

Associations of hip circumference and height with incidence of type 2 diabetes: the Isfahan diabetes prevention study

Mohsen Janghorbani · Masoud Amini

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Abstract The aim of this study was to determine the effects of hip circumference (HC) and height on diabetes incidence in non-diabetic first-degree relatives (FDRs) of patients with type 2 diabetes. A total of 1,092 (254 men and 838 women) non-diabetic FDRs ≥ 30 years old in 2003–2005 were followed through 2010 for the occurrence of type 2 diabetes. At baseline and through follow-ups, participants were underwent a standard 75 g 2-h oral glucose tolerance test. The incidence of type 2 diabetes was 17.0 (95% CI: 13.7, 20.2) (13.0 men and 18.1 women) per 1,000 person-year based on 6,015 person-years of follow-up. Height was inversely associated with diabetes incidence. The age-, gender-, and waist-adjusted relative risk (95% CI) of diabetes was 0.54 (0.31, 0.93) for highest quartile of height and 0.59 (0.25, 1.37) for highest quartile of HC compared with lowest quartile. These data indicate that height was inversely associated with diabetes incidence, independently of gender among FDRs of patients with type 2 diabetes.

Keywords Diabetes mellitus · First-degree relatives · Hip circumference · Waist circumference · Height · Risk factors

Introduction

It is well established that obesity is associated with increased prevalence of metabolic syndrome components and subsequent increased risk of type 2 diabetes [1, 2]. Epidemiological studies have demonstrated that different anthropometric measures for obesity such as body mass index (BMI), waist circumference (WC), waist-hip ratio (WHR), and waist-stature ratio (WSR) are strong and consistent predictors of type 2 diabetes [3–5]. The relationship between increased BMI, WHR, or WSR with diabetes risk could be due to direct effect of obesity or inverse effects of hip circumference (HC) or height. It is also not clear whether height affects the relationship between WC and abdominal visceral fat; height has no effect in some studies [6, 7], but has significant effect in others [8, 9]. HC appears to be inversely associated with type 2 diabetes in most [10–17] but not all studies [18]. There have also been conflicting reports about possible association between height and type 2 diabetes mellitus [10, 12, 13, 18, 19]; some studies showed a positive association [18], whereas others reported no association [20] or an inverse relation [10, 13, 19] or an association was observed only in women [12] or men [5]. However, most of these studies have been relatively small or were not able to distinguish multi-colinearity between height, WC, and HC [11].

Height [21] and glucose metabolism risk factors [22–24] such as obesity are determined by genetic and early environmental influences. First-degree relatives (FDRs) of patients with type 2 diabetes which have genetic basis are at high risk of glucose intolerance and may be more appropriate for testing this hypothesis. In order to fill some of these gaps, we used the ongoing Isfahan Diabetes Prevention Study (IDPS) to examine the effects of HC and

M. Janghorbani (✉)
Department of Epidemiology and Biostatistics,
School of Public Health, Isfahan University of Medical Sciences,
Isfahan, Iran
e-mail: janghorbani@hlth.mui.ac.ir

M. Janghorbani · M. Amini
Isfahan Endocrine and Metabolism Research Center,
Isfahan University of Medical Sciences, Isfahan, Iran

height on diabetes incidence in non-diabetic FDRs of patients with type 2 diabetes.

In this regard, it has to be noted that the relative contributions of these anthropometry indicators may vary among various ethnic groups [25–27]. Therefore, at an ethnological level, the study contributes by characterizing the occurrence of diabetes in a specific population from central Iran.

