# Progression to Type 2 Diabetes Among High-Risk Groups in Kin-Chen, Kinmen

## Exploring the natural history of type 2 diabetes

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**OBJECTIVE** — To examine the natural history of 654 high-risk subjects (340 men and 314 women) with fasting hyperglycemia (first fasting plasma glucose [FPG] level 5.6–7.8 mmol/l) who also exhibited 2-h postload glucose concentrations <11.1 mmol/l and an FPG level <7.8 mmol/l in a 75-g oral glucose tolerance test (OGTT). We were particularly interested in comparing the likelihood of developing type 2 diabetes for those with persistent fasting hyperglycemia (PFH), impaired glucose tolerance (IGT), and normal glucose tolerance (NGT). PFH is a relatively new definition, and those with PFH used to be defined as NGT according to WHO criteria.

**RESEARCH DESIGN AND METHODS** — Subjects were located in a 1992–1994 community-based population survey and followed up and reexamined during 1995–1996. An OGTT was used to determine who had progressed to type 2 diabetes. Risk factors predictive of subsequent progression to type 2 diabetes were determined by comparing baseline variables from the 1992–1994 survey with data of those who had or had not progressed to type 2 diabetes in 1995–1996.

**RESULTS** — Of 654 high-risk subjects screened in the baseline survey, 481 (73.5%, 255 men and 226 women) were followed up. Of these, 8.1% had progressed to diabetes (4.1% progression/year, 95% CI 2.3–5.9). Of 131 baseline IGT subjects, 17.6% progressed to diabetes (8.8% progression/year, 6.3–11.3), but only 7.4% of 95 PFH subjects (3.7% progression/year, 2.0–5.4) and 3.5% of 255 NGT subjects (1.8% progression/year, 0.1–3.0) progressed to diabetes.

**CONCLUSIONS** — The rates of progression to type 2 diabetes were lowest from the NGT subgroup, highest from the IGT group, with the PFH group in the middle, suggesting that PFH might be a transitional condition that precedes IGT and diabetes. Other significant predictors of subsequent diabetes were baseline BMI, baseline hyperuricemia, baseline FPG, and 2-h plasma glucose concentration.

Impaired glucose tolerance (IGT) is an intermediate condition between normal blood glucose regulation and diabetes. The National Diabetes Data Group and subsequently the World Health Organization (WHO) Expert Committee on Diabetes have developed the following definition of IGT: fasting plasma glucose (FPG) <7.8 mmol/l and a 2-h plasma glucose during the oral glucose tolerance test (OGTT) between 7.8 and 11.1 mmol/l (1–3). Since the introduction of the currently used WHO criteria for glucose tolerance, only a few prospective studies based on these criteria have been completed, including those that use the Nauru of Micronesia (4), the Pima Indians (5,6), and the Maltese (7) as study subjects. In all of these, subjects with IGT have been found to be at risk for developing type 2 diabetes, and, therefore, they constitute a possible target group for intervention (5,8). Recently, Alberti (9) suggested that IGT is a useful risk factor category not only for type 2 diabetes but also for cardiovascular disease.

Wasada et al. (10) used the criteria of the Japan Diabetes Society, which includes a category called borderline glucose intolerance, which covers a broader spectrum than IGT. Nonetheless, Wasada's study is important because he concluded that insulin resistance, as found in borderline glucose intolerance, plays an important role in the pathogenesis of diabetes.

Only limited information is available about subjects with consistent fasting hyperglycemia but normal 2-h glucose levels, a condition called persistent fasting hyperglycemia (PFH). PFH is a relatively new definition, and those with PFH used to be defined as having normal glucose tolerance (NGT) according to WHO criteria. It remains to be clarified whether PFH represents an earlier stage, preceding IGT, in the development of diabetes (11). The present study was undertaken to evaluate the natural history and risk factors associated with the development of type 2 diabetes in subjects with fasting hyperglycemia (5.6–7.8 mmol/l) and 2-h postload glucose concentrations <11.1 mmol/l, which includes the IGT, PFH, and NGT groups in the baseline survey.

