Fasting Versus Post-Load Plasma Glucose Concentration and the Risk for Future Type 2 Diabetes: Results From The Botnia Study

Running Title: Fasting versus Post-Load Glucose & Diabetes Risk

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Aim: To assess the efficacy of post-load plasma glucose concentration in predicting future risk of T2DM, compared to prediction models based upon measurement of the fasting plasma glucose (FPG) concentration.

Research Design and Methods: 2442 subjects from the Botnia Study, who were free of T2DM at baseline, received an OGTT at baseline and after 7-8 years of follow. Future risk for T2DM was assessed with area under the ROC for prediction models based upon measurement of the FPG (i) with or without 1-h PG during the OGTT and (ii) with or without the metabolic syndrome.

Results: Prediction models based on measurement of the FPG were weak predictors for the risk of future T2DM. Addition of 1-h PG markedly enhanced the prediction the risk of future T2DM. A cut point of 155 mg/dl for the 1-h PG during OGTT and presence of the metabolic syndrome were used to stratify subjects in each glucose tolerance group into low, intermediate and 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 and adds to the prediction power of models based upon measurements made during the fasting state. A plasma glucose cut point of 155 mg/dl plus the ATP III criteria for the metabolic syndrome can be used to stratify non-diabetic subjects into low, intermediate and high risk groups.
Reliable models for identification of individuals at high risk for future T2DM (T2DM) are essential and have important clinical implications for intervention programs. Because subjects with impaired glucose tolerance (IGT) are at increased risk for future T2DM (1), the oral glucose tolerance test (OGTT) has become the standard method for identifying individuals at risk for developing T2DM. Indeed, all clinical trials which have assessed strategies for T2DM prevention have recruited subjects with IGT (2). However, performance of the OGTT is time consuming, and models, based on measurement of fasting plasma glucose concentration (FPG) and plasma lipid profile, in addition to medical history and anthropometric measurements, have been developed to predict the future risk for T2DM (3-8). Of note, only ~50% of subjects with IGT convert to T2DM within 10 years of follow-up (1). Moreover, in longitudinal epidemiological studies ~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 (1).

We have demonstrated that, although NGT subjects are at relatively low risk for the future development of T2DM, a group of NGT subjects with an increased risk for diabetes can be identified based upon the relationship between their post-load and fasting plasma glucose concentrations (9), or upon the 1-hour plasma glucose concentration and presence of the metabolic syndrome (10).

Insulin resistance and impaired insulin secretion represent the characteristic pathophysiologic disturbances responsible for development of T2DM (11, 12). Although both insulin resistance and beta cell dysfunction are present long before the onset of diabetes, progressive beta cell failure is the principal factor responsible for the development of overt hyperglycemia (13). Prediction models for T2DM have been developed based upon measurement of the fasting state include fasting plasma glucose and lipid concentrations, waist circumference and blood pressure. All of these risk factors are components of the metabolic (insulin resistance) syndrome, which itself is a predictor of future T2DM in non-diabetic individuals (14). As such they would be expected to correlate strongly with the presence insulin resistance, but less well with impairment in beta cell function. In a recent publication, we demonstrated that the 1-h PG correlates strongly with indices of both insulin resistance and insulin secretion, and is a better predictor for future T2DM than either the fasting plasma glucose concentration or the 2-h PG in Mexican American individuals (15). Further, we demonstrated that 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 in Mexican Americans (15) and was able to stratify subjects with normal and impaired glucose tolerance into low, intermediate and high risk groups (10).

Because the relative contributions of insulin resistance and impaired beta cell function may vary among various ethnic groups (16), the aim of this study was to assess the ability of the 1-hour PG during OGTT to predict future risk of T2DM compared to the fasting and 2-hour plasma glucose concentrations (2-h PG) in a European Caucasian population. We also examined whether addition of the 1-h PG to models based upon fasting measurements would enhance their predictive value for development of future T2DM.

EXPERIMENTAL DESIGN AND METHODS


**Study Population**—The participants of this study were subjects who participated in the Botnia study (17), were free of diabetes at baseline, had their plasma glucose and insulin concentrations measured during an OGTT, and had a repeat OGTT after 7-8 years. Subjects were classified into various categories of glucose tolerance based upon their fasting and 2 hour plasma glucose concentrations during the OGTT, according to the ADA criteria (18).

