## Letters

## Weight Gain After Smoking Cessation and Cardiovascular Events in Young Adults

Although the adverse health effects of cigarette smoking have been publicized extensively (1), weight gain following smoking cessation remains a major concern for current smokers due to its cosmetic effect, especially in young adults. Association of post-cessation weight gain with cardiovascular disease (CVD) is controversial because weight gain is a predictor of increased CVD risk along with other lifestyle-associated risk factors (2). Furthermore, health professionals in the primary care have been constantly challenged by their patients on the potential disadvantages of weight gain attributable to cessation of smoking.

We abstracted data from a nationwide cohort of 2,044,226 men and women between the ages of 20 and 39 years free of CVD who had received 2 consecutive biennial health examinations through the National Health Insurance Service (NHIS) between 2002 and 2005. The institutional review board at the Seoul National University Hospital and NHIS approved our study (Seoul National University Hospital Institutional Review Board No: 1703-039-836/ NHIS-2017-1-143). Patient informed consent was waived because the NHIS database contains anonymous clinical data. We categorized the participants as continual smokers, quitters (further stratified by weight gain in Table 1), and never smokers according to the responses and body weight measurement data. The follow-up period began on January 1, 2006, and extended through December 31, 2015. Each participant was censored at the time of CVD events or deaths from CVD or other causes, whichever occurred first. We used Cox proportional hazards model to calculate hazard ratio and 95% confidence interval [CI] for coronary heart disease (CHD) and stroke among quitters and never smokers using continual smokers as a reference (for adjustment variables, see footnotes in Table 1). For secondary analysis, we compared the risk for CVD events

within the subpopulation of quitters using quitters with no weight gain as a reference.

The average weight gain was higher in quitters (1.92 kg) compared with continual smokers (0.94 kg). The 2 groups were predominantly male and had a similar prevalence of hypertension (2017 American College of Cardiology/American Heart Association guideline), but the proportion of diabetes and hyperlipidemia was higher in quitters. Among 2,044,226 young adults included in our cohort, there were 21,934 cases of CHD and 10,591 cases of stroke during 10 years of follow-up. After adjusting for potential confounders, the hazard ratio for CHD was 0.82 (95% CI: 0.78 to 0.87) in all quitters, 0.71 (95% CI: 0.62 to 0.83) in quitters with weight gain of more than 5.0 kg, 0.71 (95% CI: 0.64 to 0.79) in quitters with weight gain of 2.1 kg to 5.0 kg, 0.79 (95% CI: 0.70 to 0.88) in quitters with weight gain of 0.1 kg to 2.0 kg, and 0.90 (95% CI: 0.83 to 0.98) in quitters with no weight gain as compared with continual smokers. Compared with quitters with no weight gain, risk estimates for quitters with weight gain showed a protective association (Table 1). In addition, analyses for stroke showed similar results.

Young adults who had quit smoking had a lower risk of CVD as compared with those who continued to smoke, despite the severity of weight gain following smoking cessation. In addition, our data showed that quitters who gained body weight did not have an increased risk for CHD compared with those with less weight gain or no weight gain. Although we adjusted for comorbidity in our analyses, quitters with less weight gain or no weight gain may in part include those who lost weight due to pre-existing illness that attenuated the positive health effect of smoking cessation. Although weight gain is associated with an increase in CVD risk (3), our analyses showed that weight gain attributable to smoking cessation does not adversely affect the overall cardiovascular health benefits of successfully quitting smoking in young adults. Our findings were consistent with the results from the Nurses' Health Study, Health Professionals Follow-Up Study, and Framingham Offspring Cohort (1,4), but participants of these studies were mostly middle-aged Caucasians, whereas our analytic cohort was limited to young Asian adults.

	No.	Events/ Person-Years	Age, Sex Adjusted Model	Multivariable Adjusted Model*
Total population				
Continual smokers	765,094	11,777/7,555,540	1.00 (reference)	1.00 (reference)
All quitters	110,833	1,498/1,095,990	0.93 (0.89-0.98)	0.82 (0.78-0.87)
Quitters by weight gain				
Weight gain >5.0 kg	17,132	202/169,611	0.82 (0.71-0.94)	0.71 (0.62-0.83)
Weight gain 2.1-5.0 kg	29,447	358/291,544	0.76 (0.68-0.84)	0.71 (0.64-0.79)
Weight gain 0.1-2.0 kg	26,225	347/259,420	0.79 (0.71-0.88)	0.79 (0.70-0.88)
No weight gain	38,029	591/375,415	0.91 (0.84-0.99)	0.90 (0.83-0.98)
Never smokers	1,168,299	8,659/11,606,227	0.67 (0.65-0.70)	0.65 (0.62-0.67)
Quitter population				
Weight gain $>$ 5.0 kg	17,132	78/170,443	1.01 (0.87-1.12)	0.86 (0.73-1.00)
Weight gain 2.0-5.0 kg	29,447	128/292,925	0.94 (0.83-1.06)	0.89 (0.79-0.99)
Weight gain 0.1-2.0 kg	26,225	135/260,674	0.93 (0.83-1.04)	0.92 (0.82-1.03)
No weight gain	38,029	242/377,581	1.00 (reference)	1.00 (reference)

Values are hazard ratio (95% confidence interval). This population is based on medical claims records for coronary heart disease (International Classification of Diseases-10th Revision [ICD-10] codes: I20-I25) with at least 2 days of hospitalization. \*Hazard ratio and 95% confidence intervals derived from Cox proportional hazards model adjusted for age, sex (men and women), insurance premium (proxy for income status), residential area (proxy for urbanization level), alcohol consumption, physical activity, fasting serum glucose, body mass index, blood pressure, total cholesterol level, comorbidity (Charlson Comorbidity Index), and family history of cardiovascular disease.

Limitations of our study include using a selfreported questionnaire for assessing change in smoking behavior and not being able to follow up quitters who might have relapsed over time. The results of our study imply the need for promoting smoking cessation in young adults for CVD prevention, despite concern over weight gain. However, clinical intervention for those who gain substantial body weight after smoking cessation might be necessarily to sustain cardiovascular health benefit of smoking cessation.

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## Temporal Changes in Body Fat Distribution and Hypertension



Excess visceral adipose tissue (VAT) is associated with cardiovascular disease and associated risk factors, whereas subcutaneous abdominal adipose tissue (SAT) is relatively metabolically inert in obesity (1). Conversely, lower body subcutaneous fat (LBF) is associated with lower risk (1). VAT had a graded association with incident hypertension in the DHS study (Dallas Heart Study) independent of traditional risk factors (2). Gains of VAT and SAT were associated with incident hypertension in a predominantly white cohort (3). However, effects of changes in adiposity over time on hypertension are