CVD Prevention and Control (2008) **3,** 133—140

WORLD HEART FEDERATION®

www.elsevier.com/locate/precon

The association of alcohol, tea, and other modifiable lifestyle factors with myocardial infarction and stroke in Chinese men

Wanqing Wen [a,*], Yong-Bing Xiang [b], Wei Zheng [a], Wang-Hong Xu [b], Gong Yang [a], Honglan Li [b], Xiao Ou Shu [a]

[a] *Vanderbilt Epidemiology Center, Institute of Medicine & Public Health, Vanderbilt University Medical Center, Sixth Floor, Suite 600, 2525 West End Avenue, Nashville, TN 37203-1738, United States*
[b] *Department of Epidemiology, Shanghai Cancer Institute, #25 2200 Xie Tu Road, Shanghai 200032, People's Republic of China*

Received 23 July 2007; revised 31 October 2007; accepted 5 July 2008
Available online 3 August 2008

KEYWORDS
Stroke;
Myocardial infarction;
Alcohol consumption;
Tea consumption;
Weight change

Summary
Background: Studies of the lifestyle predictors of cardiovascular diseases (CVD) have been predominantly conducted in Caucasian populations. There are few data from other populations, such as Chinese men, who have different lifestyles and a different spectrum of CVD as compared with Caucasian populations.
Methods: Based on the baseline data of the Shanghai Men's Health Study during March 2002—June 2006, a matched case-control analysis including 518 myocardial infarction, 333 hemorrhagic stroke, and 1927 ischemic stroke cases was conducted. Five controls were selected for each case. The lifestyle factors under study included alcohol, tea and ginseng consumption, physical activity during adolescence, and weight change from age 20 to 40. The associations of these lifestyle factors with myocardial infarction and stroke were evaluated. To account for the misclassification of exposures and disease diagnosis, a sensitivity analysis was conducted.
Results: Alcohol consumption was inversely associated with myocardial infarction (OR = 0.63, 95% CI: 0.50, 0.80). Tea consumption was inversely associated with hemorrhagic (OR = 0.63, 95% CI: 0.49, 0.81) and ischemic stroke (OR = 0.77, 95% CI: 0.69, 0.85). Weight increase from age 20 to 40 was positively associated with myocardial infarction and stroke in a dose-response manner (trend $p < 0.001$).
Conclusions: Alcohol and tea consumption may decrease the prevalence of myocardial infarction and stroke, respectively. Weight increase from age 20 to 40 may increase the prevalence of myocardial infarction and stroke in Chinese men.

* Corresponding author. Tel.: +1 615 936 0747; fax: +1 615 936 1269.
E-mail address: wanqing.wen@vanderbilt.edu (W. Wen).

1875-4570/$ - see front matter
doi:10.1016/j.cvdpc.2008.07.001

Introduction

Studies have shown that various lifestyle factors, such as cigarette smoking, alcohol consumption, lack of exercise, and overweight, are important predictors of cardiovascular diseases (CVD). Investigation into these modifiable CVD risk factors offers great potential for CVD prevention. A large international case-control study of risk factors for myocardial infarction conducted in 52 countries (the INTERHEART study) has shown that nine easily measured and potentially modifiable risk factors, abnormal lipids, smoking, hypertension, diabetes, abdominal obesity, psychosocial factors, low consumption of fruits and vegetables, a pattern of drinking alcohol and lack of regular physical activity, account for more than 90% of the risk of myocardial infarction [1]. Although the INTERHEART study has shown that the effect of these nine risk factors is consistent across all geographic regions and ethnic groups of the world, the prevalence of these risk factors might vary [1] and other studies have indicated that the effects of lifestyle factors on CVD risk might differ across populations [2,3]. For example, the association between moderate alcohol consumption and ischemic stroke has been consistently observed for white populations, but there is little if any evidence of an association for Japanese and possibly black populations [2]. Likewise, the association between tea consumption and coronary heart disease risk was positive in the United Kingdom, but negative in continental Europe [3]. Therefore, it is important to investigate significant lifestyle risk factors for CVD in particular populations in order to encourage effective modification of these factors.

