

# Delayed Diagnosis of Incident Type 2 Diabetes Mellitus in the ARIC Study

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**Objectives:** To estimate delays to physician diagnosis of incident cases of type 2 diabetes mellitus (DM) and to identify predictors of delayed diagnosis.

**Study Design:** The Atherosclerosis Risk in Communities (ARIC) study, an ongoing population-based prospective study of 15 792 middle-aged adults.

**Methods:** The study population comprised 298 adults with incident DM. Exposures were demographic, socioeconomic, health behavior, and clinical risk factors before the onset of type 2 DM. The main outcome was the delay from onset of DM to physician diagnosis.

**Results:** Among 298 ARIC participants with incident type 2 DM at visit 2 of the study, the median delay from onset of DM to physician diagnosis was 2.4 years. More than 7% of incident cases remained undiagnosed for at least 7.5 years after the onset of disease. Compared with individuals with promptly diagnosed incident DM, those with delayed diagnosis were more likely to be obese before the onset of DM ( $P = .003$ ), less likely to have heart disease at baseline ( $P = .02$  for trend), less likely to have seen their physician in the past year ( $P = .005$  for trend), and had a slower rise in fasting hyperglycemia ( $P = .04$ ). Neither demographic characteristics nor study site predicted delayed diagnosis.

**Conclusions:** Even with a de facto screening program, diagnosis of incident type 2 DM in the community is typically delayed for 2 years and sometimes as long as 7 years or more. The associated risk factors suggest deficiencies in organizational processes, physician actions, and patient access or acceptance of the diagnosis.

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The past decade has seen an epidemic increase in the prevalence of type 2 diabetes mellitus (DM) in the United States and worldwide that is fueled by weight gain and aging.<sup>1-8</sup> Early treatment to control blood glucose levels, blood pressure, and hyperlipidemia reduces the risk of vascular complications.<sup>9-14</sup> However, the benefits of early therapy are incompletely realized, in part because of delayed diagnosis.<sup>15-17</sup> A better understanding of delayed diagnosis could guide improvements in screening and treatment programs. Prior literature about delayed diagnosis is scant and is limited to mathematical projections based on group data.<sup>16,17</sup> Therefore, we studied delayed diagnoses in the Atherosclerosis Risk in Communities (ARIC) study<sup>18</sup> of 15 792 middle-aged adults in whom fasting plasma glucose (FPG) levels and self-report of physician diagnosis of DM were deter-

mined at 4 study visits during 9 years. We focused on a subgroup of participants who did not have DM at baseline but who developed incident DM by the third year of follow-up. Our objectives were (1) to quantify delay times and (2) to identify factors that predict longer delays.

## METHODS

### Study Setting

The ARIC study is an ongoing prospective study of 15 792 persons, aged 45 to 64 years at baseline, selected by probability sampling from the following 4 US communities: Washington County, Maryland; Forsyth County, North Carolina; the northwest suburbs of Minneapolis, Minn; and Jackson, Miss. These study protocols were reviewed and approved by the ARIC constituent institutional review boards (The Johns Hopkins University, University of North Carolina, University of Minnesota, and University of Mississippi).

The methods used in the ARIC study have been described in detail elsewhere.<sup>18</sup> This prospective analysis was based on information from the baseline visit (1986-1989) and from 3 follow-up clinic visits scheduled at approximately 3-year intervals.

### Definition of DM

In addition to FPG measurements at each ARIC study visit, staff-administered interviews were conducted to collect information, including health insurance cover-

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age, time since the last physician visit, whether a physician had diagnosed DM, whether the patient had fasted, and current use of diabetes medications. At each ARIC visit, DM was determined to be present if the participant answered yes to one of the following questions: "Have you taken blood sugar-lowering medication in the past 2 weeks?" or "Has a doctor ever told you that you have diabetes?" Participants were also classified as having DM if their FPG level was at least 140 mg/dL ( $\geq 7.7$  mmol/L) or their non-FPG level was at least 200 mg/dL ( $\geq 11.0$  mmol/L).

