Predicting the Development of Diabetes in Older Adults

The derivation and validation of a prediction rule

ALKA M. KANAYA, MD[1]
CHRISTINA L. WASSEL FYR, MS[1]
NATHALIE DE REKENEIRE, PHD[2]
RONALD I. SHORR, MD[3]
ANN V. SCHWARTZ, PHD[1]
BRET H. GOODPASTER, PHD[4]
ANNE B. NEWMAN, MD, MPH[4]
TAMARA HARRIS, MD[2]
ELIZABETH BARRETT-CONNOR, MD[5]

OBJECTIVE — To create a simple prediction rule that could perform as well as the 2-h postchallenge plasma glucose (PCPG) test to predict those at risk for diabetes. We created a prediction rule in one sample and prospectively validated it for incident diabetes in a separate cohort.

RESEARCH DESIGN AND METHODS — A cross-sectional analysis with data from the Rancho Bernardo Study (age 67 ± 11 years) to derive a rule predicting abnormal PCPG ≥140 mg/dl, using demographic, clinical, and laboratory data of nondiabetic participants with fasting plasma glucose (FPG) <126 mg/dl. Data from the Health, Aging and Body Composition study (age 74 ± 3 years) were used to prospectively validate this rule for incident diabetes and compare it with the predictive ability of the PCPG test.

RESULTS — Of 1,549 RBS participants, 514 (33%) had PCPG ≥140 mg/dl. Female sex, age, triglycerides, and FPG were most significantly associated with abnormal PCPG. Based on standardized β-coefficients, we allotted 1 point for female sex, triglycerides ≥150 mg/dl, or FPG 95–104 mg/dl. Age ≥70 years or FPG 105–115 mg/dl were given 2 points, and FPG 116–125 mg/dl received 3 points. In the validation cohort, this simple prediction rule was as good as the 2-h PCPG test for predicting incident diabetes (C-statistic: 0.71 for both).

CONCLUSIONS — Advanced age, female sex, FPG, and triglycerides were able to predict adults at risk for diabetes equally well as the 2-h PCPG test. Using this rule, clinicians may better identify older persons who should receive intensive lifestyle intervention to prevent type 2 diabetes.

Diabetes Care **28:404–408, 2005**

Impaired glucose tolerance (IGT) was first described in 1979 (1) as a method to identify a category of individuals at increased risk of developing type 2 diabetes and cardiovascular disease. An individual is classified as having IGT if the glucose level 2 h after ingesting a 75-g oral glucose solution exceeds 140 mg/dl but is less than the diagnostic threshold for diabetes, 200 mg/dl (2). White adults with IGT have an estimated annual rate of deterioration to diabetes between 1 and 10% (3), while rates in U.S. ethnic minority populations are higher (4). Moreover, recent randomized trials have demonstrated that lifestyle interventions are effective in delaying or preventing the onset of type 2 diabetes among subjects with IGT (5,6).

Targeting individuals at highest risk of developing diabetes is important, but the 2-h PCPG test is cumbersome and inconvenient (7), and other methods for identifying individuals with IGT are needed. Investigators from the San Antonio study evaluated clinical and demographic variables with and without the PCPG test to detect incident diabetes (8). They developed a regression equation with eight laboratory and clinical variables that outperformed the ability of the PCPG test to predict diabetes. However, computer software is required to implement this rule, making it less practical.

We aimed to create and validate a clinical prediction rule that would be simple to use, could identify individuals with abnormal glucose tolerance, and could predict incident diabetes. We used clinical, demographic, and laboratory data from older white adults in the Rancho Bernardo Study (RBS) to develop a simple prediction rule that could identify participants with abnormal glucose tolerance. We prospectively validated this prediction rule using data from the Health, Aging and Body Composition (Health ABC) study, a separate study of older, well-functioning, black and white adults who were followed for incident diabetes for a mean 5 years.