So far, studies on CVD lifestyle predictors have been conducted predominantly in populations of European origin [1]. There are few data from other populations, such as Chinese men, who have different lifestyles and a different spectrum of CVD as compared with other populations. Chinese people, for example, often drink tea, many often use ginseng, a popular herbal medicine used to promote health in many Asian countries, and engage in some type of physical activity of slow motion and moderate intensity. In contrast with Western populations where coronary heart disease is the leading cause of death, stroke is the most common cause of death in China, and there is a relatively high proportion of hemorrhagic stroke [4].

In this report, we use the baseline data of the Shanghai Men's Health Study (SMHS) to examine particular lifestyle factors in relation to the risk of myocardial infarction, hemorrhagic stroke, and ischemic stroke in this population of Chinese men.

Methods

The SMHS is an ongoing population-based prospective cohort study conducted in eight communities of urban Shanghai, China with a primary focus on the relationship of diet to cancer and other chronic diseases. Subject recruitment began in March 2002 and was completed in June 2006. Using a roster provided by the community office, a total of 83,125 permanent male residents between 40 and 74 years of age were approached for the study by a trained interviewer (retired nurse or physician). The purpose of the study was explained and informed consent was obtained from each participant. Afterwards, the interviewer administered an interview (about 60 min long) using a structured questionnaire that covered socio-economic, anthropometric, dietary, and lifestyle information, and disease history. Of the eligible men approached for the study, 61,582 participated with a participation rate of 74.1%. Among non-participants, there were 17,513 refusals (21.1%), 1360 men who had a serious health problem (1.6%), 2252 men who were absent during the study period (3.1%), and 118 subjects who did not participate for other miscellaneous reasons (0.1%).

Data collected during the SMHS baseline interview form the basis of the current study. Information about myocardial infarction and stroke diagnosis was collected using the following questions: (1) Have you ever been diagnosed with a myocardial infarction and if yes, what was the age at diagnosis? (2) Have you ever been diagnosed with a stroke and if yes, what type of stroke was it? A total of 518 myocardial infarction patients and 2324 stroke patients (333 hemorrhagic stroke cases, 1927 ischemic stroke cases, and 64 stroke cases with unknown type; the latter were excluded from this study) were identified and were considered as cases in this study.

We used a matched case-control design to select comparable controls for cases for two reasons. The first was to control the confounding effect of some demographic factors (age and education level) and cigarette smoking, a confirmed risk factor for CVD. The second was to provide a reference time for controls in terms of the age of the cases when diagnosed in each matched set, so that exposure before the reference/diagnosis time and the same time window for exposures for both cases and controls could be attained. Because lifestyles may change as a result of disease diagnosis, only lifestyle exposures prior to disease diagnosis were evaluated.

Controls were randomly selected from the entire cohort, excluding cases, and matched to cases on

age at the baseline interview (within 5-year intervals), education level, status of cigarette smoking, and if a smoker, the age at which he started smoking (within 5-year intervals). Five controls were selected for each case, resulting in 2590 controls for 518 myocardial infarction cases, 1665 controls for 333 hemorrhagic stroke cases, and 9635 controls for 1927 ischemic stroke cases.

The lifestyle factors under study included consumption of alcohol, tea and ginseng, physical activity during adolescence (age 13–15), and weight change from age 20 to 40. Alcohol, tea, and ginseng consumption were divided into two categories (ever or never) based on whether a subject had ever consumed alcohol or tea at least 3 times a week for more than 6 months or whether a subject had ever consumed ginseng at least 5 times a year for more than 1 year. Only those who consumed alcohol, tea, and ginseng up until one year prior to diagnosis for myocardial infarction cases and up until 5 years prior to the baseline interview for stroke cases (because information on the age at diagnosis of stroke cases was not available) were considered as ever consumed. We categorized subjects' physical activity during adolescence into three levels. Subjects were categorized as inactive, if the time spent on physical activity was less than the median and the subject had never been on a sports team at any level or had never participated in any tournament during that period of time. Subjects categorized as active, if the time spent on physical activity was greater than the median and the subject had ever been on a sports team at any level or had ever participated in a tournament during that period of time. All others were categorized as moderate. Weight change was defined as the weight difference in kilograms between age 20 and 40.

The data in this study were from the SMHS baseline interview, thus, we could only evaluate the association of lifestyle factors with a history of a myocardial infarction or stroke, which was estimated with odds ratios (OR) and 95% confidence intervals (95% CI) from conditional logistic models. All the lifestyle factors of interest were included in the same model for mutual adjustment. In addition, family history of coronary heart disease and/or stroke and annual personal income were also adjusted for. Body mass index at age 20 was additionally adjusted for when analyzing the effect of weight change.