## RESEARCH DESIGN AND METHODS

### Study population

Kinmen is series of islands located in the Pacific Ocean between Taiwan and mainland China. Its population consists of Han Chinese whose ancestors came from southern China several hundred years ago. A series of community-based epidemiological surveys in Kinmen detailing characteristics of the target population have been reported (12–17).

Subjects for this study were recruited from a group that was classified as having fasting hyperglycemia (5.6–7.8 mmol/l) but who also exhibited 2-h postload glucose concentrations <11.1 mmol/l. The original

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**Abbreviations:** dBP, diastolic blood pressure; FPG, fasting plasma glucose; IGT, impaired glucose tolerance; NGT, normal glucose tolerance; OGTT, oral glucose tolerance test; OR, odds ratio; PFH, persistent fasting hyperglycemia; sBP, systolic blood pressure; WHO, World Health Organization; WHR, waist-to-hip ratio.

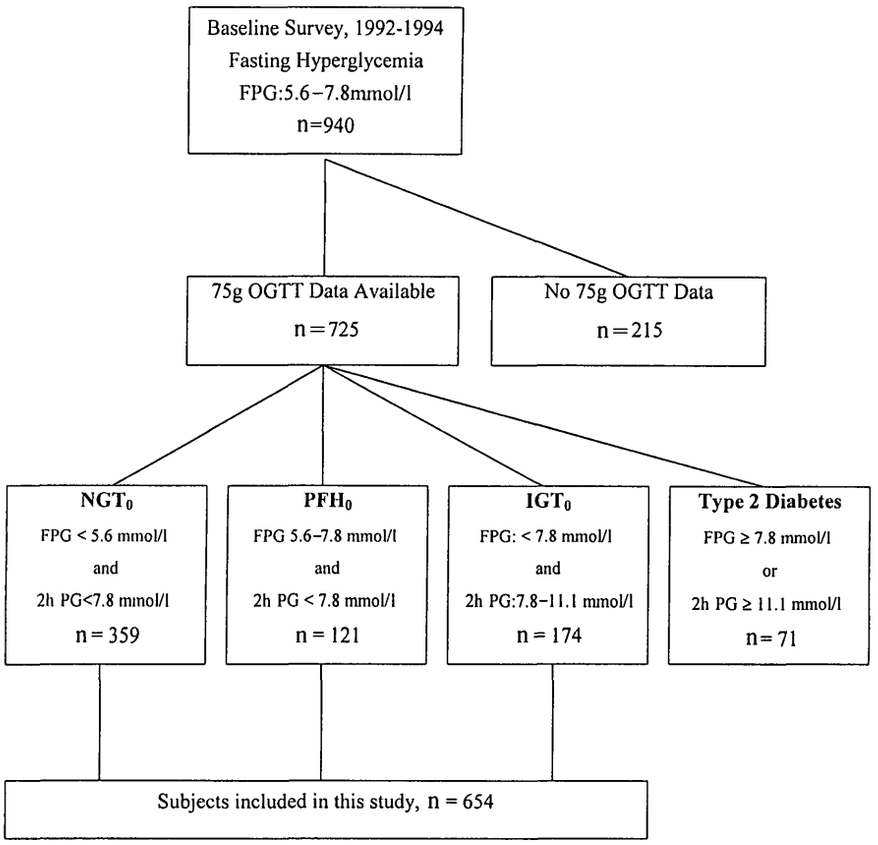


**Figure 1**—*Flow chart of diagnostic criteria and subjects included in this study, as selected from a baseline survey in Kin-Chen, Kinmen, in 1992–1994.*

classification took place during a 1992–1994 community-based population survey of glucose tolerance in Kin-Chen, the largest township in Kinmen (18). All residents over 30 years of age were included in that study. Blood glucose classifications were based on a modified version of the OGTT as defined by the WHO in 1985 (2).

