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Editorial

Weight Gain After Smoking Cessation and Atherosclerotic Low-Density Lipoprotein Marker

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Smoking is one of the major risk factors for atherosclerosis and develops coronary artery disease, cerebrovascular disease, and peripheral artery disease[1]. It adversely affects the platelet and vascular endothelial function, inflammation, and oxidative stress[2]. Moreover, it increases the incidence of atherogenic lipid profile (higher levels of triglycerides and low-density lipoprotein cholesterol (LDL-C); and lowers the levels of high-density lipoprotein cholesterol (HDL-C))[3]; insulin resistance, including type 2 diabetes[4]; and metabolic syndrome[5]. Therefore, smoking cessation (SC) is the most important matter for the prevention and treatment of atherosclerotic cardiovascular diseases (CVD). On the other hand, SC is often accompanied by weight gain. Recent meta-analysis reported that SC is associated with a mean increase of 4−5 kg in body weight after 12 months of abstinence, and most weight gain occurs within 3 months of quitting[6]. Weight gain itself is also a risk factor for CVD. SC causes an improvement in insulin sensitivity and an increase in HDL-C, whereas obesity after SC might paradoxically contribute to insulin resistance and atherogenic lipid profile[2, 7]. Therefore, the relationship between SC and weight gain could remain to be a highly relevant subject for further research for preventing and treating CVD.

The α1-antitrypsin−low-density lipoprotein complex (AT-LDL) and serum amyloid A-LDL complex (SAA-LDL) are oxidatively modified LDL complexes and are closely associated with both smoking and obesity, which induce oxidative stress and inflammation[8, 9]. One-year time course changes of these two markers and other serum metabolic and inflammatory parameters, as well as body mass index (BMI) and waist circumference, after SC are evaluated in the present study[10]. The authors show that the serum levels of AT-LDL and SAA-LDL were unchanged from baseline to 3 months after SC but these levels significantly decreased from 3 months to 1 year after SC. Although abdominal obesity progressively worsened after SC, the beneficial effect of non-smoking clearly overcomes potential vascular risks by cessation-associated obesity at 1 year after SC. The authors previously reported a significant decrease in serum AT-LDL values among patients with a BMI increase smaller than the median at 3 months after SC[11]. However, large weight gain after SC perturbs the decrease in AT-LDL at 3 months after SC.

We consider that the reduced post-cessation weight gain can bring in earlier beneficial effects of SC for oxidatively modified LDL complexes. Furthermore, post-cessation weight gain is indicated to be an essential reason why smokers, especially women, fail to initiate SC or relapse after initiating SC. It is hereafter worth considering for SC programs and therapies, including pharmacological treatment, designed for reducing post-cessation weight gain.

Conflicts of Interest

None.

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