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ORIGINAL ARTICLE

Comparison of body mass index with abdominal obesity indicators and waist-to-stature ratio for prediction of type 2 diabetes: The Isfahan diabetes prevention study

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KEYWORDS
Diabetes mellitus;
First-degree relatives;
Body mass index;
Waist circumference;
Central obesity;
Abdominal obesity;
Risk factors

Summary
Objectives: The aim of this study was to compare the ability of the body mass index (BMI), waist circumference (WC), waist-to-hip ratio (WHR) and waist-to-stature ratio (WSR) to predict progression to diabetes in non-diabetic first-degree relatives (FDRs) of patients with type 2 diabetes.
Methods: A total of 704 non-diabetics FDRs 20—70 years old in 2003—2005 were followed through 2008 for the occurrence of type 2 diabetes mellitus. At baseline and through follow-ups, participants were underwent a standard 75 g 2-h oral glucose tolerance test. Prediction of progression to type 2 diabetes was assessed with area under the receiver operating characteristic (ROC) curves based upon measurement of BMI, WC, WHR and WSR.
Results: The incidence of type 2 diabetes was 3.3% per year in men and 4.8% in women. BMI, WC and WSR were related to diabetes. These three obesity indicators have similar associations with incident diabetes. Areas under the ROC curves were 0.625 for BMI, 0.620 for WC, 0.611 for WSR and 0.538 for WHR.
Conclusions: These data indicate that BMI was as strong as WC or WSR in predicting progression to diabetes.
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Introduction

Obesity is a major independent risk factor for type 2 diabetes [1], thus the definition of obesity is important for the purpose of intervention. Epidemiological studies have demonstrated that different anthropometric measures for obesity such as body mass index (BMI), waist circumference (WC), waist-to-hip ratio (WHR) and waist-to-stature ratio (WSR) are strong and consistent predictors of type 2 diabetes [2—6]. However, uncertainty exists about the strength of the association among BMI, WC, WHR, WSR and diabetes incidence. Some studies showed a stronger association between diabetes and central obesity measures than was apparent BMI, but these findings are inconclusive [4—15]. Other studies showed no significant differences between the obesity measures [16—20], whereas other studies provide evidence that WHR or WSR are the best predictive variables [7,21—24]. However, from clinical and public health point of view it is important to clarify the role of these obesity indicators in association with diabetes. Our study contributes to this issue by comparing the ability of BMI, WC, WHR and WSR to predict the incidence of type 2 diabetes in non-diabetic first-degree relatives (FDRs) of patients with type 2 diabetes.

In this regard, it has to be noted that the relative contributions of these obesity indicators may vary among various ethnic groups [25—28]. Therefore, at an ethnological level, the study contributes by characterizing the occurrence of diabetes in a specific population from central Iran.

Patients and methods

Data collection

The Isfahan Diabetes Prevention Study (IDPS) is an ongoing cohort study in central Iran to assess the efficacy of intensive diet and exercise to prevent or delay the onset of type 2 diabetes mellitus in FDRs of patients with type 2 diabetes. The study was established in 2003—2005 when 2368 (614 men and 1754 women) FDRs of a consecutive sample of patients with type 2 diabetes attending clinics in Isfahan Endocrine and Metabolism Research Center which is affiliated to Isfahan University of Medical Sciences, Iran, completed laboratory tests including standard 75 g 2-h oral glucose tolerance test (OGTT) and a questionnaire on their health status and on various potential risk factors for diabetes. Participants receive follow-up tests according to Standard of Medical Care in Diabetes [29] to update information on demographic, anthropometric, and lifestyle factors and on newly diagnosed diabetes. Accordingly, if OGTT was normal at baseline, repeat testing was carried out at least at 3-year interval. Otherwise, repeat testing was carried out annually. The IDPS baseline methods have been described in detail elsewhere [30]. The participants included siblings and children of type 2 diabetes patients.

