

Implications of Alternative Definitions of Prediabetes for Prevalence in U.S. Adults

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OBJECTIVE—To compare the prevalence of prediabetes using A1C, fasting plasma glucose (FPG), and oral glucose tolerance test (OGTT) criteria, and to examine the degree of agreement between the measures.

RESEARCH DESIGN AND METHODS—We used the 2005–2008 National Health and Nutrition Examination Surveys to classify 3,627 adults aged ≥ 18 years without diabetes according to their prediabetes status using A1C, FPG, and OGTT. We compared the prevalence of prediabetes according to different measures and used conditional probabilities to examine agreement between measures.

RESULTS—In 2005–2008, the crude prevalence of prediabetes in adults aged ≥ 18 years was 14.2% for A1C 5.7–6.4% (A1C5.7), 26.2% for FPG 100–125 mg/dL (IFG100), 7.0% for FPG 110–125 mg/dL (IFG110), and 13.7% for OGTT 140–199 mg/dL (IGT). Prediabetes prevalence varied by age, sex, and race/ethnicity, and there was considerable discordance between measures of prediabetes. Among those with IGT, 58.2, 23.4, and 32.3% had IFG100, IFG110, and A1C5.7, respectively, and 67.1% had the combination of either A1C5.7 or IFG100.

CONCLUSIONS—The prevalence of prediabetes varied by the indicator used to measure risk; there was considerable discordance between indicators and the characteristics of individuals with prediabetes. Programs to prevent diabetes may need to consider issues of equity, resources, need, and efficiency in targeting their efforts.

Diabetes Care 34:387–391, 2011

Increased levels of glycemia in the prediabetic range, measured by fasting or postchallenge glucose or by A1C can serve as simple and reasonably accurate predictors of subsequent type 2 diabetes risk (1). Above and beyond its association with diabetes, hyperglycemia at the prediabetic level is associated with increased risk of subsequent cardiovascular disease (2–5). The value of glycemic measures is further strengthened by evidence that diabetes risk can be reduced with structured lifestyle interventions and/or metformin

among individuals with levels of glycemia above normal but below the diagnostic threshold for diabetes (6–8). This means that measures of glycemia can serve as practical indicators for referral to diabetes prevention programs.

A series of definitions to classify people at elevated glycemic risk have been recommended, including impaired glucose tolerance (IGT), impaired fasting glucose (IFG), and “prediabetes.” In 1979, the National Diabetes Data Group (NDDG) defined IGT as fasting plasma

glucose (FPG) < 140 mg/dL and 2-h plasma glucose values ranging from 140 to 199 mg/dL (9). The World Health Organization (WHO) also adopted this recommendation. The term IFG was introduced in 1997, whereby FPG values from 110 to 125 mg/dL additionally differentiated the metabolic state between normal and diabetic (10). In 2003, the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus (11) lowered the FPG cut point for IFG to 100 mg/dL, which optimized sensitivity and specificity in predicting diabetes and made the prevalence of prediabetes more comparable to IGT prevalence. The WHO did not change its previous recommendations, placing greater emphasis on 2-h plasma glucose.

Most recently, the American Diabetes Association recommended an A1C range of 5.7–6.4% to identify individuals at high risk for future diabetes (1). Because an overnight fast is not required to measure A1C, this change is expected to facilitate identification of people at risk for diabetes who could benefit from intervention.

Each change in definition has implications for the population prevalence of prediabetes, along with potential devolution of resources. Thus, we examined nationally representative data to 1) describe the prevalence of prediabetes among the U.S. population according to different glycemic measures and combinations of measures and 2) examine the degree of agreement between measures.

RESEARCH DESIGN AND METHODS

The 2005–2008 National Health and Nutrition Examination Survey (NHANES) was conducted by the National Center for Health Statistics (NCHS), Centers for Disease Control and Prevention (12,13). NHANES uses a complex, multi-stage probability sample design to collect data representative of the U.S. civilian non-institutionalized population. Survey participants are interviewed at home and invited to attend a mobile examination center to undergo medical examinations and laboratory measurements. Among participants aged 18 years and older, 11,791 completed

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Received 9 July 2010 and accepted 17 November 2010.

DOI: 10.2337/dc10-1314

This article contains Supplementary Data online at <http://care.diabetesjournals.org/lookup/suppl/doi:10.2337/dc10-1314/-/DC1>.

The findings and conclusions in this article are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention or the National Institute of Diabetes and Digestive and Kidney Diseases.

