Sleep Medicine 14 (2013) 206–210

Contents lists available at SciVerse ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Original Article

Association between sleep duration, weight gain, and obesity for long period

Masato Nagai *, Yasutake Tomata, Takashi Watanabe, Masako Kakizaki, Ichiro Tsuji

Division of Epidemiology, Department of Public Health and Forensic Medicine, Tohoku University Graduate School of Medicine, Sendai, Japan

ARTICLE INFO

Article history:
Received 29 May 2012
Received in revised form 13 September 2012
Accepted 14 September 2012
Available online 4 December 2012

Keywords:
Sleep duration
Weight gain
Obesity
Japanese
Long period

ABSTRACT

Background: Although previous studies showed the long-term effects of sleep duration on risk of weight gain, Western tends to gain weight irrespective of sleep duration over a long period. Conversely, it is showed that body mass index (BMI) decreases during a long period in Japanese and thus, the long-term effect of sleep duration on weight gain and obesity is still unclear in Asia.
Methods: We followed up 13,629 participants aged 40–79 years and prospectively collected data from 1995 to 2006. We divided the participants into five groups according to their self-reported sleep duration: ⩽5 h (short sleep), 6 h, 7 h (reference), 8 h, and ⩾9 h (long sleep). The main outcome was ⩾5 kg weight gain or BMI ⩾ 25 kg/m^2 (obesity). We used logistic regression analyses to derive odds ratios (ORs) and 95% confidence intervals (CIs), adjusted for several confounding factors.
Results: We observed no association between sleep duration and risk of ⩾5 kg weight gain and obesity. After stratification by BMI, long sleepers had a significantly increased risk of ⩾5 kg weight gain (OR: 1.36, 95%CI: 1.09–1.70) in obese participants.
Conclusions: Among community-dwelling Japanese, only obese long sleepers have a significantly increased long-term risk of ⩾5 kg weight gain.

1. Introduction

Obesity is increasing worldwide and its associated health problems are now widely recognized. According to the World Health Organization, Asia has a low prevalence of obesity in comparison to other areas of the world [1]. They recommended that obesity should be defined lower in Asia than Western [2]. In Japan, the prevalence of obesity is only 3%, and severe obesity almost never exists [3]. However, the percentage age of body fat is higher in Asian than in Western with the same body mass index (BMI) [4]. Meanwhile, according to the Organisation for Economic Co-operation and Development, Koreans have the shortest mean sleep duration in the world, followed by the Japanese [5].

The associations between sleep duration, weight gain, and/or obesity have been examined previously in seven meta-analyses and a systematic review [6–12]. However, almost all of the populations studied were Western, with longer mean sleep durations and higher prevalence of obesity than Asian populations. In Asia, three prospective studies, all from Japan, have examined the association between sleep duration and obesity [13–15]. However, the study participants were recruited from among people who were undergoing health checkups. The effect of sleep duration on weight gain and obesity has not yet been examined in an Asian population or recruited from the general community.

In addition, some previous studies have examined the long-term effects of sleep duration on weight gain and obesity from data accumulated over periods of at least 10 years [16,17]. Even though these studies showed that short sleepers were at risk of weight gain and obesity, Patel et al. showed that their study participants gained weight irrespective of sleep duration over a 16 year period [16]. In contrast, Matsushita et al. showed that mean BMI tended to decrease during a 10 year follow-up period in Japanese participants aged ⩾50 years [18]. Thus, the long-term association between sleep duration, weight gain, and obesity might differ between Western and Japanese populations, and the long-term effect of sleep duration on weight gain and obesity is still unclear in Asia.

In the present study, therefore, we examined the long-term association between sleep duration, weight gain, and obesity in Japanese subjects recruited from the general community based on data accumulated over 12 years.

2. Methods

2.1. Study cohort

Between October and December 1994, we distributed a self-administered questionnaire survey of various lifestyle habits to all National Health Insurance (NHI) beneficiaries aged 40–79 years

* Corresponding author. Address: Division of Epidemiology, Department of Public Health and Forensic Medicine, Tohoku University Graduate School of Medicine, 2-1 Seiryo-machi, Aoba-ku, Sendai, Miyagi 980-8575, Japan. Tel.: +81 22 717 8123; fax: +81 22 717 8125.
E-mail address: m-nagai@med.tohoku.ac.jp (M. Nagai).

1389-9457/$ - see front matter © 2012 Elsevier B.V. All rights reserved.
http://dx.doi.org/10.1016/j.sleep.2012.09.024

who lived in the catchment area of Ohsaki Public Health Center, Miyagi Prefecture, northeastern Japan [19]. Among 54,996 eligible individuals, 52,029 (95%) responded. We followed-up with the participants from January 1, 1995 through December 31, 2008 and recorded any mortality or migration by reviewing the NHI withdrawal history files. On December 1, 2006, we distributed another questionnaire to all community-dwelling individuals aged over 40 years in Ohsaki City [20]. Among the 77,325 eligible individuals, 40,027 individuals who participated in the survey in 1994, were included without death or lost to follow-up until December 1, 2006. By combining the data for these two surveys, we were able to use the questionnaire responses for 16,982 participants (response rate 42.4%).

For the present analysis, we excluded 3116 participants who did not provide information about body weight and height, and 237 participants who did not provide information about sleep duration. As a result, a final total of 13,629 participants were included.

The study protocol was approved by the Ethics Committee of Tohoku University School of Medicine. We considered the return of self-administered questionnaires signed by the participants to imply their consent to participate in the study.

2.2. Sleep duration

The self-administered questionnaire included question on sleep duration. Sleep duration was assessed through each participant's response to the question, "How many hours on average do you sleep per day ?" The participants entered the mean integer number representing the hours of sleep taken per day during the previous year. We divided the participants into five groups according to their sleep duration: ⩽5 h (short sleep), 6 h, 7 h (reference), 8 h, and ⩾9 h (long sleep).

2.3. Outcome measures

The main outcome measure was ⩾5 kg weight gain calculated as weight (kg) in the NHI Cohort Study minus self-reported weight (kg) recorded in the Ohsaki Cohort 2006 Study. We also assessed BMI ⩾ 25.0 kg/m^2 (obesity) calculated as weight (kg) divided by the square of height (m^2). These self-reported heights, weights, and BMI in the questionnaire were considered to be sufficiently valid [21]. The Pearson's correlation coefficient (r) between the self-reported values and measured values were $r = 0.96$ ($p < 0.01$) for weight, $r = 0.93$ ($p < 0.01$) for height, and $r = 0.88$ ($p < 0.01$) for BMI.

2.4. Statistical analysis

We used logistic regression analyses to derive odds ratios (ORs) and 95% confidence intervals (CIs) for ⩾5 kg weight gain and obesity according to each sleep duration category and to adjust for potentially confounding factors, using the SAS version 9.2 statistical software package. The 7 h sleep duration category was selected as the reference. All p values were two-tailed and differences at $p < 0.05$ were accepted as statistically significant.

We considered the following variables to be potential confounding factors: sex (men or women), age (continuous), BMI (<18.5, 18.5–24.9, 25.0–29.9, or ⩾30.0 kg/m^2), education (junior high school or less, high school, or college/university or higher), smoking status (never smoker, past smoker, current smoker consuming 1–19 cigarettes per day, or current smoker consuming at least 20 cigarettes per day), alcohol drinking (never drinker, past drinker, or current drinker), time spent walking per day (less than 1 h, or 1 h or longer), sports and physical exercise time per week (less than 1 h, 1–2 h, 3–4 h, or 5 h or longer), job status (employed, or no occupation or housewife), marital status (married or unmarried), menopausal status (premenopausal or postmenopausal), coffee consumption (never or occasionally, 1–2 cups/day, 3–4 cups/day, ⩾5 cups/day), and self-rated health (good or not good). We further adjusted for energy consumption (kcal/day) in the multivariate models 2.

In addition, we repeated analyses after excluding participants who had functional limitation, poor self-rated health, or history of disease. Physical function status was assessed using the 6 item measure of the Medical Outcomes Study (MOS) Short-form General Health Survey [22]. Participants were excluded if they stated on the MOS questionnaire that they were unable to perform moderate or vigorous activities ($n = 3576$), walk one block ($n = 25$), or perform self-care activities such as eating, dressing, bathing, or using the toilet ($n = 2$). We excluded participants who reported severe bodily pain ($n = 246$), poor self-rated health ($n = 896$), or history of cancer ($n = 160$), myocardial infarction ($n = 100$), or stroke ($n = 45$). The remaining 8579 participants were apparently healthy and their sleep duration was not affected these physical conditions.

3. Results

3.1. Baseline characteristics in terms of sleep duration categories

Table 1 shows the baseline characteristics of the study participants according to the categories of sleep duration.

The mean age was highest in the long sleep category. The proportions of women decreased linearly as the sleep duration category increased. Even though the mean weight was the lowest in the short sleep category, the mean BMI showed no significant difference. The mean weight change increased linearly as the sleep duration category increased. The proportion of participants who were employed was lowest in the short sleep category, whereas the proportions of participants who were current drinkers and consumed ⩾3 cups/day of coffee were lowest in the long sleep category. The proportions of participants who walked ⩾1 h/day and did ⩾3 h/week sports and physical exercise were highest in the long sleep category. Mean energy consumption was lowest in the short sleep category.

3.2. Weight gain and obesity by sleep duration category

Table 2 shows the numbers of participants who had ⩾5 kg weight gain and obesity and the ORs of ⩾5 kg weight gain and obesity with 95% CIs according to the sleep duration categories.

We observed no association between sleep duration, ⩾5 kg weight gain, and obesity. The multivariate OR1 for ⩾5 kg weight gain was 0.93 (95%CIs; 0.73–1.19) in short sleeper and 1.05 (0.91–1.20) in long sleepers (p for trend = 0.3087). Similarly, the multivariate OR1 for obesity was 1.08 (0.77–1.52) in short sleeper and 1.06 (0.86–1.30) in long sleeper (p for trend = 0.3712). After further adjustments of multivariate ORs1 for energy consumption, the multivariate ORs2 showed associations similar to those for ORs1 (⩾5 kg weight gain; p for trend = 0.3097, obesity; p for trend = 0.3655). In addition, after stratification by BMI, the present study also demonstrated a null association between sleep duration and ⩾5 kg weight gain in normal weight participants (p for trend = 0.6236). However, among obese participants, long sleepers had a significantly increased risk of ⩾5 kg weight gain (OR: 1.36, 95%CI: 1.09–1.70).

Table 3 shows the numbers of healthy participants who had ⩾5 kg weight and obesity and ORs with 95% CIs according to the sleep duration categories. We also observed no association between sleep duration, ⩾5 kg weight gain, and obesity in participants who had no physical limitations or history of disease. The multivariate OR1 for ⩾5 kg weight gain was 0.88 (0.61–1.26) in

Table 1
Baseline characteristics by sleep duration in 13,629 participants aged 40–79 years.