RESEARCH DESIGN AND METHODS

The RBS

The derivation sample consisted of subjects enrolled in the RBS who participated in a clinical research evaluation during

From the [1]Department of Medicine and Epidemiology and Biostatics, University of California, San Francisco, California; the [2]National Institute on Aging, National Institutes of Health, Bethesda, Maryland; the [3]Department of Preventive Medicine, University of Tennessee, Memphis, Tennessee; the [4]Department of Epidemiology, University of Pittsburgh, Pittsburgh, Pennsylvania; and the [5]Department of Family and Preventive Medicine, University of California, San Diego, California.

Address correspondence and reprint requests to Alka M. Kanaya, MD, 1635 Divisadero St., Suite 600, San Francisco, CA 94115. E-mail: alkak@itsa.ucsf.edu.

Received for publication 22 June 2004 and accepted in revised form 27 October 2004.

Abbreviations: FPG, fasting plasma glucose; Health ABC, Health, Aging and Body Composition; IFG, impaired fasting glucose; IGT, impaired glucose tolerance; PCPG, postchallenge plasma glucose; RBS, Rancho Bernardo Study; ROC, receiver operating characteristic.

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

Table 1—*Characteristics of the RBS population, 1984–1987

Variable	Normal glucose tolerance (n = 1,035)	Abnormal glucose tolerance (n = 514)	P†
Female sex	560 (54.1)	314 (61.1)	0.008
Age (years)	65.3 ± 11.2	71.3 ± 9.8	<0.001
Education			
High school or greater	985 (95.2)	500 (97.3)	0.05
Less than high school	50 (4.8)	14 (2.7)	
Family history of diabetes	153 (16.1)	68 (14.4)	0.40
BMI (kg/m^2)			
Men	25.8 ± 3.1	26.2 ± 4.7	0.54
Women	24.2 ± 4.8	24.4 ± 3.5	0.40
Waist (cm)			
Men	92.9 ± 8.5	94.2 ± 9.5	0.10
Women	76.7 ± 9.5	79.9 ± 9.5	<0.001
Systolic blood pressure (mmHg)	133.0 ± 21.3	143.6 ± 21.2	<0.001
Diastolic blood pressure (mmHg)	76.6 ± 9.4	76.2 ± 9.6	0.43
Fasting plasma glucose			
<95 mg/dl	463 (44.7)	137 (26.7)	<0.001
95–104 mg/dl	378 (36.5)	198 (38.5)	
105–115 mg/dl	159 (15.4)	130 (25.3)	
116–125 mg/dl	35 (3.4)	49 (9.5)	
Triglycerides			
<150 mg/dl	858 (82.9)	364 (70.8)	<0.001
≥150 mg/dl	177 (17.1)	150 (29.2)	
LDL (mg/dl)	135.0 ± 35.6	136.8 ± 39.0	0.37
HDL (mg/dl)	63.5 ± 19.0	60.2 ± 18.9	<0.001
Total cholesterol (mg/dl)	220.0 ± 38.3	223.3 ± 44.1	0.15
HbA_{1c} (%)	6.0 ± 0.7	6.1 ± 0.8	<0.001

Data are means ±SD or n (%). *Excluding those with known diabetes or fasting glucose ≥126 mg/dl; abnormal glucose tolerance is defined by a 2-h postchallenge glucose level ≥140 mg/dl. †Comparisons by Student's t test, Wilcoxon, or χ^2 tests where appropriate.

1984–1987. The RBS is a population-based study of white, middle or upper-middle class, community-dwelling adults in a southern California suburb. Participants with a diagnosis of diabetes or those with FPG ≥126 mg/dl were excluded. The final derivation sample population consisted of 1,549 men and women.

Demographic information (age, sex, and education), health-related behaviors (smoking and alcohol use), family history, and medication use were assessed. Weight and height were measured using standard protocols, and BMI was calculated (kg/m^2). Waist circumference was measured with a flexible tape measure at the level of minimum abdominal circumference. Two resting systolic and diastolic blood pressure measurements were taken on each subject using a mercury sphygmomanometer; the mean blood pressure was used for analyses.

