

# Effects of Changing Diagnostic Criteria on the Risk of Developing Diabetes

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**OBJECTIVE** — The American Diabetes Association (ADA) has recommended that the fasting plasma glucose (FPG) level used to diagnose diabetes be changed from 7.8 mmol/l (the level recommended by the National Diabetes Data Group [NDDG] in 1979) to 7.0 mmol/l. We examined the impact of this change on rates of progression to overt diabetes from different levels of FPG.

**RESEARCH DESIGN AND METHODS** — Using the laboratory database of Mayo Clinic, we assembled a cohort of 8,098 nondiabetic Olmsted County residents 40 years of age or older on 1 July 1983. Subjects were followed for a median of 9 years.

**RESULTS** — Among 7,567 individuals with follow-up FPG data, 778 (10.3%) progressed to ADA diabetes and 513 (6.8%;  $P < 0.0001$ ) progressed to NDDG diabetes. The risk of developing ADA diabetes was 7, 19, and 39% for individuals with initial FPG values in the ranges of <5.6, 5.6–6.0, and 6.1–6.9 mmol/l, respectively. For progression to NDDG diabetes, the respective risks were 3, 11, and 25%. A clear gradient of risk was observed within the “normal” range of FPG (<5.6 mmol/l). Among the 793 individuals who developed ADA diabetes, 222 (29%) developed NDDG diabetes simultaneously and 291 (37%) developed NDDG diabetes later. In all FPG subgroups, progression to ADA diabetes occurred ~7 years sooner than progression to NDDG diabetes.

**CONCLUSIONS** — The baseline level of FPG is a major predictor of an individual's risk of developing diabetes. The proposed change in the diagnostic criteria for diabetes will lead to earlier diagnosis among individuals who are destined to develop the disease.

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In 1979, the National Diabetes Data Group (NDDG) established criteria for the diagnosis of diabetes (1). These criteria were based on a level of fasting plasma glucose (FPG) of  $\geq 7.8$  mmol/l and/or a 2-h oral glucose tolerance test (OGTT) plasma glucose level of  $\geq 11.1$  mmol/l on more than one occasion. Two problems arise with the NDDG criteria. First, outside of pregnancy, OGTTs are seldom used in routine clinical

practice. Second, data from epidemiological studies suggest that the level of FPG associated with an increased risk of developing microvascular complications in diabetes is closer to 7.0 than to 7.8 mmol/l (2,3). Furthermore, a FPG cut point of 7.0 mmol/l has a sensitivity for diagnosing diabetes similar to a 2-h value of 11.1 mmol/l (2–4). These considerations led the American Diabetes Association (ADA) to recommend that the

FPG diagnostic cut point be changed from 7.8 to 7.0 mmol/l (5).

In a recent report using data from the Third National Health and Nutrition Examination Survey (NHANES III), Harris et al. commented on the impact of this change in the diagnostic criteria on the prevalence of diagnosed and undiagnosed diabetes in the U.S. population (6). The impact of the new diagnostic criteria on the incidence of diabetes in the community has yet to be determined. Although longitudinal studies have demonstrated that an individual's risk of developing diabetes is determined, to a large degree, by the baseline glucose level, the majority of these studies have used serial OGTT data to assess risk and to diagnose diabetes (7–14).

Population-based research is facilitated at Mayo Clinic by virtue of its unique location as the major source of medical care for residents of Olmsted County, Minnesota, with a population of ~100,000 individuals. The Rochester Epidemiology Project established a medical-records linkage system with other providers of care to local residents to support studies of the incidence and prevalence of many diseases (15), including diabetes (16–18). The Laboratory Information System database at Mayo has preserved on magnetic tape all laboratory results from blood drawn at the Clinic since 1983. These resources have previously been used in Olmsted County residents to study diseases that can be defined based on laboratory criteria alone (19,20).

The aim of the present study was to quantify in community residents the rate of progression from different baseline levels of FPG to overt diabetes using only FPG data to define diabetes (i.e., no OGTT data). In addition, we sought to determine the potential impact of a change in diagnostic criteria for diabetes on rates of progression from nondiabetic to diabetic levels of FPG.