	Sleep duration (h/day)					p Value[b]
	⩽5	6	7	8	⩾9	
No. of subjects	453	1831	4291	5265	1789	
Mean age (years) (SD[a])	57.5 (9.7)	56.3 (9.6)	56.2 (9.3)	58.4 (9.1)	61.9 (8.5)	<.0001
Women (%)	70.0	66.3	58.4	51.2	45.6	<.0001
Mean weight (kg) (SD)	56.9 (10.1)	57.6 (9.2)	58.7 (9.3)	58.8 (9.4)	58.5 (10.0)	<.0001
Mean BMI (kg/m^2) (SD)	23.6 (3.3)	23.6 (3.0)	23.7 (3.0)	23.7 (3.0)	23.7 (3.6)	NS
Mean weight change (kg) (SD)	0.7 (5.7)	0.8 (5.0)	1.0 (5.4)	1.1 (5.8)	1.8 (7.4)	<.0001
Education (%)						
Junior high school or less	48.9	42.8	46.2	56.7	69.7	<.0001
High school	40.7	43.9	42.4	35.7	25.9	
College/university or higher	10.5	13.3	11.4	7.6	4.4	
Job status (%)						
Employed	54.8	58.8	65.6	63.4	58.3	<.0001
No occupation or housewife	45.2	41.2	34.4	36.6	41.7	
Smoking status (%)						
Never smoker	63.7	62.9	60.6	53.1	49.0	<.0001
Past smoker	12.3	12.2	12.6	15.1	18.1	
Current smoker <20	10.8	11.4	9.3	12.8	15.3	
Current smoker ⩾20	13.1	13.5	17.5	19.0	17.7	
Alcohol drinking (%)						
Never drinker	48.7	48.4	48.6	50.6	54.1	<.0001
Past drinker	6.3	5.7	4.8	6.7	6.8	
Current drinker	45.0	45.9	46.7	42.8	39.2	
Time spent walking (%)						
⩾1 h/day	42.5	45.2	45.9	47.4	50.2	0.0054
<1 h/day	57.5	54.8	54.1	52.6	49.8	
Sports and physical exercise (%)						
⩾5 h/week	8.2	5.3	5.8	6.9	8.5	0.0083
3–4 h/week	6.1	6.5	6.2	6.2	7.0	
1–2 h/week	13.9	16.2	16.2	16.8	15.0	
<1 h/week	71.8	72.0	71.8	70.1	69.5	
Marital status (%)						
Married	77.1	83.4	86.7	86.6	84.2	<.0001
Unmarried	22.9	16.6	13.3	13.4	15.8	
Menopause status (%)						
Premenopausal	25.6	30.0	28.0	18.0	8.6	<.0001
Postmenopausal	74.4	70.0	72.0	82.0	91.3	
Coffee consumption (%)						
⩾5 cups/day	6.8	5.2	3.2	3.1	1.7	<.0001
3–4 cups/day	7.8	9.5	9.5	7.6	5.4	
1–2 cups/day	27.8	34.6	36.8	34.1	29.1	
<1 cup/day	57.6	50.7	50.6	55.3	63.8	
Self-rated health (%)						
Good	56.7	69.0	71.6	71.4	67.9	<.0001
Not good	43.3	31.0	28.5	28.6	32.1	
Mean energy consumption/day (kcal) (SD)	1405.2 (528.9)	1488.7 (550.8)	1554.3 (565.4)	1595.8 (598.4)	1572.4 (600.3)	<.0001

[a] BMI, body mass index; SD, standard deviation; NS, not significant.
[b] p Values were calculated by chi-squared test (categorical variables), or ANOVA (continuous variables).

short sleepers and 1.06 (0.88–1.27) in long sleepers (p for trend = 0.5539). Similarly, the multivariate OR1 for obesity was 0.96 (0.59–1.57) in short sleepers and 1.06 (0.82–1.37) in long sleepers (p for trend = 0.3267). After stratification by BMI, only obese long sleepers also had a significantly increased risk of ⩾5 kg weight gain (OR: 1.41, 95%CI: 1.04–1.92).

4. Discussion

The present results indicate that the association between sleep duration, ⩾5 kg weight gain and obesity showed no significant association over a long period in Japanese subjects recruited from the community. After stratification by BMI, obese long sleepers showed a significantly increased risk of ⩾5 kg weight gain (OR: 1.36, 95%CI: 1.09–1.70).

The mean age in the long sleep category was oldest and the proportion of women in the short sleep category was highest in the sleep duration categories, but the association between sleep duration, ⩾5 kg weight gain, and obesity also showed no significant associations after stratification by the age categories (<65 years or ⩾65 years) and sexes (data not shown).

There is a possibility that the long sleep category would have included participants who were bedridden due to physical limitation. Short sleep is associated with poor self-rated health [23]. Therefore, we conducted additional analysis after excluding participants who had functional limitation, poor self-rated health, or history of disease in order to eliminate any bias due to these effects. However, only obese long sleepers also showed a significantly increased risk of weight gain (OR: 1.41, 95%CI: 1.04–1.92).

These results were different from those of previous studies conducted in Japan [13–15]. However, the participants of those studies had been recruited from among individuals undergoing health checkups. Also, the previous studies had examined the short-term effect of sleep duration on obesity, whereas, the present study examined the long-term effect. On the other hand, the present results were also different from those of previous studies that had examined the long-term effects of sleep duration on weight gain

Table 2
The association between sleep duration, ⩾5 kg weight gain, and obesity.

	Sleep duration					
	⩽5	6	7	8	⩾9	p for trend[a]
⩾5 kg weight gain						
Total number	453	1,831	4,291	5,265	1,789	
Case	95	355	876	1113	468	
Crude	1.03(0.82–1.31)	0.94(0.82–1.08)	1.00 (reference)	1.05(0.95–1.15)	1.38(1.21–1.57)	<.0001
Age-sex adjusted ORs	0.98(0.77–1.25)	0.93(0.81–1.07)	1.00 (reference)	0.94(0.85–1.04)	1.07(0.93–1.22)	0.2032
Multivariate ORs1[b]	0.93(0.73–1.19)	0.95(0.82–1.09)	1.00 (reference)	0.94(0.84–1.04)	1.05(0.91–1.20)	0.3087
Multivariate ORs2[c]	0.93(0.73–1.20)	0.95(0.82–1.09)	1.00 (reference)	0.94(0.84–1.04)	1.05(0.91–1.20)	0.3093
Stratified analyses (BMI)						
<25 kg/m^2	0.99(0.72–1.35)	0.94(0.78–1.12)	1.00 (reference)	0.93(0.81–1.06)	0.91(0.76–1.08)	0.6236
⩾25 kg/m^2	0.86(0.58–1.29)	0.96(0.76–1.23)	1.00 (reference)	0.95(0.80–1.13)	1.36(1.09–1.70)	0.0145
Obesity[d]						
Total number	311	1,329	3,038	3,724	1,256	
Case	44	177	413	539	162	
Crude	1.05(0.75–1.47)	0.98(0.81–1.18)	1.00 (reference)	1.08(0.94–1.24)	0.94(0.78–1.14)	0.9454
Age-sex adjusted ORs	1.07(0.76–1.49)	0.98(0.81–1.18)	1.00 (reference)	1.12(0.98–1.29)	1.05(0.86–1.29)	0.3125
Multivariate ORs1	1.08(0.77–1.52)	0.99(0.82–1.20)	1.00 (reference)	1.12(0.97–1.29)	1.06(0.86–1.30)	0.3712
Multivariate ORs2	1.08(0.77–1.51)	0.99(0.81–1.19)	1.00 (reference)	1.12(0.97–1.29)	1.06(0.86–1.29)	0.3655

[a] p for trend values were calculated by sleep duration as a continuous variable.
[b] Multivariate ORs1 was adjusted for sex (men or women); age (continuous); body mass index (<18.5, 18.5–24.9, 25.0–29.9, or ⩾30.0 kg/m^2); education (junior high school or less, high school, or college/university or higher); smoking status (never smoker, past smoker, current smoker consuming 1–19 cigarettes per day, or current smoker consuming at least 20 cigarettes per day); alcohol drinking (never drinker, past drinker, or current drinker); time spent walking/day (less than 1 h, or 1 h or longer); job status (employed, or no occupation or housewife); marital status (married or unmarried); menopause (premenopausal or postmenopausal); coffee (never or occasionally, 1–2 cups/day, 3–4 cups/day, ⩾5 cups/day); self-rated health (good or not good).
[c] Multivariate ORs2 was further adjusted multivariate ORs1 for energy consumption/day (tertile category).
[d] Analyses of obesity excluded the 3971 participants who was obesity from the 13,629 participants.

Table 3
The association between sleep duration, 5 kg weight gain, and obesity in healthy participants aged 40–79 years.

	Sleep duration					
	⩽5	6	7	8	⩾9	p for trend[a]
5 kg weight gain						
Total number	228	1185	2830	3305	1031	
Case	41	222	522	652	251	
Multivariate ORs1[b]	0.88(0.61–1.26)	1.01(0.85–1.21)	1.00 (reference)	0.95(0.83–1.09)	1.06(0.88–1.27)	0.5539
Stratified analyses (BMI)						
<25 kg/m^2	1.00(0.64–1.56)	1.07(0.86–1.33)	1.00 (reference)	0.90(0.76–1.07)	0.91(0.73–1.15)	0.2253
⩾25 kg/m^2	0.71(0.38–1.33)	0.92(0.68–1.26)	1.00 (reference)	1.06(0.85–1.32)	1.41(1.04–1.92)	0.0073
Obesity[c]						
Total number	160	852	2,041	2362	747	
Case	20	123	282	341	101	
Multivariate ORs1	0.96(0.59–1.57)	1.09(0.87–1.38)	1.00 (reference)	1.08(0.90–1.28)	1.06(0.82–1.37)	0.3267

[a] p for trend values were calculated by sleep duration as a continuous variable.
[b] Multivariate ORs1 was adjusted for sex (men or women); age (continuous); body mass index (<18.5, 18.5–24.9, 25.0–29.9, or ⩾30.0 kg/m^2); education (junior high school or less, high school, or college/university or higher); smoking status (never smoker, past smoker, current smoker consuming 1–19 cigarettes per day, or current smoker consuming at least 20 cigarettes per day); alcohol drinking (never drinker, past drinker, or current drinker); time spent walking/day (less than 1 h, or 1 h or longer); sports and physical exercise time/week (less than 1 h, 1–2 h, 3–4 h, or 5 h or longer); job status (employed, or no occupation or housewife); marital status (married or unmarried); menopause (premenopausal or postmenopausal); coffee consumption (never or occasionally, 1–2 cups/day, 3–4 cups/day, ⩾5 cups/day).
[c] Analyses of obesity excluded the 2417 participants who was obesity from the 8579 participants.

and obesity [16,17]. This may have been due to differences in long-term BMI or weight change trends between Japanese and Americans; the former show a decreasing long-term trend for BMI [18], and the latter an increasing long-term trend for weight increases [16]. Over short periods, short sleep is also a risk factor for obesity in Japanese [13–15]. Thus, after short sleepers have increased weight, they might show a weight reduction in the long term. Therefore, the present study found no association between short sleep and the risk of weight gain and obesity. However, we were unable to demonstrate whether our study participants exhibited such weight changes during the observation period because we only had data for their weight at the baseline and the end of follow up.

The biological mechanism responsible for the association between short sleep, weight gain, and obesity has mainly been considered attributable to a decreased leptin level and an increased ghrelin level with shorter sleep duration [24–26]. Appetite increases as the level of the satiety-promoting hormone falls and that of the appetite-promoting hormone increases. Thus, short sleep induces an increase of daily energy consumption and, thereby, weight gain. However, although we did not obtain data on leptin and ghrelin levels, daily energy consumption was lowest in short sleepers. The proportion of women in the short sleep category was higher than that in the long sleep category, but the above trend was also observed after separation of the sexes (data not shown). Manini et al. and St-Onge et al. demonstrated no association between sleep duration and energy expenditure as measured using doubly labeled water [27,28]. Therefore, the lack of association between short sleep, weight gain, and obesity in the present study might have been attributable to the lack of a relationship between short sleep duration and increased energy consumption. However, although the Nurses' Health Study also showed that daily energy consumption was lowest in short sleepers [16], lack of sleep duration was associated with a risk of ⩾15 kg weight gain. The association between changes in hormone levels, increasing daily energy consumption, weight gain, and obesity due to short sleep duration might be related to not only the above mechanism, but also other unknown mechanisms.

A major strength of the present study was that it was the first to have investigated the long-term association between sleep duration, weight gain, and obesity in an Asian general population showing different long-term trends of weight change from Western populations [16,18]. Asians have a lower prevalence of obesity and a shorter mean sleep duration than Westerners [1,5]. To exclude the effects of physical condition on sleep duration, weight gain, and obesity, we repeated our analyses after excluding participants who had functional limitation or history of disease.

On the other hand, several limitations should also be considered. First, we had no information about sleep quality, the timing of sleep, the use of sleep medication, the presence of sleeping disorders, rotating shift work, or night work that can influence sleep duration and thereby affect the risk of weight gain and obesity. Although we had no information about rotating shift work and night work, since 18.2% of our participants were housewives, 32.6% farmers, and 14.8% retired, such details would have been unlikely to have changed the results. Second, the assessment of weight was done only at the baseline and at the end of follow-up. We had no information about the trends of weight change during the follow-up period. Third, we used self-reported weight, height, and BMI. There is a systematic bias in self-reported weight and height. Because of this bias, there is possibility that we observed no association between short sleep duration, weight gain, and obesity. However, we demonstrated a high correlation and appropriate agreement between self-reported values and measured values (weight: $r = 0.96$, height: $r = 0.93$, BMI: $r = 0.88$).

In conclusion, the present study showed that only obese long sleepers have a significantly increased risk of ⩾5 kg weight gain in the long term among Japanese recruited from the community. Short sleep did not carry a risk of weight gain and obesity. Further research is needed to clarify the long-term association between sleep duration, weight gain, and obesity in Asian populations.

Conflict of Interest

This was not an industry supported study. The authors declare that they have no conflicts of interest.

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2012.09.024.

