

**One Hour Plasma Glucose Concentration and the Metabolic Syndrome
Identifies Subjects at High Risk for Future Type 2 Diabetes**

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Objective: To assess the efficacy of 1 hour plasma glucose concentration and the metabolic syndrome in predicting future risk of T2DM.

Research Design and Methods: 1611 subjects from the San Antonio Heart Study, who were free of T2DM at baseline, who had plasma glucose and insulin concentrations measured at time 0, 30, 60 and 120 minutes during the OGTT, and who had their diabetes status determined with an OGTT after 7-8 years of follow-up, were evaluated. Two models, based on glucose tolerance status, 1-h plasma glucose (1h-PG) concentration and presence of the metabolic syndrome, were tested in predicting the risk for T2DM at 7-8 years of follow-up

Results: A cut off point of 155 mg/dl for the 1h-PG concentration during the OGTT was used to stratify subjects in each glucose tolerance group into low, intermediate and high risk for future T2DM. A model based upon 1h-PG concentration, ATP III criteria for the metabolic syndrome, and FPG, independent of 2-h plasma glucose, performed equally well in stratifying non-diabetic subjects into low, intermediate and high risk for future T2DM and identified a group of NGT subjects who were at very high risk for future T2DM.

Conclusion: The plasma glucose concentration at 1 hour during the OGTT is a strong predictor of future risk for T2DM. A plasma glucose cut off point of 155 mg/dl and the ATP III criteria for the metabolic syndrome can be used to stratify non-diabetic subjects into 3 risk groups: low, intermediate and high risk

Clinical trials have demonstrated that lifestyle intervention and pharmacological therapy in high risk individuals reduce the incidence of T2DM (1). Thus, reliable models for identification of individuals at high risk for future T2DM are essential and have important clinical implications for intervention programs. Subjects with impaired glucose tolerance (IGT) are at increased risk for future T2DM (2), and the oral glucose tolerance test (OGTT) has become the standard method for identifying individuals at risk for T2DM. Indeed, all clinical trials which have assessed strategies for T2DM prevention have recruited subjects with IGT. Although IGT subjects have increased risk for T2DM, only ~50% convert to T2DM within 10 years of follow-up (3), indicating that the future risk for diabetes is not similar amongst all individuals with IGT. Furthermore, in longitudinal epidemiological studies about 40% of subjects who develop T2DM have normal glucose tolerance at baseline, indicating that there is a population of NGT subjects who are at risk for future T2DM (2). Recently, we demonstrated that subjects with NGT, despite having relatively low risk for T2DM, can be stratified into low and high risk categories based upon the relationship between their post-load and fasting plasma glucose concentrations (3).

Several models have been proposed to improve the predictive ability for future T2DM (4-7). These models are based upon established risk factors for T2DM, e.g. obesity, FPG, lipid profile and blood pressure. All of these risk factors are components of the metabolic or insulin resistance syndrome, which is itself a predictor of future T2DM in non-diabetic individuals (8). In a recent publication, we demonstrated that the 1-h plasma glucose concentration is a better predictor for future T2DM than either the fasting plasma glucose or 2-h plasma glucose concentration (9). Further, the addition of the 1-h plasma glucose concentration to a prediction model based on clinical parameters significantly improved the ability of the model to predict future T2DM (9). In this study, we have used the classification tree model (10) to stratify the risk for future T2DM in non-diabetic subjects based upon their 1-h plasma glucose concentration during the OGTT and the

ATP III criteria for the metabolic syndrome. We demonstrate that a model based on the combination of 1-h plasma glucose concentration during the OGTT and the ATP III criteria for the metabolic syndrome improves the ability to predict the future risk for T2DM.

EXPERIMENTAL DESIGN AND METHODS

Study Population: All subjects were participants of the San Antonio Heart Study (11-13) which is a population-based, epidemiological study of T2DM and cardiovascular disease. A total of 2,616 eligible participants, who were free of T2DM at baseline, completed a 7-8 year follow-up examination and had their diabetes outcome determined with a repeat OGTT. Of these 2,616 participants, 1,610 subjects had plasma glucose measurements at 0, 30, 60, and 120 min during the baseline OGTT and constitute the study population. The study was approved by the IRB of UTHSCSA. All subjects gave their written informed consent before participation.

