Published in final edited form as:
Diabetes Care. 2000 November ; 23(11): 1619–1629.

The Diabetes Prevention Program:

Baseline characteristics of the randomized cohort

The Diabetes Prevention Program Research Group
From the Diabetes Prevention Program Coordinating Center, the Biostatistics Center, George Washington University, Rockville, Maryland.

Abstract

OBJECTIVE—The Diabetes Prevention Program (DPP) is a 27-center randomized clinical trial designed to evaluate the safety and efficacy of interventions that may delay or prevent development of diabetes in people at increased risk for type 2 diabetes.

RESEARCH DESIGN AND METHODS—Eligibility requirements were age ≥25 years, BMI ≥24 kg/m^2 (≥22 kg/m^2 for Asian-Americans), and impaired glucose tolerance plus a fasting plasma glucose of 5.3–6.9 mmol/l (or ≤6.9 mmol for American Indians). Randomization of participants into the DPP over 2.7 years ended in June 1999. Baseline data for the three treatment groups—intensive lifestyle modification, standard care plus metformin, and standard care plus placebo—are presented for the 3,234 participants who have been randomized.

RESULTS—Of all participants, 55% were Caucasian, 20% were African-American, 16% were Hispanic, 5% were American Indian, and 4% were Asian-American. Their average age at entry was 51 ± 10.7 years (mean ± SD), and 67.7% were women. Moreover, 16% were <40 years of age, and 20% were ≥60 years of age. Of the women, 48% were postmenopausal. Men and women had similar frequencies of history of hypercholesterolemia (37 and 33%, respectively) or hypertension (29 and 26%, respectively). On the basis of fasting lipid determinations, 54% of men and 40% of women fit National Cholesterol Education Program criteria for abnormal lipid profiles. More men than women were current or former cigarette smokers or had a history of coronary heart disease. Furthermore, 66% of men and 71% of women had a first-degree relative with diabetes. Overall, BMI averaged 34.0 ± 6.7 kg/m^2 at baseline with 57% of the men and 73% of women having a BMI ≤30 kg/m^2. Average fasting plasma glucose (6.0 ± 0.5 mmol/l) and HbA_{1c} (5.9 ± 0.5%) in men were comparable with values in women (5.9 ± 0.4 mmol/l and 5.9 ± 0.5%, respectively).

CONCLUSIONS—The DPP has successfully randomized a large cohort of participants with a wide distribution of age, obesity, and ethnic and racial backgrounds who are at high risk for developing type 2 diabetes. The study will examine the effects of interventions on the development of diabetes.

Abbreviations

CHD, coronary heart disease; CoC, Coordinating Center; CV, coefficient of variation; CVD, cardiovascular disease; DPP, Diabetes Prevention Program; DPS, Diabetes Prevention Study; ECG, electrocardiogram; IGT, impaired glucose tolerance; NCEP, National Cholesterol Education Program; NHANES, National Health and Nutrition Examination Survey; OGTT, oral glucose tolerance test

Address correspondence and reprint requests to the Diabetes Prevention Program Coordinating Center, the Biostatistics Center, George Washington University, 6110 Executive Blvd., Suite 750, Rockville, MD 20852. E-mail:dppmail@biostst.bsc.gwu.edu..

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

The Diabetes Prevention Program (DPP) is a randomized clinical trial being conducted in 27 centers in the U.S. The rationale and design of the study have been described in detail (1). Briefly, the purpose of the DPP is to determine whether the progression to diabetes from a state of impaired glucose tolerance (IGT) can be prevented or delayed. The DPP is examining two strategies: *1*) an intensive lifestyle intervention that includes moderate-intensity exercise to achieve and sustain at least 150 min per week of exercise together with a healthy diet to achieve and maintain at least a 7% loss of body weight or *2*) 850 mg metformin taken twice a day. These two intervention groups are compared with a group given standard lifestyle recommendations plus twice-daily placebo tablets. A fourth study intervention using troglitazone was discontinued in 1998 because of safety concerns (1).

The primary outcome of the DPP is the development of diabetes on an annual oral glucose tolerance test (OGTT) or semiannual fasting glucose measurement using the criteria of the American Diabetes Association (fasting plasma glucose ≥7.0 mmol/l or 2-h plasma glucose ≥11.1 mmol/l after a 75-g OGTT that must be confirmed with repeat testing [2]). Secondary outcomes of the DPP include the progression of defects in insulin sensitivity and diminished insulin secretion as well as the development and/or progression of vascular diseases, obesity, and cardiovascular risk factors.

IGT is conventionally defined as a plasma glucose level of 7.8–11.0 mmol/l 2 h after ingestion of 75 g glucose in the setting of nondiabetic fasting levels (2,3). Individuals with IGT are at increased risk for development of type 2 diabetes (4–6). However, IGT may revert to normal glucose tolerance and may not progress inexorably to diabetes (7–9). Indeed, a great deal of heterogeneity exists in the rates of progression to diabetes in different populations (10). Recent data suggest that the annual rates of progression to type 2 diabetes from IGT range from 2.3% per year to ~11% per year with higher rates in non-white racial/ethnic groups (2–4,10). In a recent analysis of six population-based cohorts, the average conversion rate was estimated at 5.8% per year (10).

In addition, some studies suggest that a higher fasting plasma glucose level confers an increased risk of conversion to diabetes (10–12). Based on these studies and also to maximize the chances of demonstrating prevention of diabetes with a practical sample size and time frame, eligibility for the DPP required both IGT and a fasting plasma glucose between 5.3 and 6.9 mmol/l (or ≤6.9 mmol/l for American Indians). Inclusion criteria for DPP initially (before June 1997) permitted fasting plasma glucose between 5.6 and 7.7 mmol/l (or ≤7.7 mmol/l for American Indians) (1). An additional study goal was to recruit 50% of participants from racial/ethnic groups with high prevalence rates of diabetes. The conversion rate using these combined criteria was estimated to be 7.7 per 100 person-years (10), and the final DPP sample size calculation was based on a conversion rate of 7.5 per 100 person-years (1).

IGT is not only associated with progression to type 2 diabetes but is independently a risk factor for coronary heart disease (CHD) (13), although not for microvascular complications (6). In addition, IGT and type 2 diabetes have a number of risk factors in common, including obesity, advanced age, prior gestational diabetes, family history of type 2 diabetes, a predilection for some racial/ethnic groups, dyslipidemia, and insulin resistance (14–18). This overlap plausibly argues that IGT is a predecessor of type 2 diabetes, but the specific contribution of each risk factor to progression from IGT to type 2 diabetes has not been quantified. The goal of the DPP was to include individuals with a variety of these risk factors and particularly participants from U.S. racial and ethnic minorities and older individuals (≥60 years of age).

The DPP was designed with an intention-to-treat analysis plan (1,19,20). The final sample size (3,234 participants) for the three-group study will provide >90% power to detect a 33% reduction in the primary outcome of conversion to diabetes with a level of significance of 5%

(two-sided), adjustment for losses to follow-up, and pairwise comparisons among three groups (1). Participants were randomized over a 2.7-year period and are to be followed for a total of 3.3–6.0 years. Interim results are reviewed by a data and safety monitoring committee to protect patient welfare. The study is scheduled to complete participant follow-up in June 2002.

The present report presents the baseline demographic and biomedical characteristics and describes the major outcome variables that were measured at entry. To compare the DPP participants with the general U.S. population, we present an analysis of the participants in the National Health and Nutrition Examination Survey (NHANES) III with glycemia, BMI, and age comparable to the participants in the DPP (21,22).

RESEARCH DESIGN AND METHODS

Participants

Individuals were recruited from a variety of sources including informational mailings, open screenings, advertisements, and referrals from health care professionals that were based on the individual's perceived risk for development of diabetes. Informed consent was obtained from all participants before screening, consistent with the Helsinki Declaration and the guidelines of each center's institutional review board (the APPENDIX provides a full list of the DPP Research Group membership and clinical centers). Individual consent procedures were used for each step of the study. A four-step consent procedure addressed specific phases of screening and recruitment and of study participation. The initial screening step consisted of a fasting glucose level, with eligibility to continue screening on the basis of the test method: a reflectance glucose meter using a sample of capillary blood (4.2–7.8 mmol/l) or venous blood (3.6–7.8 mmol/l), or a fasting plasma glucose test performed with a glucose analyzer (4.5–7.8 mmol/l). Participants were asked to fast for 12–14 h and to refrain from smoking, exercise, or other unusual activity before the testing of fasting plasma glucose. After this initial step, on the same day or a different day, a 75-g OGTT was administered from which final eligibility was determined.

Additional inclusion criteria were age ≥25 years and BMI ≥24 kg/m^2 (≥22 kg/m^2 for Asian-Americans because of difference in fat distribution in this population) (23). Major exclusions included a recent (i.e., within 6 months) myocardial infarction, symptoms of CHD, serious illness, or use of medications known to impair glucose tolerance, previously detailed (1). Screening of participants proceeded over 4–13 weeks before possible randomization.

Procedures

Initial screening consisted either of a fasting blood glucose test (performed on capillary or venous whole blood using a One Touch reflectance glucose meter [LifeScan, Milpitas, CA]) or, in a few clinics, a fasting plasma glucose test using a glucose analyzer. Study personnel were certified in the procedures for lancing the fingertip, drawing blood, and performing the tests. Reflectance devices and analyzers were checked for accuracy using established procedures. Biochemistry Laboratory–determined fasting plasma glucose from a venous sample before the OGTT was ultimately used to establish eligibility regardless of initial screening methods.

