

Impaired fasting glycaemia resembles impaired glucose tolerance with regard to cardiovascular risk factors: population-based, cross-sectional study of risk factors for cardiovascular disease

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Abstract

Aims To compare subjects with impaired glucose tolerance and impaired fasting glucose in relation to risk factors for developing cardiovascular disease.

Methods A total of 1374 patients (678 female, 696 male) listed with a general practice clinic in Denmark were given an oral glucose tolerance test, a physical examination, and a self-administered questionnaire. Risk factors for cardiovascular disease were assessed for 90 participants (48 female, 42 male) with impaired glucose tolerance (including 12 subjects (1 female and 11 male), who also fulfilled criteria for impaired fasting glycaemia) and 51 subjects (20 female, 31 male) with impaired fasting glycaemia (World Health Organization 1999 criteria).

Results There were no statistical differences with regard to known risk factors for cardiovascular disease between participants with isolated impaired fasting glycaemia and those with impaired glucose tolerance.

Conclusions We found noticeable similarities in the cardiovascular risk factor profile in subjects with impaired fasting glycaemia and in subjects with impaired glucose tolerance in our population. When planning screening initiatives, it seems relevant to take into account people with impaired fasting glycaemia as well as those with impaired glucose tolerance.

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Keywords cardiovascular risk factors, cross-sectional study, impaired fasting glycaemia, impaired glucose tolerance, metabolic syndrome

Abbreviations T2DM, Type 2 diabetes mellitus; 2-hBG, blood glucose 2 h after a 75-g glucose load; ADA, American Diabetes Association; BMI, body mass index; CI, confidence interval; CRP, C-reactive protein; CV, between assay coefficient of variation; CVD, cardiovascular disease; EGIR, European Group for the Study of Insulin Resistance; FBG, fasting blood glucose; HbA_{1c}, glycated haemoglobin; IFG, impaired fasting glycaemia; IGR, impaired glucose regulation; IGT, impaired glucose tolerance; IQR, interquartile range; MS, metabolic syndrome; NFG, normal fasting glucose; NGT, normal glucose tolerance; OGTT, oral glucose tolerance test; PAI-1, plasminogen activator inhibitor 1; r.p.m., rotations per minute; SD, standard deviation; TG, triglycerides; t-PA, tissue type plasminogen activator; vWF, von Willebrand factor; WHO, World Health Organization

Introduction

Impaired glucose tolerance (IGT) is a risk factor for the development of Type 2 diabetes mellitus (T2DM) and cardiovascular disease (CVD) [1–3]. Lifestyle intervention in people with IGT in terms of increased physical activity and weight reduction diminishes the risk of developing diabetes [4,5].

In 1997, an Expert Committee of the American Diabetes Association (ADA) proposed a set of revised criteria for the diagnosis of diabetes mellitus introducing a new category of abnormal glucose regulation, impaired fasting glycaemia (IFG) [6]. These criteria were later adapted by the World Health Organization (WHO) [7]. Since presentation of the new criteria, several investigators have argued for retaining the oral glucose tolerance test (OGTT) for diagnosis of impaired glucose regulation (IGR) [8–12]. So far there is little knowledge of the clinical significance of progression to CVD in the IFG group, and no evidence that interventions can reduce the progression to overt T2DM. It has been claimed that the two groups of IGR people have different risks of developing diabetes and CVD [13–16], and it has been pointed out that the risk factor profile may be population dependent [17].

Our aim was to compare risk factors for CVD in people with impaired fasting glycaemia (IFG) and those with impaired glucose tolerance (IGT) found by primary screening in a single general practice in Denmark.

Patients and methods

Study population

During the period April 1998 to June 2000, an investigation aimed at describing the metabolic syndrome (MS) was carried out in a Danish general practice.

A total of 3108 people were listed with the practice. Patients suffering from severe physical or mental illnesses were excluded, as were insulin-treated diabetic patients. Fertile women were scheduled for examination between days 20 and 25 of their menstruation cycle. Median for examination was day 21 (IQR 20–24). Of 2082 eligible people aged 20–69 years, 1374 persons (66.0%) gave written informed consent and were examined (Fig. 1). The investigation was carried out according to the declaration of Helsinki II and was approved by the regional Research Ethics Committee.