Acknowledgments

This study was supported by a Health Sciences Research Grant for Health Services (H22-Junkankitou (Seisyu)-Ippan-012, H23-Junkankitou (Seisyu)-Ippan-005, H23-Junkankitou (Seisyu)-Wakate-015), Ministry of Health, Labour and Welfare, Japan. Masato Nagai is a recipient of a Research Fellowships of the Japan Society for the Promotion of Science for Young Scientists.

References

[1] World Health Organization. Global Database on Body Mass Index. <http://apps.who.int/bmi/index.jsp>.
[2] Consultation WE. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. Lancet 2004;363:157–63.
[3] Yoshiike N, Matsumura Y, Zaman MM, Yamaguchi M. Descriptive epidemiology of body mass index in Japanese adults in a representative sample from the National Nutrition Survey 1990–1994. Int J Obes Relat Metab Disord 1998; 22:684–7.
[4] Deurenberg P, Deurenberg-Yap M, Guricci S. Asians are different from Caucasians and from each other in their body mass index/body fat per cent relationship. Obes Rev 2002;3:141–6.
[5] Organisation for Economic Co-operation and Development. Society at a glance 2009: OECD social indicators: Organization for Economic; 2009.
[6] Beccuti G, Pannain S. Sleep and obesity. Curr Opin Clin Nutr Metab Care 2011; 14:402–12.
[7] Grandner MA, Patel NP, Gehrman PR, Perlis ML, Pack AI. Problems associated with short sleep: bridging the gap between laboratory and epidemiological studies. Sleep Med Rev 2010;14:239–47.
[8] Horne J. Obesity and short sleep: unlikely bedfellows? Obes Rev 2011; 12:e84–94.
[9] Nielsen LS, Danielsen KV, Sorensen TI. Short sleep duration as a possible cause of obesity: critical analysis of the epidemiological evidence. Obes Rev 2011; 12:78–92.
[10] Patel SR. Reduced sleep as an obesity risk factor. Obes Rev 2009;10(Suppl. 2):61–8.
[11] Patel SR, Hu FB. Short sleep duration and weight gain: a systematic review. Obesity (Silver Spring) 2008;16:643–53.
[12] Magee L, Hale L. Longitudinal associations between sleep duration and subsequent weight gain: a systematic review. Sleep Med Rev 2012;16:231–41.
[13] Itani O, Kaneita Y, Murata A, Yokoyama E, Ohida T. Association of onset of obesity with sleep duration and shift work among Japanese adults. Sleep Med 2011;12:341–5.
[14] Kobayashi D, Takahashi O, Deshpande GA, Shimbo T, Fukui T. Association between weight gain, obesity, and sleep duration: a large-scale 3-year cohort study. Sleep Breath 2011.
[15] Nishiura C, Noguchi J, Hashimoto H. Dietary patterns only partially explain the effect of short sleep duration on the incidence of obesity. Sleep 2010;33:753–7.
[16] Patel SR, Malhotra A, White DP, Gottlieb DJ, Hu FB. Association between reduced sleep and weight gain in women. Am J Epidemiol 2006;164:947–54.
[17] Hasler G, Buysse DJ, Klaghofer R, Gamma A, Ajdacic V, Eich D, et al. The association between short sleep duration and obesity in young adults: a 13-year prospective study. Sleep 2004;27:661–6.
[18] Matsushita Y, Takahashi Y, Mizoue T, Inoue M, Noda M, Tsugane S. Overweight and obesity trends among Japanese adults: a 10-year follow-up of the JPHC Study. Int J Obes (London) 2008;32:1861–7.
[19] Tsuji I, Nishino Y, Ohkubo T, Kuwahara A, Ogawa K, Watanabe Y, et al. A prospective cohort study on National Health Insurance beneficiaries in Ohsaki, Miyagi Prefecture, Japan: study design, profiles of the subjects and medical cost during the first year. J Epidemiol 1998;8:258–63.
[20] Kuriyama S, Nakaya N, Ohmori-Matsuda K, Shimazu T, Kikuchi N, Kakizaki M, et al. The Ohsaki Cohort 2006 Study: design of study and profile of participants at baseline. J Epidemiol 2010;20:253–8.
[21] Kuriyama S, Tsuji I, Ohkubo T, Anzai Y, Takahashi K, Watanabe Y, et al. Medical care expenditure associated with body mass index in Japan: the Ohsaki Study. Int J Obes Relat Metab Disord 2002;26:1069–74.
[22] Stewart AL, Hays RD, Ware Jr JE. Short-form general health survey. Reliability and validity in a patient population. Med Care 1988;26:724–35.
[23] Steptoe A, Peacey V, Wardle J. Sleep duration and health in young adults. Arch Intern Med 2006;166:1689–92.
[24] Chaput JP, Despres JP, Bouchard C, Tremblay A. Short sleep duration is associated with reduced leptin levels and increased adiposity: results from the Quebec family study. Obesity (Silver Spring) 2007;15:253–61.
[25] Spiegel K, Tasali E, Penev P, Van Cauter E. Brief communication: sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. Ann Intern Med 2004;141:846–50.
[26] Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. PLoS Med 2004;1:e62.
[27] Manini TM, Everhart JE, Patel KV, Schoeller DA, Colbert LH, Visser M, et al. Daily activity energy expenditure and mortality among older adults. JAMA 2006;296:171–9.
[28] St-Onge MP, Roberts AL, Chen J, Kelleman M, O'Keeffe M, RoyChoudhury A, et al. Short sleep duration increases energy intakes but does not change energy expenditure in normal-weight individuals. Am J Clin Nutr 2011;94:410–6.

J Epidemiol 2013;23(1):55-62
doi:10.2188/jea.JE20120072

Original Article

Burden of Household Environmental Tobacco Smoke on Medical Expenditure for Japanese Women: A Population-Based Cohort Study

Toshitaka Morishima[1], Yuichi Imanaka[1], Tetsuya Otsubo[1], Kenshi Hayashida[2], Takashi Watanabe[3], and Ichiro Tsuji[3]

[1]Department of Healthcare Economics and Quality Management, Kyoto University Graduate School of Medicine, Kyoto, Japan
[2]Department of Medical Informatics and Management, University Hospital of Occupational and Environmental Health, Kitakyushu, Fukuoka, Japan
[3]Division of Epidemiology, Department of Public Health and Forensic Medicine, Tohoku University School of Medicine, Sendai, Japan

Received April 10, 2012; accepted September 18, 2012; released online November 24, 2012

ABSTRACT

Background: The economic consequences of environmental tobacco smoke (ETS) have been simulated using models. We examined the individual-level association between ETS exposure and medical costs among Japanese nonsmoking women.
Methods: This population-based cohort study enrolled women aged 40 to 79 years living in a rural community. ETS exposure in homes at baseline was assessed with a self-administered questionnaire. We then collected health insurance claims data on direct medical expenditures from 1995 through 2007. Using generalized linear models with interaction between ETS exposure level and age stratum, average total monthly expenditure (inpatient plus outpatient care) per capita for nonsmoking women highly exposed and moderately exposed to ETS were compared with expenditures for unexposed women. We performed separate analyses for survivors and nonsurvivors.
Results: We analyzed data from 4870 women. After adjustment for potential confounding factors, survivors aged 70 to 79 who were highly exposed to ETS incurred higher expenditures than those who were not exposed. We found no significant difference in expenditures between moderately exposed and unexposed women. Total expenditures were not significantly associated with ETS exposure among survivors aged 40 to 69 or nonsurvivors of any age stratum.
Conclusions: We calculated individual-level excess medical expenditures attributable to household exposure to ETS among surviving older women. The findings provide direct evidence of the economic burden of ETS, which is helpful for policymakers who seek to achieve the economically attractive goal of eliminating ETS.

Key words: secondhand smoke; tobacco smoke pollution; longitudinal study; environment and public health; health care costs

INTRODUCTION

Exposure to environmental tobacco smoke (ETS), also known as secondhand smoke, passive smoking, and involuntary smoking, is a risk factor for mortality[1,2] and morbidity from many diseases, including lung cancer[3,4] and coronary heart disease.[5,6] ETS exposure accounts for 11% of all tobacco-related deaths.[7] Öberg et al[8] reported that 1.0% of worldwide mortality is attributable to ETS. Public health concern is therefore elevated because diseases attributable to ETS are occurring among nonsmokers, and the resulting increase in mortality and morbidity is involuntary.

Medical expenditures are higher for active smokers than for nonsmokers,[9–15] and ETS is a risk factor for many of the same diseases caused by active smoking.[3–6] These facts naturally lead us to question whether ETS increases medical expenditures. Little is known, however, of the relationship between ETS exposure and medical expenditures among adults. Previous studies estimated the economic burden attributable to ETS by combining ETS-attributable incidence of diseases and medical costs associated with those diseases.[16–21] The results were obtained from simulations using economic models and did not necessarily reflect the real world.

Address for correspondence. Yuichi Imanaka, Department of Healthcare Economics and Quality Management, Kyoto University Graduate School of Medicine, Yoshida Konoe-cho, Sakyo-ku, Kyoto 606-8501, Japan (e-mail: imanaka-y@umin.net).

Japanese women have the highest ETS exposure among the 7 major industrialized nations.[22] The World Health Organization (WHO) has calculated that 49% to 62% of Japanese women are exposed to ETS.[22] WHO also reported that 42% of Japanese men smoked in 2006, which was the highest rate among the 7 nations. However, the prevalence of smoking among Japanese women was 13% in 2006—the lowest among the 7 nations.

We used individual-level observations of a single cohort of Japanese women to examine differences in direct medical expenditure among nonsmokers and those highly exposed, moderately exposed, and not exposed to ETS.

METHODS

Study design and setting

We used a prospective cohort design. The data were derived from the Ohsaki Cohort Study, the details of which have been reported elsewhere.[23] In brief, this study started in 1994. We conducted a questionnaire survey of National Health Insurance (NHI) beneficiaries aged 40 to 79 years who lived in the catchment area of Ohsaki Public Health Center (Miyagi Prefecture) between October and December 1994. Japan has a universal healthcare system, and the NHI system is operated by local governments, enrolls individuals (eg, farmers, the self-employed, the retired, part-time workers, and their families) not covered by Employees' Health Insurance, and pays for almost all medical services and medications.

This survey used a self-administered questionnaire on various health-related lifestyle factors. Among 54 996 eligible beneficiaries, 52 029 (95%) responded. We prospectively collected claims data (directly from the Miyagi NHI Organizations) on medical expenditures (inpatient and outpatient), insurance status, and survival status for all participants in the cohort during the study period of January 1995 through December 2007. This study was approved by the Ethics Committee of Kyoto University Graduate School of Medicine (Number E1059).

Study population

Among the 52 029 participants in the baseline survey in 1994, we excluded 774 who died or withdrew from NHI before January 1, 1995. Of the 51 255 remaining participants, we selected all female lifelong nonsmokers ($n = 17\,803$), who were identified by the question, "Are you a current smoker, a former smoker, or have never smoked?" All participants who selected the third option were classified as lifelong nonsmokers. We excluded women who provided incomplete information on ETS exposure at home ($n = 634$); those who made a proxy complete the baseline questionnaire (to avoid incorrect assessments made by a proxy; $n = 2081$); those exposed to ETS at work 3 or more days per week ($n = 4538$); those who were divorced or widowed (to avoid underestimating past ETS exposure at home; $n = 1926$); those who had left their job (to avoid underestimating past ETS exposure at work; $n = 3284$); those who died or withdrew in 1995, ie, the first follow-up year (to avoid extremely high or low monthly expenditures; $n = 215$); and those with a history of cancer, myocardial infarction, or stroke ($n = 255$). A total of 4870 women remained for analysis.

Assessment of ETS exposure

We conducted a baseline questionnaire survey of all participants. The item on ETS exposure at home was worded, "How often are you exposed to environmental tobacco smoke at home?" Participants were asked to choose 1 of 5 options: almost every day, 3 to 4 days per week, 1 to 2 days per week, less than once per week, and rarely. For our analyses, we defined household high-level exposure to ETS as the first and second options, moderate-level exposure as the third and fourth options, and no exposure as the fifth option.

Data analysis

With regard to baseline characteristics, differences between the 3 groups were examined using 1-way analysis of variance for duration of follow-up and the chi-square test for the other variables. The Fisher exact test was used when expected cell counts were less than 5.

