Smoking and obesity are both more common in persons with severe mental illness (SMI) than in the general population, occurring in 50%–80% and 45%–55%, respectively, of those with SMI. Importantly, both smoking and obesity contribute to the increased mortality of persons with SMI, which is 2 to 3 times higher than that of the overall United States population and primarily due to cardiovascular disease (CVD). Thus, both smoking cessation and weight management are crucial to enhancing the health of people with SMI.

Weight gain after smoking, an important complication of tobacco abstinence, does not alter the reduced risk of CVD from stopping smoking. Until now, however, this relationship has not been explored in persons with SMI. In this month's issue, Thorndike and colleagues provide important findings that postcessation weight gain does not decrease the cardiovascular benefits of tobacco abstinence among people with SMI. The authors evaluated a subgroup of 65 outpatient smokers with schizophrenia, schizoaffective disorder, or bipolar disorder who completed a 1-year randomized controlled trial of varenicline for maintenance of abstinence from smoking. Participants in the abstinent group had a greater weight gain and a greater decrease in 10-year Framingham general CVD risk scores than those in the nonabstinent group, despite high prevalences of obesity (mean body mass index [BMI] = 31 mg/kg), diabetes (31%), dyslipidemia (55%), and hypertension (34%). In other words, despite being associated with significant weight gain, smoking cessation still decreased 10-year CVD risk. These very encouraging findings suggest that people with SMI who stop smoking will reduce their risk for cardiovascular events despite gaining weight and having high baseline levels of metabolic dysregulation. However, a number of unresolved issues remain.

First, these findings cannot be generalized to periods of tobacco abstinence beyond 1 year. Thorndike and colleagues note that postcessation weight gain in people with SMI may have a more severe trajectory over time as compared to that in people without SMI. Indeed, postcessation weight gain in their study sample continued throughout the 12 months of abstinence. An important unresolved question, therefore, is whether there is a point at which continued postcessation weight gain in those with SMI might attenuate the beneficial effects of quitting smoking and actually increase CVD risk. For example, in a 3-year outcome study of 914 people who tried to stop smoking, quitting smoking was associated with an increased risk of diabetes and impaired fasting glucose.

Another issue is that the Framingham model predicts only cardiovascular events, and people with SMI have elevated mortality from other general medical disorders and suicide. Smoking cessation might reduce cardiovascular risk, but weight gain could increase the mortality risk from other disorders related to obesity (eg, diabetes, dyslipidemia, and metabolic syndrome). Thus, metabolic syndrome (of which abdominal obesity, diabetes, and dyslipidemia are components) is associated with a 2- to 3-fold increase in cardiovascular mortality and a 2-fold increase in all-cause mortality. Moreover, the presence of obesity in persons with SMI is associated with a more severe course of the mental disorder, including higher rates of suicide attempts. This has been hypothesized to be due in part to the adverse effects that obesity has on the central nervous system.

Additionally, it is unknown how well the 10-year Framingham risk score predicts cardiovascular events among those with SMI, as people with SMI were excluded from studies that led to development of the Framingham risk score equations. In a recent study of cardiovascular risk models for people with SMI in which 38,824 people with SMI were followed a median of 5.6 years, the PRIMROSE lipid model and the PRIMROSE BMI model (the latter to be used when laboratory values are not available) were each superior to the Framingham model for predicting cardiovascular events. Unlike the Framingham model, the PRIMROSE models include variables for psychiatric diagnosis, psychotropic medications at baseline, harmful use of alcohol, and a social deprivation score. Thus, psychiatric diagnosis, psychiatric medications, alcohol use, and social deprivation are important to consider in the prediction of CVD events among those with SMI. This leads to the question of how to best treat the person with SMI who has postcessation weight gain, especially if he or she is obese. In a meta-analysis of treatments for postcessation weight gain, dexfenfluramine, phenylpropanolamine, and naltrexone were found to limit weight gain shortly after stopping smoking, but not at 6 or 12 months. Also, dexfenfluramine and phenylpropanolamine have been removed from the market because of safety concerns. Exercise did not mitigate weight gain at end of treatment but did so at 12 months. Importantly, weight

See article by Thorndike et al

Commentary

The Dual Epidemic of Tobacco Dependence and Obesity Among Those With Severe Mental Illness

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