Examination

The participants were asked to avoid heavy physical activity for the 3 days immediately before examination and to avoid alcohol consumption the day before examination. After an overnight fasting period of 10 h, an OGTT with 75 g of glucose (Glucodex®, Rougier, Chambly, Québec, Canada) was performed [18]. Blood sampling during OGTT was done at scheduled times (at $t = 30$ min 99.6% were sampled within ± 2 min and at $t = 120$ min 99.6% within ± 1 min). As haemolysis considerably lowers the results of insulin analysis, it is mandatory to minimize this phenomenon. Haemolysis occurred in a total

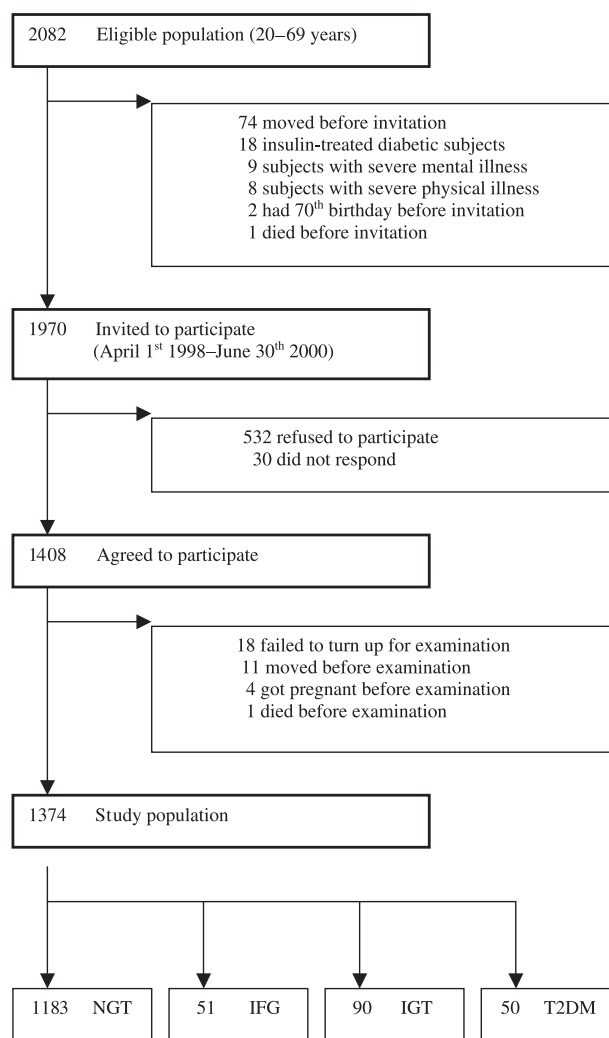


Figure 1 Inclusion and exclusion of subjects, and outcome classified according to glucose tolerance (WHO 1999).

of 15/4120 (0.36%) tubes, of which only two (0.05%) were sampled at $t = 0$ and both were above the top quartile, thus without significance for the present study.

For the purpose of standardization, the fasting period was initiated by consumption of a slice of bread without fat and a glass of tap water. The participants were told specifically not to drink, eat, smoke tobacco, or take any medication from this time until the end of examination. During the OGTT, the people were seated in an adjacent, pleasantly heated room. They were allowed to walk the few paces from here to the examination room when called upon for blood samples. Four participants were invited for examination per day. Meeting times were 06.00, 06.15, 07.00 and 07.15 h.

Anthropometric characteristics

The same investigator (PEH) carried out the physical examination of all 1374 participants. They were weighed in their underwear and the weight was registered to the nearest 100 g (Seca® Electronic 0–200 kg). Height was measured to the nearest

0.5 cm. Body mass index (BMI) was calculated as weight divided by height squared (kg/m^2). Waist circumference was measured in the umbilical plane directly on the skin with a tape measure. Blood pressure (BP) was measured in the seated position by auscultation over the brachial artery to the nearest 2 mmHg (Hawksley Random Zero Mk II Mercury Sphygmomanometer) using Korotkoff sound 1 and 5. The manometer was placed at the heart level. BP was measured on the dominant arm, and in case of ambidexterity the right arm was chosen. BP was measured three times during the examination. In order to minimize vessel damage due to stasis by the blood pressure cuff, the first BP at $t = 0$ min, i.e. after 15 min at rest, was measured just *after* the first blood sample was taken but *before* the glucose solution was swallowed. The second BP was measured at $t = 30$ min, i.e. just *after* the second blood sample was taken. The third BP at $t = 120$ min was measured just *before* the last blood sample was taken with the participant still seated in the adjacent room. The pulse rate was measured by palpation over the radial artery at the corresponding times after measuring the BP.