Subjects and methods

Data collection

The 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 3,227 (847 men and 2,380 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 [28] 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 [29, 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]. Those participants with FPG \geq 200 mg/dl (11.1 mmol/l) or pharmacological treatment were considered as diabetic. If fasting plasma glucose (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. Individuals who were not diabetic at baseline and who had at least one subsequent examination were included. 1,919 subjects were excluded as they failed to undergo subsequent examination and 216 subjects with type 2 diabetes at baseline. For the present study, analyses were limited to the 1,092 participants (254 men and 838 women, mean (SD) age 42.8 (6.4) year) in the average

5.5-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, HC, WHR, and levels of high-density lipoprotein (HDL) cholesterol, triglyceride, glycosylated hemoglobin (HbA_{1c}), blood pressure (BP), and obesity. However, non-attendees had slightly higher FPG (95.4 mg/dl vs. 94.5 mg/dl, $P <$ 0.05), levels of low-density lipoprotein (LDL) cholesterol (122.2 mg/dl vs. 115.8 mg/dl, $P <$ 0.001), and cholesterol (200.3 mg/dl vs. 192.6 mg/dl, $P <$ 0.001), but lower plasma glucose (PG) at 30 min (140.8 mg/dl vs. 144.6 mg/dl, ($P <$ 0.01), 60 min (143.5 vs. 149.7, $P <$ 0.001), and 120 min (113.2 mg/dl vs. 119.4 mg/dl, $P <$ 0.001).

Procedures

Information on age, gender, body size, 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 HC were measured using standard apparatus. Weight was measured to the nearest 0.1 kg on a calibrated beam scale. Height, waist, and HC 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. HC 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.

Determination of diabetes incidence

Incidence was expressed as the number of cases of type 2 diabetes per 1,000 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, 2010, whichever came first.

Statistical analysis

Statistical methods used included the Student's *t* test, Chi squared test, and Cox proportional hazard model. Univariate and multivariate Cox proportional hazard model 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, and WC. Variable age and WC were entered in models as continuous variable, while gender and quartiles of HC and height were categorical. Adjustment for age and gender 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. 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.

Results

The baseline characteristics of the study participants by quartile of height and HC are shown in Table 1. In age-adjusted comparisons of variables at baseline, height, weight, WC, and HC were more likely to increase across the quartiles of both height and HC. BMI, PG, cholesterol, LDL, triglyceride, blood pressure, and obesity prevalence were more likely to increase across the quartiles of HC only. Age was more likely to decrease across the quartiles of height.

Baseline characteristics of the 990 (90.7%) participants without and 102 (9.3%) with diabetes are shown in Table 2. As expected, those who developed diabetes were older and had higher age-adjusted mean weight, BMI, WC, WHR, HC, FPG, and PG at 30, 60, and 120 min,

triglyceride and HbA_{1c} at baseline, and have higher proportion of obesity. Those who developed diabetes had lower height. The age-adjusted mean (SD) height was 157.5 (7.8) cm for those with and 159.4 (8.0) cm for those without diabetes ($P < 0.05$).

During 6,015 (1,381 men and 4,634 women) person-years of follow-up, 102 (9.3%) (18 men and 84 women) incident cases of type 2 diabetes occurred. The overall incidence of subsequent diabetes was 17.0 (95% CI: 13.7, 20.2) per 1,000 person-year. Incidence rates were higher in women (18.1, 95% CI: 14.3, 22.0 per 1,000 person-year) than men (13.0%, 95% CI: 7.8, 20.6) but the difference was not statistically significant (Fisher exact test, $P = 0.243$). The incidence of diabetes was 14.3 per 1,000 person-year (95% CI: 9.1, 21.4) for participants in the highest quartile of height, and 24.9% per 1,000 person-year (95% CI: 17.5, 34.2) for the lowest quartile. The equivalent incidences for HC were 22.8 (95% CI: 16.1, 31.4) and 13.9 per 1,000 person-year (95% CI: 8.5, 21.3). The risk of type 2 diabetes decreased with increasing quartiles of height (P for trend <0.05), while the HC conferred no significant risk for diabetes. When in multivariate analysis comparing the associations in the highest with the lowest quartile, the height was inversely associated with risk of diabetes (Table 3). Age- and gender-adjusted relative risk shows increasing for HC in 3rd and 4th quartiles, whereas height is associated with decreased incidence of diabetes in 3rd and 4th quartiles. In a multivariate model, the additional adjustment for WC did not appreciably alter the relationship between height and diabetes risk compared to the model adjusted for age and gender (Table 3). The further adjustment for WC alters the relationship between HC and diabetes risk, in fact, reflects a non-significant inverse association of HC with diabetes incidence, when keeping the WC constant.