The OGTT was preceded by instructions to consume a usual diet with adequate carbohydrates and was initiated between 0700 and 1100 after an overnight fast. Blood was sampled from a vein before oral glucose (0 min) and then after 75 g flavored glucose (Trutol 75; Custom Laboratories, Baltimore, MD). Blood was drawn during the fasting state for plasma glucose, insulin, and proinsulin. Additional blood samples were obtained at 30 min (for plasma glucose and insulin) and 120 min (for plasma glucose). Fasting specimens for lipids and HbA_{1c} were

obtained in eligible participants immediately before randomization. The average time interval between OGTT specimens and HbA_{1c} was 64 ± 18 days (mean ± SD).

Blood samples were collected and processed at each DPP clinical site following the standardized manual of operations (24). Whole-blood samples for HbA_{1c} analysis were shipped to the Biochemistry Laboratory by overnight express within 24 h of sample collection. Serum and plasma samples were stored at −20°C for a few days and then shipped on dry ice in batches to the Biochemistry Laboratory.

Standardized interviewer-administered questionnaires were used to obtain self-reported data on personal medical history, employment, education, family income, prior pregnancies, smoking, medications, drug or alcohol use, and family medical history. Race/ethnicity was self-reported using the questions used in the 1990 U.S. Census questionnaire (25). Additionally, dietary intake, body fat distribution, and physical activity levels at baseline were assessed, but they are not reported here. Clinic staff were certified in the performance of a standard 12-lead electrocardiogram (ECG) as well as measurement of body weight, height, and blood pressure. Weight was measured in duplicate on a calibrated balance beam scale. Standing height was determined in duplicate with a standard stadiometer. Blood pressure was measured twice with a mercury sphygmomanometer; readings were obtained with the subject seated using procedures standardized in the manual of operations (24).

Measurements

All analytical measurements were performed at a central Biochemistry Laboratory (University of Washington, Seattle, WA). Plasma glucose was measured on a chemistry autoanalyzer by the glucokinase method (26). HbA_{1c} was measured by a dedicated ion-exchange high-performance liquid chromatography instrument (Variant; BioRad, Hercules, CA) (27). Insulin measurements were performed by a radioimmunoassay method using an anti–guinea pig antibody that measures total immunoreactive insulin. The assay is a 48-h polyethylene glycol–accelerated method with coefficients of variation (CVs) of 4.5% for high-concentration quality control samples and 6.9% for low-concentration quality control samples. The CV for masked split duplicates in this assay was <8.5%. Proinsulin was measured by a commercially available radioimmunoassay method (Linco Research, St. Louis, MO). Measurements of total plasma cholesterol and triglycerides were performed enzymatically (28) using methods standardized to the Centers for Disease Control and Prevention Reference Methods. HDL fractions for cholesterol analysis were obtained by the treatment of whole plasma with dextran sulfate-Mg^{2+} to precipitate all of the apo lipoprotein B–containing lipoproteins (29). LDL cholesterol was calculated by the equation of Friedewald et al. (30). In participants with triglycerides >4.5 mmol/l, the lipoprotein fractions were separated by preparative ultracentrifugation of plasma (31).

An ECG reading center (EPICARE Center, Wake Forest University School of Medicine, Winston-Salem, NC) analyzed and reported ECGs using the NOVACODE Program (32,33). This program also has algorithms for ECG coding according to the Minnesota Code (34). Myocardial infarction was classified using Minnesota coding and other ECG abnormalities using NOVA-CODE methodology.

Data management and analyses

Locally generated data from each participant were double-entered by certified clinic staff at each clinical site and checked for allowed ranges and internal consistency using distributed data entry software provided by the DPP Coordinating Center (CoC). Data from the network of microcomputers at the clinical sites and data from the central resource units (the Biochemistry Laboratory and the ECG reading center) were transferred electronically to a

database at the CoC. The central database was maintained, and all analyses were performed using the Statistical Analysis System (SAS Institute, Cary, NC).

The data reported here were obtained before randomization and are based on the database as of 5 January 2000. Baseline characteristics of randomized participants were examined by treatment group assignment, sex, and race/ethnicity (Caucasian, African-American, Hispanic, American Indian, and Asian-American). DPP data were also compared with the weighted NHANES III data for those participants matching the major DPP eligibility criteria (1,21).

RESULTS

Clinical and demographic characteristics

More than 158,000 individuals underwent screening, yielding 30,986 OGTTs performed across the study. Of these, 3,819 people were ultimately randomized, with 3,234 participants in the three-arm cohort.

The overall distribution by age, sex, racial/ethnic group classification, fasting glucose, BMI, and blood pressure of DPP participants by treatment group assignment is shown in Table 1. The overall mean (± SD) age at randomization was 50.6 ± 10.7 years. Nearly 68% of the participants were women. More than 45% of the DPP participants belonged to a U.S. minority racial or ethnic group; 54.7% were Caucasian, 19.9% African-American, 15.7% Hispanic, 5.3% American Indian, and 4.4% Asian-American.

Average fasting plasma glucose, BMI, and systolic and diastolic blood pressures reflected inclusion and exclusion criteria and were similar in the three treatment groups. Of note, only 54 participants (1.67%) who had been randomized before the new criteria for diabetes diagnosis would be classified as diabetic (2). These individuals had a standard assessment of glycemia at their next semiannual visit and continue to be followed in the DPP as per protocol (1).

As shown in Table 2, distributions by age and race/ethnicity differed by sex. Among women, a greater percentage was younger, and among men, a greater percentage older, although the majority of the cohort was 40–59 years of age. Nearly 31% of male and 15% of female DPP participants were 60 years of age or older. Among each racial/ethnic group, the majority of participants were women, except for Asian-Americans.

We also examined the distribution of participants by race/ethnicity, age, and sex in individual clinics (data not shown). Four clinics randomized predominantly American Indians; the total number of participants in these clinics ranged from 20 to 80 individuals per center. The remaining 23 centers randomized participants from more than one racial/ethnic group; the total number of participants in these centers ranged between 33 and 166 people per center (median 139 people). The majority of clinics enrolled >25% of participants from at least one non-Caucasian group.

The socioeconomic status of DPP participants and other demographic data at the time of randomization are also summarized in Table 2. Women were more likely than men to be unmarried upon entry into DPP, largely because a greater proportion were divorced. Of the women, 85% had had a prior pregnancy and 48% were post-menopausal upon entry into the study. Fewer men (48%) reported never having smoked cigarettes compared with women (64%).

Characteristics of participants by sex and racial/ethnic group

Other variables associated with IGT and type 2 diabetes are summarized in Table 3, stratified by sex and racial/ethnic group. Family history of type 2 diabetes among men and women by

ethnic grouping was similar, but history of gestational diabetes among women in the various racial/ethnic categories was most frequent among American Indian women. The prevalence of participant-reported history of high cholesterol and hypertension was similar for men and women and for all ethnic categories, except for the lower prevalence of high cholesterol among American Indians.

The distribution of age, BMI, blood pressure, glucose, insulin, and lipids by racial/ethnic group is shown in Tables 4 and 5. For both men and women, all of the minority groups—particularly American Indian—were younger than the Caucasians. Although inclusion criteria established the lower limits of BMI, there was no upper limit, so substantial proportions of participants (particularly women) had a baseline BMI ≥40 kg/m^2. Because Asian-Americans have lower BMIs than other racial groups, the entry criteria allowed BMI as low as 22 kg/m^2 in this group; thus, there were more Asian-American participants with BMI <30 kg/m^2. In all racial/ethnic categories, women had higher BMI levels than men. In both sexes, there were greater proportions of people in the higher BMI categories in the younger age-groups.

Mean seated systolic and diastolic arm blood pressures were 126 ± 14 and 80 ± 9 mmHg, respectively, in men and 123 ± 15 and 78 ± 9 mmHg, respectively, in women. To represent the prevalence of hypertension in the DPP cohort more accurately, we combined self-reports and measured blood pressure in participants as follows: participants were classified as having hypertension at baseline if they *1*) reported a physician diagnosis of hypertension and were taking blood pressure–lowering medication, or *2*) if their systolic blood pressure was ≥140 mmHg and/or diastolic blood pressure was ≥90 mmHg. Using these combined criteria, 27.5% of the DPP cohort had hypertension, with rates of 27.0% in Caucasians, 35.0% in African-Americans, 21.3% in Hispanics, 13.5% in American Indians, and 38.0% in Asian-Americans (data not shown).

Fasting plasma glucose values were similar across all racial/ethnic groups and by sex, although they were slightly lower in American Indians because of the different eligibility criteria for fasting plasma glucose. Two-hour plasma glucose levels after the OGTT were identical for men and women and also similar across racial/ethnic sub-groups. HbA1c averaged 5.9 ± 0.5% in both sexes, but a substantial fraction of participants had HbA_{1c} > 6.1%—the upper limit of the Biochemistry Laboratory's normal range. Overall, 30.4% of men and 28.2% of women had elevated HbA_{1c}; African-American men and women had the highest proportions with elevated values (64 and 54%, respectively), and the proportions were lowest in Caucasian men and women (22 and 19%, respectively). HbA_{1c} averages were equal by treatment group assignment (data not shown).

Fasting plasma insulin and proinsulin values in men showed no differences among the different racial/ethnic groups, although among women, both fasting insulin and proinsulin values were lowest among Asian-American women. The 30-min plasma insulin concentrations displayed greater variability, with the highest values observed among American Indian men and women relative to the other racial/ethnic groups.

Fasting lipid parameters are also shown in Tables 4 and 5. Overall, 44% of men and 60% of women had normal lipid profiles according to the National Cholesterol Education Program (NCEP) classification. Although total cholesterol values were similar among the racial/ethnic groups in men, Caucasian and Asian-American women had the highest total cholesterol. LDL cholesterol tended to be lowest among American Indians in both men and women, whereas plasma triglycerides were lower among African-American participants. HDL cholesterol levels were higher among African-Americans and Asian-Americans.