## RESEARCH DESIGN AND METHODS

### Study design

Using the Mayo laboratory database, we assembled a cohort of Olmsted County residents who were 40 years of age or older on

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**Abbreviations:** ADA, American Diabetes Association; FPG, fasting plasma glucose; NDDG, National Diabetes Data Group; NHANES III, Third National Health and Nutrition Examination Survey; OGTT, oral glucose tolerance test.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

**Table 1—Number and percent of Olmsted County, Minnesota, residents by initial FPG who progressed to diabetes using the criteria of the ADA or the FPG criteria of the NDDG**

Subgroup		ADA diabetes	NDDG diabetes	Simultaneous progression
Initial FPG [mmol/l (mg/dl)]	n			
<5.6 (<100)	5,765	384 (6.7)	197 (3.4)	117 (2.0)
5.6–6.0 (100–109)	1,122	208 (18.5)	126 (11.2)	62 (5.5)
6.1–6.9 (110–125)	521	201 (38.6)	129 (24.8)	43 (8.3)
7.0–7.7 (126–139)	159	— (—)	61 (38.4)	— (—)
Total	7,567	793 (10.7)	513 (6.8)	222 (3.0)

Data are n (%). Simultaneous progression refers to individuals who met the criteria for NDDG diabetes at the same time they met the criteria for ADA diabetes.

1 July 1983 and whose initial FPG during the interval of 1 July 1983 to 31 December 1986, was <7.8 mmol/l. By confining the analysis to specific Mayo Clinic procedure codes (2372 and 8476), we were able to exclude venous whole-blood glucose values and inpatient samples. Procedure code 2372 designates a plasma glucose measured as part of a chemistry panel; procedure code 8476 designates a plasma glucose measured separately from a chemistry panel. We also restricted the analysis to samples drawn between 6:00 and 10:00 A.M. to maximize the chances of including true FPG samples.

To identify and exclude individuals with a prior diagnosis of diabetes, we used the Rochester Epidemiology Project database to generate a list of all Olmsted County residents with known diabetes (by NDDG criteria) during the time period 1945–1983. The medical records of a subset of these individuals (the residents of Rochester, Minnesota) had previously been reviewed to confirm the diagnosis of diabetes by NDDG criteria (16–18). As part of the present study, we expanded the medical record review to Olmsted County residents living outside Rochester. In this way, we were able to exclude from our laboratory-derived cohort those Olmsted County residents with a confirmed diagnosis of diabetes before 1 July 1983.

For the remaining nondiabetic individuals, all FPG values in the laboratory database were examined between the date of the initial value and 31 December 1995. Individuals with at least two values  $\geq 7.0$  mmol/l were defined as having met ADA criteria. The date of the first FPG at or above 7.0 was assigned as the date of ADA diagnosis. Of those who met ADA diagnostic criteria, a subset also had at least two values  $\geq 7.8$  mmol/l. These individu-

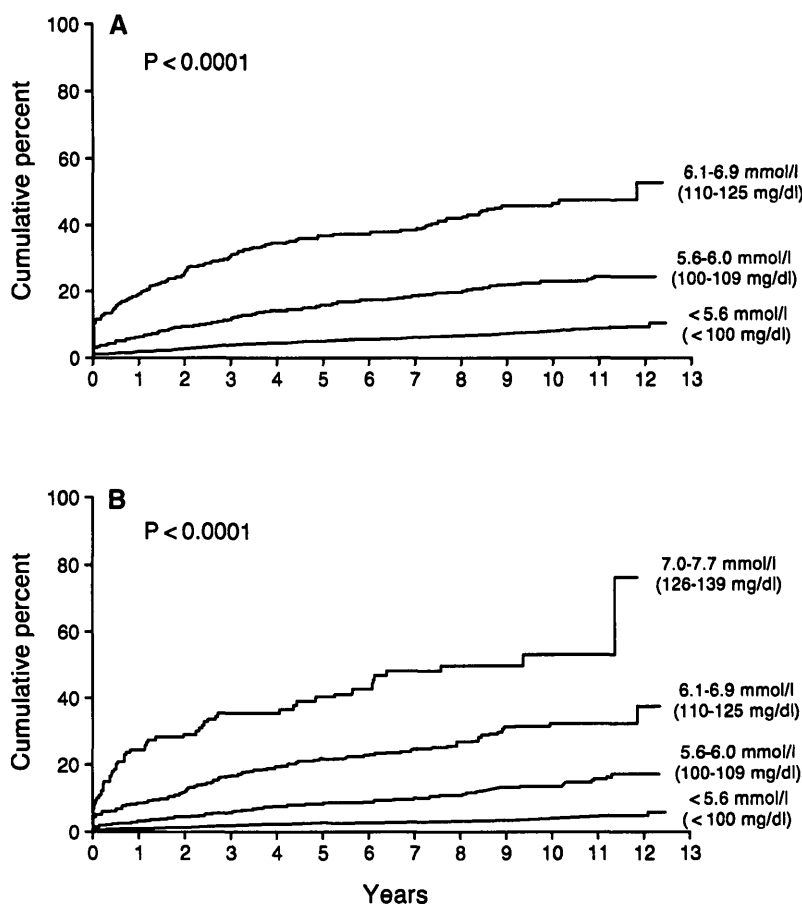
als were defined as having met NDDG criteria and, again, the date of the first FPG at or above 7.8 was assigned as the date of NDDG diagnosis. It is important to note that in the absence of OGTT data, our