**Study Design**—All subjects received a standard 75 gram OGTT following a 12-h overnight fast. Plasma glucose and serum insulin concentrations were measured at 0, 30, 60 and 120 minute. Anthropometric and lipid profile were also obtained at baseline. Subjects were followed for 7-8 years and glucose tolerance status was determined at follow-up with a repeat OGTT according to the ADA criteria (18). Detailed description of the study design has been previously published elsewhere (17).

**Analytical Methods**—Plasma glucose was measured with glucose oxidation method using a Beckman Glucose Analyzer (Beckman Instruments, Fullerton, CA). Serum insulin was measured in duplicate by radioimmunoassay (Pharmacia, Sweden).

**Calculations**—The diagnosis of diabetes was based on ADA criteria (18). The metabolic syndrome was diagnosed according to ATP III criteria (19). Areas under the glucose and insulin curves were calculated by the trapezoid rule. Matsuda index of insulin sensitivity was calculated as previously reported (20). The insulinogenic index was calculated by dividing the increment in serum insulin by the increment in plasma glucose from 0-30 and 0-120 min of the OGTT. The insulin secretion/insulin resistance (disposition) index was calculated as the product of insulinogenic index ($\Delta I_{0-30}/\Delta G_{0-30}$ or $\Delta I_{0-120}/\Delta G_{0-120}$) and the Matsuda index of insulin sensitivity. We tested the following prediction models that rely on fasting measurements: (i) a previously described, multivariate model (San Antonio Diabetes Prediction Model or SADPM) for predicting future type 2 diabetes (3); this model includes age, sex, ethnicity, BMI, blood pressure, and fasting plasma glucose, triglyceride, and HDL concentrations; (ii) the ATP III criteria for the metabolic syndrome (19); (iii) a risk score index (Score Model I) based on age, obesity measurements, use of hypertensive medications and family history for diabetes (6); and (iv) a diabetes risk score based on sex, age and measurement of fasting plasma glucose and triglyceride concentrations (8). The predictive value of these 4 models also was evaluated after adding the 1-hour plasma glucose (PG) concentration during the OGTT. We also evaluated the risk of future diabetes using a tree model analysis.

**Tree Model Analysis**—Recursively partitioned classification trees (21) were used to model the relationship between the future risk of T2DM and (i) 1-h PG during the OGTT and (ii) presence or absence of the metabolic syndrome. Subjects were classified into NGT, IGT, IFG or combined glucose intolerance (CGI) (IGT+IFG) according to ADA criteria (18). Sequential partitioning of individuals within each glucose tolerance group (NGT, IGT, IFG, CGI) based upon a 1-h PG greater than or less than 155 mg/dl and the presence or absence of the metabolic syndrome produced subgroups of individuals with homogenous risk for future T2DM. Subgroups with a risk for future T2DM that was <2.5% over 7-8 years were considered to have low risk for future T2DM. A risk between 5-10% over 7-8 years was considered to represent intermediate risk. A risk > 15% over 7-8 years was considered to represent high risk.

**Statistical Methods**—Variables are presented as the mean ± SD. The significance of the mean differences was tested with ANOVA. 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. The area under the ROC curve was used as a measure of how well a continuous variable predicts the development of type 2 diabetes. To examine whether differences between two areas under ROC curves were statistically different, the algorithm developed by DeLong et al. (22) was used. Statistical analyses were performed with the SPSS statistical software system.

RESULTS

Table 1 presents the anthropometric, laboratory and clinical characteristics of the study population. Of the 2442 study participants, 1110 had NGT, 949 had IFG, 123 had IGT and 260 had combined glucose intolerance (CGI) (IGT +IFG) at baseline. A total of 124 subjects (5.1%) developed T2DM over the 7-8 year period of follow-up. The conversion rate to T2DM was 2.4%, 5.1%, 11.5% and 13.5% for NGT, IFG, IGT and CGI subjects, respectively.