Participants with DM were classified as being undiagnosed at a given ARIC visit if they met either biochemical criterion for DM but answered no to questions about diagnosis and medication use. Otherwise, they were classified as being diagnosed. We used an FPG level of at least 140 mg/dL ( $\geq 7.7$  mmol/L) to define DM because that was the biochemical criterion recommended by the World Health Organization and by the National Diabetes Data Group during visits 1 through 4 of the ARIC study (1986-1998).<sup>19-22</sup>

**Notification of Physicians**

If participants were found to have abnormal plasma glucose levels, a nonalert letter or an alert letter was sent to the participant and to his or her physician with the laboratory results. If the FPG level was 130 to 199 mg/dL (7.7-11.0 mmol/L), a nonalert letter indicated that the result was abnormal and ought to be reviewed by the physician. If the FPG level was at least 200 mg/dL ( $\geq 11.0$  mmol/L), the alert letter indicated that the result

was abnormal and needed physician attention and follow-up. Both types of letters were sent in the same way, and neither specifically advised participants that they appeared to have DM. Confirmatory testing was left to the discretion of the participants' physicians.

**Study Population**

We applied the following approach to identify individuals who developed incident DM during the first 3 years of follow-up. Of the 15 792 participants enrolled at baseline, we excluded 48 persons who were not African American or European American and 1558 with prevalent DM, leaving 14 186 adults without DM. Of these, 483 met criteria for incident DM (based on plasma glucose level, physician diagnosis, or diabetes therapy) at visit 2 (year 3 of follow-up). After excluding 66 individuals who did not return for visit 3 (the highest proportion was in Jackson, Miss), 22 individuals who had missing data for determining diabetes status, and 97 individuals who no longer met the criteria for DM at visit 3, a total of 298 individuals with incident DM at visit 2 (and confirmed at visit 3) remained for analysis.

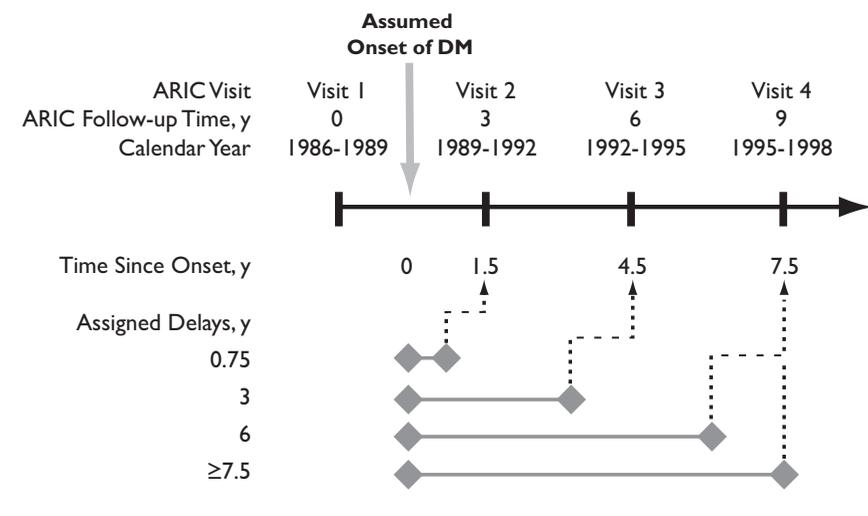
**Assignment of Delayed Time to Physician Diagnosis**

Delayed time to physician diagnosis was defined as the date of diagnosis minus the date of onset of DM, requiring knowledge of both dates. However, in large-scale epidemiological studies such as the ARIC study, this ideal is limited by infrequent visits and by reliance on recall of physician diagnosis. The data structure mandated the use of an algorithm to assign estimated delays to diagnosis using FPG level and survey data from visits at 3-year intervals (Figure 1).

All subjects in this analysis developed incident DM during the 3-year interval between baseline and visit 2. We assumed that the onset of DM occurred at a constant rate between baseline and visit 2, so that the mean time of onset was the midpoint of the interval (ie, 1.5 years before visit 2).