Patient questionnaires

Self-reported physical activity (Baecke Questionnaire of Habitual Physical Activity) [19], cardiovascular diseases (London School of Hygiene Cardiovascular Questionnaire) [20], smoking habits, dietary details, medication and family history of diabetes and CVD (parents and siblings) were all assessed by self-administered questionnaires.

Biochemical methods

Blood samples were drawn from an antecubital vein at $t = 0$, 30 and 120 min. Most analyses were carried out on fasting samples drawn at $t = 0$ min. Serum was prepared for analysis of insulin, proinsulin, c-peptide [AutoDELFIA™ two-sited (double antibody) technique using monoclonal antibodies (insulin-pro-insulin crossreactivity $< 0.4\%$)]¹, thyroidea stimulating hormone (TSH) (AutoDELFIA™ automatic immunoassay system, Wallac Oy Turku, Finland)², uric acid (Colorimetric test, VITROS Ektachem 950 IRC)², creatinine (Two-point rate test, VITROS Ektachem 950 IRC)², total cholesterol (Colorimetric test, VITROS Ektachem 950 IRC)², HDL-cholesterol (Liquid-N-geneous HDL-C, Genzyme, RA 1000)², and triglycerides (TG) (Colorimetric test, VITROS Ektachem 950 IRC)². LDL-cholesterol was calculated using Friedewald's formula: $\text{LDL-cholesterol} = \text{total cholesterol} - 0.45 \times \text{triglyceride}$.

Citrate-stabilized plasma was prepared for analysis of plasminogen-activator-inhibitor (PAI)-1 antigen (Imulyse PAI kit, Biopool, Umeå, Sweden)(CV 2.4%)³, tissue-plasminogen-activator (t-PA) antigen (Imulyse t-PA kit, Biopool) (CV 3.5%)³, C-reactive protein (CRP) (monoclonal antibodies, BN II analyser, DadeBehring, Marburg, Germany) (CV 6.3%)³ fibrinogen (modified Clauss procedure, ACL 7000, Instrumentation Laboratory, Milano, Italy)³, and von Willebrand factor (vWF) (ELISA-method, DAKO, Copenhagen, Denmark) (CV 9.5%)³. These blood samples were placed in ice water and centrifuged at 4°C and 1200 g.

Whole blood was stabilized with EDTA for analysis of glycated haemoglobin (HbA_{1c}) (HPLC VARIANT™ Bio-Rad

Laboratories, Hercules, CA, USA)². Within 5 min of drawing the sample, EDTA-stabilized whole blood was also added in duplicate to a buffer and within 5 hours analysed for glucose (Enzymatic amperometric test, EBIO Eppendorf-Netheler-Hinz GmbH) (CV 3.3%)². Each of the two samples was analysed separately, and the result is reported as mean of the two.

All blood-samples throughout the time of investigation and all preparations of blood for shipment to other laboratories together with all analyses for blood glucose were performed by the same highly skilled and thoroughly instructed medical laboratory technologist.

At $t = 30$ min and 120 min, serum was prepared for analysis of insulin¹, pro-insulin¹ and c-peptide¹, and EDTA-stabilized whole blood was analysed for glucose² according to the above mentioned standards.

Insulin resistance was calculated using the homeostasis model assessment ($\text{HOMA}_{(\text{IR})}$) according to the formula: $\text{IR} = (\text{fasting insulin} \times \text{fasting glucose})/22.5$ [21]. Insulin secretion was assessed using the change in insulin/glucose ratio over the first 30 min of the OGTT using the formula: $\Delta\text{insulin}_{30}/\Delta\text{glucose}_{30}$ [22]

Plasma was frozen to -20°C within 30 min, and serum was frozen to -20°C within 90 min. All specimens were frozen to -80°C within 5 h after start of the first OGTT. Appropriate Sarsted™ tubes were used for storage until final analysis to minimize evaporation.