Definition of Variables and Outcomes: All studies were performed in a mobile clinic following a 12-h overnight fast. A standard 75 gram glucose OGTT was performed and blood was obtained at 0, 30, 60, and 120 min for determination of plasma glucose and serum insulin concentrations. Plasma glucose and serum lipids were measured with an Abbott Bichromatic Analyzer (South Pasadena, CA). The diagnosis of diabetes was based upon World Health Organization criteria (14). Subjects on insulin or oral antihyperglycemic medications also were considered to have diabetes. The metabolic syndrome was diagnosed according to ATP III criteria (15).

Classification Tree: Recursively partitioned classification trees (16) were used to model the relationship between the future risk of T2DM and (i) 1-h plasma glucose

concentration during the OGTT and (ii) presence or absence of the metabolic syndrome. Sequential partitioning of the individuals based upon their 1-h plasma glucose concentration relative to 155 mg/dl (above or below) and the presence or absence of the metabolic syndrome produced subgroups or compartments of individuals with homogenous risk for future T2DM. Subgroups with annual risk for future T2DM < 0.5% (<3.5% risk in 7-8 years) were considered as having low risk for future T2DM. Annual risk between 1 and 2% (7-15% risk in 7-8 years) was considered intermediate risk. Annual risk > 4% (>30% risk in 7-8 years) was considered high risk.

Statistical Methods: Variables are presented as the mean \pm SD. The significance of the mean differences was tested with ANOVA. Differences between categorical variables were tested with Chi Square test. Statistical significance was considered at the level of $P < 0.05$. Assessment of the predictive discrimination of the various models was made using the receiver-operating characteristic (ROC) curve by plotting the sensitivity against the corresponding false-positive rate. Statistical analysis was performed with SPSS software package.

RESULTS

Table 1 presents the anthropometric, laboratory and clinical characteristics of the study population. Of the 1611 study participants, 1301 had NGT, 90 had IFG and 221 had IGT at baseline, respectively. 51 of the 221 subjects with IGT also had IFG and were designated as having combined glucose intolerance (CGI). The conversion rate to T2DM over the study period (7-8 years) was 5.0%, 26.1%, 30.9% and 82.3% for NGT, IFG, IGT and CGI subjects, respectively. We previously demonstrated that the 1-h plasma glucose concentration during the OGTT is a good predictor for future T2DM (9). A plasma glucose cutoff point of 155 mg/dl has

the maximal sum of sensitivity and specificity (0.75 and 0.79 for sensitivity and specificity, respectively) and for predicting future T2DM. Similarly, the ideal cut off point for fasting plasma glucose concentration in predicting future T2DM was 94.5 mg/dl. Therefore, we have used these values as cutoff points to test the prediction of future T2DM with two tree models.

The first tree model is based upon the glucose tolerance status, 1-h plasma glucose value and presence of the metabolic syndrome. The ROC for this model was 86.7%. In this model, individuals were divided, according to the ADA criteria, into four groups (NGT, IFG, IGT and CGI) based upon their fasting and 2-h plasma glucose concentration. Individuals in each group were further divided into two subgroups based upon their 1-h plasma glucose concentration (above or below 155 mg/dl). Figure 1 depicts the incidence of T2DM based upon 1-h plasma glucose concentration. Although, as a whole, subjects with NGT had a low risk for T2DM (5.0%), NGT subjects with 1-h plasma glucose > 155 mg/dl had significantly increased risk (15.3%) for future T2DM compared to NGT subjects with 1-h plasma glucose < 155 mg/dl (2.9%) ($P < 0.0001$). Further division of this group based upon the presence or absence of the metabolic syndrome demonstrated that NGT subjects with 1-h plasma glucose > 155 mg/dl and the metabolic syndrome had 32.1% incidence rate of T2DM compared to 9.4% incidence rate for subjects without the metabolic syndrome.