Cardiovascular disease

Sex-specific cardiovascular disease (CVD) data are summarized in Table 6. The absolute prevalence of a history of these major CVD events was low. The percentage with a history of myocardial infarction or stroke, or history of coronary artery bypass graft, was higher in men than in women, and ECG abnormalities were more frequent among men.

Comparison of DPP and NHANES III

To compare the DPP participants with the general U.S. population, we examined the characteristics of the NHANES III participants—a representative sample of the U.S. population. We confined this analysis to the 299 NHANES III participants with the same restrictions on the age, BMI, and fasting and 2-h plasma glucose level eligibility criteria for DPP participants. Because classification by OGTT in NHANES III was undertaken only in those subjects ≥40 years of age, we reanalyzed the DPP data to include only participants ≥40 years of age at baseline (n = 2,729). In contrast to the DPP, only 32% of the NHANES group had a first-degree relative with diabetes and 0.2% of women reported a history of gestational diabetes. As shown in Table 7, the results suggest that the DPP participants are younger, with a higher proportion of women and racial/ethnic minorities. DPP participants also self-reported a lower frequency of hypertension, and BMI and fasting plasma insulin concentrations were higher among the DPP participants. Finally, serum lipid profiles were more favorable in the DPP subset than in the NHANES group.

CONCLUSIONS

Our predetermined target was to include ~50% of participants from U.S. minority ethnic and racial groups, and >45% of the DPP cohort met those criteria. The remaining 55% self-identified as Caucasian likely represent a varied pool of individuals with ancestry from Europe as well as western Asia and the Middle East. The heterogeneity of the U.S. minority subgroups should also be noted, because the African-American designation included people of Afro-Caribbean descent, and the term “Hispanic” includes individuals from Latin America and the Caribbean without regard to racial admixture. American Indian participants in the DPP are concentrated in the Southwest because four centers recruited exclusively from regional tribes; small numbers of American Indian participants were randomized in other centers. Asian-Americans in the DPP included participants descended from Japanese, Chinese, other East Asian groups, Asian Indians, and Pacific Rim Australasian populations.

Given the paucity of data in the literature regarding the natural history of IGT in many racial/ethnic minority groups, the DPP may provide valuable data on the effectiveness of the DPP interventions in specific groups. Moreover, the baseline results already indicate that there may be significant heterogeneity in critical metabolic factors associated with an increased risk for developing type 2 diabetes. To the extent that younger age is associated with development of type 2 diabetes in populations with a strong genetic predisposition to type 2 diabetes (5,10), it is notable that the average ages of participants in those groups were lower than among Caucasian participants. In recent studies, the rate of conversion from IGT to type 2 diabetes was highest among younger adults in only three subgroups of individuals: Pima Indians, Nauruans, and Hispanics (10). Among Caucasians, the same study found that IGT progression rates increased with older age (10).

Associated risk factors for CHD were also highly prevalent in the DPP cohort. A history of and/or treatment for hypertension was present in 27% of individuals, despite the fact that individuals were excluded from participation in the DPP because of the use of antihypertensive medications such as thiazide diuretics and β-blockers. These latter exclusions may have accounted for the lower frequency of hypertension in DPP compared with NHANES III

participants, although a number of differences between these two groups were likely also because of the larger female fraction of subjects who volunteered to participate in the DPP. The presence of severe CHD in the DPP is also likely to have been constrained by excluding people with severe symptoms or recent myocardial infarction or other major vascular events (1).

Because the DPP selected participants who were overweight, this cohort has many obese individuals. Although the majority of participants had a BMI <40 kg/m^2, BMI ≥40 kg/m^2 was present in 8% of men and 21% of women. On the other hand, older participants tended to be leaner; half of the participants who were 60 years of age or older had BMI between 25 and 30 kg/m^2. These cross-sectional data do not, however, exclude a cohort effect, in that the contribution of body fat to impairment in glucose tolerance may be complex, and this older subset of DPP participants may have a differing set of risk factors for IGT than younger, more obese participants. Alternatively, "selective mortality" among people as they age may explain the difference in obesity prevalence. Given the heterogeneity in BMI, analyses of outcome results may have to be adjusted accordingly, taking into account additional factors such as age and fat distribution.

Dyslipidemia was present in a large proportion of participants. More than 37% of men and 33% of women reported a history of and/or treatment for high cholesterol. Serum lipid values fulfilled NCEP criteria for diet or drug treatment for hyper-lipidemia in 54% of men and 40% of women. These results point to the powerful association among the risk factors for cardiovascular disease in the presence of IGT.

The DPP baseline findings should be placed in the context of available information regarding the characteristics of people with IGT or comparable hyperglycemia. In two observational studies conducted in the U.K.—the Bedford (35) and Whitehall (36) studies—the average age of the subjects (~56 years) was greater than that in the DPP cohort, and the average BMI (27–28 kg/m^2) was lower. An additional observational study of 474 Mexican-Americans in San Antonio, Texas, has been reported (37): the average age (42 years) and BMI (28 kg/m^2) among these subjects were lower than those in the DPP, but among those individuals who went on to develop type 2 diabetes (n = 28), fasting plasma glucose, 2-h plasma glucose, and fasting insulin values were comparable with corresponding values in the DPP cohort. The Malmö, Sweden (38), and the Da Qing, China (39), studies were prospective interventional studies that aimed at preventing the progression from IGT to diabetes. In both studies, age (45–48 years) and BMI (~26 kg/m^2) were lower than those in the DPP. A smaller randomized clinical trial, the Malmöhus study, suggested that tolbutamide decreases progression to diabetes (11) and mortality rates caused by CVD (40).

All of the above studies included subjects with IGT, but fasting plasma glucose ranged from 5.0 mmol/l (36) to ~8.3 mmol/l (35), and mean fasting plasma insulin concentrations averaged 72–144 pmol/l (although plasma insulin values were reported only in the Mexican-Americans [37] and in the Bedford study [36]). Among 446 Mexican-Americans who did not progress to diabetes, the baseline fasting plasma insulin concentration was only 72 pmol/l (37). In a smaller cohort of individuals with IGT from Nauru, an association between fasting and 2-h plasma insulin concentrations and progression to diabetes has been reported (41); the mean fasting insulin concentration in these subjects was 126 pmol/l. Overall, the DPP cohort includes individuals who are more overweight and hyperinsulinemic and less hypertensive than the subjects in these other studies. The DPP participants may also be less susceptible to hypertension-related morbid events that may confound the secondary CVD outcomes attributed to IGT or hyperglycemia per se (42).

Two recent studies have been organized with the intention to prevent type 2 diabetes. The Diabetes Prevention Study (DPS) in Finland is a prospective trial including 523 overweight subjects with IGT that is designed to test the efficacy of a lifestyle intervention (43). The trial's published 1-year interim findings (43) and announced 4-year results (44) indicate that an intensive diet and exercise program was effective in preventing type 2 diabetes. Of note, at baseline, the average age of DPS subjects (55 years), their sex distribution (33% men), and mean BMI (31 kg/m^2) were virtually identical to those of the DPP. A second study testing the use of acarbose in preventing the conversion of IGT to type 2 diabetes is the STOP-NIDDM Trial (45). In this study, 1,418 subjects with IGT and a fasting plasma glucose between 5.6 and 7.8 mmol/l were randomized. Preliminary data in these subjects indicate some similarities to the DPP cohort, with a mean age of 54.8 years and 78% having a BMI >27 kg/m^2. Despite the absence of precise details such as the sex and ethnic distribution of this group, there also appear to be significant differences from the DPP participants; hypertension (48%), dyslipidemia (51%), and family history of diabetes (93%) were all more frequent than in the DPP.

A substantial fraction of DPP participants had HbA_{1c} > 6.1%—the upper limit of assay normal range—consistent with the enrollment criteria for fasting and postchallenge hyperglycemia. It is remarkable, however, that the proportions of participants with elevated HbA_{1c} were highest in the minority racial/ethnic groups, despite comparable fasting and 2-h plasma glucose levels across these subgroups. The difference in fasting glucose entry criteria (1) did not account for the divergence of groups with elevated HbA_{1c}, and only a small number (n = 54) of participants in the DPP were randomized under the former eligibility criteria. These observations suggest that elevated HbA_{1c} levels may precede the development of type 2 diabetes as defined by current American Diabetes Association and World Health Organization criteria (46). Alternatively, these data could be consistent with the conclusions from previous studies in subjects without diabetes (47,48) that significant variability in HbA_{1c} may reflect factors such as erythrocyte turnover rates. The large fractions of minority subgroups with an elevated HbA_{1c} are all the more puzzling, because both fasting and 2-h plasma glucose levels were similar to those in Caucasians. The observed HbA_{1c} variability among ethnic/racial groups is unlikely to reflect different glycemic exposures before randomization, because participants were carefully followed through the prerandomization run-in phase, and the average duration of the interval between the OGTT and HbA_{1c} determinations was similar for all of the groups. Notably, the groups with the highest proportion of participants with an elevated HbA_{1c} were neither more obese nor hyperinsulinemic, and preparation for the OGTT was standardized. Whether some of the low and high HbA_{1c} values were secondary to the presence of sickle cell hemoglobin, high levels of fetal hemoglobin, or other hemoglobinopathies, which were not criteria for exclusion, is not known. Finally, secondary analyses of factors such as dietary intake and physical activity along with age, sex, and ethnicity may shed light on these findings.

