syndrome returns to the level of nonsmokers after 15–20 years of smoking cessation.

Smokers often gain weight after they have quit smoking (4,6). However, which component of body fat (VFA, SFA, or both) increases after smoking cessation and how these changes influence the clustering of metabolic risk factors remain uncertain. In the present study, both the VFA and the SFA were larger in ex-smokers than in current smokers, as was the odds ratio of metabolic syndrome. The increased odds ratio of metabolic syndrome among ex-smokers for ≤14 years decreased by 35% to 55.6 % after adjustment for VFA, whereas no appreciable change was seen after adjustment for SFA, suggesting that VFA has an important contribution to the increased prevalence of metabolic syndrome after smoking cessation.

Regarding glucose metabolism, the odds ratios of hyperglycemia were higher among ex-smokers with <15 years of smoking cessation than among current smokers. The increased odds ratios among the ex-smokers (≤14 years of smoking cessation) were decreased by 15.9% to 18.2% after adjustment for VFA but remained basically unchanged after adjustment for SFA. This finding suggests that the increased prevalence of hyperglycemia after smoking cessation might only partly contribute to the increase in VFA and that hyperglycemia among ex-smokers (≤14 years of smoking cessation) might be caused by mechanisms other than those associated with an increased VFA. For example, smoking might directly increase insulin resistance (9), and this adverse effect of smoking on glucose metabolism might persist for several years after smoking cessation.

Current smokers had a higher odds ratio of having high triglyceride levels despite their relatively lower BMI or VFA, compared with the never-smoking group. This finding suggests that smoking had a strong influence on lipid metabolism that extended beyond its weight-reducing effect. The increased odds ratios for high triglyceride levels among ex-smokers with ≤4 years of smoking cessation was decreased by 38.5% after adjustment for VFA but was unchanged after adjustment for SFA. This finding suggests that an increase in VFA partially accounts for the increase in triglyceride levels after smoking cessation. The association of smoking with HDL cholesterol was similar to that for triglycerides. One exception is that the odds ratio of low HDL cholesterol was increased among current smokers, but not among ex-smokers despite their relatively higher VFA. This finding suggests that the favorable effect of smoking cessation on HDL cholesterol is much greater than the adverse effect of an increase in VFA following smoking cessation.

Current smokers had a significantly lower odds ratio of having high blood pressure, compared with never smokers, whereas the odds ratios among ex-smokers were similar to that among never smokers. Blood pressure is known to increase after smoking (14). However, as indicated by the present report and another epidemiologic study (15), smoking might have a blood pressure lowering effect over the long term. The mechanism whereby smoking decreases blood pressure is not clear.

Because development of coronary heart disease were predictive of smoking cessation (16), the inverse causality of the relationship between smoking cessation and metabolic syndrome may happen. To examine this point, we analyzed the data excluding the subjects currently receiving treatment for hyperlipidemia, hypertension, or diabetes, however, the results shown in Table 3 were materially unchanged.

The present study has several strengths and limitations. As one of its strengths, we directly assessed abdominal fat accumulation using CT scanning. This allowed the role of fat deposition in the development of metabolic syndrome and its components after smoking cessation to be examined more closely. Second, the sample size of our study was sufficiently large (>5,500 subjects). Third, we adjusted for alcohol drinking and physical activity, which might confound the association between smoking status and metabolic risk factors. Our study has some limitations. First, only men were analyzed. The prevalence of smoking among men and among women is quite different in Japan (39.4% in men, 11.0% in women) (17), and the prevalence of being overweight (>25 kg/m^2) also differs between sexes (30.4% in men, 20.2% in women) (17). Thus, the present results may not be applicable to women. Second, the present study had a cross-sectional design, and changes in the metabolic risk profile during the course of smoking cessation were not monitored. Because of the possibility of inverse causality (i.e., people who developed cardiovascular diseases may tend to quit smoking), we also analyzed data excluding subjects currently receiving medication for hyperlipidemia, hypertension, or diabetes, and confirmed the same results. Therefore, it was unlikely that the inverse causality strongly biased the relationships of smoking status with the metabolic syndrome and its components. A longitudinal study is needed to confirm the present findings. Third, we did not search the dietary intake of our participants, so we could not examine whether increased calorie intake and greater fat intake are independently associated with increased VFA and grew worse in metabolic risk factors.

Smoking cessation was associated with an increased prevalence of metabolic syndrome and its components, which could be accounted for, at least in part, by an increase in VFA but not in SFA in men. After 15 years of smoking cessation, the prevalence of metabolic syndrome returned to the level of nonsmokers. Advising individuals who try to quit smoking to adopt healthy lifestyles, including regular physical activity and a low calorie diet, should be considered to minimize the increase in VFA after smoking cessation.

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DISCLOSURE

The authors declared no conflict of interest.

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