Furthermore, we assumed that the diagnosis of the disease also occurred at a constant rate during the respective interval. Therefore, for those who were already diagnosed at visit 2, we assumed that the diagnosis

**Figure 1.** Assignment of Delay Times



Dotted lines indicate the assigned point at which it is assumed that the diagnosis was made; diamonds with solid lines, the assigned length of the delay from assumed onset to assumed time of physician diagnosis. DM indicates diabetes mellitus; ARIC, Atherosclerosis Risk in Communities study.

occurred, on average, at the midpoint of the interval between the assumed onset of disease (1.5 years before visit 2) and visit 2, which would be 0.75 year after the assumed onset of disease, with a possible range of 0 to 3 years.

For those who were undiagnosed at visit 2 but were diagnosed at visit 3, the mean time of diagnosis was assumed to be at the midpoint of the interval between visit 2 and visit 3 (1.5 years after visit 2). Therefore, their delay time would be 3 years (1.5 years from onset to visit 2 plus 1.5 years from visit 2 to diagnosis), a total delay time of 3 years, with a possible range of 0 to 6 years. For those diagnosed for the first time between visit 3 and visit 4, their delay time would be 6 years, with a possible range of 3 to 9 years. Those who remained undiagnosed at visit 4 had delays to diagnosis of at least 7.5 years, with a possible range of 6 to 9 years or more.

### Assessment of Clinical Characteristics

Information on age, race/ethnicity, sex, education, parental history of DM, and clinical profile was obtained from the baseline home and clinic interviews. Self-reported alcohol consumption was categorized into current drinker and not current drinker. Smoking was categorized into current smoker (within the past 6 months) and not current smoker.

At all visits, physical examinations, including sitting blood pressure and anthropometry, were performed following standard protocols. Hypertension was defined as diastolic blood pressure of at least 90 mm Hg, systolic blood pressure of at least 140 mm Hg, or self-reported use of antihypertensive medication. Obesity was categorized into the following 4 groups based on body mass index (BMI, calculated as weight in kilograms divided by height in meters squared): normal (BMI, <25), overweight (BMI, 25-29.9), class 1 obesity (BMI, 30-34.9), and class 2 obesity ( $\geq 35$ ). Participants were asked to fast for at least 8 hours before morning blood collection,<sup>23,24</sup> and fasting blood was drawn for clinical chemical analyses. A description of the laboratory methods is provided elsewhere.<sup>18</sup>

### Statistical Analysis

The baseline characteristics of the study population (298 adults with incident DM at ARIC visit 2) were summarized as proportions or as means with standard deviations. Two approaches were used to estimate cumulative delay times. First, a Kaplan-Meier graph was plotted based on the residual proportion remaining undiagnosed at each visit. Second, in a subgroup of 189 participants who had self-reported age at diagnosis, we estimated delay time without assuming that all diag-

noses occurred uniformly between visits.

To confirm results from the Kaplan-Meier approach, we also simulated delay times. In our simulation, we generated 2 uniform random variables for each person, one representing the time to disease onset (ie, between the baseline visit and visit 2) and the other representing the time between disease onset and disease diagnosis. These variables were then used to estimate the median delay time. The final median time was the mean of median times derived from 10 simulations. Confidence intervals (CIs) for the estimate were derived using a bootstrap procedure.<sup>25</sup> We performed 2000 bootstrap replications for the median delay time analysis.

Diagnosis was classified as delayed if a participant's physician had not diagnosed DM by ARIC visit 2. Baseline prediabetic characteristics of those who were diagnosed by their physician promptly (ie, before ARIC visit 2) were compared with those of participants who remained undiagnosed at visit 2. Simple and multiple logistic regression models were constructed to determine predictors of delayed diagnosis. To investigate graded responses, we also categorized cases of delayed diagnosis according to the duration of delay (<2 years, 2-5 years, 4-7 years, and  $\geq 7$  years).