The area under ROC (aROC) was used to evaluate the predictive power of the various prediction models. All plasma glucose concentrations (0, 30, 60, and 120 minutes) during the OGTT were significant predictors for future risk of T2DM (table 2). However, the plasma glucose concentration at 60 minutes was the strongest predictor of future risk for T2DM. The aROC for the fasting plasma glucose (FPG) concentration in this population was significantly less than the aROC for the 30 min and 60 minute plasma glucose concentrations during the OGTT. The aROC for the 120 minute plasma glucose concentration was smaller (0.688) than the aROC for both the 30 and 60 minute PG concentration during the OGTT (Table 2).

The aROC for HbA1c (0.697) was similar to that of the FPG (0.672). The incremental area under the glucose curve during the OGTT (\( \Delta G_{0-120} \)) was a strong predictor for the future risk for T2DM and had aROC (0.770) comparable to 1h glucose (0.795).

Since the 1h plasma glucose is the strongest predictor for future risk of T2DM, we tested whether the addition of 1h plasma glucose to prediction models (SADPM, ATP III criteria, Score Model I, Score Model II) based upon measurements made during the fasting state improves their predictive power. All four models performed well in predicting the future risk for T2DM and the aROC ranged from 0.64 to 0.74. However, addition of 1-h PG markedly enhanced the predictive power for each of the four models, resulting in aROC > 0.8 (Table 2).

We previously demonstrated that a one-hour plasma glucose cut point of 155 mg/dl during the OGTT and the presence of the metabolic syndrome could classify non-diabetic subjects into three risk groups, low, intermediate and high (10). In the present study, we constructed a tree model based on glucose intolerance status, the one hour plasma glucose concentration, and presence of the metabolic syndrome to classify the risk for future T2DM. The ROC for this model was 0.92. 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 PG (above or below 155 mg/dl). Figure 1 depicts the incidence of T2DM based upon the 1-h PG and the presence/absence of the metabolic syndrome in each glucose tolerant group. Although, as a whole, subjects with NGT had a low risk for developing T2DM (2.4%), NGT subjects with a 1-hour plasma glucose > 155 mg/dl had significantly increased risk (8.5%) for future T2DM compared to NGT subjects with 1-hour plasma glucose < 155 mg/dl (1.3%)
Further division of this group based upon the presence or absence of the metabolic syndrome demonstrated that NGT subjects with 1-hour plasma glucose > 155 mg/dl and the metabolic syndrome had 14.3% incidence rate of T2DM compared to 7.4% incidence rate for subjects without the metabolic syndrome.

Both IFG and IGT subjects with a 1-h plasma glucose >155 mg/dl and the metabolic syndrome had a high risk for future T2DM (15.1% and 23%, respectively), while IFG and IGT subjects with a 1-h plasma glucose concentration < 155 mg/dl without the metabolic syndrome had very low risk for future T2DM (0.8% and 0%, respectively). Subjects with a 1-hour plasma glucose < 155 with the metabolic syndrome or 1 h plasma glucose > 155 without the metabolic syndrome had an intermediate risk for future T2DM (5-10%).

Subjects with CGI and the metabolic syndrome had a very high risk for future T2DM (>20%), while none of the subjects with 1-hour plasma glucose < 155 mg/dl without the metabolic syndrome developed T2DM. Subjects with a 1-h plasma glucose > 155 mg/dl without the metabolic syndrome had an intermediate (7.2%) risk for future T2DM.

**DISCUSSION**

Clinical trials have demonstrated that life style intervention and pharmacological therapy can reduce the incidence rate of T2DM among high risk individuals (2) and an ADA Consensus Statement has recommended treatment with metformin, in addition to diet and exercise, in high risk individuals with IGT/IFG (23). This recommendation for pharmacologic intervention is “prediabetic” individuals underscores the need for models that reliably identify subjects at increased risk for future development of T2DM. The results of this study demonstrate that in a Scandinavian Caucasian population, the predictive value of future T2DM using the 1-h PG is superior to the 2-h PG and models based only on measurements taken during the fasting state. In this Scandinavian Caucasian population, as previously demonstrated in a Mexican American population, 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.

