

Association between Cigarette Smoking and New-Onset Diabetes Mellitus in 78,212 Koreans Using Self-Reported Questionnaire and Urine Cotinine (*Diabetes Metab J* 2020;44:426-35)

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The association between incidence of diabetes and cigarette smoking has been extensively examined, and there is a large amount of evidence indicating an increased risk for type 2 diabetes mellitus (T2DM) in smokers. In 2007, a meta-analysis showed that the association between smoking and incidence of T2DM was dose-dependent [1]. Cigarette smoking has also been found to lead to an increased risk of cardiovascular complications in diabetic patients [2]. Various side-effects of smoking are responsible for the link between diabetes; cigarette smoking causes visceral fat accumulation and pancreatic β -cell inflammation [3,4] and is associated with vascular damage, endothelial dysfunction, and activation of a blood-clotting cascade [5]. A recent study in rats also found that a specific protein in the brain (habenular TCF7L2) may be associated with the link between smoking and diabetes. The study also found that there may be a link between diabetes and nicotine addiction, as nicotine overrides the communication pathway between the brain and pancreas, leading to high glucose level [6].

In the article entitled, "Association between cigarette smoking and new-onset diabetes mellitus in 78,212 Koreans using self-reported questionnaire and urine cotinine," Kim et al. [7] aimed to elucidate the association between cigarette smoking and new-onset diabetes mellitus using two smoking status classification systems: a self-reported questionnaire and urine

cotinine in a cohort without baseline T2DM. The primary strength of this study is that it is the first and largest longitudinal study to assess the association between smoking and T2DM risk in an Asian population using two smoking status classification systems. Because simultaneous assessments of cotinine-verified smoking status and surveys of self-reported smoking status were performed, misclassification of smoking status was minimized. Based on the findings of this study, clinicians should emphasize the need for and importance of smoking cessation in patients with prediabetes and diabetes.

Despite its strengths, there are several issues not addressed in this study. First, the authors tried to adjust for variable clinical factors; however, a familial history of T2DM and medication history were not considered. Though this study is population based, clinical factors such as a familial history of T2DM and medication history are known to affect T2DM risk and new-onset development and should be included. This study also reported that self-classified former smokers did not have an increased risk of new-onset T2DM. Because weight gain after smoking cessation may have affected the results, it is recommended that the authors consider weight change as a factor in their study. In addition, as the authors mentioned, urine cotinine level may increase in secondhand smokers and ex-smokers. While urine cotinine level is used in many public

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health centers to evaluate smoking status, it is difficult to determine the cut-off value to distinguish between smokers and non-smokers when various values that range between 50 and 500 ng/mL are reported in systematic review [8].

In conclusion, this large, observational study demonstrates that smoking is significantly associated with an increased risk of new-onset T2DM in the Korean population without baseline T2DM. Because smoking is a modifiable risk factor for diabetes, smoking cessation should be strongly recommended to patients. Additional prospective studies should be conducted to further elucidate the association between cigarette smoking and new-onset T2DM in the Korean population.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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