Morning venous blood after a 12-h fast and 2 h after a 75-g oral glucose load were performed. Plasma glucose was measured using a glucose oxidase method. HbA_{1c} was measured using high-performance liquid chromotography. Fasting cholesterol and triglycerides were measured by enzymatic methods with an ABA-200 biochromatic analyzer (Abbott, North Chicago, IL); HDL was assayed by precipitation, and LDL was calculated using the Friedewald formula (9).

The Health ABC study

Data collected from Health ABC study participants were used to test the prediction rule developed in the RBS. Health ABC is a prospective cohort study of 3,075 community-dwelling men and women aged 70–79 years recruited at two clinical centers in Memphis, Tennessee, and Pittsburgh, Pennsylvania. To be eligible, participants had to report no difficulty walking a quarter mile or up a flight of stairs. For our analyses, we used data gathered from 2,503 (1,550 white and 953 black) participants who did not have diabetes at baseline (1997–1998), based on self-report of diabetes, use of a hypoglycemic medication, or FPG ≥126 mg/dl.

Questionnaire variables gathered from the first clinical visit included age, self-identified race, sex, and level of education completed. Participants reported smoking history and alcohol use. Participant's weight, height, and waist circumference was measured, and BMI was calculated.

Fasting serum specimens were collected from each participant. Lipid lipoproteins were measured by colorimetric technique (Vitros chemical methodology; Johnson & Johnson, New Brunswick, NJ), and fasting glucose and 2-h PCPG test were measured by glucose oxidase reaction (YSI 2300 Glucose Analyzer; YSI, Yellow Springs, OH).

Incident diabetes was defined by an algorithm using a physician diagnosis of diabetes in the past year, use of a hypoglycemic drug, or a FPG ≥126 mg/dl at the year 2, 4, or 6 examination.

Table 2—*Predictors of abnormal glucose tolerance in the RBS*

Risk factor	β	OR (95% CI)	*P*	Points allocated
Female sex	0.47	1.60 (1.26–2.02)	<0.001	1
Age ≥70 years	1.12	3.06 (2.43–3.87)	<0.001	2
Triglycerides ≥150 mg/dl	0.78	2.18 (1.66–2.87)	<0.001	1
Fasting glucose				
95–104 mg/dl	0.57	1.78 (1.36–2.33)	<0.001	1
105–115 mg/dl	1.05	1.62 (1.20–2.20)	0.002	2
116–125 mg/dl	1.69	1.89 (1.13–3.18)	0.01	3

Statistical analysis

Multivariable logistic regression models were constructed to examine which variables were most associated with an abnormal 2-h PCPG test result (≥140 mg/dl) in the RBS. Model entry criteria were $P < 0.15$. Accepted risk levels were used to categorize most variables (e.g., BMI <25.0, 25.0–29.9, ≥30.0 kg/m^2; LDL <100 vs. ≥100 mg/dl; systolic blood pressure <140 vs. ≥140 mmHg). For other continuous variables such as age, waist circumference, and fasting glucose, recursive partitioning methods were used to select categories that optimized the data selection. Backwards elimination ($P < 0.05$ to retain) was used to select the final set of risk factors and checked for interactions between sex and age with other risk factors and found none significant at $P < 0.05$.

A clinical scoring system was created by assigning points to each risk factor by dividing the β-coefficient of each variable by 0.6 and rounding to the nearest integer. A risk score was assigned to each subject by adding up the points for each risk factor present. Sensitivity, specificity, and likelihood ratio for each risk score was calculated to predict those with abnormal glucose tolerance. We also calculated the area under the receiver operating characteristic (ROC) curves for the final multivariable model built with continuous or categorical variables and the simple scoring system.

The prediction model and the scoring system were validated using data from the Health ABC study by examining the ability of the rule to predict abnormal glucose tolerance at the baseline Health ABC visit and to predict incident diabetes over 5 years of follow-up. By using a separate cohort for validation, we tested the accuracy of the model and its geographic, racial, and methodologic transportability (10). Separate ROC curves for black and white participants in Health ABC for incident diabetes were calculated and the C-statistics were compared with that of the 2-h PCPG test alone (11).

We used SAS version 8.2 (SAS Institute, Cary, NC) and S-Plus version 6.1 (Insightful, Seattle, WA) for our analyses.