working definition of NDDG diabetes was based solely on FPG data. This approach is in keeping with current clinical practice and also consistent with the recommendations of the ADA Expert Committee (5), but it is likely to underestimate the true incidence and prevalence of NDDG diabetes.

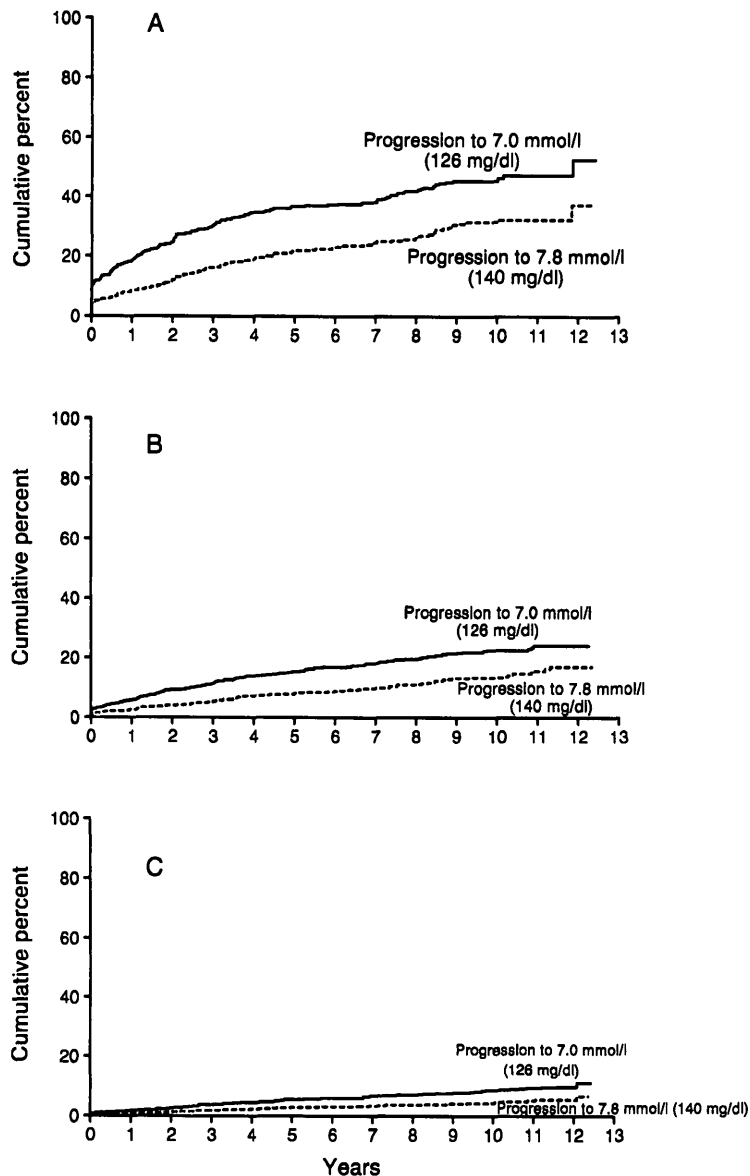
### Statistical analysis

The cohort was divided into 4 subgroups according to the level of initial FPG (<5.6, 5.6–6.0, 6.1–6.9, and 7.0–7.7 mmol/l). Individuals with an initial FPG <5.6 mmol/l (considered the upper limit of normal at our institution) were further subdivided based on quartiles of FPG within this subgroup (<4.7, 4.7–4.9, 5.0–5.1, and 5.2–5.5 mmol/l).