In conclusion, the data obtained at baseline in the participants randomized to the DPP three-group protocol indicate that the study has recruited an appropriate cohort in which to test the trial questions. These participants represent a cohort comprising older individuals as well as subjects from U.S. minority racial/ethnic groups—all of which are groups with higher risk of developing type 2 diabetes. The participants have a variety of metabolic characteristics associated with increased risk for diabetes and CVD, including obesity, hyperglycemia, hyperin-sulinemia, and dyslipidemia.

Acknowledgements

Funding was provided by the National Institutes of Health through the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK), the Office of Research on Minority Health, the National Institute of Child Health and Human Development, and the National Institute on Aging. In addition, the Indian Health Service, the Centers for Disease Control and Prevention, the American Diabetes Association, and two pharmaceutical companies—Bristol-Myers Squibb and Parke-Davis—contributed support. All support to the clinical centers and the Coordinating Center

was provided through the NIDDK using the mechanism of the Cooperative Agreement, except for the Southwestern American Indian Center, which was supported directly by the NIDDK and the Indian Health Service.

We thank Katherine M. Flegal, PhD, at the Centers for Disease Control and Prevention for assistance in obtaining the data from NHANES III. We acknowledge the General Clinical Research Centers that assisted in the conduct of the DPP. Finally, we are indebted to the thousands of people who volunteered to participate in the DPP and all of our colleagues in the health care community who provide medical services to the participants.

References

1. The Diabetes Prevention Program Research Group. The Diabetes Prevention Program: design and methods for a clinical trial in the prevention of type 2 diabetes. Diabetes Care 1999;22:623–634. [PubMed: 10189543]
2. 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. [PubMed: 9203460]
3. Alberti KGMM, Zimmet PZ. the WHO Consultation. Definition, diagnosis and classification of diabetes mellitus and its complications. I. Diagnosis and classification of diabetes mellitus: provisional report of a WHO consultation. Diabet Med 1998;15:539–553. [PubMed: 9686693]
4. Yudkin JS, Alberti KGMM, McLarty DG, Swai GPM. Impaired glucose tolerance. BMJ 1990;301:397–402. [PubMed: 2282392]
5. Saad MF, Knowler WC, Pettitt DJ, Nelson RG, Mott DM, Bennett PH. The natural history of impaired glucose tolerance in the Pima Indians. N Engl J Med 1988;319:1500–1506. [PubMed: 3054559]
6. Eriksson KF, Saltin B, Lindgärde F. Increased skeletal muscle capillary density precedes diabetes development in men with impaired glucose tolerance. Diabetes 1994;43:805–808. [PubMed: 8194667]
7. Stern MP, Valdez RA, Haffner SM, Mitchell BD, Hazuda HP. Stability over time of modern diagnostic criteria for type II diabetes. Diabetes Care 1993;16:978–983. [PubMed: 8359106]
8. Saad MF, Knowler WC, Pettitt DJ, Nelson RG, Bennett PH. Transient impaired glucose tolerance in Pima Indians: is it important? Br Med J 1988;297:1438–1441. [PubMed: 3147006]
9. King H, Zimmet P, Raper LR, Balkau B. The natural history of impaired glucose tolerance in the Micronesian population of Nauru: a six-year follow-up study. Diabetologia 1984;24:39–43. [PubMed: 6706044]
10. 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 non-insulin-dependent diabetes mellitus: an analysis of six prospective studies. Diabetes 1997;46:701–710. [PubMed: 9075814]
11. Sartor G, Scherstén B, Carlström S, Melander A, Nordén A, Persson G. Ten-year follow-up of subjects with impaired glucose tolerance: prevention of diabetes by tolbutamide and diet regulation. Diabetes 1980;29:41–44. [PubMed: 7380107]
12. Kadowaki T, Miyake Y, Hagura R, Akanuma Y, Kajinuma H, Kuzuya N, Takaku F, Kosaka K. Risk factors for worsening to diabetes in subjects with impaired glucose tolerance. Diabetologia 1984;26:44–49. [PubMed: 6368299]
13. Donahue RP, Orchard TJ. Diabetes mellitus and macrovascular complications: an epidemiological perspective. Diabetes Care 1992;15:1141–1155. [PubMed: 1396012]
14. American Diabetes Association. Screening for diabetes (Position Statement). Diabetes Care 1990;13 (Suppl1):7–9.
15. Barrett-Connor E. Epidemiology, obesity and non-insulin-dependent diabetes mellitus. Epidemiol Rev 1989;11:172–181. [PubMed: 2680554]
16. Rewers M, Hamman RF: Risk factors for non-insulin-dependent diabetes. In Diabetes in America. 2nd ed. Washington, DC, National Diabetes Data Group, 1995 (NIH publ. no. 95-1468)
17. Jarrett RJ. Epidemiology and public health aspects of non-insulin-dependent diabetes mellitus. Epidemiol Rev 1989;11:151–171. [PubMed: 2680553]
18. West KM: Epidemiology of Diabetes and its Vascular Lesions. New York, Elsevier, 1978
19. Lachin JM. Statistical considerations in the intent-to-treat principle. Control Clin Trials 2000;21:167–189. [PubMed: 10822117]

20. DPP Research Group. Diabetes Prevention Program: the U.S. type 2 diabetes prevention trial. Diabetes Care 2000;23(suppl 2):B11–B13. [PubMed: 10860185]
21. Third National Health and Nutrition Examination Survey (NHANES III), 1988–1994. CD-ROM (series 11, no. 1A and 2A). Centers for Disease Control and Prevention, Altalnta, GA
22. Harris MI, Goldstein DE, Flegal KM, Little RR, Cowie CC, Wiedmeyer H-M, Eberhard MS, Byrd-Holt DD. Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in U.S. adults. Diabetes Care 1998;21:518–524. [PubMed: 9571335]
23. Newell-Morris LL, Treder RP, Shurman WP, Fujimoto WY. Fatness, fat distribution, and glucose tolerance in second-generation Japanese American (Nisei) men. Am J Clin Nutr 1989;50:9–18. [PubMed: 2750700]
24. Manual of Operation. Diabetes Prevention Program, DPP Coordinating Center, The George Washington University Biostatistics Center, Rockville, MD
25. Bureau of the Census: Census of the Population, 1990. Washington, DC, U.S. Government Printing Office, 1990
26. Peterson JI, Young DS. Evaluation of the hexokinase-glucose-6-phosphate deydrogenase method of determination of glucose in urine. Anal Biochem 1968;23:301–316. [PubMed: 5657801]
27. Ellis G, Diamandis EP, Giesbrecht EE, Daneman D, Allen LC. An automated high pressure liquid-chromatographic assay for hemoglobin A1c. Clin Chem 1984;30:1746–1752. [PubMed: 6488517]
28. Warnick GR. Enzymatic methods for quantification of lipoprotein lipids. Methods Enzymol 1986;129:101–123. [PubMed: 3724535]
29. Warnick GR, Benderson J, Albers JJ. Dextran sulfate-Mg^{2+} precipitation procedure for quantification of high-density lipoprotein cholesterol. Clin Chem 1982;28:1279–1288.
30. 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 1972;18:499–502. [PubMed: 4337382]
31. Hainline A Jr, Karon J, Lippel K (Eds.): Manual of Laboratory Operations. 2nd ed. Lipid Research Clinics Program, Lipid and Lipoprotein Analysis, U.S. Department of Health and Human Services, Public Health Service, National Institutes of Health (updated 1983)
32. Rautaharju PM, MacInnis PJ, Warren JW, Wolf HK, Rykers PM, Calhoun HP. Methodology of ECG interpretation in the Dalhousie Program: NOVACODE ECG classification procedures for clinical trials and population health surveys. Methods Inf Med 1990;26:362–374. [PubMed: 2233384]
33. Rautaharju PM, Calhoun HP, Chaitman BR. NOVACODE serial ECG classification system for clinical trials and epidemiological studies. J Electrocardiol 1992;24:179–187. [PubMed: 1552254]
34. Prineas RJ, Crow R, Blackburn H: The Minnesota Code Manual of Electrocardiographic Findings. Littleton, MA, John Wright-PSG, 1982
35. Keen H, Jarrett RP, McCartney PM. The ten-year follow-up of the Bedford Survey (1962–1972): glucose tolerance and diabetes. Diabetologia 1982;22:73–78. [PubMed: 7060852]
36. Jarrett RP, Keen H, Fuller JH, McCartney PM. Worsening to diabetes in men with impaired glucose tolerance (“borderline diabetes”). Diabetologia 1979;16:25–30. [PubMed: 761734]
37. Haffner SM, Stern MP, Mitchell BD, Hazuda HP, Patterson JK. Incidence of type II diabetes in Mexican Americans predicted by fasting insulin and glucose levels, obesity, and body-fat distribution. Diabetes 1990;39:283–288. [PubMed: 2407581]
38. Eriksson K-F, Lindgärde F. Prevention of type 2 (non-insulin-dependent) diabetes mellitus by diet and physical exercise: the 6-year Malmö feasibility study. Diabetologia 1991;34:891–898. [PubMed: 1778354]
39. Pan X, Li G, Hu Y, Wang J, Yang W, An Z, Hu Z, Lin J, Xiao J, Cao H, Liu P, Jiang X, Jiang Y, Wang J, Zheng H, Zhang H, Bennett PH, Howard BV. 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. [PubMed: 9096977]
40. Knowler WC, Sartor G, Melander A, Scherstén B. Glucose tolerance and mortality, including a substudy of tolbutamide treatment. Diabetologia 1997;40:680–686. [PubMed: 9222648]
41. Sicree RA, Zimmet PZ, King HOM, Coventry JS. Plasma insulin among Nauruans: prediction of deterioration in glucose tolerance over 6 years. Diabetes 1985;36:179–186. [PubMed: 3542644]

