COMMENTS AND RESPONSES

Progression From Newly Acquired Impaired Fasting Glucose to Type 2 Diabetes

Response to Nichols et al.

We read with interest the report by Nichols et al. (1) on progression from impaired fasting glucose (IFG) to type 2 diabetes among 5,452 members of the Kaiser Permanente Northwest HMO. Their analysis includes a very thorough assessment of the impact of the new American Diabetes Association (ADA) criteria for IFG on future risk of diabetes. They conclude that the older criteria for IFG (110–125 mg/dl) are more predictive of future diabetes. The authors suggest that their study is the first to report diabetes incidence in routine clinical practice using the new criteria for IFG.

In 1998, shortly after the Expert Committee of the ADA recommended that the diagnostic cut point for diabetes be changed from 140 to 126 mg/dl, using the Rochester Epidemiology Project combined with the Mayo Laboratory database, we assembled a cohort of adult nondiabetic Olmsted County residents (2). We reported on the development of diabetes in this cohort over a median follow-up of 9 years. We showed that the baseline level of fasting plasma glucose (FPG) is a major predictor of an individual's future risk of developing diabetes. We estimated that adoption of the new ADA criteria would lead to recognition of diabetes ~7 years earlier.

There are a number of similarities between our study and that of Nichols et al. (1). We both assembled cohorts of nondiabetic individuals from laboratory datasets used for routine clinical practice. Both studies showed that the baseline level of FPG was a strong predictor of the future risk of diabetes and that progression occurred more rapidly from higher baseline levels of FPG. Both studies showed that while risk was greater among individuals with IFG, a sizeable number of new cases of diabetes occur among individuals with "normal" FPG at baseline (384 of 793 cases of diabetes in our study and 201 of 614 in the Nichols et al. study).

The major differences between the two reports are *1*) the availability of nonglucose variables in the Nichols et al. report and their ability to factor these into risk estimation and *2*) our analysis of those nondiabetic individuals with baseline FPG <100 mg/dl. In this subgroup, we demonstrated a clear gradation of risk similar to that observed in individuals with elevated FPG at baseline. We would be interested to know if this observation is supported by data from the Nichols et al. study.

Taken together, we feel that these two reports highlight the utility of routine clinical information in elucidating the natural history and informing the debate on screening for type 2 diabetes. It remains to be established at what level of FPG the altered homeostasis of the prediabetic state begins, but it seems likely that this is well within the currently accepted normal range for glucose.

SEAN F. DINNEEN, MD[1]
ROBERT A. RIZZA, MD[2]

From the [1]Department of Medicine, National University of Ireland, Galway, Ireland; and the [2]Division of Endocrinology, Metabolism and Nutrition, Mayo Clinic, Rochester, Minnesota.

Address correspondence to Sean F. Dinneen, MD, Department of Medicine, National University of Ireland, Newcastle Road, Galway, Ireland. E-mail: sean.dinneen@nuigalway.ie.

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References

1. Nichols GA, Hillier TA, Brown JB: Progression from newly acquired impaired fasting glucose to type 2 diabetes. *Diabetes Care* 30:228–233, 2007
2. Dinneen SF, Maldonado D, Leibson CL, Klee GG, Li H, Melton LJ, Rizza RA: Effects of changing diagnostic criteria on the risk of developing diabetes. *Diabetes Care* 21:1408–1413, 2007