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Alternative definitions of prediabetes

the household interview and 11,329 completed the examination.

During the home interview, participants were asked if they have ever been told by a doctor or other health professional that they had diabetes (other than during pregnancy). On the basis of this question, 1,272 respondents aged 18 years and older were classified as having diagnosed diabetes.

Survey participants were randomly assigned to either a morning or afternoon/evening examination session: 4,881 individuals aged 18 years and older without diagnosed diabetes were examined during a morning session. After excluding people who fasted <8 or ≥24 h (*n* = 586) and individuals with invalid FPG values (*n* = 118), the FPG subsample comprised 4,177 adults. An oral glucose tolerance test (OGTT) 2-h (± 15 min) measurement was obtained for 3639 (87%) of those in the FPG subsample. Participants within the OGTT subsample with missing A1C values (*n* = 12) were excluded resulting in a subsample of 3,627 adults. As a result of changes in laboratory equipment, a Deming regression equation ($Y = X - 1.139$) was applied to FPG and OGTT values to make 2007–2008 data comparable to 2005–2006 data. A glucose regression equation ($Y = 0.9835 \times X$) was applied to FPG and OGTT values, and a Deming regression equation ($Y = 0.4892 + 0.9277 \times X$) was applied to A1C values to make 2005–2008 data

comparable to previous years (13). A1C data were reanalyzed without the correction and no significant difference in results was observed.

Individuals without self-reported diabetes were classified as having undiagnosed diabetes if any of the following thresholds were exceeded: FPG ≥126 mg/dL, A1C ≥6.5%, or OGTT ≥200 (*n* = 276). For those without diabetes, prediabetes was defined according to different glycaemic risk strata: A1C 5.7–<6.5% (A1C5.7); FPG 100–<126 mg/dL (IFG100); OGTT 140–<200 mg/dL (IGT); FPG 110–<126 mg/dL (IFG110); A1C5.7 or IFG100 (A1C5.7-IFG100); A1C5.7 or IGT (A1C5.7-IGT); A1C5.7 or IFG110 (A1C5.7-IFG110). (Estimates for prediabetes as defined by IGT or IFG100 and by IGT or IFG110 are available in Supplementary Table 1.)

For NHANES 2005–2008, individuals with diagnosed diabetes from the interviewed sample were combined with individuals without diagnosed diabetes from the OGTT subsample. Interview and OGTT subsample weights (provided by NCHS) were used so that the sum of the sample weights from the two subsamples summed to the total U.S. noninstitutionalized population. We standardized estimates to the U.S. 2000 Census population using three age groups (i.e., 18–44, 45–64, ≥65 years). We used SAS 9.2 for Windows software (SAS Institute, Cary, NC) for data management and SUDAAN

10 software (Research Triangle Institute, Research Triangle Park, NC) to obtain point estimates and SEs, using the Taylor series linearization method.

One-sample Student *t* tests were used for testing whether differences between subgroups were significantly different from zero. A *P* value of <0.05 was considered statistically significant. Conditional probabilities were calculated to measure agreement.

RESULTS

Prevalences in 2005–2008

The crude prevalences and 95% CIs of prediabetes among U.S. adults aged ≥18 years (Table 1) were 14.2% (12.8–15.6) for A1C5.7, 26.2% (24.2–28.2) for IFG100, 13.7% (12.1–15.3) for IGT, and 7.0% (6.0–8.0) for IFG110. When the measures were combined to define prediabetes, the prevalences were 32.2% (30.4–34.0) for A1C5.7-IFG100, 23.5% (22.1–24.9) for A1C5.7-IGT, and 18.1% (16.8–19.4) for A1C5.7-IFG110. For the combination of all three measures—IFG100, A1C5.7, or IGT—prevalence was 36.7% (34.8–38.6).

Prevalence by age

Across the three age groups, prediabetes prevalence was lowest for IFG110 and highest for IFG100, with A1C5.7 and IGT falling in between (Table 1). Prevalence increased with age for most measures.