The primary outcome was total (ie, inpatient plus outpatient care) average monthly medical expenditure per capita, because total expenditures attributable to ETS exposure can be considered to impose an economic burden on society. Total average monthly expenditure for each woman was calculated by dividing the accumulated expenditures through follow-up by number of months observed. A similar approach has been used in the econometric literature to assess medical expenditures related to smoking.[9] In addition to total expenditure, we calculated and compared medical expenditures for inpatient and outpatient care. Outpatient expenditure included costs for drugs dispensed at pharmacies.

ETS exposure can cause fatal diseases, which incur considerable medical expenditures in the terminal phase.[24] Calculating average monthly expenditure incurred by a mixed population of survivors and nonsurvivors creates a bias against the group with the higher mortality risk. Hence, we performed separate analyses for survivors and nonsurvivors.

Cost data are typically skewed to the right. Therefore, generalized linear models with a gamma distribution and log-link function were used to examine the relationship between medical expenditures and ETS exposure, after controlling for other variables.[25] Gamma regression models have been shown to be multiplicative. Exponentiated coefficients were interpreted as cost ratio relative to the referent group. Gamma regression models removed zero-valued outcomes in the statistical software. Because the proportion of women with zero data for total and outpatient expenditures was small (6, 3,

Table 1. Characteristics of female nonsmokers

	Survivors				Nonsurvivors			
	Unexposed	Moderately exposed	Highly exposed	*P*-value[a]	Unexposed	Moderately exposed	Highly exposed	*P*-value[a]
No. of women	1687	1371	1494		149	77	92	
Age[b] (years)								
40–49	369 (22)	361 (26)	506 (34)	<0.001	5 (3)	9 (12)	8 (9)	0.001
50–59	456 (27)	490 (36)	442 (30)		15 (10)	19 (25)	19 (21)	
60–69	641 (38)	426 (31)	454 (30)		72 (48)	25 (32)	46 (50)	
70–79	221 (13)	94 (7)	92 (6)		57 (38)	24 (31)	19 (21)	
Mean follow-up, months (SD)	138 (40)	136 (41)	132 (45)	0.001	92 (45)	99 (37)	90 (42)	0.33
Body mass index								
<18.5 kg/m^2	63 (4)	39 (3)	43 (3)	0.14	6 (4)	4 (5)	5 (5)	0.29
18.5–24.9 kg/m^2	1148 (68)	931 (68)	977 (65)		102 (68)	52 (68)	51 (55)	
≥25.0 kg/m^2	476 (28)	401 (29)	474 (32)		41 (28)	21 (27)	36 (39)	
Alcohol drinking status								
Never	1355 (80)	1039 (76)	1151 (77)	0.06	127 (85)	61 (79)	79 (86)	0.09
Former	39 (2)	31 (2)	32 (2)		8 (5)	4 (5)	0 (0)	
Current, <3 *go* per week[c]	249 (15)	263 (19)	272 (18)		13 (9)	11 (14)	10 (11)	
Current, ≥3 *go* per week[c]	44 (3)	38 (3)	39 (3)		1 (1)	1 (1)	3 (3)	
Tertiles of dietary energy intake per day								
≤1163 kcal	592 (35)	443 (32)	452 (30)	0.07	73 (49)	28 (36)	34 (37)	0.19
1163–1440 kcal	551 (33)	461 (34)	521 (35)		40 (27)	23 (30)	25 (27)	
>1440 kcal	544 (32)	467 (34)	521 (35)		36 (24)	26 (34)	33 (36)	
Time spent walking per day								
<30 minutes	437 (26)	380 (28)	343 (23)	0.001	46 (31)	32 (42)	31 (34)	0.51
30–60 minutes	576 (34)	440 (32)	459 (31)		53 (36)	24 (31)	28 (30)	
≥60 minutes	674 (40)	551 (40)	692 (46)		50 (34)	21 (27)	33 (36)	
Self-rated health								
Good	1129 (67)	937 (68)	1019 (68)	0.60	71 (48)	36 (47)	47 (51)	0.91
Mediocre	265 (16)	210 (15)	245 (16)		25 (17)	16 (21)	16 (17)	
Poor	293 (17)	224 (16)	230 (15)		53 (36)	25 (32)	29 (32)	
Marital status								
Married	1614 (96)	1328 (97)	1472 (99)	<0.001	143 (96)	76 (99)	89 (97)	0.64
Unmarried	73 (4)	43 (3)	22 (1)		6 (4)	1 (1)	3 (3)	
Current job status								
Employed	916 (54)	784 (57)	788 (53)	0.05	57 (38)	29 (38)	40 (43)	0.67
Unemployed	771 (46)	587 (43)	706 (47)		92 (62)	48 (62)	52 (57)	
Education								
≤9 years	819 (49)	658 (48)	746 (50)	0.56	91 (61)	41 (53)	58 (63)	0.39
>9 years	868 (51)	713 (52)	748 (50)		58 (39)	36 (47)	34 (37)	

Exposure indicates exposure to environmental tobacco smoke in homes.
Values are expressed as number (column percentage), unless otherwise indicated.
Because of rounding, percentages may not add up to 100%.
[a]Differences among the 3 groups (analysis of variance for mean follow-up months and chi-square test for categorical variables).
[b]Age at baseline survey in 1994.
[c]1 *go* is equal to 180 mL of Japanese sake and contains 22.8 g of ethanol.

and 2 unexposed, moderately exposed, and highly exposed women, respectively), we added ¥1 to those who had zero expenditures to simplify the analyses. In contrast, inpatient expenditure data contained many zero-valued observations. Hence, we used logistic models to estimate the odds of any inpatient service use and then analyzed inpatient expenditures incurred only by women who had 1 or more hospitalizations.

Crude medical expenditures for moderately exposed and highly exposed women were compared with expenditures for unexposed women, which was used as the referent group. Then, analyses were age-adjusted (age strata: 40–49, 50–59, 60–69, and 70–79 years at baseline survey in 1994) and fully adjusted. The following variables were entered into the fully adjusted model: age stratum, body mass index (weight in kilograms divided by height in meters squared; <18.5, 18.5–24.9, ≥25.0),[11,26] alcohol drinking status (never, former, currently drinking <3 *go*—a traditional Japanese unit of measure equal to 180 mL of Japanese sake and containing 22.8 g of ethanol—per week, currently drinking ≥3 *go* per week), tertiles of daily dietary energy intake (≤1163, 1163–1440, >1440 kcal/day), time spent walking per day (<30, 30–60, ≥60 minutes),[27] self-rated health (good, mediocre, poor), marital status (married, unmarried), current job status (employed, unemployed), and education (≤9, >9 years). Time spent walking represented physical activity level.

The final multivariable model was similar to the fully adjusted model but included the interaction between exposure

Table 2. Monthly medical expenditures for survivors and cost ratios of expenditures for those exposed to ETS at home relative to unexposed survivors

	Median total expenditure (IQR), JPY	Total expenditure		Hospitalization		Inpatient expenditure[a]		Outpatient expenditure	
		Cost ratio (95% CI)	*P*-value	Odds ratio (95% CI)	*P*-value	Cost ratio (95% CI)	*P*-value	Cost ratio (95% CI)	*P*-value
Ratio across all age strata									
Crude ratio									
Moderately exposed		0.90 (0.83–0.97)	0.006	0.86 (0.75–0.99)	0.04	0.84 (0.74–0.96)	0.009	0.94 (0.88–1.01)	0.11
Highly exposed		0.92 (0.85–0.99)	0.02	0.83 (0.72–0.95)	0.009	1.10 (0.97–1.25)	0.14	0.89 (0.83–0.96)	0.002
Age-adjusted ratio[b]									
Moderately exposed		1.01 (0.94–1.08)	0.89	0.97 (0.83–1.12)	0.65	0.91 (0.81–1.04)	0.17	1.04 (0.97–1.11)	0.30
Highly exposed		1.05 (0.98–1.13)	0.17	0.96 (0.83–1.10)	0.53	1.15 (1.02–1.31)	0.02	1.03 (0.96–1.10)	0.38
Fully adjusted ratio[c]									
Moderately exposed		0.98 (0.91–1.05)	0.52	0.96 (0.83–1.12)	0.63	0.93 (0.82–1.06)	0.26	1.00 (0.94–1.07)	0.90
Highly exposed		1.01 (0.94–1.08)	0.87	0.94 (0.81–1.09)	0.43	1.18 (1.05–1.34)	0.007	0.98 (0.92–1.04)	0.50
Ratio in each age stratum[d]									
Age stratum 40–49 years	5900 (2800–13 100)								
Moderately exposed		0.91 (0.79–1.05)	0.20	0.89 (0.65–1.21)	0.46	0.84 (0.63–1.11)	0.21	0.97 (0.85–1.11)	0.65
Highly exposed		0.99 (0.87–1.12)	0.83	0.73 (0.54–0.98)	0.03	1.22 (0.93–1.59)	0.15	1.00 (0.89–1.13)	0.97
Age stratum 50–59 years	11 600 (4800–21 300)								
Moderately exposed		1.07 (0.94–1.21)	0.29	1.18 (0.91–1.55)	0.22	0.99 (0.78–1.26)	0.95	1.07 (0.95–1.20)	0.27
Highly exposed		0.99 (0.87–1.13)	0.91	1.07 (0.81–1.42)	0.63	1.04 (0.81–1.33)	0.77	0.98 (0.87–1.10)	0.68
Age stratum 60–69 years	21 900 (11 800–34 900)								
Moderately exposed		0.93 (0.82–1.04)	0.21	0.81 (0.63–1.03)	0.09	0.96 (0.79–1.17)	0.68	0.95 (0.85–1.06)	0.37
Highly exposed		0.94 (0.84–1.06)	0.33	0.99 (0.77–1.26)	0.92	1.06 (0.88–1.28)	0.54	0.92 (0.82–1.02)	0.12
Age stratum 70–79 years	29 900 (16 800–45 300)								
Moderately exposed		0.98 (0.78–1.24)	0.90	1.11 (0.67–1.85)	0.69	0.74 (0.53–1.03)	0.08	1.08 (0.87–1.34)	0.51
Highly exposed		1.43 (1.13–1.81)	0.003	1.18 (0.71–1.98)	0.53	1.94 (1.38–2.74)	<0.001	1.17 (0.94–1.46)	0.17

All ratios are expressed as ratio relative to women unexposed to ETS. Age indicates age at baseline survey in 1994.
ETS, environmental tobacco smoke; IQR, interquartile range; JPY, Japanese yen.
[a]Inpatient expenditures are compared among women who had 1 or more hospitalizations.
[b]Adjusted for 10-year age stratum.
[c]Adjusted for age, body mass index, alcohol drinking status, dietary energy intake, time spent walking, self-rated health, marital status, current job status, and education.
[d]Computed from a model that includes interaction term (ETS exposure level × 10-year age stratum) in addition to ETS exposure level, age, body mass index, alcohol drinking status, dietary energy intake, time spent walking, self-rated health, marital status, current job status, and education.

level and age stratum. This model with interaction enabled us to assess different ETS-attributable economic consequences by age stratum. Whether there were differences due to ETS exposure level was determined by cost ratios for each age stratum. The following SAS statements were used to calculate cost ratios of medical expenditures in the model with the interaction.

```
proc genmod data = ohsaki;
class ets age bmi alcohol diet walking
healthrating marriage job education;
model expenditure = ets age bmi alcohol diet
walking healthrating marriage job education
ets*age / dist=gamma link=log type3;
run;
```

Median total monthly expenditures per capita and interquartile ranges are presented for each age stratum. All observed expenditures were not inflation-adjusted or discounted, because medical fees were revised slightly (−3.16% to 0.80%) every 2 years during the study period. A 2-sided test was used, and a *P*-value <0.05 was considered statistically significant. All expenditures are expressed as Japanese yen (¥107 = US$1 according to the purchasing power parity rate from the Organisation for Economic Co-operation and Development National Accounts database in 2011). All statistical analyses were performed using IBM SPSS version 18 (SPSS Inc., Chicago, IL, USA) and SAS version 9.2 (SAS Institute Inc., Cary, NC, USA).

RESULTS

Participant characteristics

Table 1 shows the baseline characteristics of women not exposed, moderately exposed, and highly exposed to ETS by survival status. The age stratum 60 to 69 years had the most women ($n = 1664$), and the age stratum 70 to 79 years had the fewest women ($n = 507$). Among all women ($n = 4870$), 1836 (38%) were not exposed to ETS at home, 1448 (30%) were moderately exposed, and 1586 (33%) were highly exposed. Among survivors, the largest groups in the age strata 40 to 49, 50 to 59, 60 to 69, and 70 to 79 years were women who were highly exposed, moderately exposed, unexposed, and unexposed, respectively.