When we analyzed the relationship in men and women separately, the inverse association of height quartiles and diabetes remained in both men and women but not reached the level of statistical significant, which results in the lack of statistical power to adequately analyze this association (data not shown).

BMI, WC, HC, and height were correlated with each other, and the strongest Pearson correlation coefficients were found between HC and BMI and the weakest ones were between height and HC (Table 4). The correlation between height and BMI and HC was negative.

Discussion

This study showed that height was inversely associated with the risk of diabetes. We observed a non-significant, inverse relation between HC and diabetes after taking into

Table 1 Age, age-adjusted, and proportion baseline characteristics of first-degree relatives of patients with type 2 diabetes by quartile of height and hip circumference in the Isfahan Diabetes Prevention Study

Characteristic	1st quartile	2nd quartile	3rd quartile	4th quartile
	Height (cm)			
Number (%)	269 (24.8)	267 (24.6)	260 (23.9)	290 (26.7)
Age (year)	44.6 (0.38)	43.0 (0.38)	42.7 (0.39)	41.1 (0.37)***
Height (cm)	152.1 (0.37)	156.9 (0.37)	161.1 (0.38)	166.2 (0.36)***
Weight (kg)	67.3 (0.66)	71.3 (0.66)	74.9 (0.67)	79.1 (0.64)***
Waist circumference (cm)	87.2 (0.56)	87.9 (0.55)	89.0 (0.56)	89.8 (0.53)**
Hip circumference (cm)	105.9 (0.54)	107.3 (0.54)	108.3 (0.54)	109.4 (0.52)***
Waist-to-hip ratio	0.82 (0.004)	0.82 (0.004)	0.82 (0.004)	0.82 (0.004)
Body mass index (kg/m ²)	29.1 (0.25)	29.0 (0.25)	28.8 (0.26)	28.7 (0.25)
FPS (mg/dl)	94.6 (0.72)	95.4 (0.72)	93.6 (0.73)	94.7 (0.70)
PG 30 min (mg/dl)	146.9 (1.94)	144.7 (1.94)	140.9 (1.95)	145.3 (1.85)
PG 60 min (mg/dl)	153.4 (2.56)	148.6 (2.59)	148.1 (2.57)	147.9 (2.45)
PG 120 min (mg/dl)	124.6 (2.03)	120.6 (2.02)	116.3 (2.03)	116.3 (1.94)**
HbA _{1c} (%)	5.0 (0.05)	4.9 (0.05)	5.1 (0.06)	5.1 (0.05)**
Cholesterol (mg/dl)	194.9 (2.47)	193.0 (2.44)	193.8 (2.47)	189.5 (2.37)