Table 1—Crude and age-adjusted prevalence of prediabetes in adults aged ≥18 years, by prediabetes indicator, NHANES 2005–2008

	A1C5.7	IFG100	IGT	IFG110	A1C5.7 or IFG100	A1C5.7 or IGT	A15.7 or IFG110
<i>n</i>	696	1,074	597	303	1,395	1,069	856
Crude							
Total	14.2 (0.7)	26.2 (1.0)	13.7 (0.8)	7.0 (0.5)	32.2 (0.9)	23.5 (0.7)	18.1 (0.7)
18–44 (years)	6.7 (0.6)	18.8 (1.2)	8.4 (0.8)	4.3 (0.6)	22.0 (1.1)	13.7 (0.9)	9.7 (0.6)
45–64 (years)	19.3 (1.6)	34.0 (2.3)	15.5 (1.5)	8.9 (1.0)	41.0 (2.3)	29.3 (1.5)	24.0 (1.7)
65+ (years)	25.6 (2.3)	31.6 (1.5)	25.9 (1.9)	10.6 (1.4)	43.6 (2.1)	40.3 (2.1)	30.6 (2.2)
Male	14.3 (0.9)	33.1 (1.5)	12.8 (0.9)	8.8 (0.8)	37.9 (1.6)	23.1 (1.0)	19.7 (1.0)
Female	14.1 (1.1)	19.7 (1.1)	14.6 (1.1)	5.2 (0.6)	26.8 (1.0)	24.0 (1.1)	16.7 (0.9)
NH white	12.9 (1.0)	27.1 (1.3)	14.5 (1.1)	7.5 (0.7)	32.1 (1.4)	22.9 (1.2)	17.3 (1.0)
NH black	24.8 (1.6)	17.9 (1.4)	7.3 (0.8)	3.7 (0.8)	33.6 (1.7)	28.0 (1.5)	26.1 (1.8)
Mex-Am	13.2 (1.3)	28.3 (2.2)	15.1 (1.5)	7.1 (1.1)	33.9 (1.9)	24.0 (2.1)	17.3 (1.4)
Age-adjusted*							
Total	13.7 (0.6)	25.5 (0.9)	13.5 (0.7)	6.8 (0.5)	31.4 (0.8)	22.9 (0.6)	17.5 (0.5)
Male	14.0 (0.7)	32.3 (1.4)	12.8 (0.8)	8.7 (0.7)	37.1 (1.4)	22.7 (0.7)	19.2 (0.8)
Female	13.3 (1.0)	19.1 (1.1)	14.2 (1.0)	5.1 (0.6)	25.8 (1.0)	23.0 (1.1)	15.9 (0.9)
NH white	11.6 (0.8)	25.5 (1.2)	13.6 (0.9)	7.0 (0.6)	30.0 (1.3)	21.1 (0.9)	15.8 (0.8)
NH black	24.4 (1.6)	17.3 (1.3)	7.4 (0.8)	3.5 (0.7)	33.0 (1.7)	27.8 (1.5)	25.6 (1.8)
Mex-Am	14.6 (1.3)	27.5 (2.1)	16.1 (1.3)	7.5 (1.1)	34.0 (1.8)	25.9 (1.7)	19.0 (1.4)

Data are percent based on weighted data (SE). Estimates are based on the morning OGTT subsample. Diagnosed and undiagnosed diabetes estimates not shown. Includes racial/ethnic groups not shown separately. *Standardized to the 2000 U.S. Census population by age using three age groups (18–44, 45–64, ≥65 years). Mex-Am, Mexican American; NH, non-Hispanic; *n*, unweighted number.

However, the age-related gradient was notably steeper for IGT and A1C5.7 than for the IFG100 measure. For example, when defined by A1C5.7, IGT, A1C5.7-IGT combination, or the A1C5.7-IFG110 combination, prevalence was approximately three times as high among those ≥ 65 years of age as among those 18–44 years of age. However, when prediabetes was defined by IFG100 or the A1C5.7-IFG100 combination, older adults had only twice the prevalence, and there was no significant difference between middle-aged and older adults.

Prevalence by sex

Age-adjusted prevalence as measured by A1C5.7 or IGT did not vary significantly between men and women ($P = 0.87$ and $P = 0.14$, respectively) (Table 1). However, men were much more likely than women to have IFG100 ($P < 0.01$) and IFG110 ($P < 0.01$). Though diminished, this sex difference was also evident when IFG was combined with other indicators (A1C5.7-IFG100, $P < 0.01$; A1C5.7-IFG110, $P < 0.01$). Prevalence did not vary significantly by sex when measured by the A1C5.7-IGT combination ($P = 0.56$).