RESULTS

Of 1,549 participants in the RBS derivation sample, 514 (33.2%) subjects had elevated PCPG results (≥140 mg/dl). Those with abnormal PCPG were older and had higher systolic blood pressure and triglyceride levels compared with those with normal glucose tolerance (Table 1). There was no difference in mean BMI for those with normal and abnormal glucose tolerance. Mean waist circumference was larger in women with abnormal glucose tolerance compared with the women with normal glucose tolerance, but there was no difference in waist circumference for men in the two glucose subgroups. As expected, mean FPG was lower among participants with normal glucose tolerance (96.0 ± 10.4 mg/dl vs. 100.8 ± 10.4 mg/dl; $P < 0.001$). The mean HbA_{1c} levels were statistically but not clinically different between the two groups.

Of the nine variables that initially entered our multivariable logistic regression analysis, only two demographic and two laboratory variables remained significantly associated with an abnormal PCPG result (Table 2). Individuals aged ≥70 years had the highest odds of an abnormal PCPG test (OR 3.06; 95% CI 2.43–3.87). Women had higher odds of abnormal PCPG (1.60; 1.26–2.02) compared with men. Participants with triglycerides ≥150 mg/dl and FPG ≥95 mg/dl were at greater risk for abnormal glucose tolerance as well. The discrimination of the final model was modest in the derivation cohort (c = 0.73 with continuous variables, 0.71 with categorical variables). Results did not differ with sex-specific models.

Using β-coefficients from the final categorical multivariate model, we assigned points to each of the four risk factors for abnormal PCPG (Table 2). A risk score was calculated for each participant by summing the points of each risk factor that was present. The derivation cohort risk score ranged from 0 to 7 points (2.6 ± 1.4 [means ± SD]). Estimates of sensitivity, specificity, and positive likelihood ratios for each score for abnormal glucose tolerance are shown in Table 3. A score of 4 or higher could double the likelihood of an abnormal PCPG test.

To validate this rule, we applied the prediction models and simple scoring rule to the baseline data from Health ABC. The overall discrimination of the scoring system for abnormal PCPG was similar in both samples (c = 0.70 in the derivation sample, 0.66 in the validation sample). Next, we prospectively validated the prediction rule for incident diabetes in Health ABC. There were 143 incident cases of diabetes over 5 years (14.1 cases/1,000 person-years). The area under the

Table 3—*Prediction rule score with test characteristics for abnormal glucose tolerance from the derivation cohort*

Total score	*n* (%)	Sensitivity	Specificity	+LR
0	92 (5.9)	100.0	0	0.2
1	307 (19.8)	98.4	8.1	0.4
2	334 (21.5)	88.1	32.7	0.7
3	403 (26.0)	71.6	56.7	1.0
4	254 (16.4)	45.9	82.8	1.9
5	129 (8.3)	21.8	95.4	4.0
6	27 (1.7)	5.0	99.5	8.9
7	4 (0.3)	0.8	100.0	∞

+LR, positive likelihood ratio.