A total of 4,654 people participated in the baseline survey, which took place during February 1992 and February 1994. Those with fasting hyperglycemia (5.6–7.8 mmol/l) totaled 940, but OGTT confirmation had not been completed for 215 of these cases. The remaining 725 were classified into four groups based on the results of the OGTT: *1*) 71 (9.8%) with type 2 diabetes, 2) 174 (24.0%) with IGT, *3*) 121 (16.7%) with PFH, and 4) 359 (49.5%) with NGT. The 654 individuals in the nondiabetic groups (referred to as the $IGT_0$, $PFH_0$, and $NGT_0$ groups) were considered eligible subjects for this study (Fig. 1). Of these, 481 subjects (255 men and 226 women) were followed up and reclassified into four groups based on the results of a second OGTT in 1995–1996. These groups will be referred to as $DM_1$, $IGT_1$, $PFH_1$, and $NGT_1$.

## Survey procedure

We attempted to contact all 654 of the nondiabetic subjects from the baseline survey by mail and phone, and we invited them to go to the Kinmen Health Bureau for a second OGTT (venous plasma glucose after an overnight fast and 2 h after ingestion of 75 g of dextrose monohydrate dissolved in 300 ml water). On the day of testing, the study participants were also interviewed by members of Yang-Ming Crusade, an organization of medical students from National Yang-Ming University (19). Interview data included details of age, sex, and history of diabetes and hypertension. Height and weight were measured for calculation of BMI. Waist circumference was measured as the minimal abdominal girth between the rib cage and iliac crest; hip circumference was measured as the maximal horizontal girth between waist and thigh. (Others have suggested that obesity should be computed using waist measurement alone instead of the waist-to-hip ratio [WHR] [20]. We tried this, but the results were nearly identical, so we did not include this data.)

## Biochemical measurements

Overnight fasting serum and plasma samples (preserved with EDTA and NaF) were collected by the nurses of the Kinmen Health Bureau and kept frozen (−20°C) until analyzed. FPG concentrations were determined using the hexokinase-glucose-6-phosphate dehydrogenase method with a glucose (HK) reagent kit (Gilford, Oberlin, OH).

## Classification of glucose tolerance

Glucose tolerance classification was based on currently used WHO criteria for a 75-g OGTT (2). NGT was defined as FPG <5.6 mmol/l and a 2-h plasma glucose <7.8 mmol/l. Type 2 diabetes was defined as either FPG ≥7.8 mmol/l or a 2-h plasma glucose ≥11.1 mmol/l. IGT was defined as FPG <7.8 mmol/l and a 2-h plasma glucose ≥7.8 and <11.1 mmol/l. PFH was

**Table 1**—*Results of follow-up among nondiabetic subjects with fasting hyperglycemia in Kin-Chen, 1995–1996*

| | 1992–1994 baseline | 1995–1996 | NGT | PFH | IGT | Diabetes |
|---|---|---|---|---|---|---|
| Age (years) | | | | | | |
| 30–39 | 155 | 116 (74.8) | 77 (66.4 | 28 (24.1) | 9 (7.8) | 2 (1.7) |
| 40–49 | 168 | 129 (76.8) | 57 (44.2) | 39 (30.2) | 25 (19.4) | 8 (6.3) |
| 50–59 | 167 | 127 (76.0) | 42 (33.1) | 41 (32.3) | 25 (19.7) | 19 (15.0) |
| 60+ | 164 | 109 (66.5) | 40 (36.7) | 27 (24.8) | 32 (29.4) | 10 (9.2) |
| Men | 340 | 255 (75.0) | 126 (49.4) | 65 (25.5) | 41 (16.1) | 23 (9.0) |
| Women | 314 | 226 (72.0) | 90 (39.8) | 70 (31.0) | 50 (22.1) | 16 (7.1) |
| Total | 654 | 481 (73.5) | 216 (44.9) | 135 (28.1) | 91 (18.9) | 39 (8.1) |

Data are *n* or *n* (%).