Prevalence by race/ethnicity

The relationship between race/ethnicity and prediabetes prevalence varied according to the indicator used to measure prediabetes (Table 1). When A1C5.7 was used, age-adjusted prevalence among non-Hispanic blacks was almost twice that of non-Hispanic whites and Mexican Americans (all $P < 0.01$). However, when IFG and IGT were used as measures, prevalence among non-Hispanic whites and Mexican Americans was about twice that of non-Hispanic blacks (all $P < 0.05$). Prevalence was similar across race/ethnicity groups when defined using the

A1C5.7-IFG100 combination, but higher among non-Hispanic blacks when the A1C5.7-IGT and the A1C5.7-IFG110 combinations were used. Regardless of the measure used, prediabetes prevalence was similar between non-Hispanic whites and Mexican-Americans.

Agreement

Only 3.2% (95% CI 2.3–4.0) of adults had all three indicators (i.e., IFG100, A1C, and IGT), and an additional 11.2% (9.8–12.5) had two of the three indicators (Table 2 and Supplementary Fig. 1). The prevalence of those positive for only one of the three indicators was 22.4% (20.7–24.1); with IFG100 only (prevalence 13.2% [95% CI 11.7–14.6]) accounting for the majority. The prevalence of possessing only one indicator varied by demographic factors. The prevalence of IFG100 only was twice as large in men (17.9% [15.5–20.4]) than in women (8.7 [6.9–10.4]) and A1C5.7 only was three times larger among non-Hispanic blacks (13.8% [11.4–16.2]) than among non-Hispanic whites (3.8% [2.9–4.7]) and Mexican-Americans (3.9% [2.3–5.4]).

Among adults with IFG100, 30.6% had IGT and 31.6% had prediabetes, based on A1C5.7 (Table 3). Among those with prediabetes based on A1C5.7, 57.9, 21.3, and 31.1% had prediabetes based on IFG100, IFG110, and IGT, respectively. Among those with IGT, 58.2, 23.4, and 32.3% have IFG100, IFG110, and A1C5.7, respectively, and 67.1% had the A1C5.7-IFG100 combination.

CONCLUSIONS—In a representative sample of the U.S. adult population, the prevalence of those at high risk of diabetes varied greatly by the indicator used to measure risk. For example, the prevalence of prediabetes as measured by IFG100 (26.2%) was almost twice that measured

by A1C5.7 (14.2%) and IGT (13.7%) and over three times that of IFG110 (7.0%). The combination of either A1C5.7 or IFG100 resulted in a prevalence of almost 33%. Thus, if these indicators are used to identify the number of individuals eligible for prevention efforts, the number of individuals would range from 15.3 million (7% with IFG110) to 70.9 million (32.2% with A1C5.7-IFG100). If all three measures are combined, the number of individuals at high risk of diabetes would be 80.8 million (36.7% with A1C5.7, IFG100 or IGT).

Our study suggests that the specific choice of a prediabetes measure will yield differing associations with age, sex, and race. Although the prevalence of A1C5.7 and IGT was similar for men and women, IFG100 or IFG110 was about 1.7 times higher among men than women. These sex differences in prevalence are consistent with the findings of several international studies. Summarizing data from 13 European and 10 Asian studies, the writing committee for the International Diabetes Federation IGT/IFG consensus statement (14) found IFG110 to be consistently more common in men than women, typically 1.5–3 times higher. Although the reasons for this sex differential are unknown, some have suggested that the underlying etiologies or metabolic determinants of FPG and OGTT values may differ (14,15). Further, analysis of the Diabetes Prevention Program data suggested that progression to diabetes was more dependent upon FPG in men, whereas progression in women was more dependent on OGTT (16). Regardless of the reasons for the sex differences in IFG prevalence, epidemiological research and prevention programs solely using IFG to determine prediabetes status will identify a preponderance of men. For every 100 people eligible for enrollment in prevention programs based on IFG, about 63 would be men and 37 would be women.

In our study, prediabetes prevalence measured by A1C5.7 was about two times higher in non-Hispanic blacks than in non-Hispanic whites and Mexican Americans, but was only half that of non-Hispanic whites and Mexican Americans when measured by IGT or IFG110. Prior research comparing A1C and FPG and/or OGTT prediabetes definitions noted ethnic differences in prevalence and suggested changes to diagnostic criteria of diabetes or prediabetes may lead to substantial changes in prevalence among different ethnic groups (14,17–22). Some

Table 2—Prevalence of prediabetes in adults aged ≥ 18 years, by prediabetes indicator, NHANES 2005–2008

	IFG100	A1C5.7	IGT	Total	Men	Women	NH white	NH black	Mex-Am
All 3 Positive	+	+	+	3.2	3.1	3.2	3.4	2.2	2.5
2 Positive									
	+	+	–	5.1	6.4	3.8	4.6	6.9	5.1
	+	–	+	4.8	5.6	4.1	5.4	1.3	5.8
	–	+	+	1.3	0.9	1.6	1.2	2.0	1.7
1 Positive									
	+	–	–	13.2	17.9	8.7	13.8	7.6	14.9
	–	+	–	4.7	3.9	5.5	3.8	13.8	3.9
	–	–	+	4.5	3.1	5.8	4.8	1.9	5.0

Data are percent unless otherwise indicated. Mex-Am, Mexican American; NH, non-Hispanic.

