Type 2 diabetes mellitus (T2DM) is a serious chronic disease with disordered carbohydrate metabolism that places a heavy burden on health services and patients due to its morbidity and mortality. The prevalence of T2DM is continuously growing worldwide. Remarkably, glucose intolerance (e.g., impaired glucose intolerance [IGT] and diabetes) is frequently asymptomatic and the delay from disease onset to clinical diagnosis may exceed at least 4 to 7 years. Strikingly, tissue damage progresses before diagnosis. Therefore, early diagnosis and intervention are important in reducing the burden of diabetic complications.

In one study, it was estimated that up to 50% of persons with diabetes were undetected or newly diagnosed. One of the possible causes may be attributable to a normal fasting plasma glucose (FPG) but abnormal postprandial hyperglycemia, i.e., IGT or diabetes-on-isolated postchallenge hyperglycemia (DM-on-IPH) may also be responsible.

The prevalence of IGT in Taiwan established in 1996 was 15.5%, which was higher than the prevalence of diabetes (9.2%). However, in Taiwan, about 40% of diabetics have not yet been diagnosed. Moreover, the mortality of T2DM is growing and was the fourth
leading cause of death in Taiwan in 2002 with about a 6.3-fold increase over a period of 30 years. Thus, we are encouraged to establish a simple and efficient predictive model to reduce the incidence of T2DM by early prediction and intervention.

The diagnostic criteria of diabetes was revised by the American Diabetes Association (ADA) in 1997. The main modifications emphasized using only FPG to diagnose diabetes and lower the cutoff point to 7.0 mmol/L. Furthermore, a new category of “impaired fasting glucose” (IFG) was introduced. Subsequently, the World Health Organization (WHO) criterion for the diagnosis of diabetes was also published. It retained the lower cutoff for FPG and, at the same time, suggested that the oral glucose tolerance test (OGTT) was still a useful method for diagnosing diabetes. However, after both criteria have been reported, many studies have found that the concordance between them was not so good. Furthermore, according to ADA criteria, whether persons with normal FPG are truly non-diabetic is an emerging problem. It could be noted that people with either DM-on-IFP or diabetes-on-isolated fasting hyperglycemia are difficult to detect by the 1997 ADA criteria. To solve these problems, many authors have suggested different methods to increase the sensitivity to detect diabetes, such as the level of glycated hemoglobin or a predictive risk score model.

Metabolic syndrome (MeS) is a cluster of metabolic factors, including central obesity, hypertension, dyslipidemia, and glucose intolerance. People with MeS are found to have a high risk for cardiovascular disease and T2DM. The central pathophysiology of MeS is generally agreed to be insulin resistance, which is also central to T2DM. The term “MeS” was coined by WHO in 1998 as an attempt at early detection of subjects at high risk for diabetes and cardiovascular diseases.

Three years later, the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) had also provided another similar, but simple and clinically useful definition of MeS.

In this study, we were interested in and focused on persons with normal FPG who had either normal glucose tolerance (NGT), IGT, or DM-on-IFP. We conducted a binary logistic regression analysis to obtain a model to estimate the probability of having dysglycemia (i.e., IGT and DM-on-IFP in our study). The components of MeS were put into the proposed models. A receiver-operating characteristic (ROC) curve was used to determine the predictive discrimination power of these models with the hope of obtaining a simple and efficient predictive model that could be widely acceptable in clinical or health care settings to identify subjects at high risk for glucose intolerance.