A total of 318 (7%) women died during the follow-up period; 22 (2%) died among 1258 women aged 40 to 49, and 100 (20%) died among 507 women aged 70 to 79. Among survivors, mean follow-up was 135 months; among nonsurvivors, mean follow-up was 93 months. Among survivors, there were significant differences in time spent walking and the proportion of married women among the 3 groups.

Medical expenditures for survivors

Median total monthly medical expenditures per capita ranged from ¥5900 (age stratum 40–49) to ¥29 900 (age stratum 70–79) among survivors (Table 2).

Table 3. Monthly medical expenditures for nonsurvivors and cost ratios of expenditures for those exposed to ETS at home relative to unexposed nonsurvivors

	Median total expenditure (IQR), JPY	Total expenditure		Hospitalization		Inpatient expenditure[a]		Outpatient expenditure	
		Cost ratio (95% CI)	*P*-value	Odds ratio (95% CI)	*P*-value	Cost ratio (95% CI)	*P*-value	Cost ratio (95% CI)	*P*-value
Ratio across all age strata									
Crude ratio									
Moderately exposed		0.68 (0.53–0.87)	0.002	0.88 (0.33–2.32)	0.79	0.71 (0.52–0.96)	0.02	0.65 (0.49–0.85)	0.002
Highly exposed		0.85 (0.68–1.08)	0.18	0.49 (0.22–1.11)	0.09	0.91 (0.68–1.22)	0.52	0.88 (0.68–1.14)	0.33
Age-adjusted ratio[b]									
Moderately exposed		0.69 (0.54–0.88)	0.003	0.94 (0.35–2.54)	0.90	0.70 (0.51–0.95)	0.02	0.70 (0.53–0.92)	0.01
Highly exposed		0.84 (0.66–1.06)	0.14	0.51 (0.22–1.18)	0.12	0.89 (0.66–1.20)	0.45	0.85 (0.66–1.10)	0.22
Fully adjusted ratio[c]									
Moderately exposed		0.69 (0.54–0.87)	0.002	1.11 (0.39–3.19)	0.85	0.66 (0.48–0.90)	0.009	0.76 (0.59–0.99)	0.04
Highly exposed		0.90 (0.71–1.13)	0.36	0.57 (0.23–1.39)	0.22	0.92 (0.67–1.26)	0.60	0.93 (0.73–1.18)	0.54
Ratio in each age stratum[d]									
Age stratum 40–49 years	48 200 (16 800–72 200)								
Moderately exposed		0.73 (0.28–1.86)	0.50	NA		0.52 (0.16–1.66)	0.27	1.14 (0.42–3.09)	0.79
Highly exposed		0.97 (0.37–2.49)	0.94	NA		1.30 (0.40–4.17)	0.66	0.57 (0.21–1.55)	0.27
Age stratum 50–59 years	57 600 (22 500–111 400)								
Moderately exposed		0.58 (0.32–1.05)	0.07	1.60 (0.18–13.89)	0.67	0.70 (0.33–1.48)	0.35	0.41 (0.22–0.77)	0.005
Highly exposed		1.05 (0.58–1.92)	0.87	0.51 (0.07–3.64)	0.50	1.54 (0.70–3.38)	0.28	0.60 (0.32–1.12)	0.11
Age stratum 60–69 years	69 500 (34 600–114 100)								
Moderately exposed		0.75 (0.51–1.11)	0.15	NA		0.78 (0.48–1.26)	0.31	0.65 (0.43–0.98)	0.04
Highly exposed		0.78 (0.57–1.07)	0.12	0.61 (0.19–1.99)	0.41	0.75 (0.49–1.14)	0.17	0.91 (0.65–1.27)	0.57
Age stratum 70–79 years	52 700 (32 400–108 000)								
Moderately exposed		0.69 (0.46–1.05)	0.08	0.41 (0.08–2.18)	0.29	0.60 (0.34–1.05)	0.07	1.03 (0.67–1.59)	0.90
Highly exposed		1.03 (0.65–1.64)	0.90	0.71 (0.09–5.50)	0.74	0.87 (0.48–1.58)	0.65	1.35 (0.83–2.18)	0.23

All ratios are expressed as ratio relative to women unexposed to ETS. Age indicates age at baseline survey in 1994.
ETS, environmental tobacco smoke; IQR, interquartile range; JPY, Japanese yen; NA, not applicable.
[a]Inpatient expenditures are compared among women who had 1 or more hospitalizations.
[b]Adjusted for 10-year age stratum.
[c]Adjusted for age, body mass index, alcohol drinking status, dietary energy intake, time spent walking, self-rated health, marital status, current job status, and education.
[d]Computed from a model that includes interaction term (ETS exposure level × 10-year age stratum) in addition to ETS exposure level, age, body mass index, alcohol drinking status, dietary energy intake, time spent walking, self-rated health, marital status, current job status, and education.

Crude, age-adjusted, and fully adjusted (the model without the interaction) cost ratios for moderately exposed and highly exposed survivors, as compared with unexposed survivors, are shown in Table 2. Cost ratios of total (ie, inpatient plus outpatient care), inpatient and outpatient expenditures, and odds ratios of hospitalization were calculated for moderately exposed and highly exposed women in relation to unexposed women, which was used as the referent category.

We found significant differences in some crude total, inpatient, and outpatient expenditures associated with ETS exposure. For example, crude total expenditures were lower for moderately exposed and highly exposed women than for unexposed women. In contrast, we found no significant difference in age-adjusted or fully adjusted total expenditures, regardless of exposure, although age-adjusted and fully adjusted inpatient expenditures were higher for highly exposed women than for unexposed women.

There were significant differences in the fully adjusted model with the interaction term between exposure level and age stratum (Table 2). Total expenditure for age stratum 70 to 79 years was higher for highly exposed women than for unexposed women after adjusting for other variables (cost ratio, 1.43; 95% CI, 1.13–1.81). The difference in total expenditure between unexposed and moderately exposed women was not significant.

When inpatient and outpatient care were analyzed separately, inpatient expenditure was higher for highly exposed women than for unexposed women (cost ratio, 1.94; 95% CI, 1.38–2.74), although there was no significant association between probability of hospitalization and exposure level. We found no difference in adjusted outpatient expenditures for age stratum 70 to 79 years, regardless of exposure.

In the age strata 40 to 49, 50 to 59, and 60 to 69 years, total, inpatient, and outpatient medical expenditures did not significantly differ, regardless of exposure (Table 2). However, inpatient expenditures tended to be higher for highly exposed women than for unexposed women among all these strata.

Medical expenditures for nonsurvivors

Median total monthly medical expenditures per capita ranged from ¥48 200 (age stratum 40–49) to ¥69 500 (age stratum 60–69) among nonsurvivors (Table 3).

Crude, age-adjusted, and fully adjusted (the model without the interaction) cost ratios for moderately exposed and highly exposed nonsurvivors, as compared with unexposed nonsurvivors, are shown in Table 3. Crude, age-adjusted, and fully adjusted total expenditures were lower for moderately exposed nonsurvivors than for unexposed nonsurvivors.

Table 3 also shows cost ratios of medical expenditures for nonsurvivors in the fully adjusted model with the interaction between exposure level and age stratum. In all age strata, including age stratum 70 to 79 years, we found no significant difference in total expenditure regardless of exposure, although outpatient expenditures for the age strata 50 to 59 and 60 to 69 years were lower for moderately exposed nonsurvivors than for unexposed nonsurvivors.

DISCUSSION

To calculate expenditures attributable to ETS, we analyzed medical expenditures incurred by female nonsmokers who were not exposed, moderately exposed, and highly exposed to ETS in the home. Among highly exposed survivors aged 70 to 79 years, we found that a substantial increase in total medical expenditure was possibly attributable to ETS exposure at home. This excess expenditure suggests a significant age-specific association between ETS exposure and total medical expenditure.

The association of ETS exposure with economic impact is consistent with known clinical relationships and provides an explanation for the economic burden. Although our significant findings regarding total expenditures are limited to older survivors, they support the results of previous simulation studies[16–21] that estimated the economic burden caused by ETS-attributable diseases at the national, regional, and state levels rather than the individual level.

Interpretation of findings

High ETS exposure resulted in significantly higher inpatient but not outpatient expenditures among survivors aged 70 to 79 years, which suggests that the excess total expenditure arose from treatment for serious but nonfatal diseases rather than from treatment for relatively minor disorders.

ETS exposure significantly increased total medical expenditures only among survivors aged 70 to 79 years. One plausible explanation for this result is that it takes many years to produce a significant difference in total medical expenditures between unexposed and highly exposed adults, because the harm of ETS is subtle in comparison to active smoking. Another likely explanation is that diseases result in higher morbidity among older versus younger adults. Notably, among women aged 40 to 69 years, highly exposed women tended to incur higher inpatient expenditures than unexposed women, which suggests that serious but nonfatal diseases attributable to ETS are already present among highly exposed women in these age strata.

In contrast to survivors, we found no significant age-specific association between total medical expenditures incurred by nonsurvivors and ETS exposure, perhaps because ETS exposure does not increase costs of therapy for fatal diseases, although exposure increases the incidence of such diseases. In contrast to our findings among nonsurvivors, ETS-attributable excess total expenditures among survivors may arise from the accumulation of excess morbidities from nonfatal and near-fatal diseases.

The results for nonsurvivors in age strata 50 to 59 and 60 to 69 years showed that moderately exposed nonsurvivors incurred lower outpatient expenditures as compared with unexposed nonsurvivors, possibly because relatively low doses of toxins inhaled from ETS are pathophysiologically sufficient to elicit a strong acute effect on the cardiovascular system, whereas lung cancer is caused by long-term exposure.[28] This evidence suggests that ETS exposure causes rapid onset of coronary heart disease and subsequent death in these age strata, which may account for the lower monthly outpatient expenditure.

Strengths and limitations of the study

Our study has several advantages as compared with previous studies. To the best of our knowledge, this is the first study using directly observed long-term individual-level health insurance records to show a significant association between ETS exposure and medical expenditures among adults. Previous studies estimated population-level ETS-attributable expenditures by using economic models of mixed results from multiple databases, such as the published literature and macrodata on ETS exposure, increased morbidity, and medical costs.[16–21] We showed the individual-level economic burden imposed by ETS exposure by means of comparison within a single cohort.

Furthermore, we followed a large cohort for a long period. The expenditures in our analyses were accurate because we obtained health insurance claims data directly from the Miyagi NHI Organizations, which included information on almost all available medical services. Long-term observation allowed average monthly expenditure to be unaffected by short-term incidental use of medical services.

Our study has several limitations. First, assessment of ETS exposure was based on a questionnaire survey. Misclassification of exposure status is a concern in studies that use only questionnaires. Quantitative information on ETS exposure is less reliable in questionnaires, but information on whether exposure is heavy or light is relatively reliable.[29,30] Second, the questionnaire in our study focused on ETS exposure at 1 time point, as we assumed that exposure status at baseline was correlated with past exposure. To ensure the correctness of this assumption, we excluded women with a change in job status or marital status during their lifetime. Nevertheless, further research based on long-term continuous ETS exposure measurement is required. Third, all medical expenditures were included in our analysis. There were no available cost data for specific diseases such as lung cancer and coronary heart disease. However, diseases attributable to ETS exposure range from life-threatening diseases to relatively minor disorders (eg, respiratory tract symptoms).[31] This is similar to active smoking, which has been reported to be relevant to many diseases, including major smoking-related diseases.[32] Previous studies of the association between active smoking and medical expenditures have also addressed overall disease burden.[9,10] Furthermore, if we analyzed expenditures for specific diseases that were strongly associated with ETS exposure, we might also find significant relationships between ETS exposure and total expenditures for younger women. Future studies of real-world medical expenditures for diseases strongly associated with ETS exposure may be needed to verify the results of previous studies that simulated the excess costs attributable to ETS by combining ETS-attributable diseases and associated medical costs. Finally, long-term care (LTC) insurance claims data were not available. Japan has a LTC insurance system that supports elderly adults living at home or in nursing-care facilities. We believe that women that incur more medical expenditures for treatment of diseases due to ETS exposure may also use more LTC services than women unexposed to ETS.

We examined household ETS exposure because previous cohort studies of the health effects of ETS focused on ETS exposure at home or at work.[1,2,5] How applicable are our results to ETS exposure outside the home? Considerable evidence of increased mortality and morbidity related to ETS exposure has also been obtained from exposure in workplaces and public places.[4,5,33] We believe that ETS exposure in settings other than the home also increases medical expenditures. Medical expenditures attributable to ETS exposure in the workplace and other settings need to be explored.