At $t = 0$, a sphygmomanometer cuff was inflated for stasis, and the pressure was maintained at 40 mmHg throughout the sampling period in order to minimize mechanical damage to the vessel wall and thus prevent a rise in haemostasis parameters. Blood samples at $t = 30$ and 120 min were drawn with normal stasis.

Statistical analysis

Results are reported as means with standard deviation (SD). However, skewed data are reported as medians with interquartile range (IQR). Group comparisons were made by independent sample *t*-test for normally distributed data and by Mann-Whitney *U*-test (Wilcoxon's rank-sum test) for skewed data. Data concerning categorical variables are presented as numbers and percentages with 95% confidence intervals (95%CI). One-proportion and two-proportion tests are used where appropriate. A two-tailed *P*-value of < 0.05 was considered significant.

The hypothesis of the same risk profile in both groups was initially tested for each risk factor separately. However, as 27 variables were compared in the first analysis, an immediate concern is multiple testing. When many hypotheses are tested, the probability of finding a difference between the groups, when they are identical, exceeds the usual significance level of 0.05. In order to control the type 1 error, the *P*-values were adjusted by the Sidak step-down method [23]. The ordinary *P*-values are ordered according to their size, so that $P_1 < P_2 < \dots < P_K$, where *K* is the number of tests. They are then adjusted in the following manner, $P_{1(\text{adj.})} = 1 - (1 - P_1)^K$, and $P_{j(\text{adj.})} = 1 - (1 - P_j)^{(K-j+1)}$ for $2 \leq j \leq K$, with the restriction that also the adjusted *P*-values should be increasing. This method is less conservative than the well-known Bonferroni correction, but it still provides strong control of the type 1 error.

		ADA 1997 criteria:			Total
		Normo-glycaemia	Hyperglycaemia		
		NFG FBG < 5.6 mmol/l	IFG FBG 5.6–6.0 mmol/l	T2DM FBG ≥ 6.1 mmol/l	
WHO 1999 criteria:					
Normal glucose regulation	NGT FBG < 5.6 & 2-hBG < 6.7 mmol/l	1183			1183
Impaired glucose regulation	IFG FBG 5.6–6.0 & 2-hBG < 6.7 mmol/l		51		51
	IGT FBG < 6.1 & 2-hBG 6.7–9.9 mmol/l	78	12		90
Diabetes mellitus	T2DM FBG ≥ 6.1 & 2-hBG < 10.0 mmol/l			10	10
	T2DM 2-hBG ≥ 10.0 mmol/l	3	5	32	40
	Total	1264	68	42	1374

Table 1 A total of 1374 subjects classified according to ADA 1997 criteria of fasting blood glucose and WHO 1999 criteria of glucose tolerance

2-hBG, (whole) blood glucose at 120 min (OGTT); ADA, American Diabetes Association; DM, diabetes mellitus; FBG, fasting (whole) blood glucose; IFG, impaired fasting glycaemia; NFG, normal fasting glucose; NGT, normal glucose tolerance; WHO, World Health Organization.

We looked into possible family relations between the examined subjects and found only four sets of cases of 1st degree relatives. Two siblings had IFG, two siblings had IFG and IGT, respectively, a mother with a son and a daughter, all had IGT, and a father with a daughter, both with IGT. As most of the family relations belong to the same group, we should expect the calculated *P*-values to be greater than the actual *P*-values, thus increasing the statistical resemblance. We have chosen to ignore family relations in the present study.

Results

A total of 1374 participants 20–69 years old were examined and classified according to ADA 1997 criteria and WHO 1999 criteria (Table 1). Fifty (3.6%) subjects had T2DM, 19 (1.4%) previously diagnosed and 31 (2.3%) newly diagnosed by the screening. The patients previously diagnosed with diabetes were re-examined and the diagnosis was confirmed.