SUBJECTS AND METHODS

A total of 513 participants were enrolled and received the standard 75-g OGTT in Tri-Service General Hospital from 1998 to 2001. Subjects were either self-referred or referred by health professionals, seeking a screening for diabetes. They had no history of diabetes in the past. After excluding frank diabetes and IFG, only 424 cases were suitable for further study. Among them, 82 were classified as IGT (defined as 2-h PG during OGTT between 7.8 and 11.1 mmol/L and FPG < 6.1 mmol/L), another 6 as DM-on-IFP (defined as FPG < 6.1 mmol/L and 2-h plasma glucose (2-h PG during OGTT ≥ 11.1 mmol/L). However, due to incomplete data on family history or other parameters, only 106 subjects with NGT, 61 with IGT and 6 with DM-on-IFP were available for this study. None of the patients had significant medical or surgical history. Before the study, they were instructed by the doctors and dietitians not to receive any medication known to affect glucose or lipid metabolism and to stay on a stable diet for at least one week before the study. On the day of the visit, each subject had a complete routine work-up to rule out the presence of cardiovascular, respiratory, renal or endocrine disorders. The study had been approved by the hospital ethics committee, and the purpose and the potential risks of the study were explained to the subjects before obtaining their written consent to participate.

On the day of the test, a standard 75-g OGTT was carried out for 3 hours after a 12-h overnight fast. Blood samples were obtained for the determination of glucose and insulin concentrations at baseline (time 0 min) and 30-minute intervals for 3 hours. Other than the OGTT, homeostasis model assessment (HOMA) was also used to estimate the insulin sensitivity (HOMA-IR=fasting plasma insulin (μU/mL) × fasting plasma glucose (mmol/L)/22.5). HOMA is a mathematical model based on glucose and insulin interaction in different organs, including the pancreas, liver, and peripheral tissues. The model determines insulin sensitivity or insulin resistance. Application of HOMA has also been used in epidemiological studies.

Plasma was separated from blood within 1 hour and stored at −30°C until analyzed. Plasma glucose was determined by the glucose oxidase method (YSI 203 glucose analyzer, Scientific Division, Yellow Spring Instrument Company, Inc., Yellow Spring, Ohio, USA). Insulin was measured by a commercial radio-immunnoassay kit (Coat-A-Count insulin kit, Diagnostic Products Corporation, Los Angeles, California, USA).

Both triglycerides (TG) and total cholesterol (TC)
were measured using the dry, multilayer analytical slide method in the Fuji Drii Chem 3000 analyzer (Fuji Photo Film Corporation, Minato-iKu, Tokyo, Japan). Serum high-density lipoprotein cholesterol (HDL-C) concentration was determined by an enzymatic cholesterol assay method after dextran sulfate precipitation.

Since there were only 6 subjects with DM-on-IPH and the purpose of our study was to predict glucose intolerance only, we combined both the IGT group and DM-on-IPH group into one as a "dysglycemia group". Although there are a number of MeS definitions, the NCEP ATP III-defined MeS criteria has been widely applied in clinical and epidemiological research. Therefore, we employed these criteria in our study. However, since we did not have data on waist circumference, we used BMI instead.

Using binary logistic regression analysis we put all interesting factors into the model for model selection. Five models were proposed to identify normal and abnormal glucose metabolism. Each model included the different components of the MeS. The five models were as follows:

- Model 0 (FPG)  
- Model 1 (clinical model): family history, FPG, age and sex  
- Model 2 (the MeS model): all risk factors in Model 1 plus TG, HDL-C, BMI, systolic blood pressure (SBP) and diastolic blood pressure (DBP)  
- Model 3 (insulin was added for evaluating the effect of insulin level on the model): all risk factors in Model 2 plus fasting plasma insulin (FPI)  
- Model 4 (HOMA-IR was added): all risk factors in Model 3 plus HOMA-IR.

For Model 0, the FPG was forced into the model. The 95% confidence intervals were also calculated for the binary logistic regression analysis. The Hosmer-Lemeshow test was used to assess the goodness of fit of these models. Calculations were performed using the SPSS (10.0) statistical package (SPSS Inc., Chicago, IL, USA). A P-value (two-sided) < 0.05 was considered to be significant. For each individual in every model, an estimated probability of an abnormal event (occurrence of dysglycemia) was also calculated. We then used the estimated probability to predict whether a subject was at high risk for dysglycemia (see the appendix). A plot of the ROC curve, which is a line diagram with the sensitivity plotted vertically with the false positive rate on the horizontal axis, and is determined by the trapezoidal rule, was used to choose the cutoff of values. The diagonal line represents results no better than chance. The ROC curve is a mathematical method used to assess the predictive discrimination of a test. The statistical significance of differences in areas under ROC curves between any two models were estimated by likelihood ratio testing.