42. Balkau B, Shipley M, Jarrett RJ, Pyörälä K, Pyörälä M, Forhan A, Eschwège E. High blood glucose concentration is a risk factor for mortality in middle-aged nondiabetic men. Diabetes Care 1998;21:360–367. [PubMed: 9540016]

43. Eriksson J, Lindstrom J, Valle T, Aunola S, Hamalainen H, Ilanne-Parikka P, Keinanen-Kiukaanniemi S, Laakso M, Lauhkonen M, Lehto P, Lehtonen A, Louheranta A, Man-nelin M, Martikkala V, Rastas M, Sundvall J, Turpeinen A, Viljanen T, Uusitupa M, Tuomilehto J. Prevention of type II diabetes in subjects with impaired glucose tolerance: the Diabetes Prevention Study (DPS) in Fin-land: study design and 1-year interim report on the feasibility of the lifestyle intervention programme. Diabetologia 1999;42:793–801. [PubMed: 10440120]

44. Tuomilehto J, Lindstrom J, Eriksson J, Valle T, Louheranta A, Ilanne-Parikka P, Salminen V, Hamalainen H, Aunola S, Keinanen-Kiukaanniemi S, Laakso M, Mannelin M, Sundvall J, Uusitupa M, DPS Study Group: Type 2 diabetes can be prevented by lifestyle intervention: the final results from the randomized Finnish Diabetes Prevention Study (DPS). Poster presented at the 60th Scientific Sessions, American Diabetes Association, June 9–13, 2000, San Antonio, TX

45. Chiasson J-L, Gomis R, Hanefeld M, Josse RG, Karasik A, Laasko M. the STOP-NIDDM Trial Research Group. The STOP-NIDDM Trial: an international study on the efficacy of an alpha-glucosidase inhibitor to prevent type 2 diabetes in a population with impaired glucose tolerance: rationale, design, and preliminary screening data. Diabetes Care 1998;21:1720–1725. [PubMed: 9773737]

46. Little RR, England JD, Wiedmeyer HM, McKenzie EM, Pettitt DJ, Knowler WC, Goldstein DE. Relationship of glycosolated hemoglobin to oral glucose tolerance: implications for diabetes screening. Diabetes 1988;37:60–64. [PubMed: 3335278]

47. Kilpatrick ES, Maylor PW, Keevil BG. Biological variation of glycated hemoglobin: implications for diabetes screening and monitoring. Diabetes Care 1998;21:261–264. [PubMed: 9539993]

48. Simon D, Senan C, Balkau B, Saint-Paul M, Thibult N, Eschwège E. Reproducibility of HbA_{1c} in a healthy adult population: the Telecom Study. Diabetes Care 1999;22:1361–1363. [PubMed: 10480783]

APPENDIX

The DPP Research Group up to the end of randomization

Pennington Biomedical Research Center —G.A. Bray, MD*; I.W. Culbert, BSN, RN, CCRC**; C.M. Champagne, PhD, RD; L. Dawson, B. Eberhardt, RD, LDN; F.L. Green-way, MD; F.G. Guillory, LPN; A.A. Hebert, RD; M.L. Jeffirs, LPN; B.M. Kennedy, MPA; J.C. Lovejoy, PhD; L.E. Melancon, BA, BS; L.H. Morris, BS; L. Reed; J. Perault; D.H. Ryan, MD; D.A. Sanford, LPN; K.G. Smith, BS, RTR; L.L. Smith, BS; J.A. St. Amant, PTR; E. Tucker; R.T. Tulley, PhD; P.C. Vicknair, MS, RD; D. Williamson, PhD; and J.J. Zachwieja, PhD. *University of Chicago* — J.A. Tobian, MD, PhD*; M.J. Matulik, RN, BSN, CCRC**; B. Clarke, MD; D.A. Collins, BS, K.B. Czech, MS; C. DeSandre, BA; D.A. Ehrmann, MD; G. Geiger, MPA; B. Harding-Clay, CMA, CCRC; R.M. Hilbrich, RD; W.L. McNabb, EdD; K.S. Polonsky, MD; A.R. Semenske, MS, RD; and K.A. Stepp, MS. *Jefferson Medical College* — P.G. Watson, RN, ScD*; J.T. Mendoza, BSN, RN**; K.A. Smith, BSN, RN**; B.J. Goldstein, MD, PhD; C. Lark; R. Liberoni, MPH; L. Murphy, AS; C. Pepe, RD, MS; and J.M. Spandorfer, MD. *University of Miami* — R.B. Goldberg, MD*; P. Rowe, MPA**; J. Calles, MSEd; P. Casanova, MD; R.P. Donahue, PhD; H.J. Florez, MD; A. Giannella, RD, MS; V. McLymont, RN, BS; J. Mendez; P. O'Hara, PhD; J. Ojito; and R. Prineas, MD, PhD. *The University of Texas Health Science Center* —S.M. Haffner, MD, MPH*; M.G. Montez, RN, MSHP**; H. Miettinen, MD, PhD; C.M. Mobley, PhD; L.A. Mykkanen, MD, PhD; and M.M. Rozek, MD. *University of Colorado* — R.F. Hamman, MD, DrPH*; P.V. Nash, MS**; B.N. Calonge, MD, MPH; J.O. Hill, PhD; S.R. Hoyer, BS; B.T. Jortberg, MS, RD, CDE; M. Miller-Stone, MS, RD; J.G. Regensteiner, PhD; H. Seagle, MS, RD; S.C. Steinke, MS; L. Testaverde, MS; and B. Van-Dorsten, PhD. *Joslin Diabetes Center* — E.S. Horton, MD*; K.E. Lawton, RN**; R.A. Arky, MD; M. Bryant; J.P. Burke, BSN; E. Caballero, MD; K.M. Callaghan, BA;

O.P. Ganda, MD; T. Franklin; S.D. Jackson, MS, RD; A.M. Jacobsen, MD; L.M. Kula, RD; M. Kocal, RN, CDE; M.A. Malloy, BS; M. Nicosia, MS, RD; C.F. Oldmixon, RN; J. Pan, BS, MPH; M. Quitingon; S. Rubtchinsky, BS; E.W. Seely, MD; D. Schweizer, BSN; D. Simonson, MD; F. Smith, MD; C.G. Solomon, MD, MPH; and J. Warram, MD. *University of Washington* — S.E. Kahn, MB, ChB*; B.K. Montgomery, RN, BSN**; W.Y. Fujimoto, MD; R.H. Knopp, MD; E.W. Lip-kin, MD; M. Marr; B.S. McCann, PhD; T.T. Nguyen, BA; J.P. Palmer, MD; and R.S. Schwartz, MD. *University of Tennessee* —A.E. Kitabchi, PhD, MD*; M.E. Murphy, RN, MS, CDE, MBA**; W.B. Applegate, MD, MPH; M. Bryer-Ash, MD, MRCP (U.K.), FRCP (C); A. Crisler; G. Cunningham; A.W. Franklin, RN, BSN; S.L. Frieson, RN; D.L. Green, RN; R. Imseis, MD; C.L. Kennedy, PhD; H.C. Lambeth, RN, BSN; L.C. Lichtermann, RN, BSN; H. Oktaei, MD; M.L. O'Toole, PhD; H. Ricks; L.M.K. Rutledge, RN, BSN; A.R. Sherman, MS, RD, CDE; C.M. Smith, RD, MPH; J.E. Soberman, MD; and B.J. Williams-Cleaves, MD. *Northwestern University Medical School* — B.E. Metzger, MD*; M.K. Johnson, MS, RN**; C. Behrends, BS; M.L. Cook, MS; M. Fitzgibbon, PhD; D. Heard, MA; D. McPherson, MD; M.E. Molitch, MD; M. Moore, MS, RD; T. Pitts, MD; S. Roston, RN, RD; and P.A. Schinleber, RN, MS. *Massachusetts General Hospital* — D.M. Nathan, MD*; C. McKitrick, BSN**; K. Abbott; E. Anderson, MS, RD; L. Bissett, MS, RD; E. Cagliero, MD; S. Crowell, BSN; L. Delahanty, MS, RD; S. Fritz, RN; E. Levina, BA; T. Michel, MS, PT; D. Norman, PhD; J. O'Keefe, PhD; A. Poulos, BA; L. Ronan, MD; M. Rosal, PhD; M. Salerno, BA; C. Shagensky, BA; B. Steiner, EdM; H. Turgeon, RN, BSN; and A. Young, MPH, CHES. *University of California, San Diego* — J.M. Olefsky, MD*; M.L. Carrion-Petersen, RN, BSN**; E. Barrett-Connor, MD; M. Beltran, RN, BSN, CDE; K. Caenepeel-Mills; S.V. Edelman, MD; R.O. Ford; J. Garcia; R.R. Henry, MD; M. Hill; J. Horne, RD; D. Leos; S. Mudaliar, MD; A. Pollard; and J. Torio-Hurley. *St. Luke's-Roosevelt Hospital* — F.X. Pi-Sunyer, MD*; J.E. Lee, MS**; D.B. Allison, PhD; N.J. Aronoff, MS, RD; I.M. Barreras; J.P. Crandall, MD; S.T. Foo, MD; S.J. Orlando, BA; K. Parkes, RN; E.S. Rooney, BA; G.E.H. Van Wye, MA; and K.A. Viscovich, ANP. *Indiana University* — D.G. Marrero, PhD*; M.S. Kukman-Kelly, RN, CDE**; Y.F. Dotson, BS; S.E. Fineberg, MD; J.C. Guare, PhD; A. Hadden; J.M. Ignaut, MA; M.A. Jackson; M.S. Kirkman, MD; B.D. Porter, MSN, RNC, CDE; M.J. Prince, MD; and M.L. Wheeler, MS, RD, CDE. *MedStar Research Institute* — R.E. Ratner, MD*; G. Youssef, RD, CDE**; A. Bonar, MD; M. Bronsord, MS, RD, CDE; E. Brown; W.W. Cheatham, MD; S. Cola; A. Comfort; G. Boggs, MSN, RN; C. Evans; C. Levetan, MD; T. Kellum, MS, RD, CDE; A.K. Nair, BS; M.D. Passaro, MD; R. Rubin, PhD; and S. Shapiro, BS, RN. *UCLA Medical School* — M.F. Saad, MD*; M. Budgett**; S.D. Jinagouda, MD**; A. Aziz, MD; B. Bernaba, MD; S.L. Bodkin; V. Ciobanu; C. Cosenza, RD; J.E. Hagar, BS; A. Khan, MD; D. Kumar, MD; Q. Khawaja, MD; G. Lui; A.R. Marston, PhD; V. Mehta, MD; A.R. Sharma, MD; K. Szamos, RD; A. Vargas; B. Vargas; and N. Zambrana. *Washington University, St. Louis* — S. Dagogo-Jack, MD*; A.S. Santiago, RN**; J.S. Brendle, MT; E.B. Fisher Jr., PhD; D.C. Gherardini, RN; J.M. Heins, RD; J.M. Marsala, RN; C.C. Raspberry, MSW; and J.V. Santiago, MD. *Johns Hopkins School of Medicine* — C.D. Saudek, MD*; V.L. Bradley, BA**; F.L. Brancati, MD, MHS; S. Cappelli, BA; J.B. Charleston, RN, MSN; R.R. Rubin, PhD; K.J. Stewart, EdD; and E. Sullivan, MEd, RN. *University of New Mexico School of Medicine* — D.S. Schade, MD*; K.S. Adams, RN, MSN**; L.F. Atler, PhD; D.A. Bowling; P.J. Boyle, MD; M.R. Burge, MD; L. Butler, BUS; J.L. Canady, RN, CDE; L. Chai, RN; M. Guillen, RD; M. Gutierrez, RD; D. Hornbeck, RN; C. Johannes, RN, CDE; P. Katz, LPN; C. King; R. McCalman, RD; D.A. Montoya, RN; A. Rassam, MD; and W. Senter, RD. *Albert Einstein College of Medicine* — H. Shamoon, MD*; J.O. Brown, RN, MPH, MSN**; J. Adames, BS; E. Blanco, BA; L. Cox, MS, RD; H. Duffy, MS, C-ANP; S. Engel, MD; A. Friedler, BS; T. Harroun, BS; C.J. Howard-Century, MA; S. Kloiber, RN; N. Longchamp, LPN; D. Pompi, BA; E.A. Walker, RN, DNSc; J. Wylie-Rosett, EdD, RD; and J. Zonszein, MD. *University of Pittsburgh* — R.R. Wing, PhD*; M.K. Kramer, BSN, MPH**; S. Barr, BS; L. Clifford, BS; R. Culyba, BS; M. Frazier; R. Gilligan, BS; L. Harris, RN; S. Harrier, MLT; W. Henderson, RN, BSN; S. Jeffries, BSN, MSN; G. Koenning, MS, RD; K. Maholic, BS; M.