Subjects with IFG and a 1-h plasma glucose >155 mg/dl had a 37.3% incidence of T2DM, while IFG subjects with a 1-h plasma glucose concentration < 155 mg/dl had a 10.8 % incidence rate. Table 2 presents the odds ratio for having diabetes for the various glucose tolerance groups.

Subjects with IGT and a 1-h plasma glucose > 155 mg/dl had a 35.5% diabetes incidence rate, while IGT subjects with a 1-h

plasma glucose < 155 mg/dl had a 17.8% diabetes incidence rate.

The second model includes the 1-h plasma glucose concentration, the metabolic syndrome and fasting plasma glucose concentration. The ROC for this model was 85.4%. In this model subjects were divided into two groups based upon their 1-h plasma glucose concentration (above or below 155 mg/dl) and each group was further divided into two sub-groups based upon the presence or absence of the metabolic syndrome. Figure 2 depicts the 7-8 year risk for T2DM for each subgroup. In general, non diabetic subjects with 1-h plasma glucose < 155 mg/dl had a low risk (3.9%) for future development of T2DM compared to subjects with a 1-h plasma glucose > 155 mg/dl (31.0%) ($P < 0.0001$). When subjects with 1-h plasma glucose < 155 mg/dl were divided according to the presence or absence of the metabolic syndrome, subjects with a 1-h plasma glucose < 155 mg/dl without the metabolic syndrome had a 3.2% risk for future T2DM, while those with the metabolic syndrome had 7.8% risk for future diabetes. Subjects with a 1-h plasma glucose concentration > 155 mg/dl and the metabolic syndrome had a 51.6% risk for future diabetes. Subjects with a 1-h plasma glucose > 155 mg/dl without the metabolic syndrome but with a fasting plasma glucose > 95 mg/dl had a 44.7% risk for future diabetes, while subjects with a 1-h plasma glucose > 155 mg/dl without the metabolic syndrome and fasting plasma glucose < 95 mg/dl had a 10.8% risk for future T2DM.

Because the waist circumference is rarely measured in clinical practice and is part of the ATP III definition of the metabolic syndrome, we also examined the predictive value of the triglyceride to HDL cholesterol ratio greater than 3.5 in place of the metabolic syndrome (Table 2). Although the metabolic syndrome was a better predictor compared to the triglycerides to HDL

cholesterol ratio, a model based on 1h plasma glucose concentration and triglycerides to HDL cholesterol ratio could classify subjects to three risk groups: low, intermediate and high risk (table 2).

DISCUSSION

The ADA Consensus Statement has recommended metformin, in addition to diet and exercise, in individuals with IGT/IFG to reduce their risk for future diabetes (18). This recommendation for pharmacologic intervention underscores the need for models that reliably identify individuals at increased risk for future development of T2DM. The results of this study demonstrate that the plasma glucose concentration at 1-h during the OGTT is a useful tool that can be used to stratify the risk of future T2DM into three groups: low, intermediate and high risk. In general, subjects with NGT have low risk for progression to T2DM (~0.67% annual rate) (2). However, ~40% of individuals who develop T2DM have normal glucose tolerance at baseline (2) and, in the present study, 16.7% of NGT subjects with a 1-hour plasma glucose concentration (OGTT) >155 mg/dl developed T2DM over a 7-8 year period. In this group of NGT subjects, the annual risk for future T2DM was significantly greater (2.2% per year) compared to subjects whose 1-hour plasma glucose concentration did not exceed 155 mg/dl (0.39% per year, $P < 0.00001$). Further, NGT subjects' with a 1-hour plasma glucose > 155 mg/dl, who fulfill the ATP III criteria for the metabolic syndrome, had a 4.3% annual risk for future T2DM. Thus, the group of NGT subjects with 1-hour PG > 155 mg/dl plus the metabolic syndrome is at very high risk for the development of T2DM, and their risk exceeds that of subjects with IFG or IGT and their odds ratio for developing diabetes is double that of IGT subjects with a 1-hour plasma glucose < 155 mg/dl (Table 2). Consistent with the ADA

Consensus Conference Statement (18), this group of high risk NGT individuals could benefit from an intervention program employing diet, exercise, and pharmacotherapy (metformin) to reduce future risk for diabetes.