The analyses in this paper are confined to 51 (3.7%) subjects with isolated IFG (I-IFG) and 90 (6.6%) subjects with IGT according to WHO 1999 criteria. This latter group consists of 78 subjects with isolated IGT (I-IGT) and 12 subjects fulfilling the combined criteria for IFG and IGT (C-IFG/IGT) (Table 2).

Consistent with other investigations [24,25] we found IFG more often in males, and IGT more often in females, however, the difference was not statistically significant (*P* adj. = 0.92).

Triglycerides tended to be higher, although not statistically significant, in the IGT group than in the IFG group (*P* adj. = 0.053). We found no differences in total cholesterol, HDL-cholesterol, or LDL-cholesterol between groups. CRP tended to be higher in the IGT group. No differences were found in fibrinogen, PAI-1, t-PA or von Willebrand factor.

No differences in age, waist circumference, BMI, or blood pressure were found between IGT and IFG groups.

We found no differences in insulin resistance (HOMA) or insulin secretion between the IGT and the IFG groups. Nor did we find any differences in physical activity, tobacco smoking or family history of diabetes or CVD (Table 2).

To examine combinations of CVD risk factors, we analysed the components of the metabolic syndrome (for definitions see legend to Table 3). Dyslipidaemia and high fasting insulin were found more often in the IGT group and a tendency towards a more frequent presentation of full MS was found in the IGT group. Statistically significant differences disappeared after adjustment for multiple testing (Table 3).

Table 2 CVD risk factors in subjects with impaired fasting glycaemia and impaired glucose tolerance (analyses on fasting blood)

	IFG (n = 51)		IGT (n = 90)		P	P adjusted‡
Male	31	(60.8%)	42	(46.7%)	0.118	0.944
Age (years)*	52.1	(9.8)	49.0	(14.4)	0.176	0.983
BPsyst (mmHg)†	126	(118–144)	126	(116–141)	0.690	1.000
BPdia (mmHg)†	78	(70–88)	78	(70–88)	0.785	1.000
BMI (kg/m ²)†	27.7	(25.9–29.8)	27.4	(24.2–30.4)	0.506	1.000
Waist (male) (cm)†	100	(94–104)	100	(91–110)	0.721	1.000
Waist (female) (cm)†	91	(85–99)	88	(82–103)	0.576	1.000
HbA _{1c} (%)*	5.9	(0.45)	5.7	(0.44)	0.011	0.250
Fasting insulin (pmol/l)†	45	(30–60)	52	(31–89)	0.061	0.779
Total cholesterol (mmol/l)*	5.56	(0.90)	5.55	(1.11)	0.975	1.000
HDL (mmol/l)*	1.40	(0.32)	1.36	(0.33)	0.591	1.000
male						
HDL (mmol/l)*	1.56	(0.43)	1.59	(0.36)	0.776	1.000
female						
LDL (mmol/l)*	3.34	(0.90)	3.19	(0.98)	0.392	1.000
TG (mmol/l)†	1.33	(0.96–1.66)	1.71	(1.27–2.16)	0.002	0.053
Uric acid (mmol/l)*	0.33	(0.07)	0.33	(0.09)	0.814	1.000
PAI-1 (ng/ml)†	11.6	(4.8–19.2)	10.1	(2.9–23.9)	0.828	1.000
t-PA (ng/ml)†	8.8	(6.2–13.0)	10.1	(6.9–13.8)	0.405	1.000
CRP (mg/l)†	1.14	(0.63–3.11)	2.23	(0.09–4.87)	0.045	0.684
Fibrinogen (mg/dl)†	324	(282–380)	342	(302–395)	0.152	0.973
vWF (%)†	110	(83–133)	112	(88–136)	0.690	1.000
Physical activity*	8.1	(1.13)	7.9	(1.21)	0.460	1.000
Tobacco smoking	14		29		0.575	1.000
	27.5%	(16–42%)	32.2%	(23–43%)		
Cardiovascular disease (CVD)	5		13		0.451	1.000
	9.8%	(3–21%)	14.4%	(8–23%)		
Family history of diabetes	20		32		0.718	1.000
	39.2%	(26–54%)	35.6%	(26–46%)		
Family history of CVD	34		52		0.370	1.000
	66.7%	(52–79%)	57.8%	(47–68%)		
HOMA _{IR} †	1.68	(1.11–2.23)	1.69	(0.99–2.91)	0.665	1.000
Insulin secretion index (pmol/mmol)†	67.0	(45.7–110.6)	75.9	(45.8–106.1)	0.686	1.000

Data are means (SD) or medians (interquartile range) or numbers (per cent) (95% confidence interval).