The mean FPG values and corresponding 95% CIs at each visit were calculated. The mean FPG values were plotted by visit for each major delay time group. To test the hypothesis that a steeper climb in FPG is associated with earlier diagnosis, the difference between FPG values from 2 consecutive visits was compared between those who were diagnosed before the subsequent visit and those who remained undiagnosed at the subsequent visit. For example, to compare individuals who were diagnosed by visit 4 with those who remained undiagnosed at visit 4, a random-effects model using the generalized estimating equation was fitted to assess the association between categories of delay time to diagnosis and changes in FPG levels between visit 1 and visit 3 of the study. All statistical analyses were performed using the STATA version 7 statistical package (StataCorp LP, College Station, Tex).

## RESULTS

### Baseline Characteristics

Baseline (visit 1) characteristics of the 298 adults who developed type 2 DM between visit 1 and visit 2 and had DM at visit 3 are summarized in **Table 1**. About one third of these individuals were African American, 50% had hypertension, 89% had health insurance, and 76% had seen a physician in the past 12 months. Their mean BMI of 32.1 was squarely in the obese range, and 91% were overweight (BMI, >25).

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**Table 1.** Selected Baseline Characteristics of 298 Atherosclerosis Risk in Communities Study Participants With Incident Type 2 Diabetes Mellitus (DM) at Visit 2 of the Study by Estimated Delay Time of DM Diagnosis\*

Characteristic	Total (N = 298)	Delay Time, y		P
		0-1.5 (Diagnosed by Visit 2) (n = 115)	>1.5 Undiagnosed at Visit 2) (n = 183)	
<b>Demographic</b>				
Age, mean ± SD, y	54.5 ± 5.6	54.8	54.1	.33
African American race/ethnicity	35.9	40.0	33.3	.24
Male sex	46.3	49.0	44.8	.51
Education ≥12 y	70.5	72.2	69.4	.61
Has health insurance	88.9	90.4	88.0	.51
Maternal history of DM	27.5	28.7	26.8	.72
Paternal history of DM	13.1	13.0	13.1	.99
<b>Behavioral</b>				
Saw physician in past 12 mo	75.5	81.0	72.1	.09
Current drinker of alcohol	48.0	43.5	50.8	.22
Current smoker of cigarettes	24.2	27.8	21.9	.24
<b>Clinical</b>				
Hypertension	50.0	49.1	50.5	.81
Coronary heart disease	5.0	9.7	4.4	.08
Myocardial infarction	4.2	8.8	3.8	.08
Body mass index, mean ± SD <sup>†</sup>	32.1 ± 5.7	30.8	32.9	.003
Fasting glucose concentration, mM	6.59 ± 0.69			
	(119 ± 13 mg/dL) <sup>‡</sup>	6.51	6.64	.12
High-density lipoprotein cholesterol concentration, mM	1.14 ± 0.36			
	(44.0 ± 14 mg/dL) <sup>§</sup>	1.17	1.11	.16
Low-density lipoprotein cholesterol concentration, mM	3.62 ± 0.97			
	(140 ± 37 mg/dL) <sup>§</sup>	3.56	3.65	.44
Triglycerides concentration, mM	1.88 ± 0.99			
	(73.0 ± 38 mg/dL) <sup>  </sup>	1.85	1.90	.67

\*Data are given as percentages unless otherwise indicated.

<sup>†</sup>Calculated as weight in kilograms divided by height in meters squared.

<sup>‡</sup>To convert glucose to millimoles per liter, multiply by 0.0555.

<sup>§</sup>To convert cholesterol to millimoles per liter, multiply by 0.0259.

<sup>||</sup>To convert triglycerides to millimoles per liter, multiply by 0.0113.