RESULTS

Table 1 shows the demographic data of the study subjects. From the selection criteria, it is not surprising that all subjects had normal FPG. The dysglycemic group was older and had a higher BMI, SBP, TG, 2-h PG, FPI and HOMA-IR than the NGT group. All data were adjusted for age and BMI.

Model 0 had only one risk factor (FPG) and was regarded as the "baseline model". The area under the ROC curve was significantly greater than the area under the curve of the diagonal reference line (Panel A, Figure 1), which means that the prediction rate could be improved significantly even with use of FPG alone.

Table 2 presents the areas under the ROC curves, their Hosmer-Lemeshow goodness-of-fit statistics, and tests of the statistical significance of the differences between various models by the likelihood ratio test. When compared to Model 0, all of the other 4 models have a larger area under the ROC curve (better prediction rate). Moreover, by putting more variables into the
DISCUSSION

This is the first study to examine the performance of a simple multivariable risk score model using routinely collected data related to MeS as a screening tool for undetected glucose intolerance in Taiwanese persons. Persons with normal FPG may be considered “non-diabetic”. However, several studies have reported that up to half of diabetics are undiagnosed. Compared to diabetes, IGT is even more difficult to diagnose since OGTT is not routinely done. Although IGT is generally recognized as a "pre-diabetic" state, it still carries an increased risk for developing cardiovascular complications similar to diabetes. Therefore, in practice, to identify individuals with dysglycemia it is important for clinicians so that preventive interventions can be given early.

OGTT is costly, time consuming, inconvenient and rarely used in an ordinary clinical setting, but it can identify a subject with normal FPG who may have glucose intolerance (e.g. IGT and DM-on-IPH). Therefore, there is a need to develop a widely accepted method for identifying subjects at high risk for glucose intolerance so that early intervention with lifestyle and/or pharmacologic management can be implemented to prevent or delay diabetes.

In our study, we proposed five models with multiple variables related to the components of the MeS, with Model 0, the baseline model, including FPG only. All five models (Figure 1, panel B), including Model 0, were statistically significant (P<0.05) meaning that all models for prediction would improve diagnostic performance. We did not use WC in our study because we did not measure it at that time. Although waist circumference is suggested by NCEP ATP III as a tool to define adiposity, its superiority can be questioned. First, Ford et al showed that the correlation coefficient between WC and BMI is high, up to 0.88 in different sex, age or ethnic groups. Secondly, measurement of height and weight is more easily done in a routine clinical setting and is more accurate than measurement of WC. Indeed, there were many different methods to measure WC, each of which would yield various absolute values. Thirdly, one study using the measurement of insulin-mediated glucose disposal has demonstrated a similar relationship between insulin resistance and adiposity, regardless of whether assessed by BMI or WC. Finally, both BMI and WC have been shown to be closely related to the cardiovascular risk factors in Taiwanese persons. For these reasons, we could justify the use of BMI instead of WC in our study and we believe that the findings are not substantially altered.

From the ROC curve of Model 1, the optimum cut-off point we arbitrarily selected was 5.43 mmol/L for FPG, which is less than the ADA criteria (5.6 mmol/L). This yields a sensitivity and specificity of 55.2% and 81.1%, respectively. Interestingly, the predictive discrimination of all of the other multivariable models outperforms Model 0. That is, each of the areas under ROC curves of Model 1 to 4 was greater than the area under the ROC curve of Model 0. The most significant increment in the area was noted between Model 1 and Model 2 (67.3% to 74.8%). This implies that adding components of MeS into the model will significantly increase the predictive discrimination power.