Mullen, MPH, RD; A. Noel, BS; T.J. Orchard, MBBCh, MedSci; A. Otto, MS, RD; M. Smith, BSN; V. Stapinski, BS; J. Viteri, MS; T. Wilson, BA; K.V. Williams, MD, MPH; and J. Zgibor, MPH, PhD. *University of Hawaii* — R.F. Arakaki, MD*; R.W. Latimer, BSN, MPH**; N.K. Baker-Ladao, BS; R.M. Bed-dow, MD; L.M. Dias; D.A. Dupont; L.L. Fukuhara, BSN, RN; M.K. Mau, MD; S.K. Odom, MPH, RD; R.U. Perry; and J.S. Tokunaga, BS. *Southwest American Indian Center for Diabetes Prevention* — W.C. Knowler, MD, DrPH*; N.J. Cooeyate**; M.A. Hoskin, MS, RD**; C.A. Percy, RN, MS**; K.J. Acton, MD, MPH; V.L. Andre, RN, FNP; S. Antone; N.M. Baptisto; R. Barber; P.H. Bennett, MB, FRCP; M.B. Benson, RN, BSN; E.C. Bird, MPH, RD; B.A. Broussard, RD, MPH, MBA, CDE; M. Chavez, RN, AS; T.S. Dacawyma, PTR; R. Duncan, RD; J.M. Ghahate, MPH; M. Glass, MD; D. Gohdes, MD; G. Grant, MD; R.L. Hanson, MD; E. Horse; G. Hughte; L.E. Ingraham, MS, RD, LN; M.C. Jackson, RMA, RPT; P.A. Jay; R.S. Kaskalla; D. Kessler, MD; K.M. Kobus, RNC-ANP; T. Morgan; Y. Nashboo; S. Poirier, MD; M. Reidy, MD; S. Ridpath, BS; J. Roumain, MD, MPH; D.H. Rowse, MD; R.J. Roy; S. Sangster, BS; J. Sewemaenewa; C. Wilson, MD; and M. Yazzie, BA. *Data Coordinating Center (George Washington University Biostatistics Center)* — S. Fowler, PhD*; R. Bain, PhD; J. Bamdad; T. Brenneman; J. Callaghan; S.L. Edelstein, ScM; K.L. Grimes; S. Jones; T.L. Jones; J.M. Lachin, ScD; P. Mucik; R. Orlosky; C.E. Stimpson; C. Suiter; E. Temprosa, MS; and C. Van Aerden. National Institute of Diabetes and Digestive and Kidney Diseases Program Office — R. Eastman, MD, and S. Garfield, PhD. National Institute on Aging — R. Andres, MD. Centers for Disease Control and Prevention — M.M. Engelgau, MD; K.M. Venkat Narayan, MD; and D.F. Williamson, PhD. University of Michigan — W.H. Herman, MD, MPH. Central Units: Central Biochemistry Laboratory — S.M. Marcovina, PhD, ScD*; A. Aldrich, BA; and W.L. Chandler, MD. *Epicare Center (Wake Forest University School of Medicine)* — P.M. Rautaharju, MD, PhD*; T. Alexander, ACS; N.T. Pemberton, BS; R. Prineas, MD, PhD; F.S. Razack Rautaharju, PhD; and Z. Zhang, MD. *Nutrition Coding Center (University of South Carolina)* — E.J. Mayer-Davis, PhD*, and M. Martin, MSPH. *Central Carotid Ultrasound Unit (New England Medical Center)* — D.H. O'Leary, MD*; L.R.C. Funk, MS; K.A. O'Leary, BS; and J.F. Polak, MD. *CT-scan Reading Unit* (*University of Colorado Health Sciences Center)* — E.R. Stamm, MD*, and A.L. Scherzinger, PhD. *Lifestyle Resource Core (University of Pittsburgh)* — R.R. Wing, PhD*; B.P. Gillis, MS, RD; A.M. Kriska, PhD; C. Huffmyer; and E.M. Venditti, PhD. *Quality of Well- Being Reading Unit* (*University of California, San Diego)* — R.M. Kaplan, PhD*; K.M. David, MPH; and W.J. Sieber, PhD.

*Principal investigator; **program coordinator.

Table 1

Participant characteristics by treatment group assignment

	Overall	Lifestyle	Metformin	Placebo
n	3,234	1,079	1,073	1,082
Age (years)*	50.6 ± 10.7	50.6 ± 11.3	50.9 ± 10.3	50.3 ± 10.4
Sex				
Male	1,043 (32.3)	345 (32.0)	363 (33.8)	335 (31.0)
Female	2,191 (67.7)	734 (68.0)	710 (66.2)	747 (69.0)
Race/ethnicity				
Caucasian	1,768 (54.7)	580 (53.8)	602 (56.1)	586 (54.2)
African- American	645 (19.9)	204 (18.9)	221 (20.6)	220 (20.3)
Hispanic	508 (15.7)	178 (16.5)	162 (15.1)	168 (15.5)
American Indian	171 (5.3)	60 (5.6)	52 (4.8)	59 (5.5)
Asian-American	142 (4.4)	57 (5.3)	36 (3.4)	49 (4.5)
Fasting glucose (mmol/l)*	5.9 ± 0.5	5.9 ± 0.4	5.9 ± 0.5	5.9 ± 0.5
BMI (kg/m^2)*	34.0 ± 6.7	33.9 ± 6.8	33.9 ± 6.6	34.2 ± 6.8
Blood pressure (mmHg)*				
Systolic	123.7 ± 14.7	123.7 ± 14.8	124.0 ± 14.9	123.5 ± 14.4
Diastolic	78.3 ± 9.3	78.6 ± 9.2	78.3 ± 9.5	78.0 ± 9.2

Data are means ± SD or *n* (%) unless otherwise stated. Percentages may not add up to 100 because of rounding.

* Eligibility criteria: age ≥25 years, fasting plasma glucose 5.3–6.9 mmol/l (95–125 mg/dl) (≤6.9 mmol/l [<125 mg/dl] for American Indians), 2-h plasma glucose 7.8–11.0 mmol/l (149–199 mg/dl), BMI ≥24 kg/m^2 (≥22 kg/m^2 for Asian-Americans), and systolic/diastolic blood pressure ≤180/105 mmHg.