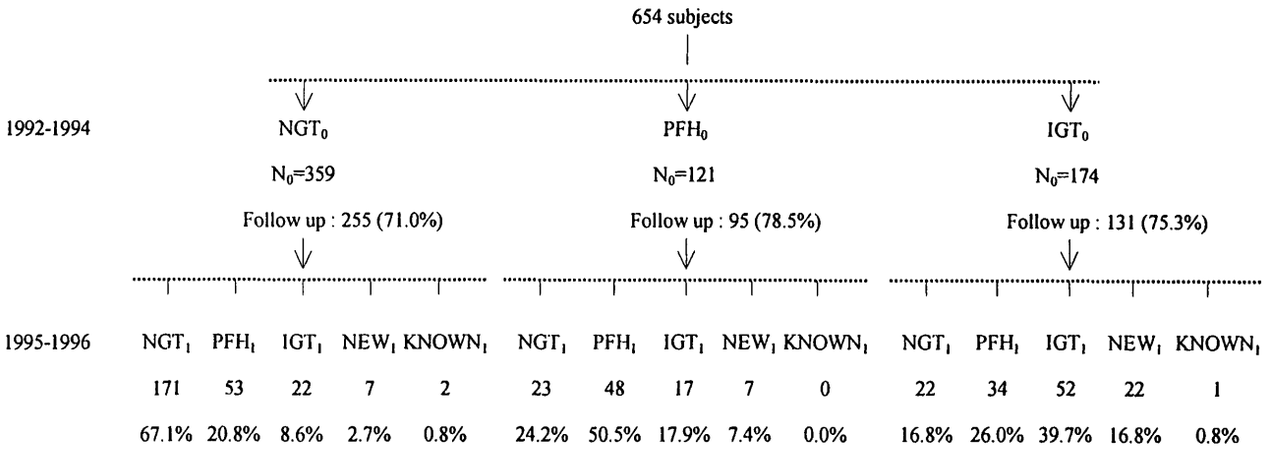


**Figure 2**—*Results of follow-up study. "New" and "known" are both type 2 diabetes groups. Those in the "known" group were diagnosed with diabetes before the 1995–1996 survey and were on medication. Those in the "new" group were discovered through testing in the 1995–1996 survey.*

defined as FPG of 5.6–7.8 mmol/l and a 2-h plasma glucose <7.8 mmol/l.

### Statistical analyses

In the univariate analysis, the means of selected baseline clinical and biochemical variables of the group that progressed to type 2 diabetes ($DM_1$) and the groups that did not ($IGT_1$, $PFH_1$, and $NGT_1$) were compared using either the Student's *t* test or the $\chi^2$ test. In the multivariate analysis to evaluate the effect of various risk factors, a stepwise logistic regression model was fitted to predict progression to type 2 diabetes. Statistical Analysis System (SAS) software was used.

**RESULTS** — Of the 654 subjects eligible for this study (i.e., nondiabetic subjects with fasting hyperglycemia from the baseline survey), 481 (73.5%) were followed up, and 173 were lost to follow-up. Of the 481 subjects, 39 (8.1%) developed diabetes, 91 (18.9%) had IGT, 135 (28.1%) had PFH, and 216 (44.9%) had NGT (Table 1, Fig. 2). The crude rate of progression to diabetes was 4.1%/year (95% CI 2.3–5.9). When the 173 nonrespondents were included in the analysis, the minimum proportion that progressed to diabetes was 6.0% (39 of 654) with a rate of progression of 3.0%/year (1.5–4.5).