Subjects with IGT (2-hour plasma glucose =140-199 mg/dl) are at increased risk for future T2DM, and intervention studies systematically have recruited IGT subjects with to test the efficacy of interventions aimed at reducing the conversion rate of IGT to T2DM. Because performance of the OGTT in routine clinical practice is complicated and time consuming, investigators have developed prediction models based upon measurements made during the fasting state to predict the risk of future T2DM (3-8). These models perform equally well in predicting future T2DM compared to 2-h PG during the OGTT. Furthermore, addition of the 2-hour plasma glucose value to these models did not improve their predictive power (3). In this study we demonstrate that the 1-h PG is superior to both the 2-h PG and predictive models based only on measurements made during the fasting state to predict the future risk for type 2 diabetes. Furthermore, addition of the 1-h PG to models based only on fasting measurements markedly improved their predictive power (Table 2).

HbA1c, which reflects the mean plasma glucose concentration over the prior 3 months, has similar predictive power to that of the FPG, and it was much weaker than the 1h plasma glucose in predicting the future risk for T2DM. Of note, the optimal HbA1c cut point for predicting future T2DM was 5.6%, which is well within the range considered normal.

The parameters used in the fasting models (BMI, waist circumference, lipid profile, fasting glucose, blood pressure) are
components of the metabolic or insulin resistance syndrome and, as such, they have greater sensitivity in assessing insulin resistance than beta cell function. Similarly, HbA1c correlated poorly with indices of beta cell function (Table 2). Conversely, measurement of the post-load plasma glucose concentrations (e.g. 1h-PG and $\Delta G_{0-120}$) correlated well with indices of beta cell function (Table 2). With regard to this, it should be emphasized that progressive beta cell failure is the principal factor responsible for the progressive decline in glycemic control as individuals progress from NGT to IGT to T2DM (11, 16, 24-25). In this study we have shown that in Caucasian population, similar to Mexican Americans, the 1-h PG correlates better with OGTT-derived indices of beta cell function than the 2-h PG or prediction models based upon fasting measurements. The strong correlation between the 1-h PG and beta cell function could explain the superior performance of the 1-hour plasma glucose in predicting the risk of future T2DM compared to 2-h PG and especially compared to models based only on fasting measurements. Similarly, $\Delta G_{0-120}$ which is highly dependent on beta cell function, also is a strong predictor for future risk of T2DM. These results indicate that the assessment of future risk for T2DM requires that the beta cell be “stressed” in order to accurately assess its function. In contrast, prediction models based upon measurements made during the fasting states do not have the ability to assess beta cell function, and therefore, have modest predictive power for future diabetes risk compared to the 1-hour plasma glucose concentration following a glucose challenge.

In this study we also demonstrate that the 1-h PG is a useful measure to stratify Caucasian subjects into risk categories: low, intermediate and high. In general, subjects with NGT have low risk for progression to T2DM (<1% annual rate) (2). However, ~30-40% of individuals who develop T2DM have normal glucose tolerance at baseline (2) and, in the present study, 22% of subjects who developed T2DM had NGT at baseline. In this study we demonstrate that NGT subjects with a 1-h PG >155 mg/dl have a greater risk for future T2DM (8.5%) compared to NGT subjects with a 1-hour plasma glucose < 155 mg/dl (1.3%) ($P< 0.00001$). Further, NGT subjects with a 1-hour plasma glucose > 155 mg/dl, who fulfill the ATP III criteria for the metabolic syndrome, had a 15% risk for future T2DM. Thus, the group of NGT subjects with 1-hour PG > 155 mg/dl plus the metabolic syndrome is at high risk for the development of T2DM, and their risk exceeds that of subjects with IFG or IGT. Consistent with the ADA Consensus Conference Statement (23), this group of high risk NGT individuals could benefit from an intervention program employing diet and exercise, and possibly pharmacotherapy, to reduce future risk for diabetes.