For both ADA and NDDG criteria, analyses were performed comparing the



**Figure 1—Cumulative incidence of diabetes according to the initial FPG level. A: Progression to a first value of 7.0 mmol/l (126 mg/dl) among individuals with two or more FPG values above this cut point (ADA criteria). B: Progression to a first value of 7.8 mmol/l (140 mg/dl) among individuals who had two or more FPG values above this cut point (NDDG criteria). Log-rank tests were used to compare the cumulative incidence rates. These tests indicated that the higher the initial FPG, the greater the probability of developing diabetes.**



**Figure 2**—Cumulative incidence of diabetes according to NDDG (“Progression to 7.8”) and ADA (“Progression to 7.0”) criteria by subgroup based on the initial FPG level. A: Subgroup with initial FPG 6.1–6.9 mmol/l (110–125 mg/dl). B: Subgroup with initial FPG 5.6–6.0 mmol/l (100–109 mg/dl). C: Subgroup with initial FPG <5.6 mmol/l (<100 mg/dl).

subgroups for cumulative incidence of diabetes. Follow-up began with initial FPG on or after 1 July 1983. Those who met one of the diagnostic criteria were assigned an event date as the date of the first value at or above the cut point (i.e., 7.0 or 7.8 mmol/l). People who failed to meet either diagnostic criterion were censored at last FPG value on or before 31 December 1995. The cumulative incidence of diabetes was estimated for both criteria, and subgroups were compared graphically using Kaplan-Meier curves. Tests of significant differences between subgroups were performed using log-rank tests (21). A *P* value of

<0.05 was taken to represent statistical significance.

## RESULTS

### Cohort characteristics

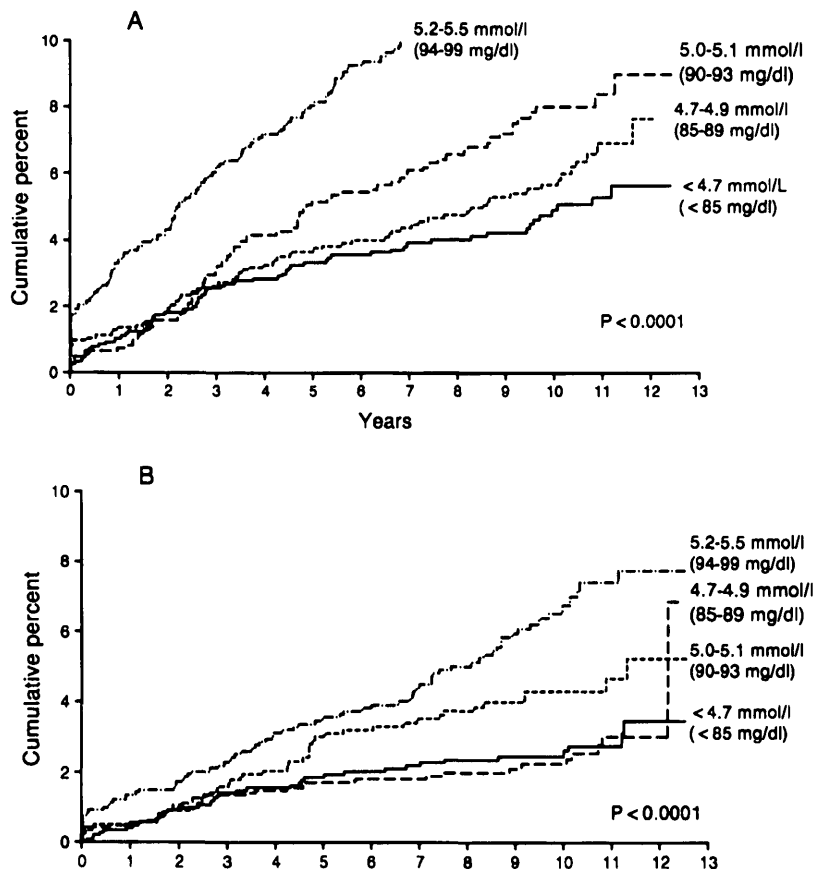
During the 30-month period of cohort assembly, the Mayo laboratory database recorded a total of 118,668 glucose readings among 26,796 Olmsted County residents. Of these, 12,748 were from Olmsted County residents 40 years of age or older on 1 July 1983. Based on census data interpolated to 1985, this latter number indicates that the laboratory-derived data set

captured 39% of the relevant Olmsted County population during the period of cohort assembly. After imposing the restrictions of procedure code, draw time, and exclusion of prior diabetes (see METHODS), the size of the cohort was reduced to 8,098 individuals. The mean ( $\pm$  SD) age of the cohort was  $61 \pm 13$  years, and it was composed of 39% male and 61% female residents. The median duration of follow-up was 9 years, with a range of 0–13 years. After their initial FPG value, 531 patients had no follow-up, and these individuals were excluded from further analysis. Of the plasma glucose measurements used in the study 88% were done as part of a chemistry panel.