To account for the misclassification of exposures and disease diagnosis, we conducted a probabilistic sensitivity analysis using a recently proposed method by Fox et al. [5]. The method involves reconstructing the data with bootstrapping that would have been observed had the misclassified variable been correctly classified, given the sensitivity and specificity of the classification. Because the true sensitivity and specificity were seldom known with certainty, we drew the sensitivity and specificity from a trapezoidal distribution, as suggested by Fox, which is specified by four points: the lower and upper bounds and the lower and upper modes between which the probability density is flat. We allowed for differential misclassification of the exposure of interest by drawing the sensitivity and specificity from separate trapezoidal distributions for cases and controls. Similarly, we allowed for differential misclassification of the disease diagnosis by drawing the sensitivity and specificity from separate trapezoidal distributions for the exposed and unexposed. A logistic model adjusted for the covariates was used for each reconstructed dataset to obtain an association point estimate ($OR_{corrected\text{-}1}$). In each data reconstruction, a random error was also taken into account by estimating $OR_{corrected\text{-}2}$, an association point estimate accounting for both the systematic and random error, as $OR_{corrected\text{-}2} = OR_{corrected\text{-}1}/\exp(z \times s)$, where z is a randomly chosen standard normal deviate and s is the standard error of the $\log(OR_{observed})$. After 3000 reconstructions of the data for each variable under study, we calculated the medians of $OR_{corrected\text{-}1}$ and $OR_{corrected\text{-}2}$, and 2.5 percentile and 97.5 percentile (for 95% simulation confidence interval) in each case. The present method can only handle binary variables. SAS version 9.1.3 (SAS Institute Inc., Cary, NC) was used for all the statistical analyses.

Results

Table 1 presents comparisons of basic characteristics of myocardial infarction cases, stroke cases, and control groups. The cases and controls were well matched on age, education level, and smoking status. Compared with their controls, the hemorrhagic and ischemic stroke patients had a lower income ($p < 0.05$). As expected, the myocardial infarction patients were more likely to have a family history of coronary heart disease and the stroke patients were more likely to have a family history of stroke ($p < 0.01$). These factors were adjusted for in subsequent analyses.

Shown in Table 2 are the odds ratios ($OR_{observed}$) and their 95% CI for the association of lifestyle factors with the occurrence of a myocardial infarction and stroke. Compared with those who never drank

Table 1 Case-control comparisons of the basic characteristics

	Myocardial infarction		Hemorrhagic stroke		Ischemic stroke	
	Case (%) (N = 518)	Control (%) (N = 2590)	Case (%) (N = 333)	Control (%) (N = 1665)	Case (%) (N = 1927)	Control (%) (N = 9635)
Age (mean ± SD)						
	64.7 ± 8.0	64.5 ± 8.2	62.3 ± 8.8	62.2 ± 8.8	65.9 ± 7.5	65.9 ± 7.5
t-Test	p = 0.513		p = 0.880		p = 0.241	
Ever smoked (%)						
Yes	70.7	70.7	63.7	63.7	20.8	20.2
No	29.3	29.3	36.3	36.3	6.7	7.3
Chi-square test	p = 1.000		p = 1.000		p = 1.000	
Starting age of smoking (mean ± SD) (among those ever smoked)						
	24.8 ± 8.2	25.2 ± 8.7	24.0 ± 7.5	24.6 ± 8.8	25.2 ± 8.8	25.2 ± 8.7
t-Test	p = 0.370		p = 0.337		p = 0.939	
Education (%)						
<Middle school	14.2	14.2	20.4	20.4	21.6	21.6
=Middle school	28.8	28.8	35.1	35.1	33.7	33.7
=High school	26.0	26.0	24.9	24.9	22.8	22.8
>High school	31.0	31.0	19.5	19.5	22.0	22.0
Chi-square test	p = 1.000		p = 1.000		p = 1.000	
Income (yuan per month per person) (%)						
<500	6.8	7.4	12.3	8.5	8.7	8.1
500—1000	46.9	47.1	55.0	51.3	55.8	51.2
1000—2000	37.7	37.6	27.0	33.0	30.8	33.6
2000—3000	7.4	6.4	4.2	5.9	4.2	6.0
>3000	1.2	1.5	1.5	1.3	0.5	1.0
Chi-square test	p = 0.888		p = 0.048		p < 0.001	
Family history of coronary heart diseases (%)						
Yes	29.3	14.5	14.1	14.5	16.2	13.3
No	70.7	85.5	85.9	85.5	83.8	86.7
Chi-square test	p < 0.001		p = 0.842		p = 0.001	
Family history of stroke (%)						
Yes	25.9	21.0	26.7	18.6	28.4	18.8
No	74.1	79.0	73.3	81.4	71.6	81.2
Chi-square test	p = 0.014		p < 0.001		p = 0.001	