LDL (mg/dl)	117.2 (2.27)	118.3 (2.24)	114.8 (2.28)	113.7 (2.17)
HDL (mg/dl)	46.3 (0.75)	44.8 (0.74)	46.1 (0.75)	44.6 (0.72)
Triglyceride (mg/dl)	164.5 (5.83)	162.2 (5.78)	163.2 (5.88)	161.0 (5.65)
Systolic BP (mm Hg)	113.1 (0.99)	113.9 (0.98)	116.9 (1.00)	115.1 (0.95)
Diastolic BP (mm Hg)	72.7 (0.76)	73.4 (0.75)	75.3 (0.76)	75.0 (0.73)
Women, no. (%)	208 (76.8)	210 (78.4)	195 (74.1)	226 (77.9)
Obesity (BMI ≥ 30), no. (%)	112 (41.3)	100 (37.5)	94 (36.2)	92 (32.1)
	Hip circumference (cm)			
Number (%)	266 (24.7)	250 (23.2)	278 (25.8)	283 (26.3)
Age (year)	42.9 (0.39)	42.6 (0.40)	42.9 (0.38)	42.8 (0.38)
Height (cm)	157.4 (0.48)	159.2 (0.50)	159.9 (0.47)	160.1 (0.47)***
Weight (kg)	60.9 (0.44)	69.8 (0.45)	75.3 (0.43)	85.8 (0.43)***
Waist circumference (cm)	79.8 (0.39)	86.0 (0.41)	90.3 (0.38)	97.2 (0.38)***
Hip circumference (cm)	97.5 (0.25)	104.5 (0.26)	109.3 (0.24)	118.7 (0.24)***
Waist-to-hip ratio	0.82 (0.004)	0.82 (0.004)	0.83 (0.004)	0.82 (0.004)
Body mass index (kg/m ²)	24.6 (0.15)	27.6 (0.16)	29.5 (0.15)	33.6 (0.15)***
FPS (mg/dl)	93.3 (0.71)	93.3 (0.74)	94.6 (0.70)	96.9 (0.69)***
PG 30 min (mg/dl)	142.0 (1.91)	141.1 (1.98)	143.5 (1.87)	150.7 (1.88)**
PG 60 min (mg/dl)	145.2 (2.54)	146.6 (2.62)	147.5 (2.47)	157.9 (2.45)**
PG 120 min (mg/dl)	120.9 (2.01)	119.6 (2.08)	116.9 (1.97)	120.5 (1.96)
HbA _{1c} (%)	5.0 (0.05)	5.0 (0.06)	5.1 (0.05)	5.1 (0.05)
Cholesterol (mg/dl)	185.5 (2.39)	187.8 (2.45)	201.1 (2.32)	194.4 (2.32)***
LDL (mg/dl)	111.0 (2.20)	111.7 (2.27)	123.5 (2.11)	115.5 (2.11)***
HDL (mg/dl)	46.8 (0.75)	44.9 (0.77)	45.7 (0.72)	44.3 (0.72)
Triglyceride (mg/dl)	145.6 (5.79)	163.0 (5.97)	167.0 (5.66)	173.9 (5.61)**
Systolic BP (mm Hg)	110.5 (0.95)	112.9 (0.98)	115.0 (0.93)	119.0 (0.94)***
Diastolic BP (mm Hg)	70.7 (0.73)	72.9 (0.76)	75.2 (0.72)	77.3 (0.72)***
Women, no. (%)	208 (77.9)	190 (76.0)	214 (76.2)	223 (78.2)
Obesity (BMI ≥ 30), no. (%)	4 (1.5)	26 (10.5)	110 (39.3)	253 (89.7)***