*Normally distributed data; †skewed data; ‡adjusted for multiple testing using Sidak's step down method (27 variables).

Physical activity, Baecke Index of Physical Activity (answers from self-administered questionnaire); Tobacco smoking, daily and occasional smoking (answers from self-administered questionnaire); Cardiovascular disease (CVD), arterial hypertension, myocardial infarction, angina pectoris, stroke or intermittent claudication, present or previous to the examination (answers from self-administered questionnaire); Family history of diabetes, 1st degree relatives (parents and/or siblings) with diabetes mellitus (answers from self-administered questionnaire); Family history of CVD, 1st degree relatives (parents and/or siblings) with cardiovascular disease (answers from self-administered questionnaire); HOMA_{IR}; Homeostasis Model Assessment of Insulin Resistance (IR, fasting insulin × fasting glucose/22.5); Insulin secretion index, (Δ insulin₃₀/ Δ glucose₃₀). Insulin conversion, microunits/ml = 6.945 pmol/l (http://www.unc.edu/~rowlett/units/scales/clinical_data.html).

Discussion

We found great similarities in the CVD risk factor profile between participants with IFG and participants with IGT. Only fasting triglycerides, dyslipidaemia and fasting insulin above the top quartile were found more frequently in the IGT group than in the IFG group, but the differences were non-significant after adjustment for multiple statistical testing.

Even the best measurement of glucose has an error of about $\pm 2\%$, and the biological variation is about $\pm 5\%$, which makes misclassification of IFG subjects in particular unavoidable [26]. One of the major strengths of the present study is the precision of glucose analyses. We minimized errors connected to pre-analytic conditions, and the same person performed all

blood sampling, handling, and shipping. This person also did all glucose analyses in the central laboratory.

As recommended, the participants were classified according to only one OGTT [18], but a considerable variation in classification after repeated OGTTs has previously been demonstrated by others [27,28].

Classification problems in the fasting and non-fasting conditions prompt one to look upon IFG and IGT, not as well-defined categories, but rather as 'dynamic stages' in the natural course of diabetes. IFG and IGT may represent two major 'pathways' from NGT to T2DM, one leading through a state of mainly insulin secretory deficiency and one leading through a state of insulin resistance combined with insulin secretory deficiency [29].

	IFG (<i>n</i> = 51)		IGT (<i>n</i> = 90)		<i>P</i>	<i>P</i> adjusted‡
Dyslipidaemia	11 22%	(6–18) (11–35%)	37 41%	(28–47) (31–52%)	0.026	0.100
Abdominal obesity	38 75%	(23–44) (60–86%)	67 74%	(58–75) (64–83%)	1.000	1.000
Hypertension	20 39%	(13–27) (26–54%)	32 36%	(23–42) (26–46%)	0.469	0.720
Fasting insulin in top quartile	14 27%	(8–21) (16–42%)	44 49%	(34–54) (38–60%)	0.014	0.068
Metabolic syndrome	9 18%	(4–16) (8–31%)	29 32%	(20–39) (23–43%)	0.076	

Data are numbers (95% confidence interval). Fisher's exact test.

‡Adjusted for multiple testing using Sidak's step down method (four variables).

Dyslipidaemia, HDL-cholesterol < 1.0 mmol/l or triglycerides = 2.0 mmol/l; Abdominal obesity, waist circumference = 94 cm for men, = 80 cm for women, and/or BMI = 30 kg/m²; Hypertension, systolic blood pressure = 140 mmHg and/or diastolic blood pressure = 90 mmHg; Fasting insulin, 75th quartile = 55 pmol/l; Metabolic syndrome, fasting insulin in top quartile combined with any two of the following: impaired glucose regulation (IFG, IGT or T2DM), dyslipidaemia, abdominal obesity or hypertension (EGIR).