Table 2

Demographic and socioeconomic characteristics by sex

	Overall	Male	Female
n	3,234	1,043	2,191
Age (years)			
25 to <40	505 (15.6)	113 (10.8)	392 (17.9)
40 to <50	1,137 (35.2)	286 (27.4)	851 (38.8)
50 to <60	945 (29.2)	325 (31.2)	620 (28.3)
≥60	647 (20.0)	319 (30.6)	328 (15.0)
Race/ethnicity			
Caucasian	1,768 (54.7)	608 (58.3)	1,160 (52.9)
African-American	645 (19.9)	165 (15.8)	480 (21.9)
Hispanic	508 (15.7)	167 (16.0)	341 (15.6)
American Indian	171 (5.3)	20 (1.9)	151 (6.9)
Asian-American	142 (4.4)	83 (8.0)	59 (2.7)
Employment status			
Employed (full- or part-time)	2,401 (74.2)	771 (73.9)	1,630 (74.4)
Retired	420 (13.0)	217 (20.8)	203 (9.3)
Homemaker	204 (6.3)	1 (0.1)	203 (9.3)
Not employed	121 (3.7)	33 (3.2)	88 (4.0)
Seasonally employed	25 (0.8)	8 (0.8)	17 (0.8)
Student	21 (0.6)	2 (0.2)	19 (0.9)
Other	37 (1.1)	11 (1.1)	26 (1.2)
Never worked	5 (0.2)	0 (0.0)	5 (0.2)
Education (years)			
<13	834 (25.8)	221 (21.2)	613 (28.0)
13–16	1,556 (48.1)	488 (46.8)	1,068 (48.7)
≥17	844 (26.1)	334 (32.0)	510 (23.3)
Prior pregnancy	—	—	1,869 (85.3)
Postmenopausal	—	—	1,049 (47.9)
Annual family income			
<$20,000	446 (13.8)	110 (10.5)	336 (15.3)
$20,000 to <$35,000	561 (17.4)	146 (14.0)	415 (18.9)
$35,000 to <$50,000	641 (19.8)	207 (19.8)	434 (19.8)
$50,000 to <$75,000	646 (20.0)	218 (20.9)	428 (19.5)
≥$75,000	682 (21.1)	281 (26.9)	401 (18.3)
Refused	257 (7.9)	81 (7.8)	176 (8.0)
Marital status			
Never married	420 (13.0)	115 (11.0)	305 (13.9)
Living together	125 (3.9)	31 (3.0)	94 (4.3)
Married	1,999 (61.8)	765 (73.3)	1,234 (56.3)
Separated	91 (2.8)	26 (2.5)	65 (3.0)
Divorced	448 (13.9)	75 (7.2)	373 (17.0)
Widowed	151 (4.7)	31 (3.0)	120 (5.5)
Smoking			
Never	1,897 (58.7)	497 (47.7)	1,400 (63.9)
Former	1,111 (34.4)	471 (45.2)	640 (29.2)
Current	226 (7.0)	75 (7.2)	151 (6.9)

Data are *n* (%) unless otherwise stated. Percentages may not add up to 100 because of rounding.

Table 3

Self-reported characteristics of participants by sex and race/ethnicity

	All	Caucasian	African-American	Hispanic	American Indian	Asian-American
Men						
n	1,043	608	165	167	20	83
Family history of type 2 diabetes	690 (66.2)	390 (64.3)	117 (70.9)	112 (67.1)	13 (65.0)	58 (69.9)
History of high cholesterol	389 (37.3)	234 (38.5)	65 (39.4)	53 (31.7)	3 (15.0)	34 (41.0)
History of hypertension	302 (29.0)	171 (28.1)	58 (35.2)	49 (29.3)	5 (25.0)	19 (22.9)
Women						
n	2,191	1,160	480	341	151	59
Family history of type 2 diabetes	1,553 (70.9)	799 (68.9)	360 (74.8)	243 (71.3)	116 (76.8)	35 (60.3)
History of gestational diabetes	353 (16.1)	191 (16.5)	63 (13.1)	55 (16.2)	36 (23.8)	8 (13.8)
History of high cholesterol	730 (33.3)	429 (37.0)	147 (30.6)	114 (33.4)	22 (14.6)	17 (29.3)
History of hypertension	569 (26.0)	303 (26.1)	144 (30.0)	68 (19.9)	40 (26.5)	15 (25.9)

Data are *n* (%) unless otherwise stated. Percentages may not add up to 100 because of rounding.

Table 4

Clinical characteristics in male subjects by racial/ethnic group

	All	Caucasian	African-American	Hispanic	American Indian	Asian-American
n	1,043	608	165	167	20	83
Age at randomization (years)	53.8 ± 11.2 (26.5–85.2)	55.3 ± 11.2 (27.5–85.2)	54.3 ± 10.3 (26.5–79.3)	50.4 ± 10.9 (26.5–77.7)	44.2 ± 10.5 (27.5–66.1)	50.7 ± 10.7 (29.1–72.1)
25 to <40	113 (10.8)	53 (8.7)	12 (7.3)	28 (16.8)	7 (35.0)	13 (15.7)
40 to <50	286 (27.4)	149 (24.5)	50 (30.3)	51 (30.5)	8 (40.0)	28 (33.7)
50 to <60	325 (31.2)	186 (30.6)	58 (35.2)	56 (33.5)	3 (15.0)	22 (26.5)
≥60	319 (30.6)	220 (36.2)	45 (27.3)	32 (19.2)	2 (10.0)	20 (24.1)
BMI (kg/m^2)	32.0 ± 5.7 (22.7–70.9)	32.5 ± 5.8 (24.0–70.9)	32.5 ± 6.0 (24.4–64.9)	31.7 ± 5.0 (24.4–54.4)	31.2 ± 4.1 (24.3–40.1)	28.3 ± 3.7 (22.7–44.0)
<30	453 (43.4)	246 (40.5)	66 (40.0)	72 (43.1)	8 (40.0)	61 (73.5)
30 to <40	505 (48.4)	305 (50.2)	84 (50.9)	84 (50.3)	11 (55.0)	21 (25.3)
≥40	85 (8.1)	57 (9.4)	15 (9.1)	11 (6.6)	1 (5.0)	1 (1.2)
Blood pressure (mmHg)						
Systolic	125.8 ± 13.9 (80.0–176.0)	125.9 ± 13.7 (80.0–175.0)	128.2 ± 14.2 (100.0–176.0)	123.9 ± 13.6 (95.0–169.0)	119.6 ± 11.4 (102.0–138.0)	125.9 ± 14.3 (92.0–164.0)
Diastolic	79.9 ± 9.3 (25.0–105.0)	79.2 ± 9.6 (25.0–105.0)	80.9 ± 8.9 (55.0–105.0)	80.2 ± 8.0 (60.0–102.0)	79.2 ± 8.5 (63.0–94.0)	83.2 ± 9.1 (54.0–101.0)
Glycemia (mmol/l)						
Fasting plasma glucose	6.0 ± 0.5 (5.2–7.7)	6.0 ± 0.5 (5.3–7.7)	6.0 ± 0.4 (5.3–7.3)	6.0 ± 0.5 (5.3–7.7)	5.8 ± 0.4 (5.2–6.6)	6.0 ± 0.4 (5.3–7.5)
2-h plasma glucose	9.1 ± 0.9 (7.8–11.0)	9.2 ± 0.9 (7.8–11.0)	9.1 ± 1.0 (7.8–11.0)	9.1 ± 1.0 (7.8–11.0)	9.1 ± 0.9 (7.9–10.5)	9.1 ± 0.9 (7.8–11.0)
HbA_{1c} (%)	5.9 ± 0.5 (4.0–7.7)	5.8 ± 0.4 (4.0–7.2)	6.2 ± 0.7 (4.2–7.7)	5.9 ± 0.5 (4.4–7.2)	5.8 ± 0.5 (4.5–6.7)	6.0 ± 0.4 (4.8–6.8)
HbA_{1c} > 6.1%	316 (30.4)	133 (22.0)	105 (64.0)	47 (28.1)	5 (25.0)	26 (31.3)
Insulinemia (pmol/l)						
Fasting insulin	158.3 ± 98.6 (26.4–1,104)	156.6 ± 100.8 (27.0–684)	147.5 ± 73.5 (26.4–510)	178.3 ± 118.2 (42.6–1,104)	151.0 ± 70.1 (48.0–288)	155.3 ± 88.2 (36.0–480)
30-min insulin	589.6 ± 423.1 (27.0–4,854)	555.4 ± 423.7 (31.2–4,854)	527.2 ± 317.3 (66.0–1,812)	710.8 ± 413.5 (27.0–2,190)	819.5 ± 760.1 (294.0–3,480)	660.6 ± 441.0 (78.0–2,280)
Fasting proinsulin	20.7 ± 16.7 (2.0–144.0)	20.5 ± 17.3 (2.0–131.0)	20.5 ± 17.3 (4.8–144.0)	22.8 ± 16.8 (3.8–100.0)	19.4 ± 12.1 (6.8–43.0)	19.0 ± 12.1 (3.5–67.0)
Lipids (mmol/l)						
Total cholesterol	5.2 ± 0.9 (2.0–9.0)	5.2 ± 0.9 (2.9–8.1)	5.2 ± 0.9 (2.0–9.0)	5.2 ± 0.9 (2.8–7.9)	4.9 ± 1.0 (2.5–6.3)	5.4 ± 1.0 (2.9–7.7)
HDL cholesterol	1.0 ± 0.2 (0.5–2.2)	1.0 ± 0.2 (0.5–2.2)	1.1 ± 0.2 (0.6–1.9)	1.0 ± 0.2 (0.5–1.7)	1.0 ± 0.1 (0.7–1.2)	1.1 ± 0.2 (0.7–1.6)
LDL cholesterol	3.3 ± 0.8 (0.9–7.1)	3.2 ± 0.8 (1.0–6.0)	3.4 ± 0.9 (0.9–7.1)	3.2 ± 0.9 (1.0–6.3)	2.9 ± 1.0 (1.1–4.4)	3.4 ± 0.8 (1.6–5.3)
Triglycerides	2.0 ± 1.1 (0.3–6.8)	2.1 ± 1.1 (0.3–6.8)	1.5 ± 0.9 (0.5–5.9)	2.2 ± 1.2 (0.5–6.5)	2.1 ± 1.1 (0.3–5.1)	2.0 ± 1.1 (0.5–5.3)

Data are means ± SD (range) or *n* (%) unless otherwise stated. Percentages may not add up to 100 because of rounding. Insulin results were available for 848 men.