Ascertainment of diabetes

Cases of diabetes were identified from baseline and follow-up OGTTs according to American Diabetes Association criteria [31]. Individuals who were not diabetic at baseline and who had at least one subsequent examination were included. Pregnant women were excluded. For the present study, analyses were limited to the 704 participants (151 men and 553 women, mean (SD) age 42.7 (6.4) years) in the average 2.3-year follow-up for whom complete data were available. Attendees at the follow-up visit did not differ significantly from non-attendees regarding most baseline characteristics: age, height, weight, BMI, WC, hip circumference, WHR and levels of FPG, cholesterol, high-density lipoprotein (HDL) cholesterol, triglyceride, systolic blood pressure (BP) and obesity. However, non-attendees had slightly lower diastolic BP (73.5 mmHg versus 74.6 mmHg, $P < 0.05$), HbA_{1c} (5.0% versus 5.1%, $P < 0.05$), and plasma glucose (PG) at 30 min (142.2 mg/dl versus 147.0 mg/dl ($P < 0.01$), 60 min (145.1 versus 155.5, $P < 0.01$) and 120 min (115.0 mg/dl versus 127.8 mg/dl, $P < 0.01$)), but higher levels of low-density lipoprotein (LDL) cholesterol (118.5 mg/dl versus 115.3 mg/dl, $P < 0.05$).

Procedures

Information on age, gender, body size, glycosylated hemoglobin (HbA_{1c}), cholesterol, HDL, and LDL, triglyceride and BP, family and personal medical history was collected at the baseline and through follow-ups. The same methodology was used at baseline and follow-ups. Participants reported to clinics in the morning after an overnight fast. Subjects were asked to abstain from vigorous exercise in the evening before and in the morning of the investigations. Smokers were encouraged to abstain from smoking in the morning of the investigations. First on arrival at the clinic, the information given by the participants in the questionnaire on family history was verified. Then, with the subjects in light clothes and without shoes height, weight, waist and hip circumference were measured using standard apparatus. Weight was measured to the

nearest 0.1 kg on a calibrated beam scale. Height, waist, and hip circumference were measured to the nearest 0.5 cm. Waist was measured midway between the lower rib margin and the iliac-crest at the end of a gentle expiration. Hip circumference was measured over the greater trochanters directly over the underwear. Resting BP was measured after subjects had been seated for 10 min by using a mercury sphygmomanometer and appropriately sized cuffs, using standard techniques. FPG was measured using the glucose oxidase method. Subjects with FPG <126 mg/dl (7.0 mmol/L) underwent a standard OGTT (75 g glucose 2-h) at baseline and the follow-ups. Venous blood was sampled at fasting, 30, 60, and 120 min after oral glucose administration. Plasma samples obtained after centrifugation were analyzed the same day.

HbA_{1c} (measured by ion-exchange chromatography), total cholesterol, triglyceride, HDL, and LDL (calculated by the Friedewald Equation [32] provided total triglycerides did not exceed 400 mg/dl) were also assessed. All the blood sampling procedures were performed in the central laboratory of the Isfahan Endocrine and Metabolism Research Center using enzyme-linked method. Tenets of the Declaration of Helsinki were followed, institutional ethical committee approval was granted, and an informed consent form was signed by each participant.

Definitions

We calculated BMI as the ratio of weight (kg) to height squared (m^2), the latter being assessed at baseline only. Abdominal obesity was defined by WC or by the WHR. WHR was calculated as WC divided by hip circumference and WSR was calculated as WC divided by height in centimetres. Those participants with FPG $\geq$200 mg/dl (11.1 mmol/L) or pharmacological treatment were considered as diabetic. If FPG was $\geq$126 mg/dl (7.0 mmol/L) and <200 mg/dl (11.1 mmol/L), a second FPG was measured on another day. If the second FPG was also $\geq$126 mg/dl (7.0 mmol/L), participants were considered as diabetic. FPG $\geq$126 mg/dl (7.0 mmol/L) (or 2-h PG of $\geq$200 mg/dl (11.1 mmol/L)) defined diabetes mellitus. Impaired glucose tolerance (IGT) was defined as FPG <126 mg/dl (7.0 mmol/L), but with 2-h PG concentration $\geq$140 mg/dl (7.8 mmol/L) and <200 mg/dl (11.1 mmol/L). If FPG was in the range of 100 mg/dl (5.6 mmol/L) to 126 mg/dl (7.0 mmol/L) and 2-h PG was <140 mg/dl (7.8 mmol/L), it was considered as impaired fasting glucose (IFG). If the FPG was below 100 mg/dl (5.6 mmol/L) and 2-h PG smaller than 140 mg/dl (7.8 mmol/L), it was considered a sign of normal glucose tolerance (NGT) [31]. The NCEP-ATP III [33] definition was used for the metabolic syndrome (MetS) by the presence of three or more of the five abnormalities: (i) BP$\geq$130/85 mmHg or a history of hypertension and current use of antihypertensive treatment; (ii) waist girth >102 cm for men and >88 cm for women, (iii) serum triglyceride $\geq$150 mg/dl (1.7 mmol/L) and/or (iv) HDL cholesterol (<40 mg/dl (0.9 mmol/L) for men and <50 mg/dl (1.0 mmol/L) for women), and (v) FPG levels $\geq$110 mg/dl (6.1 mmol/L).