### Progression to diabetes

A total of 793 individuals (10.7% of the cohort) progressed to ADA diabetes, and 513 individuals (6.8%) progressed to NDDG diabetes during the period of follow-up. Table 1 shows the number of individuals who progressed to overt diabetes (by either criterion) during the course of the study according to the initial level of FPG. These data are illustrated in Fig. 1, where it is evident that regardless of the criterion used to define diabetes, the initial FPG level was a major determinant of the subsequent risk of developing the disease. The higher an individual's initial FPG, the greater the probability of developing diabetes during follow-up. Of note, although the risk of progression to diabetes was lowest among those with initial FPG <5.6 mmol/l, this large subgroup contributed the greatest number of patients who went on to develop diabetes of any subgroup. Among the 793 individuals who developed ADA diabetes, 222 (29%) developed NDDG diabetes simultaneously, i.e., their first two FPG values >7.0 mmol/l were also >7.8 mmol/l. An additional 291 subjects (37%) developed NDDG diabetes subsequently. Table 1 shows the number and percent of individuals with simultaneous progression according to subgroup.

Figure 2 shows the cumulative incidence of progression to ADA and NDDG diabetes for each of the individual subgroups. The subgroup with an initial FPG between 7.0 and 7.7 mmol/l is not included because it was assumed that many of these individuals would have met the criteria for diagnosis of ADA diabetes before the study had they been given a confirmatory test. In all other subgroups, progression to ADA diabetes occurred



**Figure 3**—Cumulative incidence of diabetes according to the initial FPG level for the subgroup with initial FPG  $<5.6$  mmol/l ( $<100$  mg/dl). The 25th, 50th, and 75th percentiles of initial FPG were used to divide individuals into groups. A: Progression to a first value of  $7.0$  mmol/l ( $126$  mg/dl) among individuals with two or more FPG values above this cut point (ADA criteria). B: Progression to a first value of  $7.8$  mmol/l ( $140$  mg/dl) among individuals who had two or more FPG values above this cut point (NDDG criteria). Log-rank tests were used to compare the cumulative incidence rates. These tests indicated that the risk of progressing to diabetes was greater for individuals in the upper compared with the lower quartiles.

sooner than progression to NDDG diabetes. For instance, among individuals with an initial FPG between  $6.1$  and  $6.9$  (Fig. 2A), almost 40% had progressed to ADA diabetes within 5 years, whereas it took almost 12 years for a similar proportion to progress to NDDG diabetes. Similarly, among individuals with an initial FPG between  $5.6$  and  $6.0$  mmol/l (Fig. 2B), almost 10% had progressed to ADA diabetes by 2 years, whereas it took almost 9 years for a similar proportion to progress to NDDG diabetes.

The above data indicate that any elevation of glucose concentration above the “normal” range is associated with an increased risk of progression to diabetes, regardless of the criteria. Data in Fig. 3 indicate that the same relationship also holds for individuals with glucose concentrations within the so-called “normal”

range. Although the overall risk of progressing to diabetes was low in these individuals, it was still greater ( $P < 0.0001$ ) for individuals in the upper compared with the lower quartiles. This was true for progression both to ADA and to NDDG diabetes.

**CONCLUSIONS**—Our study confirms previous reports (7–14) that the level of FPG is a major determinant of an individual’s subsequent risk of developing diabetes. In addition, our data indicate that the risk of diabetes increases with the degree of elevation of the FPG level, even among individuals with a FPG level within the so-called “normal” range. Our findings are also relevant to the recently proposed change in diagnostic criteria for diabetes. If FPG levels are to be the main means of diagnosing diabetes and if the cut point for diagnosis is to be reduced from  $7.8$  to  $7.0$

mmol/l, then our data indicate that the primary effect will be earlier diagnosis of the disease. This is consistent with the observations of Harris et al. (6) based on NHANES III data that the new diagnostic criteria will reduce the number of individuals in the U.S. population with undiagnosed diabetes.