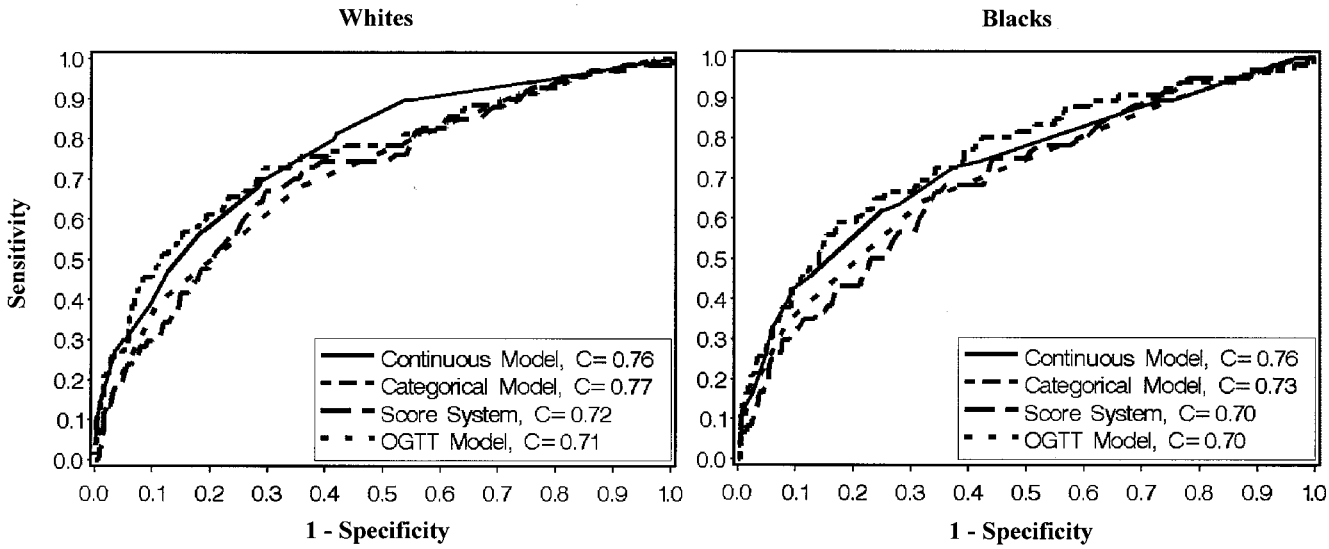

Figure 1—*ROC curves comparing the continuous multivariable model, the categorical model, the prediction rule scoring system, and the 2-h postchallenge glucose test alone for the prediction of diabetes among whites and blacks in the Health ABC cohort.*

ROC curve for the multivariable model using continuous or categorical variables was not different than the scoring system (c = 0.75 for both multivariable models, 0.71 for the scoring system). Thus, the scoring system was able to discriminate incident diabetes cases equally well as the PCPG test. When we stratified the Health ABC cohort by race, similar discrimination in both the black and white participants using the continuous model, categorical model, or scoring system was observed (Figure 1). There was no significant difference among the three models and the PCPG test in discrimination of diabetes ($P > 0.10$ for each comparison).

CONCLUSIONS — We have developed a clinical prediction rule to identify persons with abnormal glucose tolerance that predicted diabetes equally well as the 2-h PCPG test. Two demographic variables, age and sex, and two laboratory variables, FPG and triglyceride levels, were most strongly associated with abnormal glucose tolerance in a cohort of older white adults. Using a simple scoring system, a score of ≥4 points at least doubled the likelihood of an abnormal 2-h PCPG test result. Moreover, this scoring system, validated in an independent cohort of black and white older adults, was able to predict future diabetes with similar accuracy as the PCPG test. Thus, patients with a score of ≥4 points with this simple scoring rule should receive appropriate lifestyle or pharmacologic therapies in order to prevent the onset of type 2 diabetes.

Recent reports that the incidence of diabetes can be prevented or delayed in individuals with IGT by lifestyle intervention (5,6) or medication therapy (5,12,13) has highlighted the need for identification of these high-risk individuals. The American Diabetes Association (ADA) does not recommend the 2-h PCPG test for screening or diagnosis of diabetes because the test is costly, inconvenient, and has poor reproducibility (7). The ADA adopted the term of "prediabetes" to identify persons with IGT or impaired fasting glucose (IFG; FPG levels between 110–125 mg/dl) collectively as those at highest risk for developing diabetes. In 2003, the ADA revised the definition for IFG to include persons with fasting glucose between 100 and 125 mg/dl (14,15). The use of IFG alone to identify those with incident diabetes in our validation cohort had a sensitivity of 54% and a specificity of 82% (ROC area of 0.68). We found that the addition of age, sex, and triglyceride level to elevated FPG results increased the predictive accuracy for diabetes.