alcohol, alcohol drinkers ($OR_{observed}$ = 0.63, 95% CI: 0.50, 0.80) had a decreased risk of myocardial infarction. Men who started drinking alcohol at an older age had a lower risk of myocardial infarction ($OR_{observed}$ = 0.65, 95% CI: 0.46, 0.90 for those who started drinking alcohol at age 25—39 and $OR_{observed}$ = 0.30, 95% CI: 0.18, 0.51 for those who started drinking alcohol at age 40 or older, trend p < 0.001). Alcohol consumption was not significantly associated with a hemorrhagic stroke ($OR_{observed}$ = 1.20, 95% CI: 0.92, 1.57) or ischemic stroke ($OR_{observed}$ = 1.04, 95% CI: 0.93, 1.17). Tea consumption, on the other hand, was not significantly associated with myocardial infarction ($OR_{observed}$ = 0.89, 95% CI: 0.72, 1.09), but was associated with a decreased risk of hemorrhagic stroke ($OR_{observed}$ = 0.63, 95% CI: 0.49, 0.81) and ischemic stroke ($OR_{observed}$ = 0.77, 95% CI: 0.69, 0.85). The age at which men started drinking tea did not appear to modify the association with stroke. The effect of ginseng on the risk of myocardial infarction and stroke was not evident. Physical activity during adolescence seemed to be consistently associated with a decreased risk of myocardial infarction and stroke, although the association was not statistically significant. Weight increase from age 20 to 40 was consistently associated with an increased risk of myocardial infarction and stroke in a dose-response manner (trend p < 0.001 for each case) with adjustment for other lifestyle factors and BMI at age 20. We did a similar analysis using the difference in BMI between ages 20 and 40, instead of weight change, and found a very similar association pattern (data not shown).

Table 2 Associations of lifestyle factors with the risk of myocardial infarction and stroke