**Time to Delayed Diagnosis of Incident Type 2 DM**

Overall, the proportions of the total study population remaining undiagnosed were 61% at visit 2, 21% at visit 3, and 7% at visit 4. The Kaplan-Meier approach (Table 2) summarizes the pattern of diagnosis among 298 ARIC participants with incident type 2 DM at visit 2. Of these 298 individuals, 115 had already been diagnosed by their physician before visit 2 (prompt diagnosis). The remaining 183 (61%) of 298 adults with incident DM had not been diagnosed by visit 2 (initial delays). Some of these participants with initial delays also went on to have longer delays to diagnosis. By visit 3, 121 of these 183 undiagnosed participants had been diagnosed by their physicians, while 62 (34%) of 183 remained undiagnosed. By visit 4, 40 of these 62 individuals with undiagnosed

type 2 DM had been diagnosed by their physicians, but 22 (35%) of 62 remained undiagnosed at visit 4, approximately 7.5 years after the onset of type 2 DM. Therefore, more than 7% (22/298) of incident cases remained undiagnosed for at least 7.5 years after the onset of disease.

In the subgroup of 189 participants who had self-reported age at diagnosis, the median delay time was 2.5 years (95% CI, 2.3-2.7 years), and the mean ± SD self-reported delay times corresponding to our estimated categories of delay of 0.75, 3, and 6 years were 2.4 ± 1.5, 3.1 ± 1.3, and 4.3 ± 1.4 years, respectively. The 22 persons who remained undiagnosed after visit 4 (delay of ≥7.5 years) did not self-report an age at diagnosis.

Using a modified Kaplan-Meier approach and joining the midpoints of the steps (Figure 2), we estimated that

**Table 2.** Life Table Proportion of Atherosclerosis Risk in Communities Study Participants With Incident Diabetes Mellitus (DM) Remaining Undiagnosed

Interval of Diagnosis	Time From Onset of DM, y	No. at Start of Interval	No. (%) Diagnosed During Interval	No. (%) Undiagnosed at End of Interval	Fasting Glucose Concentration of Undiagnosed Participants at End of Interval, mean ± SD, mM
Visit 1 to visit 2	0-1.5	298	115 (38.6)	183 (61.4)	9.8 ± 2.8
Visit 2 to visit 3	1.5-4.5	183	121 (66.1)	62 (33.9)	11.2 ± 3.0
Visit 3 to visit 4	4.5-7.5	62	40 (64.5)	22 (35.5)	12.0 ± 5.7
Visit 4	≥7.5	22	—	—	—

the median delay from onset of type 2 DM to physician diagnosis was 2.4 years. To determine the median delay and the 95% CI empirically, we simulated failure times. Under our simulation, the estimated median delay time was 2.4 years (95% CI, 2.0-2.7 years). This simulation-based estimate was validated by the estimate of 2.5 years (95% CI, 2.3-2.7 years) derived from the 189 participants self-reporting the time of DM diagnosis.

**Prediabetic Predictors of Delayed Physician Diagnosis**

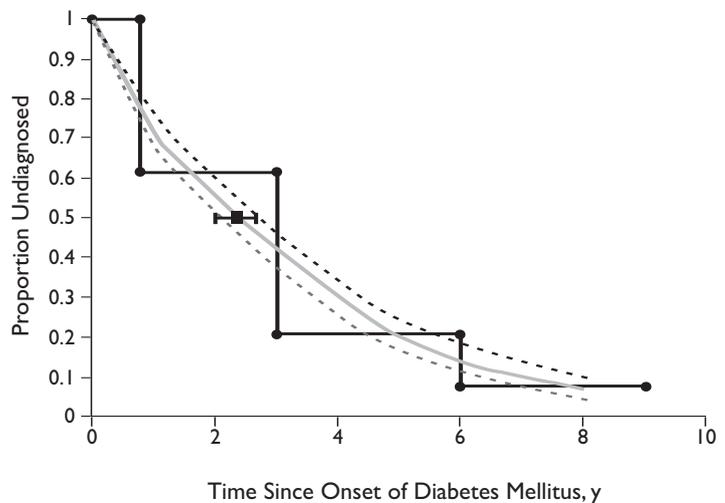
We next examined the associations between prediabetic characteristics and subsequent diagnosis status at visit 2 (Table 1). Obesity at baseline was the strongest predictor of initial delay to diagnosis. The mean BMI at baseline of individuals who were diagnosed by visit 2 was 30.8 compared with 32.9 in those who were undiagnosed at visit 2 ( $P = .003$ ). Body mass index was also treated as a categorical variable with the following 4 levels: BMI less than 25 (reference), overweight, class 1 obesity, and at least class 2 obesity. Compared with the reference group, overweight individuals were 2.9 times (95% CI, 1.2-7.4 times) more likely to have initial delayed diagnosis of type 2 DM, individuals with class 1 obesity were 2.8 times (95% CI, 1.1-6.8 times) more likely, and individuals with class 2 obesity were 5.2 times (95% CI, 2.0-13.5 times) more likely. This graded association remained significant after adjustment for potential confounders (Figure 3).

