Letter

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The Association between Pulmonary Functions and Incident Diabetes: Longitudinal Analysis from the Ansung Cohort in Korea (*Diabetes Metab J* 2020;44: 699-710)

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Diabetes is a chronic systematic metabolic disease that effects multiple organs and systems, including the lungs [1]. The pulmonary system has rich vascularity and abundant connective tissue [2]. Long-standing hyperglycemia can lead to microvascular damage, non-enzymatic glycation, and proliferation of extracellular connective tissue, which result in declining lung function [3,4]. Oxidative stress, endothelial micro-injuries, platelet activation, and inflammation also can affect diabetes-related lung dysfunction [5,6].

Several studies have suggested that impaired pulmonary function can predict the development of diabetes or insulin resistance [7-9]. Lazarus et al. [10] reported that forced vital capacity (FVC), forced expiratory volume in 1 second (FEV_1), and maximal mid-expiratory flow rate were associated with insulin resistance over a period of 20.9 years. Identifying individuals who are at risk of developing diabetes may allow for earlier precision interventions that would manage or prevent diabetes.

In the article entitled "The association between pulmonary functions and incident diabetes: longitudinal analysis from the Ansung cohort in Korea," Choi et al. [11] evaluated the potential role of reduced pulmonary function as a risk factor for incident diabetes in Koreans. The authors clearly showed that FVC and FEV_1 were independent risk factors for diabetes in Koreans aged 40 to 60 years. They proposed several potential possibilities, although determining the exact biologic mechanism underlying these findings was not possible. They suggested that pulmonary factors were possible risk factors for insulin resistance and diabetes. Considering the ethnic differences in diabetes pathogenesis, this study is highly relevant to Asians with diabetes. The strength of this study is that it was a prospective community-based cohort study of pulmonary function as a risk factor for incident diabetes using 10-year follow-up data and assessing diabetes incidence using both oral glucose tolerance tests and glycosylated hemoglobin levels in order to avoid misclassification of diabetes.

In my opinion, these finding may reflect early pulmonary functional change in the course of glucose intolerance before diabetes appears. Pulmonary function may be a marker reflecting subclinical glucose intolerance status leading to lung dysfunction, and not risk factor for developing diabetes. Although the authors sought to adjust for all confounding variables, this issue remains a concern. Extending this study to longer term follow-up and other age groups would also be useful. Lastly, evaluation of the association with other chronic diabetes complications including cardiovascular disease is needed. This study's findings appear especially relevant during the current coronavirus disease 2019 pandemic. I hope the authors further explore the association between lung dysfunction and diabetes.

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CONFLICTS OF INTEREST

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

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