Determination of diabetes incidence

Incidence was expressed as the number of cases of type 2 diabetes per 100 person-years of follow-up beginning on the date of completion of the baseline examination in 2003—2005 and continuing until the occurrence of diabetes, the date of the last completed follow-up, death, or end of follow-up on December 31, 2007, whichever came first. For ease of interpretability, we report the incidence rates in terms of percent per year.

Statistical analysis

Statistical methods used included the Student's *t*-test, chi squared test, and binary logistic regression. Univariate and multivariate binary logistic regressions were fitted to identify predictors of new-onset diabetes using the SPSS for Windows (SPSS Inc., Chicago, IL, USA). We considered the following covariates in the multivariate-adjusted analyses: age, gender, BMI, WC, WHR, and WSR. Variable age, was entered in models as continuous variable, while gender and quartiles of BMI, WC, WHR and WSR were categorical. Adjustment for age was examined in separate models. Age-adjusted means were calculated and compared using general linear models. All anthropometric measures were not included simultaneously in regression analysis to avoid co-linearity that these independent variables may have. The ability of BMI, WC, WHR and WSR to predict incidence diabetes was examined by receiver operating characteristic (ROC) curve and their respective areas under the curve, in which sensitivity is plotted as a function of 1-specificity. Areas under the ROC curves were compared by the algorithm developed by DeLong et al. [34]. Analyses were initially stratified by gender, but as the findings were similar, the results are presented for both gender combined to increase statistical power. All tests for statistical significance were two-tailed, and performed assuming a type I error probability of <0.05.

Table 1 Age, age-adjusted means (SE) and proportions of selected baseline characteristics between 72 first-degree relatives of patients with type 2 diabetes who did and 632 who did not develop diabetes.

Variables	Developed diabetes	Not developed diabetes	Difference (95% CI)
	Mean (SE)	Mean (SE)	
Age (years)	43.6 (0.74)	42.6 (0.25)	1.0 (−0.44, 2.65)
BMI (Kg/m^2)	30.9 (0.48)	28.9 (0.16)	2.0 (1.01, 2.90)***
Waist circumference (cm)	92.0 (1.04)	88.3 (0.35)	3.7 (1.84, 6.16)**
Waist-to-hip ratio	0.83 (0.007)	0.82 (0.003)	0.01 (−0.004, 0.02)
Waist-to-stature ratio	0.58 (0.006)	0.56 (0.002)	0.02 (0.008, 0.03)***
Systolic BP (mmHg)	119.5 (1.92)	114.8 (0.64)	4.7 (1.24, 9.36)*
Diastolic BP (mmHg)	78.6 (1.45)	74.3 (0.49)	4.3 (1.36, 7.44)**
Baseline fasting glucose (mg/dl)	106.1 (1.44)	93.5 (0.49)	12.6 (9.80, 15.80)***
Plasma glucose 30 min (mg/dl)	168.5 (3.75)	144.8 (1.23)	23.7 (16.7, 32.3)***
Plasma glucose 60 min (mg/dl)	193.8 (4.95)	151.3 (1.64)	42.5 (33.50, 54.10)***
Plasma glucose 120 min (mg/dl)	156.8 (4.16)	124.8 (1.40)	32.0 (24.10, 41.50)***
HbA1c (%)	5.4 (0.12)	5.1 (0.04)	0.3 (0.05, 0.55)*
Triglyceride (mg/dl)	170.8 (12.35)	169.1 (4.19)	1.7 (−22.60, 28.80)
Cholesterol (mg/dl)	202.6 (4.83)	192.6 (1.62)	10.0 (1.03, 21.40)*
HDL cholesterol (mg/dl)	45.2 (1.41)	45.3 (0.48)	−0.1 (−3.02, 2.82)
LDL cholesterol (mg/dl)	123.0 (4.25)	114.4 (1.47)	8.6 (0.35, 18.30)
Variables	Developed diabetes	Not developed diabetes	Difference (95% CI)
	%	%	
Men	16.4	21.9	−5.5 (−14.50, 3.00)
Obesity (BMI $\geq$ 30)	52.8	35.7	17.1 (4.97, 29.21)**
Normal glucose tolerance	5.6	51.9	−46.3 (−52.90, −39.80)***
Impaired fasting glucose	9.7	7.6	2.1 (−5.00, 9.30)
Impaired glucose tolerance	84.7	40.1	44.6 (35.50, 53.80)***
Metabolic syndrome	32.4	24.4	8.0 (−3.15, 19.20)

Age-adjusted means were calculated using general linear models. The difference in the mean or percentage of the variables between diabetes and no diabetes. CI, confidence interval

* $P < 0.05$.