Previous cohort studies have analyzed the risk of progression from a state of impaired glucose tolerance to overt diabetes (7–14). Pooled data from six of these cohort studies, involving 16,775 person-years of follow-up, showed that the level of FPG, the 2-h post-OGTT glucose, and the baseline BMI were the most important predictors of progression from impaired glucose tolerance to diabetes (22). The FPG data in that report are consistent with our observations and demonstrate an increasing incidence of diabetes going from the lowest to the highest quartile of FPG. Although the 2 h post-OGTT data demonstrated a steeper gradient of risk than the FPG data, this likely related to the fact that in most studies, OGTTs were used both to assess baseline risk and to diagnose diabetes. In our data set, the level of FPG was used exclusively to assess risk and diagnose disease. This approach better reflects current clinical practice, where OGTTs are seldom used except during pregnancy.

The use of electronically stored FPG data to assemble and follow our cohort offered several advantages. We were able to retrospectively assemble a cohort with 63,164 person-years of follow-up. Even confining our analysis to those individuals with fasting hyperglycemia at baseline (FPG  $>5.6$  mmol/l) left us with 15,241 person-years of follow-up, which compares well with the combined cohort from the Edelstein et al. report (22). Another strength of our study related to the selection of clinically relevant cut points to define our subgroups. These were chosen to represent what, in our estimation, equates to mild ( $5.6$ – $6.0$ ), moderate ( $6.1$ – $6.9$ ), and severe ( $7.0$ – $7.7$ ) fasting hyperglycemia. In addition, these cut points conform to the levels of FPG recommended by the expert committee (5). By choosing these cut points, our data should be helpful to clinicians assessing future diabetes risk among patients with characteristics similar to those in our cohort. For example, data in Table 1 indicate that individuals with “impaired fasting glucose” (FPG  $6.1$ – $6.9$ ) have an approximately sixfold higher risk of progressing to

ADA diabetes over the subsequent 9 years compared with individuals with initial FPG <5.6.

The Mayo laboratory uses a cut point of 5.6 mmol/l as the upper limit of normal for FPG, whereas the expert committee of the ADA proposed an upper limit of normal of 6.1 mmol/l (5). In our study, individuals with an initial FPG between 5.6 and 6.0 mmol/l demonstrated an approximately threefold higher risk of progressing to overt diabetes than did individuals with an initial FPG <5.6 mmol/l. This suggests that the choice of 6.1 mmol/l for the upper limit of normal should be reconsidered. Furthermore, in our study, even a FPG level of <5.6 mmol/l was associated with a clear gradient of risk; individuals in the highest quartile had a significantly increased risk of progressing to overt diabetes than individuals in the lowest quartile. Thus, our data indicate that, at least in terms of progressing to future diabetes, the lower an individual's FPG, the better.

As with most epidemiological studies, ours has a number of limitations. Our sample was not a truly random sample, but the distribution of individuals with normal and impaired fasting glucose as well as undiagnosed diabetes (by ADA criteria) in our study closely approximated a recent national sample of the U.S. population age 40 to 74 years (6; data not shown). Ethnicity was not assessed in this study, but nearly 98% of Olmsted County residents 40 years of age and older are Caucasian. For this reason, care must be taken in extrapolating our findings to other ethnic groups. Because the risk of developing diabetes appears, if anything, to be greater in non-Caucasian populations (22), the present data are likely to represent minimal estimates of the risk of progression. The lack of OGTT data in our study means that we may have diagnosed diabetes in some individuals who have normal oral glucose tolerance, and we may have missed individuals in the community with normal FPG levels but diabetic glucose tolerance tests. Although this makes it difficult to compare our data with previous epidemiology studies that have relied primarily on OGTT data, the use of FPG levels reflects current clinical practice and is consistent with the recommendations of the ADA expert committee (5). By crossing our laboratory-derived data set with the existing Rochester Epidemiology Project diabetes incidence and prevalence cohort, we were able to minimize the likelihood of recruiting indi-

viduals with diabetes into our cohort. Our study design was based on a univariate approach to assessing risk of future diabetes in our cohort. We do not know the extent to which other factors (e.g., obesity, sedentary lifestyle, family history) modify this risk. This is an area of interest, particularly in the subgroup with "normal" FPG levels at baseline. Although this subgroup was at low risk of progressing to diabetes, it contributed the largest number of individuals who went on to develop diabetes of all subgroups. This observation is relevant to the planning of population-based diabetes prevention strategies.

In conclusion, our study confirms previous reports of the importance of an elevated FPG level as a predictor of an individual's future risk of developing diabetes. Our data further suggest that use of the new ADA criteria will result in diabetes being diagnosed several years earlier than with the NDDG criteria. Hopefully, this will increase the opportunity to prevent the devastating microvascular and macrovascular complications that lead to so much morbidity in this disease.

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