Table 5

Clinical characteristics in female subjects by racial/ethnic group

	All	Caucasian	African-American	Hispanic	American Indian	Asian-American
n	2,191	1,160	480	341	151	59
Age at randomization (years)	49.1 ± 10.1 (25.5–84.4)	50.3 ± 10.2 (26.1–84.4)	48.8 ± 9.8 (25.5–83.2)	47.3 ± 9.6 (25.8–79.1)	44.5 ± 9.7 (26.1–79.1)	48.9 ± 8.5 (32.0–70.1)
25 to <40	392 (17.9)	167 (14.4)	88 (18.3)	77 (22.6)	52 (34.4)	8 (13.8)
40 to <50	851 (38.8)	442 (38.1)	187 (39.0)	138 (40.5)	57 (37.7)	27 (45.8)
50 to <60	620 (28.3)	341 (29.4)	136 (28.3)	90 (26.4)	35 (23.2)	18 (30.5)
≥60	328 (15.0)	210 (18.1)	69 (14.4)	36 (10.6)	7 (4.6)	6 (10.2)
BMI (kg/m^2)	34.9 ± 7.0 (22.1–71.5)	35.0 ± 7.1 (23.9–71.5)	36.3 ± 7.1 (24.1–65.1)	34.0 ± 6.0 (22.6–64.9)	33.9 ± 6.3 (24.0–55.4)	30.7 ± 6.5 (22.1–50.4)
<30	593 (27.1)	325 (28.0)	101 (21.0)	94 (27.6)	38 (25.2)	35 (59.3)
30 to <40	1,134 (51.8)	585 (50.4)	248 (51.8)	194 (56.9)	90 (59.6)	16 (28.8)
≥40	464 (21.2)	250 (21.6)	131 (27.3)	53 (15.5)	23 (15.2)	7 (11.9)
Blood pressure (mmHg)						
Systolic	122.7 ± 15.0 (84.0–179.0)	122.6 ± 14.3 (84.0–175.0)	126.4 ± 15.9 (89.0–175.0)	121.1 ± 14.7 (90.0–179.0)	115.6 ± 12.3 (88.0–152.0)	123.4 ± 18.4 (89.0–173.0)
Diastolic	77.5 ± 9.2 (47.0–110.0)	77.2 ± 8.7 (48.0–103.0)	79.4 ± 10.3 (47.0–105.0)	76.5 ± 8.7 (57.5–103.0)	74.8 ± 8.6 (53.0–110.0)	81.0 ± 11.0 (59.0–104.0)
Glycemia (mmol/l)						
Fasting plasma glucose	5.9 ± 0.4 (4.2–7.7)	5.9 ± 0.4 (5.3–7.7)	6.0 ± 0.5 (5.3–7.5)	5.8 ± 0.4 (5.3–7.3)	5.5 ± 0.5 (4.2–6.8)	5.9 ± 0.4 (5.3–6.8)
2-h plasma glucose	9.1 ± 0.9 (7.8–11.0)	9.2 ± 0.9 (7.8–11.0)	9.1 ± 1.0 (7.8–11.0)	9.1 ± 0.9 (7.8–11.0)	9.1 ± 1.0 (7.8–11.0)	9.4 ± 0.9 (7.8–11.0)
HbA_{1c} (%)	5.9 ± 0.5 (3.2–8.5)	5.8 ± 0.4 (3.6–7.4)	6.2 ± 0.6 (3.2–8.5)	5.9 ± 0.5 (4.4–7.5)	6.0 ± 0.4 (5.0–7.6)	5.9 ± 0.4 (4.5–7.1)
HbA_{1c} > 6.1%	616 (28.2)	215 (18.6)	259 (53.8)	76 (22.4)	52 (34.4)	15 (25.4)
Insulinemia (pmol/l)						
Fasting insulin	158.1 ± 86.1 (14.4–720)	150.7 ± 80.3 (14.4–552)	166.9 ± 91.1 (18.0–576)	168.0 ± 91.1 (32.4–720)	170.0 ± 89.4 (34.2–534)	147.5 ± 103.1 (36.0–576)
30-min insulin	606.6 ± 367.5 (18.0–3,600)	557.4 ± 323.1 (36.0–3,600)	617.0 ± 415.6 (18.0–3,024)	680.8 ± 378.0 (51.6–2,100)	809.7 ± 444.1 (78.0–2,436)	568.8 ± 276.2 (132.0–1,248)
Fasting proinsulin	16.7 ± 12.2 (2.0–101.0)	15.7 ± 11.4 (2.0–101.0)	18.6 ± 13.1 (3.0–93.0)	17.8 ± 12.7 (3.0–68.0)	17.9 ± 14.0 (4.0–75.0)	13.2 ± 8.3 (2.5–43.0)
Lipids (mmol/l)						
Total cholesterol	5.3 ± 1.0 (2.1–9.3)	5.4 ± 0.9 (2.5–9.3)	5.2 ± 1.0 (2.1–8.3)	5.2 ± 0.9 (3.2–8.3)	4.8 ± 1.0 (2.8–8.3)	5.4 ± 0.9 (2.7–7.4)
HDL cholesterol	1.2 ± 0.3 (0.5–2.7)	1.2 ± 0.3 (0.5–2.6)	1.3 ± 0.3 (0.6–2.7)	1.2 ± 0.3 (0.6–2.7)	1.2 ± 0.3 (0.6–2.1)	1.3 ± 0.3 (0.6–1.9)
LDL cholesterol	3.2 ± 0.9 (0.6–6.5)	3.2 ± 0.8 (0.6–6.4)	3.3 ± 0.9 (0.6–6.1)	3.2 ± 0.9 (1.2–6.5)	2.8 ± 0.8 (1.3–5.4)	3.2 ± 0.9 (1.3–5.0)
Triglycerides	1.7 ± 0.9 (0.2–6.6)	1.9 ± 1.0 (0.2–6.6)	1.2 ± 0.6 (0.3–5.4)	1.8 ± 0.9 (0.5–5.4)	1.7 ± 0.8 (0.5–5.4)	2.0 ± 1.2 (0.6–6.3)

Data are means ± SD (range) or *n* (%) unless otherwise stated. Note: Percentages may not add up to 100 because of rounding. Insulin results were available for 1,720 women.

Table 6

Cardiovascular disease characteristics by sex

	Overall	Men	Women
n	3,234	1,043	2,191
History			
Myocardial infarction	32 (1.0)	23 (2.2)	9 (0.4)
Stroke	34 (1.1)	18 (1.7)	16 (0.7)
Bypass surgery			
CABG/PTCA	13 (0.4)	11 (1.1)	2 (0.1)
Leg	3 (0.1)	2 (0.2)	1 (0.0)
ECG results			
Minnesota Code MI	60 (2.0)	34 (3.4)	26 (1.3)
Left ventricular hypertrophy	29 (1.0)	14 (1.4)	15 (0.7)
Major abnormality	225 (7.3)	113 (11.3)	112 (5.4)
Minor abnormality	607 (19.8)	233 (23.4)	374 (18.1)

Data are *n* (%) unless otherwise stated. Percentages may not add up to 100 because of rounding. Centrally read ECG results were available for 3,069 participants. CABG, coronary artery bypass graft; PTCA, percutaneous transluminal coronary angioplasty .

Table 7

Comparison of DPP and NHANES III participant characteristics

	DPP-IGT*	NHANES III*
n	2,729	299
Age in years (%)		
40 to <50	41.7	30.8
50 to <60	34.6	23.4
≥60	23.7	45.8
Female sex (%)	65.9	51.5
Race/ethnicity (%)		
Caucasian	56.7	75.6
African-American	20.0	5.9
Hispanic	14.8	13.8
Asian-American	4.4	NA
American Indian	4.1	NA
Other	0.0	4.7
BMI (kg/m^2)	33.6	29.9
Education in years (%)		
<13	25.7	68.7
13–16	47.1	23.9
≥17	27.2	7.4
Smoking status (%)		
Former	37.4	44.9
Current	6.5	14.4
Hypertension (%)	29.0	44.6
Lipids (mmol/l)		
Total cholesterol	5.3	5.7
HDL cholesterol	1.2	1.2
LDL cholesterol	3.3	3.6
Triglycerides	1.8	2.1
Glycemia		
HbA_{1c} (%)	5.9	5.5
Fasting insulin (pmol/l)	153.0	87.0

Data are % or means (lipids, HbA_{1c}, fasting insulin). NHANES III participants met the DPP fasting glucose, age, and BMI eligibility criteria. NA, not available.

*Since glucose tolerance tests in NHANES were obtained only on those who were ≥40 years of age, DPP subjects aged ≥40 years were analyzed (*n* = 2,729).