HOMA-IR is an important quantitative method for

### Table 2. Area under the receiver-operating characteristic (ROC) curve and their comparisons for models predicting dysglycemia.

| Models* and model comparisons | Area under the ROC curve (95% CI),% | P value (Hosmer-Lemeshow)** | P values for model comparisons (likelihood ratio tests)*** |
|-------------------------------|-----------------------------------|-----------------------------|----------------------------------------------------------|
| Models                        |                                   |                             |                                                          |
| 0                             | 64.8 (56.1-73.6)                  | 0.001                       |                                                          |
| 1                             | 67.3 (59-75.6)                    | 0.303                       |                                                          |
| 2                             | 74.8 (67.3-82.3)                  | 0.045                       |                                                          |
| 3                             | 76.8 (69.5-84.1)                  | 0.658                       |                                                          |
| 4                             | 76.6 (69.3-83.8)                  | 0.767                       |                                                          |
| Comparisons between models    |                                   |                             |                                                          |
| 0-1                           |                                   | 0.01                        |                                                          |
| 1-2                           |                                   | 0.003                       |                                                          |
| 2-3                           |                                   | 0.02                        |                                                          |
| 3-4                           |                                   | 0.414                       |                                                          |

*Model 0: FPG; Model 1 (clinical data): family history, FPG, age and sex; Model 2 (the MeS model): all risk factors in Model 1 plus TG, HDL-C, BMI, SBP and DBP; Model 3 (insulin was added for evaluating the effect of insulin level on the model): all risk factors in Model 2 plus FPI; Model 4 (HOMA-IR was added): all risk factors in Model 3, plus HOMA-IR.

**P values calculated by using the Hosmer-Lemeshow goodness-of-fit test.

***P values for test of difference in areas under two ROC curves; calculated by the likelihood ratio test.
Fasting plasma glucose 5.43 mmol/L

Figure 1A. Receiver operating characteristic curve of Model 0 (see methods for a description of the model). The optimum cutoff (5.43 mmol/L) is shown with an arrow (sensitivity: 55.2%, specificity: 81.1%; area: 67.3%).

Risk score = 0.39

Figure 1B. Receiver operating characteristic curves of the five models, Models 0-4 (see METHODS for a description of the models). The arrow indicates the arbitrarily selected risk score cutoff (0.39) of Model 3 (sensitivity, 70.1%; specificity, 73.6%; area, 76.8%).
predicting glucose intolerance.

In our study, no significant change in the area under the ROC curve was noted after adding the parameter of HOMA-IR into Model 4. This is not surprising because the HOMA-IR is derived from a simple equation multiplying FPI and FPG. Since Model 3 has both of FPI and FPG, Model 4 with HOMA-IR should not be significantly different from Model 3.

Although putting the FPI level into Model 3 will further increase the area under the ROC curve up to 76.8%, it should be noted that FPI is not routinely measured in a routine health check-up. Additionally, the cost is also too high. Thus, considering that there is no significant difference between Model 2 and Model 3 in performance and the simplicity of Model 2, Model 2 seems to be the most appropriate predictive model to be used in the current clinical and health care settings. Nevertheless, it should be noted that Model 3 has the largest area under the ROC curve. The cutoff will vary depending on workload and attitudes to false positives and false negatives. In Model 3, a cutoff of the risk score of 0.39 is suggested, which gives a sensitivity and specificity of 70.1% and 73.6%, respectively, with the area under the ROC curve of 76.8% (95% CI, 69.5-84.1%).