Of those in the $IGT_0$ group, 131 (75.3%) completed the study. Of the 131, 23 (17.6%) progressed to diabetes (8.8%/year, 6.3–11.3), 52 (39.7%) remained with IGT, 34 (26.0%) had PFH, and 22 (16.8%) reverted to NGT. Of those in the $PFH_0$ group, 95 (78.5%) completed the study. Of the 95, 7 (7.4%) progressed to diabetes (3.7%/year; 2.0–5.4), 17 (17.9%) were reclassified as having IGT, 48 (50.5%) remained with PFH, and 23 (24.2%) reverted to NGT. Of those in the $NGT_0$ group, 255 (71.0%) completed the study. Of the 255, 9 (3.5%) progressed to diabetes (1.8%/year; 0.1–3.0), 22 (8.6%) were reclassified as having IGT, 53 (20.8%) had PFH, and 171 (67.1%) remained with NGT.

To assess which factors were associated with subsequent progression to diabetes in subjects with fasting hyperglycemia (5.6–7.8 mmol/l) and 2-h postload glucose concentrations <11.1 mmol/l (1992–1994), the

**Table 2**—*Results of clinical and biochemical characteristics in 1995–1996 among 481 nondiabetic subjects with fasting hyperglycemia at baseline in 1992–1994 in Kin-Chen*

| | Diabetic | Normal | *P* value |
|---|---|---|---|
| *n* | 39 | 442 | |
| Age (years) | 54.92 ± 1.69 | 49.82 ± 0.58 | 0.0118 |
| Sex (men) | 58.97 | 52.49 | NS |
| WHR | 0.88 ± 0.009 | 0.86 ± 0.003 | 0.0294 |
| Obese 1 (yes) | 25.64 | 25.11 | NS |
| BMI | 26.17 ± 0.56 | 24.03 ± 0.16 | 0.0001 |
| Obese 2 (yes) | 51.28 | 26.03 | 0.001 |
| Previous hypertension (yes) | 20.51 | 14.48 | NS |
| dBP (mmHg) | 88.62 ± 1.82 | 85.05 ± 0.62 | NS |
| sBP (mmHg) | 141.88 ± 3.59 | 135.92 ± 1.03 | NS |
| Hypertension/borderline hypertension | 41.03/20.51 | 25.06/23.23 | NS |
| Uric acid (mmol/l) | 0.39 ± 0.015 | 0.34 ± 0.004 | 0.0024 |
| Hyperuricemia (yes) | 45.95 | 21.20 | 0.001 |
| Cholesterol (mmol/l) | 5.28 ± 0.16 | 5.37 ± 0.06 | NS |
| Triglycerides (mmol/l) | 1.08 ± 0.09 | 0.93 ± 0.02 | NS |
| HDL cholesterol (mmol/l) | 1.25 ± 0.05 | 1.42 ± 0.02 | 0.0081 |
| Fasting insulin (pmol/l) | 126.58 ± 14.69 | 109.60 ± 2.96 | NS |
| Fasting C-peptide (nmol/l) | 0.63 ± 0.09 | 0.50 ± 0.01 | NS |
| FPG (mmol/l) | 6.18 ± 0.10 | 5.97 ± 0.02 | 0.0375 |
| 120-min plasma glucose (mmol/l) | 8.17 ± 0.27 | 6.54 ± 0.08 | <0.0001 |
| $IGT_0$ (yes) | 58.97 | 24.43 | <0.001 |

Data are *n*, means ± SEM, or %. *P* values are for comparisons between the two groups by Student's *t* test or $\chi^2$ test; $P > 0.05$. Obese 1 subjects are in the upper 75th percentile of WHR distribution: WHR ≥0.916 for men; ≥0.878 for women. Obese 2 subjects have a BMI ≥27 for men; ≥25 for women. Hypertension is sBP/dBP ≥160/95. Borderline hypertension is 160/95 > sBP/dBP ≥ 140/90. Hyperuricemia is uric acid ≥0.42 for men, ≥0.36 for women. IGT includes subjects belonging to the $IGT_0$ group at baseline.