Subjects with CGI have the greatest risk (13.5%) for future T2DM, 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 identifies high risk individuals. Thus, IFG and IGT subjects with a 1-h plasma glucose < 155 mg/dl have a <2% risk compared to a >10% risk for a 1-hour plasma glucose > 155 mg/dl. Thus, the plasma glucose concentration at 1 hour during the OGTT, independent of the glucose tolerance status, is a strong predictor for future T2DM. NGT, IFG and IGT subjects who fulfill the ATP III criteria for the metabolic syndrome and have a 1-hour plasma glucose > 155 mg/dl are at the greatest risk (>15%) for future type 2 diabetes, and. In addition to lifestyle intervention, pharmacological therapy should be considered in these subjects. Conversely, subjects with a 1-hour plasma glucose <155 mg/dl without the metabolic syndrome are at very low risk for future diabetes (<1%) and,
therefore, no intervention is necessary in this group.

In summary, measurement of post-glucose load plasma glucose concentration has additive value to models based only on fasting measurements in predicting the future risk for T2DM. Similar to Mexican Americans, the plasma glucose concentration at 1 hour during the OGTT is a strong predictor of future risk for T2DM in Caucasians and a 1-hour plasma glucose cut point of 155 mg/dl plus the ATP III criteria for the metabolic syndrome can be used to stratify non-diabetic subjects into low, intermediate and high risk categories.

ACKNOWLEDGEMENTS

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FIGURE LEGENDS:

**Figure 1:** Tree model based on the glucose tolerance status (NGT, IFG, IGT, IFG + IGT) of the subjects, 1 hour plasma glucose concentration greater than or less than 155 mg/dl, 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.
REFERENCES


24) Abdul-Ghani MA, Jenkinson C, Richardson D, and DeFronzo RA. Insulin secretion and insulin action in subjects with impaired fasting glucose and impaired glucose tolerance: results from the Veterans Administration Genetic Epidemiology Study (VEGAS). Diabetes 55:1430-5; 2006

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

<table>
<thead>
<tr>
<th></th>
<th>NGT</th>
<th>IFG</th>
<th>IGT</th>
<th>CGI</th>
<th>Total Population</th>
<th>ANOVA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>1110</td>
<td>949</td>
<td>123</td>
<td>260</td>
<td>2442</td>
<td></td>
</tr>
<tr>
<td>Sex (F\M)</td>
<td>661/449</td>
<td>427/522</td>
<td>72/51</td>
<td>155/105</td>
<td>1315/1127</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Age (years)</td>
<td>45±1</td>
<td>46±1</td>
<td>50±2</td>
<td>52±1</td>
<td>46±0.3</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>24.1±0.7</td>
<td>23.5±0.3</td>
<td>24.9±0.2</td>
<td>25.6±0.2</td>
<td>25.8±0.08</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Waist (cm)</td>
<td>84.5±0.4</td>
<td>88.4±0.4</td>
<td>90.1±1.1</td>
<td>92.7±0.8</td>
<td>87.2±0.24</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>FPG (mg/dl)</td>
<td>92±1</td>
<td>108±1</td>
<td>93±1</td>
<td>110±1</td>
<td>100±0.2</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>2-h PG (mg/dl)</td>
<td>99±1</td>
<td>109±1</td>
<td>158±2</td>
<td>158±1</td>
<td>112±0.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>T Chol (mM)</td>
<td>5.4±0.03</td>
<td>5.6±0.04</td>
<td>5.6±0.09</td>
<td>5.6±0.07</td>
<td>5.6±0.02</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>HDL Chol (mM)</td>
<td>1.42±0.01</td>
<td>1.37±0.03</td>
<td>1.33±0.03</td>
<td>1.29±0.02</td>
<td>1.38±0.01</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Triglyceride (mM)</td>
<td>1.15±0.02</td>
<td>1.31±0.03</td>
<td>1.51±0.08</td>
<td>1.65±0.06</td>
<td>1.28±0.02</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>BP (mmHg)</td>
<td>125/77</td>
<td>129/78</td>
<td>132/81</td>
<td>139/83</td>
<td>128/78</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>% with MS</td>
<td>8.1</td>
<td>30.5</td>
<td>22.8</td>
<td>55.4</td>
<td>22.6</td>
<td>&lt;0.0001</td>
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<tr>
<td># converted to diabetes</td>
<td>27</td>
<td>48</td>
<td>14</td>
<td>35</td>
<td>124</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>% converted to diabetes</td>
<td>2.4</td>
<td>5.1</td>
<td>11.5</td>
<td>13.5</td>
<td>5.1</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