Exposure	Myocardial infarction		Hemorrhagic stroke		Ischemic stroke	
	Case/Cont[a]	$OR_{observed}$ (95% CI)[b]	Case/Cont[a]	$OR_{observed}$ (95% CI)[b]	Case/Cont[a]	$OR_{observed}$ (95% CI)[b]
Alcohol consumption						
No	394/1752	1.00 (reference)	217/1132	1.00 (reference)	1325/6645	1.00 (reference)
Yes	124/838	0.63 (0.50–0.80)	116/533	1.20 (0.92–1.57)	602/2990	1.04 (0.93–1.17)
Starting age of alcohol drinking						
<25	60/292	0.90 (0.66–1.24)	48/198	1.38 (0.95–2.01)	240/1038	1.21 (1.03–1.42)
25–39	48/327	0.65 (0.46–0.90)	50/202	1.36 (0.95–1.94)	228/1105	1.08 (0.93–1.27)
40+	16/219	0.30 (0.18–0.51)	18/133	0.76 (0.45–1.28)	134/847	0.82 (0.67–1.01)
Tea consumption						
No	208/967	1.00 (reference)	168/667	1.00 (reference)	890/3892	1.00 (reference)
Yes	310/1623	0.89 (0.72–1.09)	165/998	0.63 (0.49–0.81)	1073/5742	0.77 (0.69–0.85)
Starting age of tea drinking						
<25	127/592	1.01 (0.78–1.31)	79/366	0.82 (0.59–1.14)	354/1899	0.79 (0.68–0.91)
25–39	128/683	0.89 (0.69–1.15)	55/418	0.51 (0.36–0.72)	430/2377	0.78 (0.68–0.89)
40+	55/348	0.72 (0.52–1.02)	31/214	0.55 (0.36–0.85)	253/1467	0.73 (0.63–0.85)
Ginseng use						
No	397/2007	1.00 (reference)	266/1253	1.00 (reference)	1431/7239	1.00 (reference)
Yes	121/583	1.03 (0.82–1.29)	67/412	0.77 (0.57–1.03)	496/2396	1.07 (0.95–1.20)
Physical activity during adolescence						
Inactive	100/432	1.00 (reference)	73/296	1.00 (reference)	400/1948	1.00 (reference)
Moderate	375/1917	0.84 (0.66–1.08)	231/1238	0.76 (0.56–1.03)	1398/6986	0.97 (0.86–1.10)
Active	43/241	0.74 (0.49–1.12)	29/131	0.83 (0.50–1.38)	129/701	0.91 (0.72–1.14)
Weight change from age 20 to 40						
<0 kg	22/158	0.73 (0.44–1.21)	15/97	0.74 (0.40–1.36)	86/589	0.85 (0.66–1.09)
0 to <5 kg	115/703	1.00 (reference)	79/467	1.00 (reference)	443/2841	1.00 (reference)
5 to <10 kg	112/564	1.21 (0.91–1.61)	55/344	1.00 (0.69–1.47)	385/2030	1.26 (1.08–1.46)
10 to <15 kg	83/397	1.34 (0.97–1.84)	53/239	1.45 (0.98–2.16)	293/1179	1.73 (1.46–2.04)
⩾15 kg	81/277	2.01 (1.43–2.82)	51/201	1.77 (1.17–2.67)	248/911	1.98 (1.65–2.37)
Weight change from age 20 to 40						
<10 kg	249/1425	1.00 (reference)	149/908	1.00 (reference)	914/5460	1.00 (reference)
⩾10 kg	164/674	1.49 (1.18–1.88)	104/440	1.63 (1.22–2.18)	541/2090	1.68 (1.48–1.90)

[a] Number of cases/controls.
[b] Adjusted for family history of coronary heart disease and/or stroke and annual personal income. All the lifestyle factors of interest were also included in the same model for mutual adjustment. Body mass index at age 20 was additionally adjusted for when analyzing the effect of weight change.

As shown in Table 2, alcohol and tea consumption and weight change were the most significant predictors. We selected these three factors for the sensitivity analysis and divided weight change into two categories (<10 and ⩾10 kg). The odds ratios for these three factors after correcting for only misclassification ($OR_{corrected\text{-}1}$) and for both misclassification and random error ($OR_{corrected\text{-}2}$) and their 95% CI are presented in Table 3. The corrected associations, without assuming a non-differential misclassification, were in the same directions but were stronger than the associations shown in Table 2. The 95% CI of the $OR_{corrected\text{-}1}$, however, were in general wider than the 95% CI of the $OR_{observed}$ (Table 2), and the 95% CI of the $OR_{corrected\text{-}2}$ were even wider. The 95% CI for correcting misclassification of disease diagnosis were wider than those for correcting misclassification of exposure even if higher sensitivity and specificity were specified for the former than for the latter (a minimum of 90%, modes of 93% and 97%, and a maximum of 100% for diagnosis misclassification versus a minimum of 80%, modes of 85% and 95%, and a maximum of 100% for exposure misclassification). We further conducted the sensitivity analysis on disease misclassification by specifying the sensitivity and specificity distribution with a minimum of 85%, modes of 90% and 95%, and a maximum of 100% for each, and a minimum of 80%, modes of 85% and 95%, and a maximum of 100%

Table 3 Results of sensitivity analysis correcting for misclassification of the selected exposures and for disease misclassification