This study analyzed the self-employed, part-time workers, the unemployed, and their families. These groups may spend more time at home than corporate employees, who are covered by Employees' Health Insurance. Thus, the effects of household ETS might be more severe in the present study population than in corporate employees and their families.

Conclusions

We found that severe household ETS exposure results in excess total medical expenditures. Surviving female nonsmokers aged 70 to 79 years who were highly exposed to ETS at home incurred significantly higher total medical expenditures than those living in smoke-free households. The present study provides information on the economic burden of ETS, although significant findings regarding total expenditure are limited to surviving older women. This information should help policymakers to develop strategies that reduce secondhand smoke and hasten the economically attractive goal of eliminating ETS. Further research is required to examine the association between accumulated ETS exposure and medical expenditure.

ACKNOWLEDGMENTS

This work was supported by a Health Sciences Research Grant from the Ministry of Health, Labour and Welfare of Japan. The funder had no role in the study design; the collection, analysis, and interpretation of data; the writing of the report; or in the decision to submit the article for publication.

Conflicts of interest: None declared.

ONLINE ONLY MATERIALS

The Japanese-language abstract for articles can be accessed by clicking on the tab labeled Supplementary materials at the journal website **http://dx.doi.org/10.2188/jea.JE20120072.**

REFERENCES

1. Hill S, Blakely T, Kawachi I, Woodward A. Mortality among "never smokers" living with smokers: two cohort studies, 1981–4 and 1996–9. BMJ. 2004;328:988–9.
2. McGhee SM, Ho SY, Schooling M, Ho LM, Thomas GN, Hedley AJ, et al. Mortality associated with passive smoking in Hong Kong. BMJ. 2005;330:287–8.
3. Hackshaw AK, Law MR, Wald NJ. The accumulated evidence on lung cancer and environmental tobacco smoke. BMJ. 1997;315:980–8.
4. Boffetta P, Agudo A, Ahrens W, Benhamou E, Benhamou S, Darby SC, et al. Multicenter case-control study of exposure to environmental tobacco smoke and lung cancer in Europe. J Natl Cancer Inst. 1998;90:1440–50.
5. Steenland K, Thun M, Lally C, Heath C Jr. Environmental tobacco smoke and coronary heart disease in the American Cancer Society CPS-II cohort. Circulation. 1996;94:622–8.
6. Barnoya J, Glantz SA. Cardiovascular effects of secondhand smoke: nearly as large as smoking. Circulation. 2005;111: 2684–98.
7. Centers for Disease Control and Prevention (CDC). Smoking-attributable mortality, years of potential life lost, and productivity losses—United States, 2000–2004. MMWR Morb Mortal Wkly Rep. 2008;57:1226–8.
8. Öberg M, Jaakkola MS, Woodward A, Peruga A, Prüss-Ustün A. Worldwide burden of disease from exposure to second-hand smoke: a retrospective analysis of data from 192 countries. Lancet. 2011;377:139–46.
9. Izumi Y, Tsuji I, Ohkubo T, Kuwahara A, Nishino Y, Hisamichi S. Impact of smoking habit on medical care use and its costs: a prospective observation of National Health Insurance beneficiaries in Japan. Int J Epidemiol. 2001;30:616–21.
10. Miller VP, Ernst C, Collin F. Smoking-attributable medical care costs in the USA. Soc Sci Med. 1999;48:375–91.
11. Sturm R. The effects of obesity, smoking, and drinking on medical problems and costs. Health Aff (Millwood). 2002;21: 245–53.
12. Bartecchi CE, MacKenzie TD, Schrier RW. The human costs of tobacco use (1). N Engl J Med. 1994;330:907–12.
13. Barendregt JJ, Bonneux L, van der Maas PJ. The health care costs of smoking. N Engl J Med. 1997;337:1052–7.
14. Rasmussen SR, Prescott E, Sørensen TI, Søgaard J. The total lifetime costs of smoking. Eur J Public Health. 2004;14:95–100.
15. Sung HY, Wang L, Jin S, Hu TW, Jiang Y. Economic burden of

smoking in China, 2000. **Tob Control. 2006;15 suppl 1:i5–11.**
16. Hauri DD, Lieb CM, Rajkumar S, Kooijman C, Sommer HL, Röösli M. Direct health costs of environmental tobacco smoke exposure and indirect health benefits due to smoking ban introduction. **Eur J Public Health. 2011;21:316–22.**
17. Zollinger TW, Saywell RM Jr, Overgaard AD, Jay SJ, Holloway AM, Cummings SF. Estimating the economic impact of secondhand smoke on the health of a community. **Am J Health Promot. 2004;18:232–8.**
18. Behan DF, Eriksen MP, Lin Y [homepage on the Internet]. Economic Effects of Environmental Tobacco Smoke. Schaumburg, IL: Society of Actuaries; 2005 [cited 2012 April 4]. Available from: **http://www.soa.org/research/research-projects/life-insurance/research-economic-effect.aspx.**
19. McGhee SM, Ho LM, Lapsley HM, Chau J, Cheung WL, Ho SY, et al. Cost of tobacco-related diseases, including passive smoking, in Hong Kong. **Tob Control. 2006;15:125–30.**
20. Waters HR, Foldes SS, Alesci NL, Samet J. The economic impact of exposure to secondhand smoke in Minnesota. **Am J Public Health. 2009;99:754–9.**
21. Plescia M, Wansink D, Waters HR, Herndon S. Medical costs of secondhand-smoke exposure in North Carolina. **N C Med J. 2011;72:7–12.**
22. Öberg M, Woodward A, Jaakkola MS, Peruga A, Prüss-Ustün A [homepage on the Internet]. Global estimate of the burden of disease from second-hand smoke. Geneva: World Health Organization; 2011 [cited 2012 April 4]. Available from: **http://www.who.int/tobacco/publications/second_hand/global_estimate_burden_disease/en/index.html.**
23. Tsuji I, Nishino Y, Ohkubo T, Kuwahara A, Ogawa K, Watanabe Y, et al. A prospective cohort study on National Health Insurance beneficiaries in Ohsaki, Miyagi Prefecture, Japan: study design, profiles of the subjects and medical cost during the first year. **J Epidemiol. 1998;8:258–63.**
24. Emanuel EJ, Fairclough DL, Slutsman J, Emanuel LL. Understanding economic and other burdens of terminal illness: the experience of patients and their caregivers. **Ann Intern Med. 2000;132:451–9.**
25. Manning WG, Basu A, Mullahy J. Generalized modeling approaches to risk adjustment of skewed outcomes data. **J Health Econ. 2005;24:465–88.**
26. Kuriyama S, Ohmori K, Miura C, Suzuki Y, Nakaya N, Fujita K, et al. Body mass index and mortality in Japan: the Miyagi Cohort Study. **J Epidemiol. 2004;14 Suppl 1:S33–8.**
27. Tsuji I, Takahashi K, Nishino Y, Ohkubo T, Kuriyama S, Watanabe Y, et al. Impact of walking upon medical care expenditure in Japan: the Ohsaki Cohort Study. **Int J Epidemiol. 2003;32:809–14.**
28. Raupach T, Schäfer K, Konstantinides S, Andreas S. Secondhand smoke as an acute threat for the cardiovascular system: a change in paradigm. **Eur Heart J. 2006;27:386–92.**
29. Öberg M, Jaakkola MS, Prüss-Ustün A, Schweizer C, Woodward A [homepage on the Internet]. Second hand smoke: Assessing the burden of disease at national and local levels. Geneva: World Health Organization; 2010. Environmental burden of disease series; vol 18 [cited 2012 April 4]. Available from: **http://www.who.int/quantifying_ehimpacts/publications/ebd18/en/index.html.**
30. Coultas DB, Peake GT, Samet JM. Questionnaire assessment of lifetime and recent exposure to environmental tobacco smoke. **Am J Epidemiol. 1989;130:338–47.**
31. Bascom R, Kulle T, Kagey-Sobotka A, Proud D. Upper respiratory tract environmental tobacco smoke sensitivity. **Am Rev Respir Dis. 1991;143:1304–11.**
32. Warner KE, Hodgson TA, Carroll CE. Medical costs of smoking in the United States: estimates, their validity, and their implications. **Tob Control. 1999;8:290–300.**
33. Sargent RP, Shepard RM, Glantz SA. Reduced incidence of admissions for myocardial infarction associated with public smoking ban: before and after study. **BMJ. 2004;328:977–80.**

日衛誌 (Jpn. J. Hyg.), 67, 50–55 (2012)

総　説

ミニ特集　第81回日本衛生学会
健康増進・地域医療・医療費適正化計画とデータ活用
～生活習慣病の予防・治療システムの戦略的構築へ～

喫煙者と非喫煙者の生涯医療費

林田　賢史[*1], 村上　玄樹[*2], 高橋　裕子[*3], 辻　一郎[*4], 今中　雄一[*5]

[*1] 産業医科大学病院医療情報部
[*2] 広島大学大学院医歯薬学総合研究科公衆衛生学研究室
[*3] 奈良女子大学保健管理センター
[*4] 東北大学大学院医学研究科公衆衛生学分野
[*5] 京都大学大学院医学研究科医療経済学分野

Lifetime Medical Expenditures of Smokers and Nonsmokers

Kenshi HAYASHIDA[*1], Genki MURAKAMI[*2], Yuko TAKAHASHI[*3], Ichiro TSUJI[*4] and Yuichi IMANAKA[*5]

[*1] *Department of Medical Informatics and Management, University Hospital of Occupational and Environmental Health*
[*2] *Department of Public Health and Health Policy, Hiroshima University Graduate School of Biomedical Sciences*
[*3] *Health Administration Center, Nara Women's University*
[*4] *Department of Public Health and Forensic Medicine, Tohoku University Graduate School of Medicine*
[*5] *Department of Healthcare Economics and Quality Management, Kyoto University Graduate School of Medicine*

Abstract　**Objectives:** The aim of this study was to examine which of the two groups have higher lifetime medical expenditures; male smokers or male nonsmokers. We conducted this investigation using a Japanese single cohort database to calculate long-term medical expenditures and 95% confidence intervals.

Methods: We first constructed life tables for male smokers and male nonsmokers from the age of 40 years after analyzing their mortality rates. Next, we calculated the average annual medical expenditures of each of the two groups, categorized into survivors and deceased. Finally, we calculated long-term medical expenditures and performed sensitivity analyses.

Results: The results showed that although smokers had generally higher annual medical expenditures than nonsmokers, the former's lifetime medical expenditure was slightly lower than the latter's because of a shorter life expectancy that resulted from a higher mortality rate. Sensitivity analyses did not reverse the order of the two lifetime medical expenditures.

Conclusions: In conclusion, although smoking may not result in an increase in lifetime medical expenditures, it is associated with diseases, decreased life expectancy, lower quality of life (QOL), and generally higher annual medical expenditures. It is crucial to promote further tobacco control strategically by maximizing the use of available data.