In 1997, ADA recommended the abolition of the OGTT. In this study, omission of OGTT would have misclassified 78 of 90 IGT subjects (87%) as having NGT, and 12 (13%) of 90 IGT subjects would have been classified as IFG. Three of 31 newly diagnosed diabetic patients (9%) would have been undiagnosed and five of 31 newly diagnosed diabetic patients (16%) would have been misclassified as having IFG.

A number of recent papers have demonstrated IGT to be a risk factor for CVD, whereas IFG is not. A survey of a Japanese cohort of 2534 subjects aged 40 and over, who underwent an OGTT from 1990 to 1996 and were followed up after 7 years with outcome determined by death certificates, showed a significantly higher death rate from CVD for subjects with IGT at baseline than for NGT subjects, but a non-significant difference between IFG subjects and NFG subjects [14]. This study used two sets of diagnostic criteria (WHO 1985 and ADA 1997), but they did not compare directly the survival rates in subjects with IGT and IFG, respectively.

A survey among 3229 non-diabetic Mauritians performed in 1987 and 1992 gave a similar result, but this survey has some weaknesses. The IGT and IFG groups are not exclusive. A total of 118 subjects fulfilling the combined IGT/IFG criteria were analysed within both groups. No information was given on variation coefficients in glucose analysis, which may raise some questions as to the classification of IFG subjects, bearing in mind the very narrow interval for FPG from 6.1 to 6.9 mmol/l [9].

In a recent meta-analysis it was claimed that the combined IGT/IFG group represents a more advanced stage towards diabetes and CVD than does isolated IFG and IGT [17].

We found the same trend in our material, where only 12 subjects were classified as having combined IGT/IFG. Our analyses were primarily performed on isolated IFG, isolated

IGT and combined IFG/IGT groups, but no significant deviation was found between these three groups (data not shown). It seemed justified then to include combined IFG/IGT in the IGT group instead of introducing a third group consisting of only 12 subjects. Hereby, we gained statistical strength, and the decision was in concordance with placing the diabetic subjects also having IFG in the T2DM group. Unwin *et al.* have recently suggested that a person falling into two categories should be placed in the higher [17].

In a German case-control cross-sectional study including 104 pairs of 40–70 years old subjects, IFG was found to be a risk factor for CVD (using the carotid intima-media thickness as a measure of CVD), only in combination with IGT [16].

In contrast, it was shown that subjects with IFG and subjects with IGT among 3568 Asians share the same risk of developing CVD, but again the combined group had a greater risk as assessed by the incidence of cardiovascular dysmetabolic syndrome [30]. This cohort is slightly younger but otherwise comparable with ours concerning CVD risk profile despite differences in ethnicity.

After having demonstrated great similarities in CVD risk factor profile, glucose values have to be taken into consideration. As anticipated from definitions, IFG was related to higher values of FBG than IGT, and conversely IGT was related to higher values of 2-hBG than IFG.

The DECODE study has shown the 2-hBG to be a better predictor of death than the FBG [31]. In people with IGT, a high post-load glucose level predicted conversion to diabetes [32]. 2-hPG has been shown to be more related to carotid intima-media thickness than FPG [33], and it has been hypothesized that the effect is procured by a noxious effect of hyperglycaemia directly on the vessel wall [34].

Table 3 Components of the metabolic syndrome in subjects with impaired fasting glycaemia and impaired glucose tolerance

IFG is characterized mainly by insulin resistance resulting in higher FBG, whereas IGT is connected mainly with β -cell deficiency, resulting in higher post-load glucose values [24].

In the Botnia Study, they found significantly lower insulin secretion index in IGT compared with IFG [24]. We found no difference, maybe because our IGT subjects were younger. Other risk factors were comparable.

Our values involving insulin are generally low due to the method of insulin analysis, which has a very low cross-reactivity with pro-insulin.

Conclusions

We conclude that individuals with IFG and IGT have similar CVD risk profiles. Based on this finding, the risk of developing CVD was expected to be similar in both groups and possible differences in CVD outcome might be explained by differences in fasting or post-load glucose, the latter being the more important. Prospective studies are needed to observe the actual risk of developing CVD in the two groups. Until such data are available it seems appropriate to take into account both IFG and IGT when planning screening initiatives.

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