	OR (%95 CI)[a]		
	Myocardial infarction	Hemorrhagic stroke	Ischemic stroke
Exposure misclassification[b]			
Alcohol			
$OR_{corrected\text{-}1}$	0.60 (0.35—0.93)	1.31 (0.89—1.92)	1.05 (0.73—1.51)
$OR_{corrected\text{-}2}$	0.60 (0.32—1.00)	1.31 (0.82—2.12)	1.05 (0.72—1.55)
Tea			
$OR_{corrected\text{-}1}$	0.88 (0.63—1.20)	0.60 (0.44—0.84)	0.70 (0.52—0.91)
$OR_{corrected\text{-}2}$	0.88 (0.61—1.25)	0.61 (0.41—0.92)	0.70 (0.51—0.92)
Weight change			
$OR_{corrected\text{-}1}$	1.62 (1.15—2.39)	1.80 (1.26—2.65)	1.90 (1.31—2.93)
$OR_{corrected\text{-}2}$	1.61 (1.05—2.53)	1.80 (1.13—2.90)	1.90 (1.28—3.02)
Disease misclassification[c]			
Alcohol			
$OR_{corrected\text{-}1}$	0.61 (0.37—0.90)	1.32 (0.94—1.91)	1.05 (0.80—1.43)
$OR_{corrected\text{-}2}$	0.60 (0.36—0.94)	1.33 (0.84—2.10)	1.06 (0.77—1.47)
Tea			
$OR_{corrected\text{-}1}$	0.87 (0.63—1.19)	0.59 (0.39—0.84)	0.71 (0.52—0.95)
$OR_{corrected\text{-}2}$	0.87 (0.59—1.28)	0.59 (0.36—0.92)	0.71 (0.51—0.97)
Weight change			
$OR_{corrected\text{-}1}$	1.61 (1.01—2.27)	1.82 (1.28—2.97)	1.88 (1.39—2.70)
$OR_{corrected\text{-}2}$	1.63 (0.80—2.74)	1.85 (1.14—3.15)	1.89 (1.36—2.78)

[a] Adjusted for family history of coronary heart disease and/or stroke and annual personal income. All the lifestyle factors of interest were also included in the same model for mutual adjustment. Body mass index at age 20 was additionally adjusted for when analyzing the effect of weight change.

[b] Sensitivity and specificity among cases and controls were drawn from trapezoidal distributions independently with a minimum of 80%, modes of 85% and 95%, and a maximum of 100%.

[c] Sensitivity and specificity among the exposed and unexposed were drawn from trapezoidal distributions independently with a minimum of 90%, modes of 93% and 97%, and a maximum of 100%.

for each (the same specification as for exposure misclassification). We found that the association patterns remained similar, but the 95% CI become wider (and contained 1) as lower sensitivity and specificity were specified (data not shown).

Discussion

Light to moderate alcohol consumption has consistently been associated with a lower risk of myocardial infarction in Western populations [6—9]. The INTERHEART study found a protective effect on myocardial infarction of alcohol consumption of three or more times per week [1]. A recent cohort study reported that risk of myocardial infarction or coronary death decreased in a dose-response manner as alcohol consumption increased [10]. The relationship of alcohol with stroke is more complicated. Studies have shown that alcohol has a positive dose-response association with hemorrhagic stroke risk [11—13]. However, reports were less consistent about alcohol-ischemic stroke relations. Several studies have shown evidence for a protective effect of alcohol consumption on ischemic stroke risk [14,15], but others failed to show any effect [12,13]. A recent cohort study reported a U-shaped association between alcohol consumption and ischemic stroke risk [16]. Consistent with most previous studies, our study found that alcohol was associated with a decreased risk of myocardial infarction, but was not significantly associated with hemorrhagic or ischemic stroke.

One meta-analysis showed a consistent reverse association of tea consumption with myocardial infarction risk [3], but the association with stroke risk varied across the geographic region, where the studies were conducted. Findings from the United Kingdom and Australia indicated a positive association; whereas a negative association with stroke risk was found in studies conducted in the

United States, continental Europe, and Asia [3,17]. One explanation for the marked heterogeneity of the association may be due to residual confounding of socio-economic status. For example, tea consumption is more common among people of low socio-economic status in the United Kingdom, whereas higher tea consumption might be a surrogate for a healthier lifestyle in the United States [3,18,19]. A recent Japanese study found that green tea consumption was associated with reduced mortality due to cardiovascular disease, particularly due to stroke [20]. Consistent with previous reports, our findings show that tea consumption was inversely associated with both hemorrhagic and ischemic stroke after adjustment for socio-economic status and other lifestyle factors.

No study has published results on the effect of ginseng on CVD risk. In our study, ginseng did not seem to have a significant impact on the risk of myocardial infarction or stroke.