Several recent studies have evaluated the utility of laboratory and demographic variables for predicting incident diabetes. Stern et al. (8) reported the effectiveness of eight clinical variables at predicting those at risk of incident diabetes among the San Antonio Heart Study participants. The prediction equation they created was based mostly on continuous variables and requires the use of a computer or personal digital assistant to implement. Additionally, being Mexican American explained most of the predictive value of their equation. This clinical model was tested in a cohort of Japanese Americans and was useful in predicting diabetes risk in younger Japanese Americans after 5–6 years but was not useful in older participants or with longer follow-up (16). Another study that calculated a diabetes risk score utilized data from a Finnish population sample (17). The authors found seven demographic and questionnaire variables that predicted future drug-treated diabetes with ROC curve area of 0.85 and 0.87 in two consecutive cohorts. As with the San Antonio study clinical model, the Finnish model requires the use of a computer to calculate the risk score and has not been validated in different ethnic groups. The present study differs from these prior studies in that it was first a cross-sectional evaluation of predictors of abnormal glucose tolerance in one sample and then was prospectively validated for diabetes prediction in a separate cohort. Moreover, this prediction rule was derived and validated in separate samples of different racial makeup and was based on only four variables that could be calculated easily without the use of a computerized program.

Clinical decision rules attempting to refine the accuracy of clinicians' diagnostic and prognostic ability are widely re-

ported. Using criteria developed to grade the evidence for clinical decision rules (10), our study is limited in its applicability because we validated it only in the Health ABC study with well-functioning adults older than age 70. The age criterion for our prediction rule was met by every Health ABC participant, so age was uninformative. This may have weakened both the sensitivity and specificity of the rule in the validation cohort. This prediction rule needs to be validated prospectively in broader populations. Clinicians may consider using this rule if the patients in their clinical setting are older, well-functioning, black or white adults. However, the risk of a lifestyle intervention for those identified with abnormal glucose tolerance is low. Lifestyle modification has been recommended as the first-line intervention to prevent or delay diabetes, with the recommended goals of modest weight loss (5–10% of body weight) and modest physical activity (30 min daily) (18). Because this intervention is likely to have a variety of other benefits, the ADA has urged health care providers to counsel all overweight or sedentary individuals to adopt these changes.

We found that measures of adiposity such as BMI or waist circumference were not independently associated with abnormal glucose tolerance. We did not expect to find an association between BMI and glucose intolerance because BMI is a poor marker for total adiposity in older adults (19). But abdominal adiposity has been more closely linked with adverse metabolic consequences and has been suggested to precede insulin resistance (20). Waist circumference is an accepted criterion in the diagnosis of the metabolic syndrome, but waist circumference may reflect total body fat better than visceral fat in older adults (21). It is possible that in our derivation cohort waist circumference did not adequately represent visceral obesity, explaining the lack of association of waist circumference with glucose intolerance.

In conclusion, advanced age, female sex, FPG, and triglycerides had good ability to predict those with abnormal glucose tolerance. A score of 4 or higher doubled the likelihood of an abnormal postchallenge glucose test result. This rule could help clinicians to better identify individuals with abnormal glucose tolerance, who should be targeted for interventions to prevent diabetes.

Acknowledgments— A.M.K. was funded by the BIRCWH (5 K12 AR47659) and by grant P30-AG15272 under the RCMAR program. E.B.-C., PI for the RBS, was funded by NIA grant 5R01 AG07181 and NIDDK grant 5R01 DK31801. The Health ABC study was funded by NIA contracts N01-AG-6-2101, N01-AG-6-2103, and N01-AG-6-2106.

The authors thank Dr. Eric Vittinghoff for his statistical help with recursive partitioning and the development of models.