We next divided subjects according to duration of delays (0.75, 3, 6, or ≥7.5 years) to diagnosis to assess for graded

responses across time. Graded associations with delay time included history of myocardial infarction, coronary heart disease, having seen a physician in the past 12 months, and current smoking. Among subjects with the shortest delays, 9% had myocardial infarction and 10% had coronary heart disease vs 0% of subjects with the longest delays ( $P = .03$  and  $P = .02$ , respectively, for trend), while 80% of subjects with the shortest delays had seen their physician vs 50% of subjects with the longest delays ( $P = .005$  for trend). In the shortest delay group, 28% were smokers vs 9% in the longest delay group ( $P = .03$  for trend). These trends persisted after adjustment for BMI and years since the last physician visit (data not shown).

To test the hypothesis that gradual increases in glucose levels may delay DM diagnosis, we conducted an

**Figure 2.** Kaplan-Meier Survival Curves for Remaining Undiagnosed



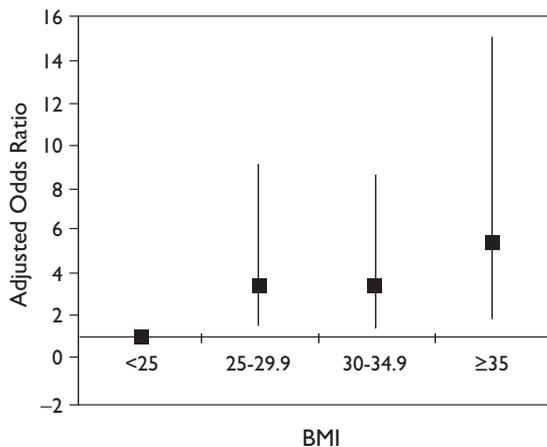
Points represent the assumed time of diagnosis; square and bars, the median time of delay with 95% confidence interval; and dashed lines, the 95% confidence intervals from the simulations.

analysis of FPG trend (Figure 4) according to the duration of delays to diagnosis, distinguishing between trends before and after confirmed diagnosis. Compared with subjects who remained undiagnosed at visit 4, subjects diagnosed between visit 3 and visit 4 had a steeper increase in blood glucose levels between visit 1 and visit 3. After adjusting for age, race/ethnicity, sex, and obesity, the change in glucose concentration was 0.77 mM per visit higher ( $P = .04$ ) among subjects who were diagnosed by visit 4 compared with subjects who remained undiagnosed at visit 4. The results were similar when we compared subjects diagnosed with subjects remaining undiagnosed at visit 3.

DISCUSSION

These data support the following conclusions in this atypical best-case scenario. First, even with a de facto screening program in a volunteer population, physician diagnosis of DM was commonly delayed, with a median delay of more than 2 years and some remaining undiagnosed for more than 7 years. Second, greater adiposity was most strongly associated with initial delays, while not smoking cigarettes, lack of recent physician visits, absence of prevalent heart disease, and shallow glucose trends each predicted longer delays. In contrast, neither age, race/ethnicity, sex, parental history of DM, education, income, insurance, nor study site predicted initial or longer delays to physician diagnosis.