Key words: smoking（喫煙）, lifetime medical expenditure（生涯医療費）, mortality（死亡率）, cohort study（コホート研究）, tobacco control（たばこ規制）

I．緒　言

喫煙はがん，心疾患，脳血管障害，糖尿病（2型），高血圧などの生活習慣病と深く関連しており (1–4)，わが国における喫煙による死亡数は2005年の時点で年間19.6万人と推定されている (5)。欧米先進国では，喫煙は予防しうる最大の疾病・早死の原因との認識のもと，種々

受付2011年10月31日，受理2011年12月21日
Reprint requests to: Kenshi HAYASHIDA
Department of Medical Informatics and Management, University Hospital of Occupational and Environmental Health, 1-1 Iseigaoka, Yahatanishi-ku, Kitakyusyu, Fukuoka 807-8556, Japan
TEL: +81(93)603-1611 (ext7005), FAX: +81(93)601-2098
E-mail: kenshi@clnc.uoeh-u.ac.jp

の喫煙対策が実施され成果を上げている（6, 7）。近年医療費が高騰するなか，我が国でも喫煙の健康への影響だけでなく，医療費への影響，特に長期的影響（生涯医療費）に大きな関心が寄せられている。これは疾病予防と健康増進によって医療費節減効果がもたらされるかに関する一例といえるからである。

喫煙者の生涯医療費が非喫煙者に比べて高額か否かについては，これまで国内外を問わず多くの議論がなされてきている。わが国においては，短期間での医療費は喫煙者の方が非喫煙者に比べて高額であるという先行研究（8）はあるものの，喫煙者の年間医療費は高額である反面余命は短いため，喫煙者と非喫煙者のどちらの生涯医療費が高額かはっきりしていない。

諸外国ではこれまで，喫煙と生涯医療費の関係についていくつかの研究が実施されており，それらの研究結果では，Manningら（9），Hodgsonら（10），Mackenzieら（11），Rasmussenら（12）は喫煙者のほうが高額であるとしたが，Leuら（13），Lippiatt（14），Barendregt（15）は非喫煙者のほうが高額であるとした。つまり生涯医療費に関しては，喫煙者の方が高い結果もあれば低い結果もあり，一致した結果が示されていないのが現状である。

ただしこれらの研究には，計算の正確性における限界があることは否めない。というのも，これらの研究の多くは，生涯医療費を算出するために構築した経済学的なモデルに，喫煙者と非喫煙者の国レベルで集約されたマクロデータを適用することで，あるいは喫煙と発症が強く関連している疾患に限定しその医療費増加データを用いることで，生涯医療費を推計したものであり，複数のデータベースのミックスによる計算結果，あるいは限定した疾患に関する医療費にすぎないからである。

そこで本研究では，わが国で実施されている単一のコホート研究（追跡研究）によって収集された個人レベルのデータに単純な計算モデルを用いることで，喫煙者と非喫煙者の余命，年間平均や中長期の医療費について，より直接的に算出することを目的とした。

II．研究方法

1．解析データ

本研究では，宮城県の「大崎コホート研究」で得られた40～79歳（1994年8月31日時点）の男性の国民健康保険加入者24,573人の11年間にわたるデータを用いた。また，大崎国保コホート研究のデータベースに含まれない高齢の死亡率の推定のため，平成17年の完全生命表である第20回生命表のデータを用いた。

この「大崎コホート研究」は東北大学社会医学講座公衆衛生学分野が，宮城県大崎保健所管内1市3町（当時）に住む国民健康保険加入者で，1994年8月31日時点で40～49歳の全員56,294名を対象に同年10～12月に自記式アンケート調査による生活習慣などに関するベースライン調査を実施し，1995年1月以後の医療利用状況についてレセプトデータ等を用いて追跡しているものである（16）。ベースライン調査の項目は性別，年齢などの基本的情報や病歴，身体機能，喫煙や食習慣等，健康に関する生活習慣である。ベースライン調査の有効回答者52,029名のうち，1995年1月のレセプトデータ追跡開始時までに死亡または転出したものを除外した51,255人について，1995年1月から毎月，宮城県国民健康保険団体連合会からデータの提供をうけて国民健康保険レセプトとレコードリンケージをおこない，受診状況，医療費を継続して把握している。また対象者の死亡や転出による異動に関しても，1995年1月からの国民健康保険の喪失異動データとのレコードリンケージにより追跡している。

2．解析方法

（1）生命表（喫煙者・非喫煙者）の作成

①各年齢の死亡確率の推定

喫煙状況別の各年齢における死亡確率についてロジスティック回帰モデルを用いて推定した。

②シミュレーションによる生命表の作成

40歳を起点とした喫煙者と非喫煙者それぞれの10万人のコホートを設定し，推定された各年齢における死亡確率を用いてシミュレーションすることで生命表を作成した。なお，大崎国保コホート研究のデータベースに含まれない高齢の死亡率に関しては，喫煙状況にかかわらず同じ値を用いることとし，完全生命表における死亡率を用いた。

（2）1年間医療費の平均の算出

年齢別・喫煙状況別・生存死亡別にデータを区分し，年間医療費（入院・入院外）の平均（1人あたりの年間医療費の平均）を算出した。

（3）生涯医療費を含めた中長期の累積医療費（1人あたりの換算値）の算出シミュレーション

中長期の累積医療費（1人あたりの換算値）を算出するにあたり，まずコホート全体の累積医療費を算出するために，「各年齢の生存・死亡者数」と「1人あたりの年間医療費の平均（年齢別・喫煙状況別・生存死亡別）」から各年齢の医療費全体（喫煙状況別）を算出した後，ある年齢までの総和（コホート全体の医療費）を算出した。その後今回は10万人のコホートを設定したため，コホート全体の医療費を10万で除すことで1人あたりに換算した。将来の金銭的価値は現在価値に換算すると低い評価となるため，経済的評価においては，未来の金銭的価値について割り引く必要がある。そのため本研究では，ベースシナリオとして累積医療費（全体，入院，入院外）に対して，近年一般的な3%の割引率を設定した。また感度分析のために，3%の割引率に加えて，なし（0%），1%，5%の全部で4種類の割引率を設定するとともに，平均余命や生涯医療費の95%信頼区間をブートストラップ法を用いて算出した。

解析・集計ソフトは，SPSS 17.0J for WindowsとEXCEL 2007 for Windowsを用いた。

表1　年齢・喫煙状況が生死に与える影響（男性）

説明変数		係数	オッズ比	95% 信頼区間	p 値
年齢		0.0915	1.10	(1.09–1.10)	0.000
喫煙経験	なし（基準）		1.00		
	あり	0.417	1.52	(1.38–1.67)	0.000
切片		−10.6			
Hosmer-Lemeshow test			χ^2 値＝7.126		p＝0.52
C-statistics			0.743	(0.735–0.750)	

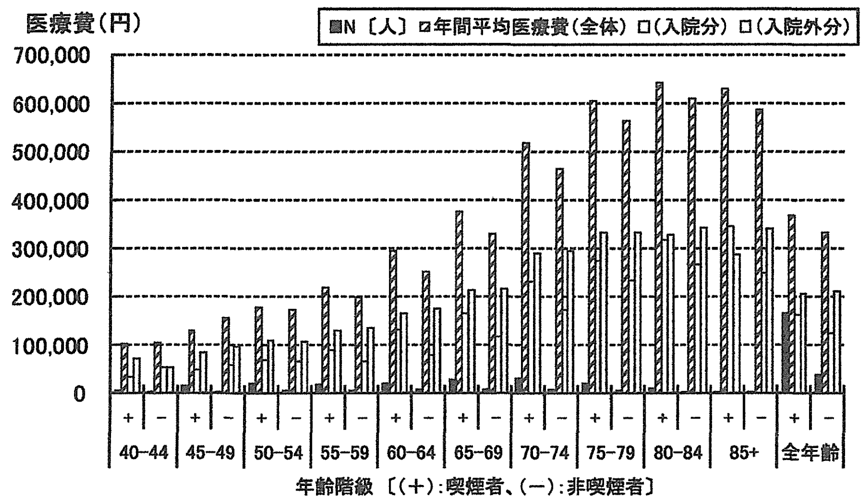


図1　年齢区分・喫煙状況別1年間平均医療費（入外別）（男性：全体（生存者・死亡者））

III．研究結果

(1) 生命表（喫煙者・非喫煙者）の作成

ロジスティック回帰分析の結果，年齢が上がるほど，また喫煙者であるほど死亡のリスクが高かった。オッズ比はそれぞれ1.10（95% 信頼区間：1.09–1.10），1.52（1.38–1.67）であり，統計的に有意な結果であった（表1）。それらの値から算出される各年齢での死亡確率を用いて生命表を作成したところ，40歳平均余命は喫煙者で39.6歳，非喫煙者で43.1歳と非喫煙者の方が長かった。

(2) 1人あたり年間医療費の平均の算出

1人あたり年間医療費（年齢階級別）の平均を生存者死亡者あわせた全体で算出したところ，40歳代を除き喫煙者の方が高額であった（図1）。生存者・死亡者で区別すると生存者の場合，全体の結果と同様に40歳代を除き喫煙者の方が高額であった（図2）が，死亡者の場合特にはっきりした傾向はなかった（図3）。

(3) 生涯医療費の算出シミュレーション

生涯医療費については，非喫煙者621万円，喫煙者600万円となり，非喫煙者の方が3.5%高額であった（図4）。しかし，入院医療費に限ってみると喫煙者群の方が高額であった。

複数の割引率（なし，1%，3%，5%）を用いた累積医療費を把握したところ，生涯医療費に関しては，すべての割引率において非喫煙者の方が喫煙者より高額（割引率なし：89万円差，割引率1%：54万円差，割引率3%：21万円差，割引率5%：9万円差）であった。また，中長期の累積医療費に関しては，割引率なしの場合22年後となる62歳時点から43年後となる83歳時点までの期間に関して，割引率1%の場合22年後となる62歳時点から41年後となる81歳時点までの期間に関して，割引率3%の場合24年後となる64歳時点から41年後となる81歳時点までの期間に関して，割引率5%の場合24年後となる64歳時点から40年後となる80歳時点までの期間に関しては，喫煙者群の累積医療費の方が非喫煙者群のそれより高額という結果であった（図5）。ブートストラップ法を用いた喫煙者と非喫煙者間の平均余命や生涯医療費の95% 信頼区間に関しては，表2の通りであった。

IV．考　察

本研究では単一のコホート研究によって収集された個人レベルのデータを用いて，喫煙状況別の各年齢における死亡確率や1人あたり年間医療費の平均を算出し，現在40歳である男性の喫煙者と非喫煙者の余命，生涯医療費を含む中長期の累積医療費を算出した。その結果，(1)