Subjects with CGI have the greatest risk for future T2DM, with an annual risk > 10% per year, while subjects with isolated IFG or IGT have an intermediate risk between CGI and NGT. However, within the IFG and IGT groups, the 1-hour plasma glucose during the OGTT also stratifies the future diabetes risk into intermediate and high risk. Thus, IFG and IGT subjects with a 1-h plasma glucose < 155 mg/dl have an annual risk of ~1.5% compared to an annual risk of ~5% for IGT and IFG subjects with a 1-h plasma glucose > 155 mg/dl. It is noteworthy that every CGI subject had a 1-h plasma glucose concentration above 155 mg/dl. Thus, the plasma glucose concentration at 1 hour during the OGTT is a strong predictor for future T2DM, independent of the glucose tolerance status, and a 155 mg/dl cutoff point divides individuals with NGT, IFG and IGT into low, intermediate and high risk groups.

A predictive model based on the plasma glucose concentration at 1-h during the OGTT and the presence or absence of the metabolic syndrome, independent of the 2-h plasma glucose concentration, performs equally well in stratifying subjects for future risk of T2DM compared to the model which includes the 2-h plasma glucose concentration. The earlier model had 0.82 sensitivity and 0.63 specificity compared to 0.82 and 0.67 sensitivity and specificity, respectively, for the model based on 1-h plasma glucose concentration. Moreover, the later model (individuals with 1-h plasma glucose > 155 mg/dl plus the metabolic syndrome or FPG > 95 mg/dl) reduces the number of subjects in the very high risk group (>6.5% incidence per year), who are candidates for pharmacological intervention,

from 18% (based on the model which includes the 2 h plasma glucose concentration) to 14% of the total study population. Furthermore, the model with the 1-h plasma glucose concentration plus the metabolic syndrome performs better in predicting future diabetes than does the ADA criteria of IGT or IFG. Most importantly, ~17% of NGT subjects, who have intermediate and high risk for future T2DM and who were identified with the 1-h plasma glucose plus metabolic syndrome, would have been missed with the ADA criteria alone. These observations underscore the importance of obtaining the plasma glucose concentration at 1 hour during the OGTT.

Substituting the metabolic syndrome with triglycerides to HDL cholesterol ratio in the second model slightly reduces its predictability. However, the second model with triglyceride to HDL cholesterol ratio is a good predictor for future risk of T2DM and classifies subjects into three risk groups. Because measurement of triglyceride and HDL cholesterol is part of the routine clinical practice, the second model could be used in routine clinical practice to assess the risk of non diabetic subjects for future risk of T2DM.

Why is the 1-hour plasma glucose concentration a better predictor for future T2DM than the 2 hour plasma glucose? It could be argued that the high predictability for 1-h plasma glucose is due to its high correlation with the 2-h plasma glucose ($r=0.58$, $p<0.0001$). However, the one hour plasma glucose stratifies subjects with NGT, as well as subjects with IGT, into two risk groups, high and low. Thus, it is unlikely that its predictability is secondary to its correlation with the 2-h plasma glucose. Subjects who are destined to develop T2DM manifest 2 major defects: (i) insulin resistance in liver and skeletal muscle and (ii) impaired beta cell function (19). Previous studies have demonstrated that subjects with hepatic insulin resistance have an increased fasting

plasma glucose concentration and impaired suppression of hepatic glucose production during the OGTT, resulting in an excessive rise in plasma glucose concentration at 30 and 60 minutes (20). In non-diabetic subjects, the decline in plasma glucose concentration at 30-60 minutes during the OGTT is dependent on insulin sensitivity in skeletal muscle and beta cell function (21,22). Thus, insulin resistance in liver and skeletal muscle, as well as impaired beta cell function, would result in an increase in 1-h plasma glucose concentration. This renders the 1-h plasma glucose a good indicator for the major metabolic abnormalities which lead to the development of T2DM. Consistent with this, we previously demonstrated that the plasma glucose concentration at 1-hour during the OGTT has a stronger correlation with surrogate measures of hepatic and muscle insulin resistance and beta cell dysfunction compared to the 2-hour plasma glucose value (9).