** $P < 0.01$.

*** $P < 0.001$.

Results

During 1630 (354 men and 1276 women) person-years of follow-up, 72 (10.3%) (11 men and 61 women) incident cases of type 2 diabetes occurred. The overall incidence of subsequent diabetes was 4.4% (95% CI: 3.5, 5.5) per year. Incidence rates were higher in women (4.8%, 95% CI: 3.7, 6.1 per year) than men (3.3%, 95% CI: 1.7, 5.8) but the difference was not statistically significant. Of the 315 participants who had IGT at initial registration, 61 subsequently developed diabetes, giving an incidence of 10.0% (95% CI: 7.7, 12.6) per year. This was much higher than the incidence rates seen for NGT, 0.5% per years (95% CI: 0.1, 1.2) ($P < 0.001$). Of the 55 participants who had IFG at initial registration, 7 subsequently developed diabetes, giving an incidence of 5.1% (95% CI: 2.1, 10.2) per year. MetS was present in over a quarter of the participants (25.2%; 95% CI: 22.0, 28.4). Incidence of type 2 diabetes was 6.0% (95% CI: 3.9, 8.8) per year in those with MetS. This was higher than the incidence rates seen for those without MetS, 4.0% per year (95% CI: 3.0, 5.3) but this difference was not statistically significant (Table 1).

Baseline characteristics of the 632 (89.8%) participants without and 72 (10.3%) with diabetes are shown in Table 2. As expected, those who developed diabetes had higher age-adjusted mean BMI, WC, hip circumference, WSR, FPG, and PG at 30, 60 and 120 min, and HbA_{1c} at baseline and have higher proportion of obesity and IGT. The mean (SD) age was 43.7 (7.0) years for those with and 42.6 (6.3.) years for those without diabetes.

The incidence of diabetes was 6.6% per year (95% CI: 4.33, 9.47) for participants in the highest quartile of BMI, and 2.6% per year (95% CI: 1.29, 4.56) for the lowest quartile. The equivalent incidences for WC were 6.6% (95% CI: 4.34, 9.64) and 1.8% per year (95% CI: 0.80, 3.57). For individuals in the highest quartile of WSR, the incidence of diabetes was 7.0% per year (95% CI: 4.60, 10.00) and for the low-

Table 2 Incidence rates and relative risks (95% CI) of type 2 diabetes by quartiles of anthropometric parameters, the Isfahan Diabetes Prevention Study, 2003–2008.

Variables	Cases (no.)	Incidence/100 person-year	Age-adjusted relative risk (95% CI)	Age and gender-adjusted relative risk (95% CI)[a]
BMI				
1st quartile (<26.2)	11	2.6	1.00	1.00
2nd quartile (26.2–28.6)	15	3.8	1.36 (0.60, 3.04)	1.35 (0.60, 3.03)
3rd quartile (28.7–31.5)	20	5.0	1.88 (0.87, 4.06)	1.84 (0.85, 3.92)
4th quartile (>31.5)	26	6.6	2.59 (1.24, 5.43)*	2.4 (1.16, 5.19)*
WC				
1st quartile (<82.0)	8	1.8	1.00	1.00
2nd quartile (82.0–88.5)	19	4.7	2.39 (1.02, 5.63)*	2.42 (1.03, 5.70)*
3rd quartile (88.5–94.5)	20	5.1	2.69 (1.14, 6.32)*	3.06 (1.29, 7.25)*
4th quartile (>94.5)	25	6.6	3.45 (1.51, 7.91)**	4.22 (1.81, 9.86)**
WHR				
1st quartile (<0.77)	12	2.8	1.00	1.00
2nd quartile (0.77–0.81)	20	4.9	1.67 (0.79, 3.56)	1.68 (0791, 3.57)
3rd quartile (0.82–0.86)	26	6.7	2.27 (1.10, 4.69)*	2.30 (1.11, 4.77)*
4th quartile (>0.86)	14	3.7	1.11 (0.49, 2.51)	1.38 (0.48, 3.95)
WSR				
1st quartile (<0.52)	11	2.5	1.00	1.00
2nd quartile (0.52–0.55)	15	3.8	1.32 (0.59, 2.97)	1.36 (0.60, 3.07)
3rd quartile (0.56–0.59)	20	5.2	1.90 (0.88, 4.12)	1.97 (0.91, 4.27)
4th quartile (>0.59)	26	7.0	2.51 (1.19, 5.30)*	2.52 (1.19, 5.34)*