Age-adjusted means were calculated using general linear models. Data are expressed as mean (SE) or number (%). ** $P < 0.01$, *** $P < 0.001$ comparison across all four groups

Table 2 Age, age-adjusted means (SE) of selected baseline characteristics between 102 first-degree relatives of patients with type 2 diabetes who did and 990 who did not develop diabetes

Variables	Developed diabetes Mean (SE)	Not developed diabetes Mean (SE)	Difference (95% CI)
Age (year)	44.0 (0.63)	42.7 (0.20)	1.3 (−0.002, 2.60)*
Weight (kg)	75.6 (1.15)	73.0 (0.37)	2.6 (0.13, 4.87)*
Height (cm)	157.5 (0.78)	159.4 (0.25)	−1.9 (−3.63, −0.37)*
Hip circumference (cm)	110.2 (0.87)	107.5 (0.28)	2.7 (0.92, 4.48)**
BMI (kg/m ²)	30.6 (0.41)	28.7 (0.13)	1.9 (1.05, 2.75)***
Waist circumference (cm)	91.0 (0.90)	88.3 (0.29)	2.8 (1.04, 4.76)**
Waist-to-hip ratio	0.86 (0.007)	0.85 (0.002)	0.01 (−0.004, 0.02)
Systolic BP (mmHg)	113.6 (1.61)	114.6 (0.51)	−1.0 (−3.47, 3.47)
Diastolic BP (mmHg)	74.9 (1.22)	74.0 (0.39)	−0.9 (−1.25, 3.85)
Baseline fasting glucose (mg/dl)	104.7 (1.12)	93.5 (0.36)	11.2 (9.27, 13.90)***
Plasma glucose 30 min (mg/dl)	165.8 (3.16)	142.4 (0.97)	23.4 (17.60, 30.60)***
Plasma glucose 60 min (mg/dl)	188.8 (3.92)	145.4 (1.26)	43.4 (36.90, 53.30)***
Plasma glucose 120 min (mg/dl)	149.4 (3.12)	116.3 (1.00)	33.1 (27.30, 40.10)***
HbA _{1c} (%)	5.5 (0.09)	5.0 (0.03)	0.5 (0.31, 0.69)***
Triglyceride (mg/dl)	183.1 (9.4)	161.0 (3.00)	22.1 (4.57, 43.60)*
Cholesterol (mg/dl)	195.8 (4.01)	192.3 (1.27)	3.5 (−27.30, 37.30)
HDL cholesterol (mg/dl)	44.1 (1.21)	45.5 (0.39)	−1.4 (−3.89, 1.09)
LDL cholesterol (mg/dl)	118.0 (3.76)	115.6 (1.16)	2.4 (−3.96, 11.80)
	No. (%)	No. (%)	
Obesity (BMI ≥ 30)	50 (49.5)	348 (35.4)	14.1 (3.94, 24.3)***

Age-adjusted means were calculated using general linear models. The difference in the mean or percentage of the variables between diabetes and no diabetes. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$. CI Confidence interval

Table 3 Incidence rates and relative risks (95% CI) of type 2 diabetes by quartiles of height and hip circumference, the Isfahan Diabetes Prevention Study, 2003–2010

Variables	Cases (No.)	Incidence/1,000 person-year	Age-adjusted relative risk (95% CI)	Age- and gender-adjusted relative risk (95% CI)†	Age-, gender-, and WC-adjusted relative risk (95% CI)†
Hip circumference					
1st quartile (<100.0)	20	13.9	1.00	1.00	1.00
2nd quartile (100.0–104.0)	19	13.8	0.96 (0.51, 1.79)	0.94 (0.50, 1.77)	0.70 (0.36, 1.34)
3rd quartile (104.1–108.4)	26	17.2	1.25 (0.70, 2.25)	1.22 (0.68, 2.19)	0.71 (0.36, 1.39)
4th quartile (>108.4)	36	22.8	1.63 (0.94, 2.82)	1.58 (0.91, 2.73)	0.59 (0.25, 1.37)
Height					
1st quartile (<165.2)	36	24.9	1.00	1.00	1.00
2nd quartile (165.2–169.5)	24	16.3	0.67 (0.40, 1.13)	0.67 (0.40, 1.13)	0.65 (0.38, 1.10)
3rd quartile (169.6–174.0)	19	13.0	0.49 (0.28, 0.86)*	0.49 (0.26, 0.86)*	0.50 (0.28, 0.87)*
4th quartile (>174.0)	23	14.3**	0.59 (0.35, 1.02)	0.58 (0.34, 0.99)*	0.54 (0.31, 0.93)*

† Relative risks (with 95% CI) calculated by Cox proportional hazard model. CI Confidence interval. * $P < 0.05$, ** P for trend < 0.05

account the effect of age, gender, and waist. Few studies have assessed risks of diabetes with height and the results are inconsistent [5, 12, 19, 20, 33]. The Tehran Lipid and Glucose Study [12] and Finmark Study [19] have shown that height was inversely associated with diabetes incidence in women but not in men. In contrast, the Epic-

Potsdam Study [5] found that height was inversely related to the risk of diabetes in men, but not in women. In the San Antonio Heart study [20] and Nurses' Health Study [33], height predicted diabetes in neither men nor women. In another study, the relation of height and type 2 diabetes not retain statistical significance after accounting for the effect

Table 4 Pearson correlation coefficients between anthropometric indicators, the Isfahan Diabetes Prevention Study, 2003–2010

Variables	Waist circumference	Height	Hip circumference
Body mass index	0.727**	−0.187**	0.884**
Waist circumference	1	0.282**	0.654**
Height		1	−0.060*

* $P < 0.05$, ** $P < 0.01$

of other risk factors [34]. Our findings does not support the negative findings of the San Antonio Heart Study [20] and Nurses' Health study [33]. The results of this study are not consistent with the EPIC-Potsdam Study [5] and the Tehran Lipid and Glucose Study [12] which showed height is inversely associated with diabetes in men or women only.