Although many other screening tools for undiagnosed diabetes have also been developed in earlier studies, our study specifically attempts to focus on those persons with normal FPG and evaluates their chance of developing glucose intolerance (dysglycemia). The Herman et al. study was the first one that tried to prospectively identify individuals at increased risk for diabetes by using a simple questionnaire. They proposed a classification tree incorporating age, sex, history of delivery of a macrosomic infant, obesity, sedentary lifestyle, and family history of diabetes. In their study, the sensitivity was 79% and the specificity was 65%, which were somewhat better than our model. This might be due to some of the risk factors used in their tree model being not included in our models. In this study, our main purpose was to develop a simple multivariable model consisting of readily available clinical measurements, especially related to the MeS, most of which are routinely obtained anyway. Therefore, we did not put parameters such as macrosomic infant and sedentary lifestyle into our models because they are not routinely collected in the clinical setting. Also, in their study, they tried to identify diabetes, which represents more severe abnormal glucose tolerance than IGT in our study. When a larger range of the risk factors (in this case, blood glucose) are put into the model, the sensitivity and specificity are more accurate and the area under ROC curve larger.

Griffin et al. developed the Cambridge risk score, another means of determining risk of diabetes. Other than common risk factors (age, gender, BMI), they considered a history of smoking, and steroid and antihypertensive medication use as risk factors. This model yielded an area under ROC curve of 80%. In another study done by Park et al. with the same model, the area under the ROC curve was 65.7%. However, steroid use is not a common condition in Taiwan and, again, this history is not available in a routine health check-up. Therefore, we did not put this risk factor into our model. Although the area under the ROC curve in our Model 2 (74.8%) did not have as good a performance as in the Griffin’s study, it is better than the Park study.

Of all the literature reviewed, Stern’s model was most similar to ours. In their study, the full model had the same risk factors as our model except for TC, low-density lipoprotein cholesterol (LDL-C), 2-h PG and sibling history. Their prediction model had an area under the ROC curve of around 85%, which is higher than that of our model. The area under the ROC curve in other similar studies using different models ranged from 67% to 80%. In the above comparisons, our model has also a relatively better performance in sensitivity and specificity. Since most general practical clinics have been computerized, it would be convenient to identify persons with a positive score on Model 2 or 3 with the assistance of a personal computer.

Our study has some limitations. We have to stress that the case cohort in our study was not selected independently and randomly and the population size was also relatively small. Therefore, further randomized and prospective larger-scale studies are needed to validate our predictive model. Nevertheless, to the best of our knowledge, our study is the first one to suggest that using an MeS-related multivariable model could predict subjects with glucose intolerance, including IGT. We hope our study can stimulate the initiation of a larger population-based study.

In conclusion, we have demonstrated that the predictive performance of Model 2 and 3 using the components of the MeS, which are routinely available data, and FPI is significantly better than FPG and/or family history alone. Without much effort, subjects with normal FPG at high risk for glucose intolerance could be identified early in the clinical setting, and then followed by the 75-g OGGT for further diagnosis. We hope that our predictive model can be validated by a large-scale longitudinal cohort study in the future and accepted widely by general practitioners.
PREDICTING GLUCOSE INTOLERANCE

Appendix:

The following are parameter estimates for the model

\[ p = \frac{1}{1 + e^{-\beta_0 + \beta_1 FPG + \beta_2 FPI + \beta_3 HDL-C + \beta_4 BMI + \beta_5 age}} \]

where \( X = 0.094 + 0.132(FPG) + 0.24 \times (sex) - 0.091(FH) + 0.039(age) + 0.07(BMI) + 0.014(SBP) - 0.001(DBP) + 0.474(TG) - 0.116(HDL-C) + 0.008(FPI) \). In this equation, \( \beta \) is the probability of developing diabetes; FPG, fasting plasma glucose in mmol/L; sex = 1 if female, 0 if male; FH = 0 if neither parent has diabetes, 1 if one of the parents has diabetes; age is in years; BMI, body mass index in kg/m²; SBP, systolic blood pressure in mm Hg; DBP, diastolic blood pressure in mm Hg; TG, triglycerides in mmol/L; LDL-C, low-density lipoprotein cholesterol in mmol/L; FPI, fasting plasma insulin in pmol/L.

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