CI, confidence interval.
[a] Relative risks (with 95% CI) calculated by binary logistic regression.
* $P < 0.05$.
** $P < 0.01$.

est quartile 2.5% per year (95% CI: 1.24, 4.39). The incidence of diabetes was 3.7% (95% CI: 2.04, 6.15) for individuals in the highest quartile of WHR, and for the lowest quartile 2.8% (95% CI: 1.42, 4.74). The association among BMI, WC, WSR and type 2 diabetes was similar and the risk of type 2 diabetes increased with increasing quartiles of these three obesity indicators. When in multivariate analysis comparing the associations in the highest with the lowest quartile, the WC relative risk was stronger than the BMI or WSR relative risks (Table 3). Age-, gender-adjusted relative risk shows increasing for WC in all the quartiles, whereas BMI is associated with diabetes only in higher quartiles.

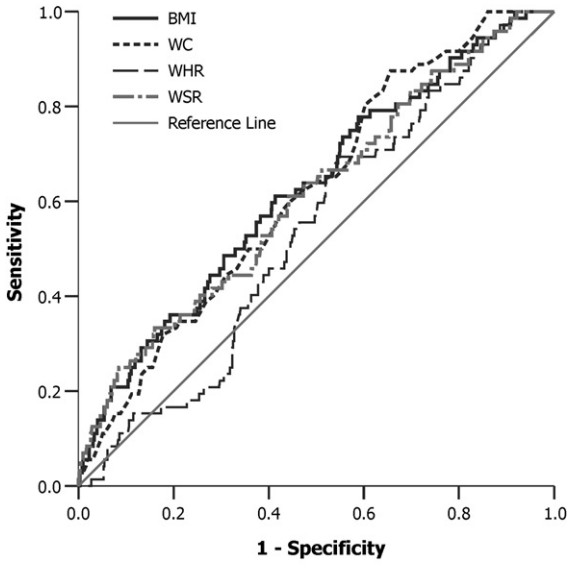
The ROC curves for the incidence of type 2 diabetes for BMI, WC, WHR and WSR are shown in Fig. 1. The areas under the ROC curves were 0.625 (95% CI: 0.556, 0.693) for BMI, 0.620 (95% CI: 0.557, 0.683) for WC, 0.538 (95% CI: 0.474, 0.601) for WHR, and 0.611 (95% CI: 0.541, 0.680) for WSR. All anthropometric parameters, except WHR, were significant predictors for future risk of type 2 diabetes ($P < 0.001$). BMI and WC had similar area. BMI and WC had areas slightly but not significantly larger than that of WHR and WSR. However, it is apparent that in this population of FDRs of patients with type 2 diabetes, the BMI was similar to WC and WSR to predict future risk for type 2 diabetes.

All four anthropometric indicators, except BMI and WHR, were correlated with each other and the strongest Pearson correlation coefficients were

Table 3 Pearson correlation coefficients between anthropometric indicators, the Isfahan Diabetes Prevention Study, 2003–2008.

Variables	Waist circumference	Waist-to-hip ratio	Waist-to-stature ratio
Body mass index	0.746*	0.034	0.845*
Waist circumference	1	0.602*	0.883*
Waist-to-hip ratio		1	0.401*

* $P < 0.01$.



Area under the curve (95% confidence interval)

Body mass index	0.625 (0.556, 0.693)
Waist circumference	0.620 (0.557, 0.683)
Waist-to-hip ratio	0.538 (0.474, 0.601)
Waist-to stature ratio	0.611 (0.541, 0.680)

Figure 1 Receiver operating characteristic curves for body mass index (BMI), waist circumference (WC), waist-to-hip ratio (WHR) and waist-to-stature ratio (WSR) for prediction of type 2 diabetes in non-diabetic first-degree relatives of patients with type 2 diabetes. The estimates of the area under the ROC curves and their 95% confidence intervals are shown.

found between WC and WSR and the weakest ones were between WSR and WHR (Table 3).

