* **25**, 79–99.
36. Kurahashi N, Sasazuki S, Iwasaki M, *et al.* (2008) Green tea consumption and prostate cancer risk in Japanese men: a prospective study. *Am J Epidemiol* **167**, 71–77.
37. Sasazuki S, Kodama H, Yoshimasu K, *et al.* (2000) Relation between green tea consumption and the severity of coronary atherosclerosis among Japanese men and women. *Ann Epidemiol* **10**, 401–408.
38. Kuriyama S, Shimazu T, Ohmori K, *et al.* (2006) Green tea consumption and mortality due to cardiovascular disease, cancer, and all causes in Japan: the Ohsaki study. *JAMA* **296**, 1255–1265.
39. De Bacquer D, Clays E, Delanghe J, *et al.* (2006) Epidemiological evidence for an association between habitual tea consumption and markers of chronic inflammation. *Atherosclerosis* **189**, 428–435.
40. Gardner EJ, Ruxton CH & Leeds AR (2007) Black tea—helpful or harmful? A review of the evidence. *Eur J Clin Nutr* **61**, 3–18.
41. Carlson JR, Bauer BA, Vincent A, *et al.* (2007) Reading the tea leaves: anticarcinogenic properties of (–)-epigallocatechin-3-gallate. *Mayo Clin Proc* **82**, 725–732.
42. Beltz LA, Bayer DK, Moss AL, *et al.* (2006) Mechanisms of cancer prevention by green and black tea polyphenols. *Curr Med Chem Anticancer Agents* **6**, 389–406.
43. Beitz R, Mensink GB, Hintzpetter B, *et al.* (2004) Do users of dietary supplements differ from nonusers in their food consumption? *Eur J Epidemiol* **19**, 335–341.
44. Lee SA, Xu WH, Zheng W, *et al.* (2007) Physical activity patterns and their correlates among Chinese men in Shanghai. *Med Sci Sports Exerc* **39**, 1700–1707.
45. Cherkas LF, Aviv A, Valdes AM, *et al.* (2006) The effects of social status on biological aging as measured by white-blood-cell telomere length. *Aging Cell* **5**, 361–365.
46. Aviv A, Shay J, Christensen K, *et al.* (2005) The longevity gender gap: are telomeres the explanation? *Sci Aging Knowledge Environ* **2005**, pe16.
47. Leung SSF, Ho SC & Woo J (1997) *Hong Kong Adult Dietary Survey 1995*. Hong Kong: Department of Paediatrics, The Chinese University of Hong Kong.
48. Moreno DA, Lopez-Berenguer C & Garcia-Viguera C (2007) Effects of stir-fry cooking with different edible oils on the phytochemical composition of broccoli. *J Food Sci* **72**, S064–S068.
49. Dung CH, Wu SC & Yen GC (2006) Genotoxicity and oxidative stress of the mutagenic compounds formed in fumes of heated soybean oil, sunflower oil and lard. *Toxicol in Vitro* **20**, 439–447.
50. Wu SC, Yen GC & Sheu F (2001) Mutagenicity and identification of mutagenic compounds of fumes obtained from heating peanut oil. *Journal of Food Prot* **64**, 240–245.
51. Dai Q, Shu XO, Jin F, *et al.* (2002) Consumption of animal foods, cooking methods, and risk of breast cancer. *Cancer Epidemiol Biomarkers Prev* **11**, 801–808.
52. Kotsopoulos J, Liede A, De Matsuda ML, *et al.* (2006) Method of cooking and risk of breast cancer in the Philippines. *Cancer Causes Control* **17**, 341–348.
53. Graakjaer J, Pascoe L, Der-Sarkissian H, *et al.* (2004) The relative lengths of individual telomeres are defined in the zygote and strictly maintained during life. *Aging Cell* **3**, 97–102.
54. Barwell J, Pangon L, Georgiou A, *et al.* (2007) Is telomere length in peripheral blood lymphocytes correlated with cancer susceptibility or radiosensitivity? *Br J Cancer* **97**, 1696–1700.
55. Brouillette SW, Moore JS, McMahon AD, *et al.* (2007) Telomere length, risk of coronary heart disease, and statin treatment in the West of Scotland Primary Prevention Study: a nested case-control study. *Lancet* **369**, 107–114.
56. Trkova M, Prochazkova K, Krutilkova V, *et al.* (2007) Telomere length in peripheral blood cells of germline TP53 mutation carriers is shorter than that of normal individuals of corresponding age. *Cancer* **110**, 694–702.
57. Aviv A, Valdes AM & Spector TD (2006) Human telomere biology: pitfalls of moving from the laboratory to epidemiology. *Int J Epidemiol* **35**, 1424–1429.