Table 3—Conditional probability of prediabetes in adults aged ≥18 years without diabetes, NHANES 2005–2008

	IFG100	IFG110	IGT	A1C5.7	A1C5.7 or IFG100	A1C5.7 or IFG110	A1C5.7 or IGT
IFG100	—	100	30.6	31.6	—	26.6	49.8
IFG110	26.6	—	46.3	43.8	—	—	66.3
IGT	58.2	23.4	—	32.3	67.1	43.7	—
A1C	57.9	21.3	31.1	—	—	—	—

Data are percent.

warned that using A1C alone to screen for type-2 diabetes risks misdiagnosing large numbers of patients, especially those of African, Mediterranean, or south-east Asian heritage (23,24) and/or may be particularly insensitive in diagnosing whites (19–21). Further, studies have raised the concern that the relationship between A1C and other glycemic markers differs by race and ethnicity (21,22,25). For example, compared with their Caucasian counterparts, African Americans in particular have higher A1C levels at any given level of FPG. This may be influenced in part by genetic traits influencing erythrocyte turnover, or alternatively, by nutritional or metabolic factors. Whether this variation in the relationship among different glycemic markers affects prediction of morbid outcomes or whether the benefit that can be expected from interventions is strong enough to warrant ethnic-specific disease thresholds is not yet clear. However, a recent analysis of prospective cohort data from the Atherosclerosis Risk in Communities study did not support the use of race-specific cut points in A1C for identifying individuals at risk for diabetes (5).

In NHANES, limited data were available for some race/ethnic groups at high risk for diabetes, such as Native Americans. However, the major strengths of this study are the nationally representative data and the inclusion of standardized laboratory, clinical, and physical measurements.

Our findings confirm prior observations that considerable discordance between A1C, IFG, and IGT exists, and that the characteristics of individuals identified at risk for diabetes vary by risk indicator. This has implications for prevention programs concerning who will be identified and enrolled in prevention efforts. A1C5.7 may disproportionately identify non-Hispanic blacks, while IFG may disproportionately identify men. Due to the lack of a gold standard for comparison, it is unknown whether these

measures are biased or whether these groups are actually at a higher or lower risk of developing type 2 diabetes. The use of a combination of either A1C5.7 or IFG100 approximately equalizes prevalence by race/ethnicity, somewhat lessens sex differences, and identifies the majority with IGT (upon whom the prevention trials were based). However, using this combination identifies nearly a third of the U.S. population as at risk for developing diabetes. Because within each risk indicator the risk of diabetes increases across a continuum (6,7), it could be argued that intervention resources should be targeted toward those at greatest risk. Programs to prevent diabetes may need to consider issues of equity, resources, need, and efficiency in targeting their efforts.

Acknowledgments—No potential conflicts of interest relevant to this article were reported.

C.J. researched data and wrote the manuscript. D.B.R. and C.C.C. researched data, contributed to discussion, and reviewed and edited the manuscript. K.M.B. researched data and reviewed and edited the manuscript. E.W.G, L.S.G, D.E.W., and A.A. contributed to discussion and reviewed and edited the manuscript.

References

1. American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diabetes Care* 2010;33(Suppl. 1):S62–S69
2. Coutinho M, Gerstein HC, Wang Y, Yusuf S. The relationship between glucose and incident cardiovascular events. A meta-regression analysis of published data from 20 studies of 95,783 individuals followed for 12.4 years. *Diabetes Care* 1999;22:233–240
3. Meigs JB, Nathan DM, D'Agostino RB Sr, Wilson PW; Framingham Offspring Study. Fasting and postchallenge glycemia and cardiovascular disease risk: the Framingham Offspring Study. *Diabetes Care* 2002;25:1845–1850
4. Smith NL, Barzilay JI, Shaffer D, et al. Fasting and 2-hour postchallenge serum

glucose measures and risk of incident cardiovascular events in the elderly: the Cardiovascular Health Study. *Arch Intern Med* 2002;162:209–216