Discussion

This study showed that the discriminating ability of BMI was as good as that of WC and WSR, further emphasizing the utility of WC alone in predicting diabetes. The WHR is a weaker diabetes risk predictor than BMI, WC or WSR. Similar to our findings, the Iowa Women's Health Study [19], the Health Professionals Follow-Up Study (among men only) [35], the Tehran Lipid and Glucose Study (among men only) [7], Jamaica study [20] and Mauritius Non-communicable Disease Study [18] have shown that BMI and central obesity indicators were equally well associated with diabetes incidence but in the former two studies diabetes incidence was self-reported and anthropometric indicators were self-measured by the participants. A recent meta-analysis of 32 studies of the association of BMI, WC, and WHR and incidence of type 2 diabetes found similar results [2]. Similar to our findings, in the Nurses' Health Study, all three measurements were useful for predicting diabetes incidence; however, the relative risk for BMI was 3—4 times higher than the relative risk for WHR [36]. Our findings, does not support the findings of the San Antonio Heart Study [4], the EPIC-Potsdam Study (for women only) [5] and Shanghai Women's Health Study [6], the Diabetes Epidemiology: Collaborative Analysis of Diagnostic criteria in Asia [37] and others [4,14,15] which showed WC or WHR is a stronger than BMI in predicting future risk for type 2 diabetes, however, the differences observed in some of these studies were not statistically significant. Other studies [21—23] provide evidence that WHR has a positive effect independent of BMI. However, some have argued against use of WHR or WSR as a measure of obesity because of its ambiguous biologic interpretation, its lesser sensitivity to weight gain, its greater variability across age, gender, and ethnic groups, and its greater computational complexity and interpretation in public health context [38].

On the basis of our overall findings, both WC and BMI have the approximately same predictive discrimination. Because WC is strongly correlated with BMI, they are unlikely to yield different answers and the two measures yield similar information, with the correlation coefficient above 0.7. However, visceral adipose tissue is known to generate diabetogenic substances [3] and WC may be more informative than general obesity for diagnostic evaluation. Abdominal obesity has been associated with decrease glucose tolerance, alterations in glucose insulin homeostasis, reduced metabolic clearance of insulin, and decreased insulin-stimulated glucose disposal. In addition, a simple WC measurement is a better predictor of progression to diabetes than the BMI or WSR due to the easy to measure, reliability and conveniences, although it does require some training and standardization. Whereas accurate weighing requires removal of shoes and most clothing, and correction for occasional appliances or casts, and the use of a high-quality scale that is periodically recalibrated. But, WC requires only the removal (or loosening) of clothing around the waist and an inexpensive tape measure made of non-stretchable material. The standardized landmark for waist measurement is usually simple to identify after a short training period, and WC measurement can be highly reproducible. Both BMI and WSR require measurement of height. However, if height measurement is inaccurate, the error will be squared in the computing of BMI. However, ratios are more difficult to interpret biologically, are less sensitive to weight gain, and have statistical limitations [38].

Our study has strengths and limitations. The strengths include the prospective cohort design, the sample consisting of both men and women of a wide age range, diagnosis of diabetes based on standard OGTT, and information on potential determinants of diabetes. Selection and information bias is considered unlikely by virtue of the prospective design. Even though the study included more than 700 participants who were thoroughly examined and followed up, the follow-up period of 3 years may be controversial. Due to the still conflicting results in assessing diabetes prediction a long-term follow-up of 3—6 years in a large cohort could therefore further contribute to a clarification of the question. At follow-up, non-attendees of the entire population did not differ from attendees by major risk factors for progression to diabetes, although a difference too small to explain the high progression rate to diabetes in our study was seen in the mean levels of LDL, HbA_{1c}, and PG. Albeit we have not carried out any special studies of the validity or reliability of data for this analysis, a clerk was employed to check consistency and, where possible, to ensure completeness of data. Our experience with other parts of the data set gives us some confidence that data quality is sufficient for this type of study and that our results provide useful additional evidence on the ability of the BMI, WC, WHR and WSR to predict progression to diabetes in non-diabetic FDRs of patients with type 2 diabetes.

The high risk of developing type 2 diabetes in FDRs with high BMI or WC underlines the importance of prevention of type 2 diabetes in these individuals. Recent clinical trials demonstrate that lifestyle [39—42] and pharmaceutical [39] interventions in high-risk individuals can prevent the development of diabetes, providing a rationale for the identification of high-risk subjects so as to institute early lifestyle or pharmacological interventions.

In conclusion, our study indicates that the BMI and WC are very highly correlated and likely to behave similarly in diabetes prediction. WSR showed almost the same discriminating ability.

Conflicts of interest

None.

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