**Table 3—*Multiple logistic regression model predicting progression to type 2 diabetes in 1995–1996 among nondiabetic subjects with fasting hyperglycemia at baseline in 1992–1994***

| | Parameter ± SEM | OR (95% CI) | *P* value |
|---|---|---|---|
| Intercept | −12.778 ± 3.043 | — | 0.0001 |
| Obese 2 (yes vs. no) | 1.058 ± 0.452 | 2.881 (1.188–6.986) | 0.0191 |
| Hyperuricemia (yes vs. no) | 1.254 ± 0.455 | 3.504 (1.436–8.549) | 0.0058 |
| FPG (continuous) | 0.948 ± 0.443 | 2.581 (1.083–6.149) | 0.0322 |
| 120-min plasma glucose (continuous) | 0.502 ± 0.135 | 1.652 (1.268–2.152) | 0.0002 |

Dependent variable: current diabetes vs. nondiabetes; independent variables: age, sex, obese 1 (WHR), obese 2 (BMI), previous hypertension, definite hypertension, borderline hypertension, hyperuricemia, cholesterol, triglycerides, HDL cholesterol, fasting insulin, fasting C-peptide, FPG, 120-min plasma glucose, membership in the $IGT_0$ group at baseline. Obese 2 subjects have a BMI ≥27 for men, ≥25 for women. Hyperuricemia is uric acid ≥0.42 mmol/l for men; ≥0.36 mmol/l for women.

means of selected baseline clinical and biochemical variables were compared. For this univariate analysis, all subjects were divided into two groups: *1*) those who progressed to diabetes (the $DM_1$ group) and 2) those who did not (the combined $IGT_1$, $PFH_1$, and $NGT_1$ groups) (Table 2).

Those who progressed to diabetes were significantly older. Mean WHR (0.88 vs. 0.86, $P < 0.05$) and mean BMI (26.17 vs. 24.03 kg/m$^2$, $P < 0.01$) were significantly higher in the $DM_1$ group. The obesity prevalence in both groups was similar (25.64 vs. 25.11%, $P > 0.05$) according to WHR but significantly different (51.28 vs. 26.03%, $P < 0.01$) according to BMI. Mean HDL cholesterol (1.25 vs. 1.42 mmol/l, $P < 0.01$) was significantly lower in the $DM_1$ group compared with group 2. Mean uric acid (0.39 vs. 0.34 mmol/l, $P < 0.01$), FPG (6.18 vs. 5.97 mmol/l, $P < 0.05$), and 2-h plasma glucose (8.17 vs. 6.54 mmol/l, $P < 0.01$) values were significantly higher in the $DM_1$ group. The prevalence of hyperuricemia (uric acid 0.42 mmol/l for men; 0.36 mmol/l for women) after adjusting for sex was 45.95 vs. 21.20% ($P < 0.01$). The proportion of subjects with IGT at baseline were higher in the $DM_1$ group (58.97 vs. 24.43%, $P < 0.01$).

To assess their independent contribution to the probability of progression to diabetes, the baseline variables were further examined using multiple logistic regression analysis (Table 3). We found that obesity (BMI), hyperuricemia, FPG, and 2-h plasma glucose were significant predictors of subsequent diabetes in the final model.

**CONCLUSIONS** — In this study, the proportion of subjects with IGT who progressed to diabetes was 17.6% over 2 years (rate: 8.8%/year, 6.3–11.3). This is consistent with most published prospective studies, which report that in the majority of people with IGT, glucose tolerance returns to NGT or persists unchanged (4–7,21–24) (Table 4). The risk of progression in this study is lower than that found in South African Indians (50.4% over 4 years), which is one of the highest recorded in the literature (25), but it is higher than that found in Pima Indians (31% over 1.6–11 years; rate: 5–6%/year) (5) and Nauruans (26% over 6 years; rate: 4%/year) (4). The proportion of subjects who remained with IGT in this study (39.7% over 2 years) is higher than that found in South African Indians (24.8% over 4 years). We also found a larger percentage of subjects who reverted to NGT (42.8%) compared with the South African Indians (24.8%) (25).