In summary, the plasma glucose concentration at 1 hour during the OGTT is a strong predictor of future risk for T2DM. A cut off point at 155 mg/dl plus the ATP III criteria for the metabolic syndrome can be used to stratify non-diabetic subjects into 3 risk groups: low, intermediate and high risk, independent of the 2-hour plasma glucose concentration.

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Table 1: Anthropometric, clinical, and laboratory characteristics of the study population

| | <u>NGT</u> | <u>IFG</u> | <u>IGT</u> | <u>CGI</u> | <u>ANOVA</u> |
|---------------------------|------------|------------|------------|------------|--------------|
| Number | 1301 | 90 | 169 | 51 | |
| Sex (% female) | 57 | 39 | 63 | 53 | <0.0001 |
| Age (years) | 42 ± 11 | 47 ± 10 | 49 ± 10 | 51±2 | < 0.0001 |
| BMI (kg/m ²) | 27.1 ± 5.1 | 29.8 ± 5.9 | 30.3 ± 5.6 | 31.1±0.8 | <0.0001 |
| Waist (cm) | 87.9 ± 1.3 | 96.3 ± 1.2 | 96.4 ± 1.6 | 102.5 ±1.8 | <0.0001 |
| FPG (mg/dl) | 83 ± 8 | 106 ± 5 | 93 ± 12 | 109 ±1 | <0.0001 |
| 2-h PG (mg/dl) | 95 ± 23 | 105 ± 22 | 165 ± 16 | 173±5 | <0.0001 |
| # with 1-h PG > 155 mg/dl | 217 | 52 | 171 | 50 | <0.0001 |
| % with MS | 14.3% | 66.7 | 49.3 | 84.3 | <0.0001 |
| # converted to diabetes | 65 | 23 | 52 | 42 | <0.0001 |

FPG = fasting plasma glucose; 2-h PG = plasma glucose at 2 hours during the OGTT; 1-h PG: plasma glucose at 1-hour during OGTT; MS = metabolic syndrome; DM = diabetes mellitus; CGI=combined glucose intolerance.

Table 2: Odds ratio and 95% confidence intervals for the risk of developing type 2 diabetes for the prediction models

| | Odds Ratio | 95% CI |
|--|------------|-----------|
| Model 1 | | |
| NGT, 1h PG <155 mg/dl | 1 | |
| NGT, 1h PG > 155, MS - | 3.4 | 1.8-6.4 |
| NGT, 1h PG>155, MS + | 15.2 | 7.8-29.3 |
| IFG, 1h PG <155 | 4.0 | 1.3-11.9 |
| IFG, 1h PG > 155 | 19.5 | 10.0-38.0 |
| IGT, 1h PG < 155 | 7.1 | 3.0-16.5 |
| IGT, 1h PG>155 | 18.1 | 10.8-30.1 |
| Model 2(A) | | |
| 1h PG <155 and MS- | 1 | |
| 1h PG <155 and MS+ | 2.4 | 1.2-4.8 |
| 1h PG >155 and MS- and FPG <95 | 3.6 | 2.0-6.2 |
| 1h PG >155, and MS+ or FPG >95 | 30.0 | 19.4-46.3 |
| Model 2(B) | | |
| 1h PG<155 and TRIG/HDL <3.5 | 1 | |
| 1h PG< 155 and TRIG/HDL >3.5 | 2.3 | 1.3-4.2 |
| 1h PG>155 and TRIG/HDL <3.5 and FPG < 95 | 4.3 | 2.4-7.9 |
| 1h PG>155, and TRIG/HDL>3.9 or FPG>95 | 22.4 | 14.2-35.3 |

NGT = normal glucose tolerance; IFG = impaired fasting glucose; IGT = impaired glucose tolerance; 1-h PG =1 hour plasma glucose concentration during OGTT; MS = metabolic syndrome; FPG = fasting plasma glucose concentration; TRIG=plasma triglyceride concentration.