5. Selvin E, Steffes MW, Zhu H, et al. Glycated hemoglobin, diabetes, and cardiovascular risk in nondiabetic adults. *N Engl J Med* 2010;362:800–811
6. Pan XR, Li GW, Hu YH, et al. Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. *Diabetes Care* 1997;20:537–544
7. Tuomilehto J, Lindström J, Eriksson JG, et al.; Finnish Diabetes Prevention Study Group. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 2001;344:1343–1350
8. Knowler WC, Barrett-Connor E, Fowler SE, et al.; Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 2002;346:393–403
9. National Diabetes Data Group. Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. *Diabetes* 1979;28:1039–1057
10. 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* 1997;20:1183–1197
11. 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* 2003;26(Suppl. 1):S5–S20
12. Centers for Disease Control and Prevention National Center for Health Statistics. National Health and Nutrition Examination Survey 2005–2006. Questionnaires, Datasets and Related Documentation. Available from http://www.cdc.gov/nchs/nhanes/nhanes2005-2006/nhanes05_06.htm. Accessed 20 September 2009
13. Centers for Disease Control and Prevention National Center for Health Statistics. National Health and Nutrition Examination Survey 2007–2008. Questionnaires, Datasets and Related Documentation. Available from http://www.cdc.gov/nchs/nhanes/nhanes2007-2008/nhanes07_08.htm. Accessed 20 September 2009
14. Unwin N, Shaw J, Zimmet P, Alberti KG. Impaired glucose tolerance and impaired fasting glycemia: the current status on definition and intervention. *Diabet Med* 2002;19:708–723
15. Williams JW, Zimmet PZ, Shaw JE, et al. Gender difference in the prevalence of impaired glycaemia and impaired glucose tolerance in Mauritius. Does sex matter? *Diabet Med* 2003;20:915–920

16. Perreault L, Ma Y, Dagogo-Jack S, et al.; Diabetes Prevention Program. Sex differences in diabetes risk and the effect of intensive lifestyle modification in the Diabetes Prevention Program. *Diabetes Care* 2008;31:1416–1421
17. Mann DM, Carson AP, Shimbo D, Fonseca V, Fox CS, Muntner P. Impact of A1C screening criterion on the diagnosis of pre-diabetes among U.S. adults. *Diabetes Care* 2010;33:2190–2195
18. Christensen DL, Witte DR, Kaduka L, et al. Moving to an A1C-based diagnosis of diabetes has a different impact on prevalence in different ethnic groups. *Diabetes Care* 2010;33:580–582
19. Lorenzo C, Wagenknecht LE, Hanley AJG, Rewers MJ, Karter AJ, Haffner SM. A1C between 5.7 and 6.4% as a marker for identifying pre-diabetes, insulin sensitivity and secretion, and cardiovascular risk factors: the Insulin Resistance Atherosclerosis Study (IRAS). *Diabetes Care* 2010;33:2104–2109
20. Olson DE, Rhee MK, Herrick K, Ziemer DC, Twombly JG, Phillips LS. Screening for diabetes and pre-diabetes with proposed A1C-based diagnostic criteria. *Diabetes Care* 2010;33:2184–2189
21. Mostafa SA, Khunti K, Srinivasan BT, Webb D, Gray LJ, Davies MJ. The potential impact and optimal cut-points of using glycated haemoglobin, HbA1C, to detect people with impaired glucose regulation in a UK multi-ethnic cohort. *Diabetes Res Clin Pract* 2010;90:100–108
22. Ziemer DC, Kolm P, Weintraub WS, et al. Glucose-independent, black-white differences in hemoglobin A1C levels: a cross-sectional analysis of 2 studies. *Ann Intern Med* 2010;152:770–777
23. Kilpatrick ES, Bloomgarden ZT, Zimmet PZ. Is haemoglobin A1C a step forward for diagnosing diabetes? *BMJ* 2009;339:b4432
24. Borch-Johnsen K, Colagiuri S. Diagnosing diabetes—time for a change? *Diabetologia* 2009;52:2247–2250
25. Herman WH, Ma Y, Uwaifo G, et al.; Diabetes Prevention Program Research Group. Differences in A1C by race and ethnicity among patients with impaired glucose tolerance in the Diabetes Prevention Program. *Diabetes Care* 2007;30:2453–2457