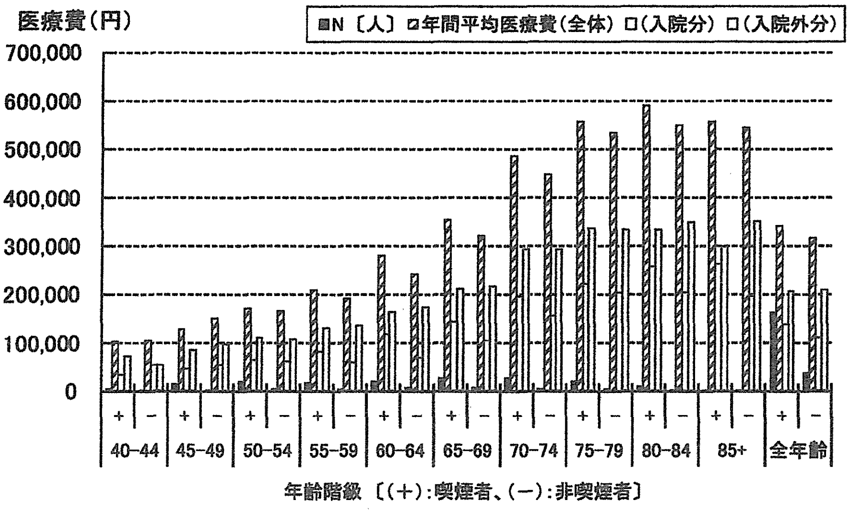


図2　年齢区分・喫煙状況別1年間平均医療費（入外別）（男性：生存者）

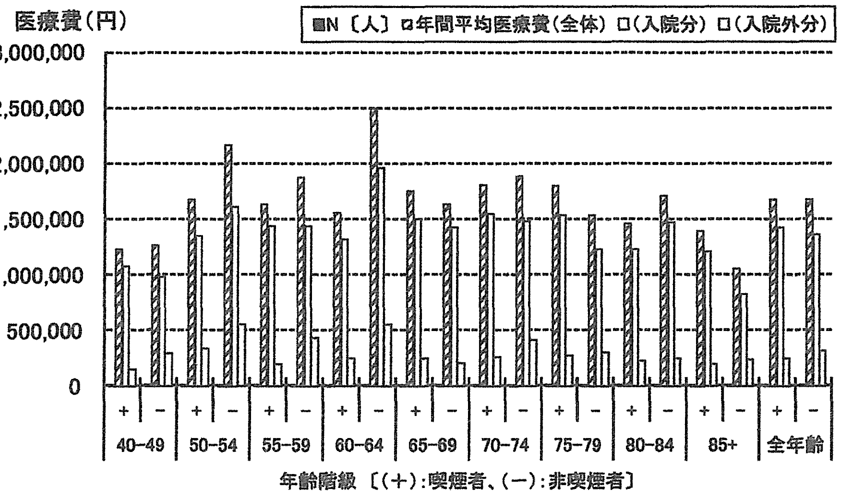


図3　年齢区分・喫煙状況別1年間平均医療費（入外別）（男性：死亡者）

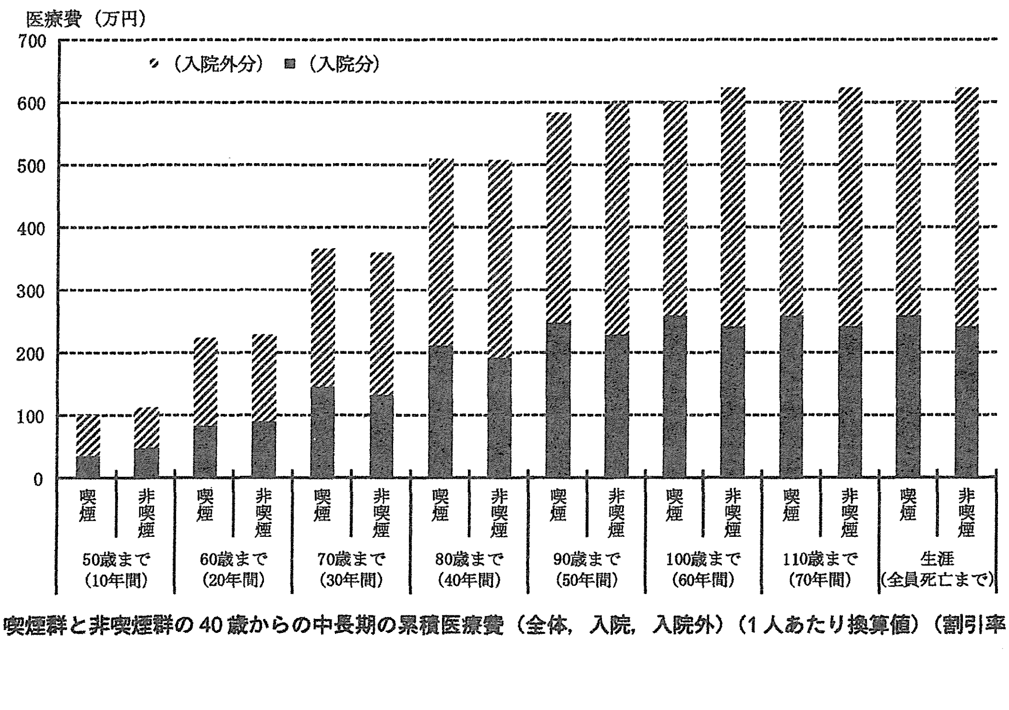


図4　喫煙群と非喫煙群の40歳からの中長期の累積医療費（全体，入院，入院外）（1人あたり換算値）（割引率：3%）

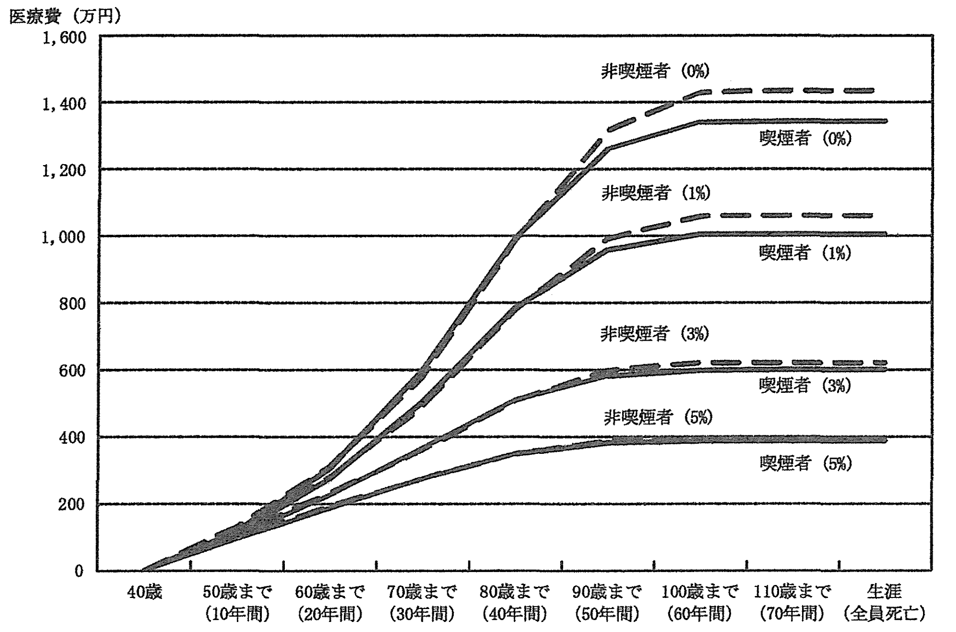


図 5　喫煙群と非喫煙群の 40 歳からの中長期の累積医療費（1 人あたり換算値）（割引率：なし，1，3，5%）

表 2　40 歳からの平均余命と生涯医療費（男性）

平均余命（年）		喫煙者	39.8	（39.5–40.1）
		非喫煙者	43.3	（42.6–43.5）
生涯医療費（万円）	割引率 0%	喫煙者	1,355	（1,334–1,366）
		非喫煙者	1,444	（1,408–1,482）
	割引率 1%	喫煙者	1,013	（999–1,020）
		非喫煙者	1,067	（1,043–1,098）
	割引率 3%	喫煙者	602	（597–608）
		非喫煙者	624	（616–644）
	割引率 5%	喫煙者	388	（386–394）
		非喫煙者	397	（394–412）

喫煙者が非喫煙者より死亡のリスクが高く短命であること，（2）1 人あたり年間医療費の平均は，全体で見ると喫煙者が非喫煙者より高額であること，（3）中長期の累積医療費は，60 ～ 80 歳頃までは喫煙者が非喫煙者より高額であったが，生涯医療費で見ると非喫煙者が喫煙者より高額であること，（4）感度分析を実施しても，喫煙者と非喫煙者の生涯医療費の関係が逆転するような影響はないことがわかった。

（1）喫煙者と非喫煙者の医療費

本研究の結果，生涯医療費に関しては喫煙者が非喫煙者よりわずかに低いと示唆され，いくつかの先行研究（13–15）の結果と一致した。これは喫煙者の方が年間の医療費は高額であるが非喫煙者と比較して短い余命であるためだと考えられる。また割引率を変化させて感度分析を実施したところ，割引率が大きいほど非喫煙者と喫煙者の生涯医療費の差は縮まったが，前者の方が高額であることに変わりはなく，結果が大きく変わるような影響はなかった。おそらく喫煙は，年間医療費を高額にする影響はあるものの，生涯医療費を高額にする影響はないと考えられる。しかし中長期の累積医療費に関しては，60 ～ 80 歳代（40 歳開始時点の 20 ～ 40 年後）までの場合，非喫煙者の医療費が喫煙者よりわずかに低額であった。

（2）喫煙者と非喫煙者の平均余命

本研究では，40 歳男性の平均余命は喫煙者 39.6 年，非喫煙者 43.1 年であり，非喫煙者の方が 3.5 年長かった。この結果は他のコホート集団を用い別の方法で算出した先行研究の結果（17）とほぼ一致しており，40 歳の平均余命は喫煙者と非喫煙者において約 3.5 年の違いが出ると考えられる。この 3.5 年という長さはイギリスでの研究結果（1）と比較すると短い結果であったが，これは（1）日本では分煙が不十分であり，受動喫煙により非喫煙者もたばこの影響を受けていた可能性，（2）1994 年の一時点でのみの喫煙状況であったので喫煙状況に変化が起こっていた可能性，（3）質問紙票による喫煙状況の調査に正確に回答していなかった対象者がいた可能性等が考えられる。

（3）生涯医療費算出における単一コホートデータの活用（たばこを例として）

本研究は，長期間追跡されている単一のコホートデータを用いて分析が実施された研究であり，従来研究のような国家レベルのマクロデータなどを結合し経済モデルを適用する研究（9–13）に比して，生涯医療費の算出の正確性が上がったと考えられる。それは，コホートのデータを用いることで，個人ベースのデータにアクセスする

ことができ，年齢や喫煙状況による死亡確率の推定，あるいは年齢・喫煙状況・生死の状態による年間の医療費の平均を分析することが可能となったからである。そのため例えば，喫煙者は同じ年齢区分の非喫煙者より全体でみると高い年間医療費であったが，それを生存者と死亡者に区分したり，入院患者と入院外患者で区分したりすると，わずかながらとはいえ反対の傾向であることが導き出された。今まで喫煙者群と非喫煙者群に関するコホートデータがなかったため，こうした生存者と死亡者を区分した医療費に関する分析はほとんど行われてこなかったが，今回生存者と死亡者を層別した分析の重要性も示唆された。

V. 結　語

今回の分析結果により，非喫煙者が喫煙者より生涯医療費はわずかに高く平均余命は長いことが示唆された。これらから喫煙で生涯医療費が上がることはないと考えられるが，喫煙は様々な疾患の発症に関係していることも明らかであり，そのため余命を短くし生活の質 (QOL) を低下させ，また年間の医療費を高額にする。健康でQOLの高い生活を送ることは皆の願いであり，今後も喫煙対策を進めることは重要であろう。

文　献

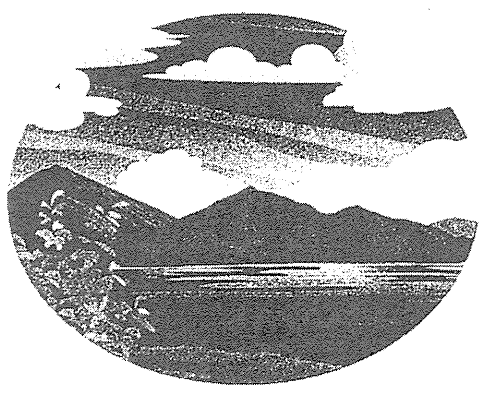
(1) Doll R, Peto R, Boreham J, Sutherland I. Mortality in relation to smoking: 50 years' observations on male British doctors. Br Med J. 2004;328:1519.
(2) Ezzati M, Lopez AD. Estimates of global mortality attributable to smoking in 2000. Lancet. 2003;362:847–852.
(3) Jacobs Jr DR, Adachi H, Mulder I, Kromhout D, Menotti A, Nissinen A, et al. Cigarette smoking and mortality risk: twenty-five-year follow up of the Seven Countries Study. Arch Intern Med. 1999;159:733–740.
(4) LaCroix AZ, Lang J, Scherr P, Wallace RB, Cornoni-Huntley J, Berkman L, et al. Smoking and mortality among older men and women in three communities. N Engl J Med. 1991;324:1619–1625.
(5) Katanoda K, et al., An official American Thoracic Society public policy statement: Novel risk factors and the global burden of chronic obstructive pulmonary disease. Am J Respir Crit Care Med. 2010;182:693–718.
(6) Eriksen MP, Cerak RL. The diffusion and impact of clean indoor air laws. Annu Rev Public Health. 2008;29:171–185.
(7) Fong GT, Cummings KM, Borland R, Hastings G, Hyland A, Giovino GA, et al. The conceptual framework of the International Tobacco Control (ITC) Policy Evaluation Project. Tob Control. 2006;15(Suppl.3):iii3–11.
(8) Izumi Y, Tsuji I, Ohkubo T, Kuwahara A, Nishino Y, Hisamichi S. Impact of smoking habit on medical care use and its costs: a prospective observation of National Health Insurance beneficiaries in Japan. Int J Epidemiol. 2001;30: 616–621.
(9) Manning WG, Keeler EB, Newhouse JP, Sloss EM, Wasserman J. The taxes of sin. Do smokers and drinkers pay their way? JAMA. 1989;261:1604–1609.
(10) Hodgson TA. Cigarette smoking and lifetime medical expenditures. Milbank Q. 1992;70:81–125.
(11) MacKenzie TD, Bartecchi CE, Schrier RW. The human costs of tobacco use (2). New Engl J Med. 1994;330:975–980.
(12) Rasmussen SR, Prescott E, Sorensen TI, Sogaard J. The total lifetime costs of smoking. Eur J Public Health. 2004;14:95–100.
(13) Leu RE, Schaub T. Does smoking increase medical care expenditure? Soc Sci Med. 1983;17:1907–1914.
(14) Lippiatt BC. Measuring medical cost and life expectancy impacts of changes in cigarette sales. Prev Med. 1990;19: 515–532.
(15) Barendregt JJ, Bonneux L, van der Maas PJ. The health care costs of smoking. New Engl J Med. 1997;337:1052–1057.
(16) Tsuji I, Nishino Y, Ohkubo T, Kuwahara A, Ogawa K, Watanabe Y, et al. A prospective cohort study on National Health Insurance beneficiariesin Ohsaki, Miyagi Prefecture, Japan: study design, profiles of the subjects and medical cost during the first year. J Epidemiol. 1998;8:258–263.
(17) Murakami Y, Ueshima H, Okamura T, Kadowaki T, Hozawa A, Kita Y, et al. Life expectancy among Japanese of different smoking status in Japan: NIPPON DATA80. J Epidemiol. 2007;17:31–37.

事例で学ぶ

禁煙治療のためのカウンセリングテクニック

【エキスパート編】

著 谷口千枝　編集 田中英夫



看護の科学社