**Figure 3.** Adjusted Relative Odds of Delayed Diagnosis (>1.5 vs ≤1.5 Years) By Categories of Body Mass Index (BMI) at Baseline in 298 Middle-aged Adults With Incident Type 2 Diabetes Mellitus



The bars indicate 95% confidence intervals ( $P = .008$  for trend). BMI was calculated as weight in kilograms divided by height in meters squared. (Adjusted for age, race, and sex.)

It is well established that 30% to 50% of prevalent cases of type 2 DM are undiagnosed in the United States, but few data are available on delay times. Two published estimates<sup>16,17</sup> projected estimates of delays to diagnosis of 5 to 12 years. We found no previously published studies with empiric determination of delays. In comparison to the previously published estimates, estimates obtained in this analysis are artificially optimized, likely related to (1) de facto screening and notification of physician and patient, (2) volunteers in a longitudinal study, and (3) the age-restricted ARIC population. Therefore, these results set a conservative limit, giving an estimate of the minimum delay time to diagnosis, with screening and notification at 3-year intervals.

The prospective nature of this study afforded a unique opportunity to empirically examine the natural history of the clinical diagnosis of type 2 DM and to determine the mean delay time to diagnosis in a community setting. Nonetheless, several limitations deserve comment.

First, because assessments of diabetes status in the ARIC study occurred at 3-year intervals, we were compelled to make assumptions about the exact time of onset of DM and about the time of diagnosis by the physician. We assumed that the onset of disease occurred uniformly during the interval between visit 1 and visit 2. This is a valid point estimate, regardless of the method of ascertaining the estimated time of onset, but the CIs around this estimate will vary depending on the method of estimation. We performed 2 sets of analyses (categorical and continuous) to check the robustness of our uniformity assumption. Results from the subgroup analysis of participants with self-reported age at diagnosis revealed delay time estimates within the 95% CI of the estimate based on the entire data set using the uniformity assumption. Because physicians and patients were notified, this could have precipitated an earlier diagnosis. Therefore, assuming that the mean time from visit to diagnosis is 0.5 year, the estimate of the median delay time is 1.6 years (95% CI, 1.4-1.8 years). When the average is 1 year, the estimated median is 2.0 years (95% CI, 1.7-2.2 years). When the average is 1.5 years (the original assumption), the estimated median is 2.4 years (95% CI, 2.0-2.7 years).

Second, although hyperglycemia was ascertained by a single glucose measurement at each visit with no confirmatory testing by the ARIC study until the next scheduled follow-up visit, the analysis reduced the possibility of misclassification by requiring the study population to have been nondiabetic at baseline and diabetic at both visit 2 and visit 3. Therefore, 119 adults who were nondiabetic at baseline, diabetic at visit 2, and nondiabetic or with missing data at visit 3

were excluded. This excluded proportion corresponds to a previous report of 12% to 40% of patients who seem to revert to nondiabetes status after positive screening.<sup>26</sup>

Third, we lacked some information about the ARIC participants. This included when participants visited their primary care physician, whether the physician or the patient actually received and read the ARIC notification letters, whether there was any patient vs physician difference in the response to the letters, whether confirmatory blood tests were performed, whether patients understood that elevated FPG levels suggested the presence of type 2 DM, and whether physicians counseled about diet and exercise without explicitly diagnosing DM.