The benefits of physical activity have been well documented. Guidelines endorsed by the Centers for Disease Control and Prevention and the National Institutes of Health recommend at least 30 min of moderately intense physical activity a day [2]. Many studies have demonstrated a protective effect of physical activity on myocardial infarction and stroke risk [1,21—26]. Our study found a nonsignificant association of physical activity during adolescence with a decreased risk of myocardial infarction and stroke later in life, although this association could be explained, at least in part, by physical activity during adulthood for people who maintain healthy habits.

Many previous studies have shown that obesity is an independent risk factor for myocardial infarction and stroke [1,27—30]. Consistent with previous reports, our study suggests a dose-response relation between weight increase during adulthood (from age 20 to 40) and the risk of myocardial infarction and stroke. It is likely that weight increase may act as a proxy for high blood pressure, a known major risk factor for both myocardial infarction and stroke. Our recent report derived also from SMHS data indicated that both systolic and diastolic blood pressure measured at the baseline survey were closely related with weight gain since age 20 [31]. We could not directly use blood pressure as a predictor in the current analysis since it was measured after the onset of self-reported myocardial infarction and stroke.

Several methodological limitations need to be kept in mind when interpreting our results. First, our study is based on prevalent cases, only those patients who were alive at the time of the SMHS baseline interview were included in the study. The exclusion of deceased patients, particularly hemorrhagic stroke patients who have a high fatality rate, could possibly have induced a selection bias. Second, the residual confounding of unmeasured factors, such as blood pressure, serum levels of lipids, and physical activity during adulthood, could affect the association estimates of weight change and physical activity during adolescence. Third, we did not have information on the type, duration, or amount of alcohol consumption during the year prior to the diagnosis of the study cases. Previous studies have indicated that the effect of alcohol is probably dependent on dose, only light to moderate alcohol consumption has beneficial effects, and the protective effect of alcohol is limited to wine versus other liquors. It has been suggested that the polyphenols found in wine have beneficial effects on the cardiovascular system [19]. However, the inclusion of heavy drinkers in the alcohol drinker category would bias our estimate towards the null, and some studies have found no difference between the effects of wine, beer, and liquor [14]. In our study, we did find an association between men who started drinking at an older age with a lower risk of myocardial infarction. Our data show that, among controls, those who started drinking alcohol at an older age consumed significantly less alcohol at the baseline survey than those who started drinking at a younger age (data not shown). Finally, the diagnosis of myocardial infarction and stroke was based on self-reports, and thus, may be subject to disease misclassification. To address the last two limitations, we conducted a sensitivity analysis and found that the association patterns held.

In summary, our findings were consistent with most previous reports and provide population-based evidence that in this Chinese population, alcohol consumption may decrease the risk of myocardial infarction, tea consumption may decrease the risk of stroke, and weight increase from age 20 to 40 may increase the risk of myocardial infarction and stroke in a dose-response manner.

Conflict of interest statement

None declared.

Acknowledgement

This study was supported by USPHS Grant No. RO1 CA82729.

References

[1] Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet 2004;364(9438):937—52.

[2] Boden-Albala B, Sacco RL. Lifestyle factors and stroke risk: exercise, alcohol, diet, obesity, smoking, drug use, and stress. Curr Atheroscler Rep 2000;2:160—6.

[3] Peters U, Poole C, Arab L. Does tea affect cardiovascular disease? A meta-analysis. Am J Epidemiol 2001;154: 495—503.

[4] Zhang LF, Yang J, Hong Z, et al. Proportion of different subtypes of stroke in China. Stroke 2003;34:2091—6.

[5] Fox MP, Lash TL, Greenland S. A method to automate probabilistic sensitivity analysis of misclassified binary variables. Int J Epidemiol 2005;34:1370—6.

[6] Maclure M. Demonstration of deductive meta-analysis: ethanol intake and risk of myocardial infarction. Epidemiol Rev 1993;15:328—51.

[7] Gaziano JM, Buring JE, Breslow JL, et al. Moderate alcohol intake, increased levels of high-density lipoprotein and its subfractions, and decreased risk of myocardial infarction. New Engl J Med 1993;329:1829—34.

[8] Corrao G, Rubbiati L, Bagnardi V, et al. Alcohol and coronary heart disease: a meta-analysis. Addiction 2000;95:1505—23.