FIGURE LEGENDS:

Figure 1: Tree model based on the glucose tolerance status of the subjects, 1 hour plasma glucose concentration, and presence or absence of the metabolic syndrome. The numbers in each nodule represent the number of subjects converting to diabetes/total number of subjects in each particular group and the incidence rate of conversion to diabetes over 8 years; NGT = normal glucose tolerance; IFG = impaired fasting glucose; IGT = impaired glucose tolerance. 1-h PG = 1 hour plasma glucose concentration during the OGTT. MS+ = metabolic syndrome present; MS- = metabolic syndrome absent.

Figure 2: Tree model based on 1 hour plasma glucose concentration, presence or absence of the metabolic syndrome, and fasting plasma glucose concentration. 1-h PG = 1 hour plasma glucose concentration during the OGTT. The numbers in each nodule represent the number of subjects converting to diabetes/total number of subjects in each particular group and the incidence rate of conversion to diabetes over 8 years; MS+ = metabolic syndrome present; MS- = metabolic syndrome absent. FPG = fasting plasma glucose concentration.

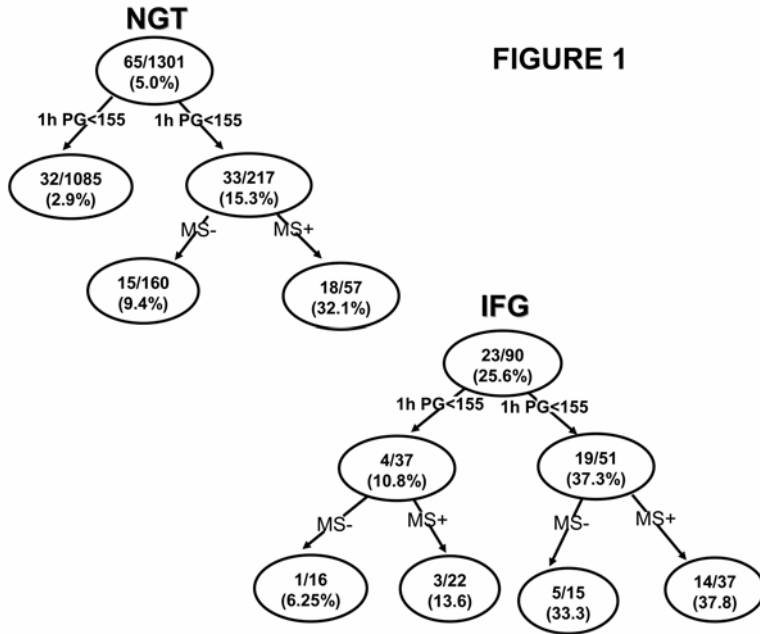


FIGURE 1

FIGURE 1

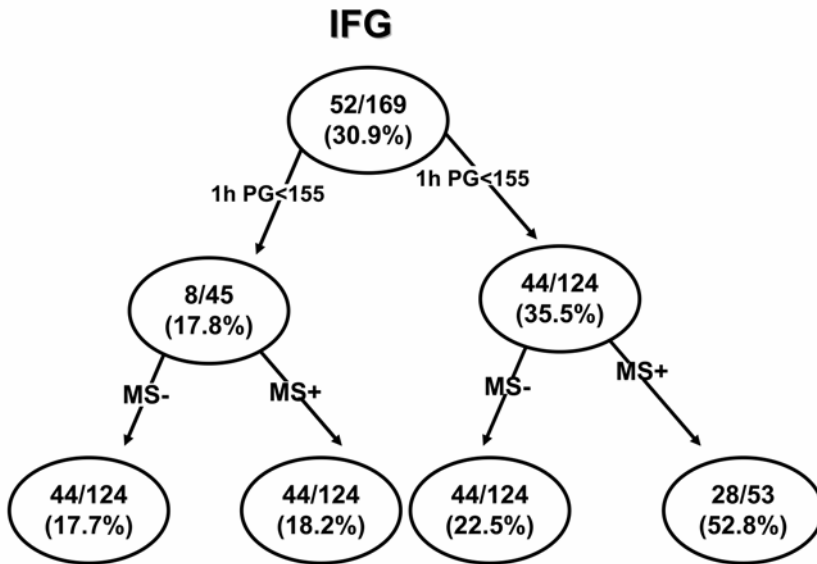


FIGURE 2