References

1. National Diabetes Data Group: Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. *Diabetes* 28:1039–1057, 1979
2. World Health Organization: *Diabetes Mellitus: Report of a WHO Study Group*. Geneva, World Health Organization, 1985 (Tech Rep. Ser., no. 727)
3. Valle TT, Tuomilehto J, Eriksson JG: Epidemiology of NIDDM in Europoids. In *International Textbook of Diabetes Mellitus*. 2nd ed. Alberti K, Zimmet PZ, De Fronzo RA, Keen H, Eds. Chichester, New York, Wiley, 1997, p. 125–142
4. Edelstein SL, Knowler WC, Bain RP, Andres R, Barrett-Connor EL, Dowse GK, Haffner SM, Pettitt DJ, Sorkin JD, Muller DC, Collins VR, Hamman RF: Predictors of progression from impaired glucose tolerance to NIDDM: an analysis of six prospective studies. *Diabetes* 46:701–710, 1997
5. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, Nathan DM: Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 346:393–403, 2002
6. Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Hamalainen H, Ilanne-Parikka P, Keinanen-Kiukaanniemi S, Laakso M, Louheranta A, Rastas M, Salminen V, Uusitupa M: Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 344:1343–1350, 2001
7. Expert Committee on the Diagnosis and Classification of Diabetes Mellitus: Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. *Diabetes Care* 20:1183–1197, 1997
8. Stern MP, Williams K, Haffner SM: Identification of persons at high risk for type 2 diabetes mellitus: do we need the oral glucose tolerance test? *Ann Intern Med* 136: 575–581, 2002
9. Friedewald WT, Levy RI, Fredrickson DS: Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* 18:499–502, 1972
10. McGinn TG, Guyatt GH, Wyer PC, Naylor CD, Stiell IG, Richardson WS: Users' guides to the medical literature. XXII. How to use articles about clinical decision rules: Evidence-Based Medicine Working Group. *JAMA* 284:79–84, 2000
11. DeLong ER, DeLong DM, Clarke-Pearson DL: Comparing the areas under two or more correlated receiver operating characteristic curves: a nonparametric approach. *Biometrics* 44:837–845, 1988
12. Chiasson JL, Josse RG, Gomis R, Hanefeld M, Karasik A, Laakso M: Acarbose for prevention of type 2 diabetes mellitus: the STOP-NIDDM randomised trial. *Lancet* 359:2072–2077, 2002
13. Buchanan TA, Xiang AH, Peters RK, Kjos SL, Marroquin A, Goico J, Ochoa C, Tan S, Berkowitz K, Hodis HN, Azen SP: Preservation of pancreatic β-cell function and prevention of type 2 diabetes by pharmacological treatment of insulin resistance in high-risk hispanic women. *Diabetes* 51: 2796–2803, 2002
14. Genuth S, Alberti KG, Bennett P, Buse J, Defronzo R, Kahn R, Kitzmiller J, Knowler WC, Lebovitz H, Lernmark A, Nathan D, Palmer J, Rizza R, Saudek C, Shaw J, Steffes M, Stern M, Tuomilehto J, Zimmet P: Follow-up report on the diagnosis of diabetes mellitus. *Diabetes Care* 26:3160–3167, 2003
15. American Diabetes Association: Diagnosis and classification of diabetes mellitus. *Diabetes Care* 27 (Suppl. 1):S5–S10, 2004
16. McNeely MJ, Boyko EJ, Leonetti DL, Kahn SE, Fujimoto WY: Comparison of a clinical model, the oral glucose tolerance test, and fasting glucose for prediction of type 2 diabetes risk in Japanese Americans. *Diabetes Care* 26:758–763, 2003
17. Lindstrom J, Tuomilehto J: The diabetes risk score: a practical tool to predict type 2 diabetes risk. *Diabetes Care* 26:725–731, 2003
18. American Diabetes Association: The prevention or delay of type 2 diabetes. *Diabetes Care* 25:742–749, 2002
19. Gallagher D, Visser M, Sepulveda D, Pierson RN, Harris T, Heymsfield SB: How useful is body mass index for comparison of body fatness across age, sex, and ethnic groups? *Am J Epidemiol* 143:228–239, 1996
20. Lemieux S: Contribution of visceral obesity to the insulin resistance syndrome. *Can J Appl Physiol* 26:273–290, 2001
21. Harris TB, Visser M, Everhart J, Cauley J, Tylavsky F, Fuerst T, Zamboni M, Taaffe D, Resnick HE, Scherzinger A, Nevitt M: Waist circumference and sagittal diameter reflect total body fat better than visceral fat in older men and women: the Health, Aging and Body Composition Study. *Ann N Y Acad Sci* 904:462–473, 2000