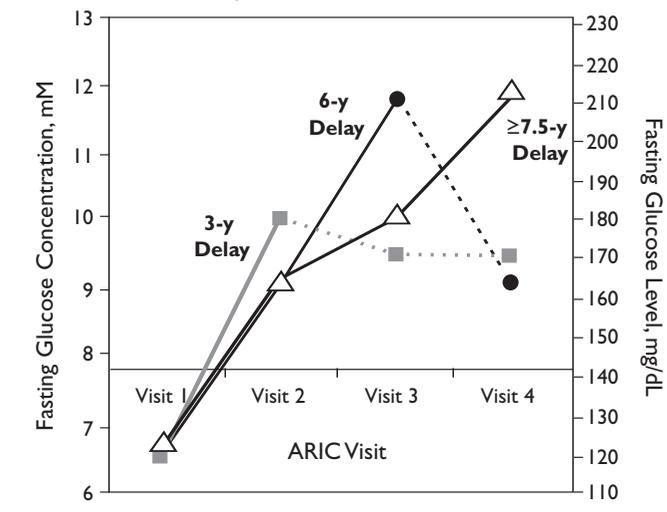
Fourth, the participants may have had faulty recall and, although reporting no diagnosis, may have been diagnosed (or less likely, they might inaccurately state that they had been diagnosed). No data on the participants' knowledge or attitude about their disease were collected, so participant denial of the diagnosis is possible. The incidence rate for DM in this population was similar to that of the US population during this period, given that the US incidence rates would not have included undiagnosed disease and the ARIC rates excluded subjects who failed to return for follow-up.

As expected, there was a strong graded association between categories of delay to diagnosis and fewer recent physician visits. However, among those participants with delays to diagnosis of at least 7.5 years, 55% had visited their physician in the 12 months before visit 2, suggesting that physicians often had the opportunity to make the diagnosis.

However, the finding that greater obesity predicted longer delayed time to diagnosis was unexpected. It is likely that weight loss preceding diagnosis is associated with symptoms, with diagnosis being prompted by symptoms and not by serum glucose results. Alternatively, obese patients may be less likely to receive screening or preventive health care services for all diagnoses,<sup>27,28</sup> despite having more physician visits than their leaner counterparts.

We demonstrated that delay to physician diagnosis is associated with the rate of change of the fasting glucose concentration. The rate of change could be affecting diagnosis of the disease, with a shallow trend seeming to be reasonably constant from year to year, while a steep trend looks more ominous. In addition, most newly diagnosed patients with DM are asymptomatic,<sup>29,30</sup> and physicians tend to be reluctant to diagnose and treat DM when there are no symptoms or when the diagnosis is made by screening and may simply recommend lifestyle

**Figure 4.** Fasting Plasma Glucose Trends in 298 Middle-aged Adults With Incident Type 2 Diabetes Mellitus by Atherosclerosis Risk in Communities (ARIC) Study Visit and Estimated Delay Time



Shaded line with squares indicates delay time of 3 years; black line with circles, delay time of 6 years; and thick black line with triangles, delay time of at least 7.5 years. Solid lines indicate trends before physician diagnosis; dotted lines, trends after physician diagnosis.  $P = .04$  for the difference in the rate of change in fasting blood glucose measurements before diagnosis at visits 1 through 3 for 6-year vs  $\geq 7.5$ -year delay. To convert glucose to millimoles per liter, multiply by 0.0555.

changes.<sup>31</sup> However, the presence of additional risk factors, such as smoking or prior heart disease, precipitated physician diagnosis.

Now that the criterion for diagnosing DM has been reduced from an FPG level of 140 mg/dL (7.8 mmol/L) to 126 mg/dL (7.0 mmol/L),<sup>32,33</sup> it is possible that delays to physician diagnosis may be longer. In settings of de facto screening, diagnosis is delayed by factors that might betray health system, physician, or patient factors. However, the magnitude of delays is much less than the previous estimates, suggesting that even weak screening programs should decrease delay times. The American Diabetes Association recommends opportunistic screening at 3-year intervals for patients older than 45 years or with risk factors as being cost-effective.<sup>34-36</sup> However, even with recommended screening of high-risk patients, the findings of this study suggest that stronger letters, physician follow-up, and patient activation may also be required. Managed care organizations may consider specifying the fasting condition in serum glucose laboratory requests, then mining the data for undiagnosed cases of DM. Obese patients should be encouraged to ask their physician about earlier or more frequent screening tests.

In advance of such programs, physicians should be careful not to miss DM diagnosis in the most obese

patients and in those with insidiously slow increases in fasting glucose concentrations. Patients should be encouraged to have regular physician consultations and should be assisted in accepting the diagnosis of DM.

#### Acknowledgments

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