There has recently been one other study of progression of IGT to type 2 diabetes in an ethnic Chinese population (26). In that study, there was a cumulative incidence of diabetes at 6 years of 46.0% (37.3–54.7) in the study group (diet plus exercise) and 67.7% (59.8–75.2) in the control group. We computed the average annual progression to diabetes for that study in order have a more relevant comparison with our data. Average annual progression to diabetes for those with IGT in our study (8.8%/year) was lower than the control group in the other study (11.2%/year) and higher than the study group (7.7%/year). This is justified because our study population included people with various types of dietary and exercise habits.

The definition of PFH used in this study is relatively new, and people with PFH used to be classified as having NGT according to WHO criteria, so there is very little relevant data that can be compared. This study found that the rate of progression from PFH to diabetes (7.4% over 2 years, 3.7%/year, 2.0–5.4) was between the rates of progression from NGT to diabetes (1.8%/year, 0.1–3.0) and from IGT to diabetes (8.8%/year, 6.3–11.3). This rate of progression from PFH to diabetes was also lower than the rate of progression from IGT to type 2 diabetes in Pima Indians (5–6%/year) and Nauruans (4%/year). The above findings, as well as other findings of ours (18), indicate that people with PFH are less likely to develop diabetes than are people with IGT and more likely to develop diabetes than are those with NGT, which suggests that PFH may precede IGT in the progression toward diabetes.

In this study, the rate of progression to diabetes over 2 years for those in high-risk groups was 8.1%, but in another study in Pu-Li, the rate of progression over 4 years was 7.2% (21). The reason for this difference is that the general population was used as the study population in the Pu-Li study, and this included both high-risk and low-risk individuals.

Those who developed diabetes in our study were significantly older, were more likely to have belonged to the $IGT_0$ group, and had significantly higher WHR, BMI, uric acid, FPG, and 2-h glucose values. They also had significantly lower HDL cholesterol values. After controlling for covariates, stepwise logistic regression analysis showed that

**Table 4—*Summary of studies of the progression from IGT to type 2 diabetes***

| Study population | Participants with IGT (*n*) | Follow-up (years) | IGT to type 2 diabetes progression [*n* (%)] | Progression rate per year (%) |
|---|---|---|---|---|
| South African Indians (25) | 113 | 4 | 57 (50.4) | 12.6 |
| Pima Indians (5) | 384 | 1.6–11.5 | 118 (30.7) | 5–6 |
| Nauruans (4) | 51 | 6 | 13 (26) | 4 |
| Da Qing, China (26) | | | | |
| Control group | 133 | 6 | 90 (67.7) | 11.2 |
| Diet and exercise group | 126 | 6 | 58 (46.0) | 7.7 |
| Kin-Chen, Kinmen | 131 | 2 | 23 (17.6) | 8.8 |

obesity (a BMI of ≥27 for men, ≥25 for women), hyperuricemia, baseline FPG, and 2-h plasma glucose were significant predictors of subsequent diabetes.

Particularly, both the baseline FPG and 2-h plasma glucose levels were consistent with the majority of studies of subjects with IGT (4–7,27). The observation that obesity is a risk factor is also in agreement with other studies (28–30). In other studies with IGT subjects (4,5), BMI was not found to be a significant predictor in multivariate analysis, but it is significant in multivariate analysis in this study. It is particularly interesting that in this study, subjects with hyperuricemia (uric acid ≥0.42 mmol/l for men; ≥0.36 mmol/l for women) had a 3.50 times higher rate of developing diabetes.

In conclusion, this study has demonstrated that those with PFH are more likely to develop diabetes than those with NGT, but less likely than those with IGT, suggesting that PFH may precede IGT in the progression toward type 2 diabetes. Other significant predictors of subsequent diabetes include baseline BMI, baseline hyperuricemia, baseline FPG, and 2-h plasma glucose concentrations.

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