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: Area under the ROC and simple correlation (Pearson) between plasma glucose concentration during the OGTT and log transformation of insulin secretion and insulin sensitivity indices

<table>
<thead>
<tr>
<th>Variable</th>
<th>aROC</th>
<th>Matsuda Index</th>
<th>ΔI₀-30/ΔG₀-30</th>
<th>ΔI₀-120/ΔG₀-120</th>
<th>ΔI₀-30/ΔG₀-30X Matsuda</th>
<th>ΔI₀-120/ΔG₀-120X Matsuda</th>
</tr>
</thead>
<tbody>
<tr>
<td>FPG</td>
<td>0.672</td>
<td>0.38*</td>
<td>0.06**</td>
<td>0.02</td>
<td>0.33*</td>
<td>0.24*</td>
</tr>
<tr>
<td>PG at 30</td>
<td>0.735</td>
<td>0.45*</td>
<td>0.42*</td>
<td>0.39*</td>
<td>0.73*</td>
<td>0.62*</td>
</tr>
<tr>
<td>PG at 60</td>
<td>0.795</td>
<td>0.50*</td>
<td>0.40*</td>
<td>0.55*</td>
<td>0.73*</td>
<td>0.72*</td>
</tr>
<tr>
<td>PG at 120</td>
<td>0.688</td>
<td>0.46*</td>
<td>0.11*</td>
<td>0.34*</td>
<td>0.42*</td>
<td>0.58*</td>
</tr>
<tr>
<td>HbA1c</td>
<td>0.679</td>
<td>0.18*</td>
<td>0.06*</td>
<td>0.1**</td>
<td>0.19*</td>
<td>0.19*</td>
</tr>
<tr>
<td>ΔG₀-120</td>
<td>0.77</td>
<td>0.48*</td>
<td>0.42*</td>
<td>0.6*</td>
<td>0.73*</td>
<td>0.83*</td>
</tr>
<tr>
<td>Score Model I</td>
<td>0.646</td>
<td>0.36*</td>
<td>0.02</td>
<td>0.015</td>
<td>0.05**</td>
<td>0.07**</td>
</tr>
<tr>
<td>Score Model II</td>
<td>0.74</td>
<td>0.40*</td>
<td>0.04</td>
<td>0.07**</td>
<td>0.01</td>
<td>0.07**</td>
</tr>
<tr>
<td>SADPM</td>
<td>0.743</td>
<td>0.51*</td>
<td>0.025</td>
<td>0.042</td>
<td>0.333*</td>
<td>0.351*</td>
</tr>
<tr>
<td>MS</td>
<td>0.72</td>
<td>0.56*</td>
<td>0.017</td>
<td>0.015</td>
<td>0.032</td>
<td>0.316*</td>
</tr>
<tr>
<td>MS + G60</td>
<td>0.813</td>
<td>0.53*</td>
<td>0.23*</td>
<td>0.272*</td>
<td>0.53*</td>
<td>0.53*</td>
</tr>
<tr>
<td>Model I + G60</td>
<td>0.805</td>
<td>0.38*</td>
<td>0.13*</td>
<td>0.09**</td>
<td>0.11*</td>
<td>0.22*</td>
</tr>
<tr>
<td>Model II + G60</td>
<td>0.822</td>
<td>0.43*</td>
<td>0.11*</td>
<td>0.09**</td>
<td>0.11*</td>
<td>0.22*</td>
</tr>
<tr>
<td>SADPM + G60</td>
<td>0.832</td>
<td>0.52*</td>
<td>0.214*</td>
<td>0.256*</td>
<td>0.52*</td>
<td>0.51*</td>
</tr>
</tbody>
</table>

$p<0.001$ compare to aROC for plasma glucose concentration at 60 minutes; $$* p<0.05$$ versus aROC for plasma glucose concentration 60 minutes; $p<0.0001$ compared to aROC of the same model without G60; * $p<0.0001$; ** $p<0.05$. MS = metabolic syndrome; SADPM = San Antonio Diabetes Prediction Model; PG = plasma glucose concentration; G60 = plasma glucose concentration at 60 minutes during OGTT
Figure 1.