The mechanisms whereby height exerts negative effects on diabetes risk are not clear. Height [21] and glucose metabolism risk factors [22–24] such as obesity are determined by genetic and early environmental influences. Hormonal factors relevant to growth [35], the intrauterine environment, and childhood nutrition have been previously suggested as a potential pathway linking impaired peripheral growth to the risk of diabetes in adulthood [34]. The risk of developing diabetes has also been observed to be increased among individuals with vitamin D insufficiency [36]. Vitamin D insufficiency may increase visceral fat [37]. Leg length, a component of height and a marker of prepubertal growth, also related to diabetes [34], but this anthropometric measure is not available in the IDPS. The ability of leg length to predict diabetes requires further research.

In our study, the non-significant association of a greater HC with lower risk of developing type 2 diabetes only emerged after controlling for WC. We believe that the absence of statistically significant in our study population was most likely due to the limited statistical power. More studies have examined the effect of HC than height on type 2 diabetes. HC appears to be inversely associated with type 2 diabetes once overall obesity is controlled for [10–18, 38], although this association is not always observed [16]. Most of the studies that have examined association of HC with risk of diabetes were cross-sectional [13–16, 38]. Few prospective studies have looked at HC as an independent predictor of diabetes [10, 11, 17, 18, 39]. The Atherosclerosis Risk in Community Study [11] and the Tehran Lipid and Glucose Study [12] demonstrated that after controlling for WC and BMI, HC was inversely associated with incident diabetes only in women. The Hoorn Study [10] and Shanghai Health Study [39] reported that after controlling for WC and BMI, large HC was associated with a lower risk of diabetes in both men and women. In a Swedish female cohort, Lessner et al. [17] found that larger

HC was associated with a lower incidence of several cardiovascular end-points and diabetes, independent of WC. In a prospective study of Chinese men and women, HC was positively associated with the incidence of type 2 diabetes [18]. However, neither WC nor BMI was taken into account. However, in three later prospective studies the presence of diabetes was not examined on the basis of an OGTT.

Although the exact mechanisms of negative effects of HC on diabetes risk are not entirely clear, the association of a larger HC with a lower risk of developing type 2 diabetes may be attributed to greater muscle mass at the gluteo-femoral region [40]. Larger body fat mass, regardless of location, is a risk factor for diabetes. However, a number of studies have observed that more peripheral fat accumulation in the hip and thigh, for a given amount of abdominal fat, may be associated with a more favorable metabolic profile [41–45]. Both fat and lean tissue from the hip and thigh may contribute, and regional differences in lipolysis may be involved in reduced diabetes risk associated with relatively large HC [42]. Large HC is associated with greater muscle mass at the gluteal region. Skeletal muscle mass is the main tissue target for insulin action and therefore a major site for insulin resistance. These depots may protect the liver and muscle from high exposure to free fatty acids through uptake and storage.

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. Anthropometric variables collected by using direct measurement rather than self-report. Selection and information bias is considered unlikely by virtue of the prospective design. Even though the study included more than 1,000 participants who were thoroughly examined and followed up, the follow-up period of 5.5 years may be controversial. Due to the still conflicting results in assessing diabetes prediction, a long-term follow-up 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, cholesterol, and PG.

In conclusion, these data provide further evidence that the higher height is associated with a lower risk of type 2 diabetes, independently of gender. These findings may represent a more severe metabolic disturbance in a short person than a taller one. To better understand the association of height with incidence diabetes, future research should investigate large and more diverse samples of multiethnic population of men and women.

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Conflict of interest The authors declared no conflict of interest.

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