[9] Koppes LL, Dekker JM, Hendriks HF, et al. Meta-analysis of the relationship between alcohol consumption and coronary heart disease and mortality in type 2 diabetic patients. Diabetologia 2006;49:648—52.

[10] Mukamal KJ, Chung H, Jenny NS, et al. Alcohol consumption and risk of coronary heart disease in older adults: the Cardiovascular Health Study. J Am Geriatr Soc 2006;54: 30—7.

[11] Stampfer MJ, Colditz GA, Willett WA, et al. A prospective study of moderate alcohol consumption and the risk of coronary disease and stroke in women. New Engl J Med 1998;319:267—73.

[12] Mazzaglia G, Britton AR, Altmann DR, et al. Exploring the relationship between alcohol consumption and non-fatal or fatal stroke: a systematic review. Addiction 2001;96: 1743—56.

[13] Reynolds K, Lewis LB, Nolen JDL, et al. Alcohol consumption and risk of stroke: a meta-analysis. JAMA 2003;289: 579—88.

[14] Sacco RL, Elkind ME, Boden-Albala B. The protective effect of moderate alcohol consumption on ischemic stroke. JAMA 1999;281:53—60.

[15] Elkind MS, Sciacca R, Boden-Albala B, et al. Moderate alcohol consumption reduces risk of ischemic stroke: the Northern Manhattan Study. Stroke 2006;37:13—9.

[16] Mukamal KJ, Chung H, Jenny NS, et al. Alcohol use and risk of ischemic stroke among older adults: the Cardiovascular Health Study. Stroke 2005;36:1830—4.

[17] Chen Z, Li Y, Zhao LC, et al. A study on the association between tea consumption and troke. Zhonghua Liu Xing Bing Xue Za Zhi 2004;25:666—70.

[18] Sesso HD, Gaziano JM, Buring JE, et al. Coffee and tea intake and the risk of myocardial infarction. Am J Epidemiol 1999;149:162—7.

[19] Vita JA. Polyphenols and cardiovascular disease: effects on endothelial and platelet function. Am J Clin Nutr 2005; 81(1 Suppl):292S—7S.

[20] Kuriyama S, Shimazu T, Ohmori K, et al. Green tea consumption and mortality due to cardiovascular disease, cancer, and all causes in Japan: the Ohsaki study. JAMA 2006;296:1255—65.

[21] Haheim LL, Holme I, Hjermann I, et al. Risk factors of stroke incidence and mortality: a 12-year follow-up of the Oslo Study. Stroke 1993;24:1484—9.

[22] Abbott RD, Rodriguez BL, Burchfiel CM, et al. Physical activity in older middle-aged men and reduced risk of stroke: the Honolulu Heart Program. Am J Epidemiol 1994;139:881—93.

[23] Kiely DK, Wolf PA, Cupples LA, et al. Physical activity and stroke risk: The Framingham Study. Am J Epidemiol 1994;140:608—20.

[24] Gillum RF, Mussolino ME, Ingram DD. Physical activity and stroke incidence in women and men: The NHANES I Epidemiologic Follow-up Study. Am J Epidemiol 1996;143: 860—9.

[25] Sacco RL, Gan R, Boden-Albala B, et al. Leisure-time physical activity and ischemic stroke risk: the Northern Manhattan Stroke Study. Stroke 1998;29:380—7.

[26] Fischer HG, Koenig W. Physical activity and coronary heart disease. Cardiologia 1998;43:1027—35.

[27] Calle EE, Thun MJ, Petrelli JM, et al. Body-mass index and mortality in a prospective cohort of US adults. New Engl J Med 1999;341:1097—105.

[28] Wilson PWF, D'Agostino RB, Sullivan L, et al. Overweight and obesity as determinants of cardiovascular risk, the Framingham experience. Arch Intern Med 2002;162: 1867—72.

[29] Wannamethee SG, Shaper AG, Walker M. Overweight and obesity and weight change in middle aged men: impact on cardiovascular disease and diabetes. J Epidemiol Community Health 2005;59:134—9.

[30] Tsai SP, Donnelly RP, Wendt JK. Obesity and mortality in a prospective study of a middle-aged industrial population. J Occup Environ Med 2006;48:22—7.

[31] Yang G, Xiang YB, Zheng W, et al. Body weight and weight change in relation to blood pressure in normotensive men. J Human Hypertens 2007;21:45—52.