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Skinfold thickness and the incidence of type 2 diabetes mellitus and hypertension: an analysis of the PERU MIGRANT Study

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Abstract

Objective—To determine the association between excess body fat, assessed by skinfold thickness, and the incidence of type 2 diabetes mellitus (T2DM) and hypertension (HT).

Design—Data from the ongoing PERU MIGRANT Study was analyzed. The outcomes were T2DM and HT, and the exposure was skinfold thickness measured in bicipital, tricipital, subscapular, and suprailiac areas. The Durnin-Womersley formula and SIRI equation were used for body fat percentage estimation. Risk ratios (RR) and population attributable fractions (PAF) were calculated using Poisson regression.

Settings—Rural (Ayacucho) and urban shantytown district (San Juan de Miraflores, Lima) in Peru

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Conflict of Interest

The authors declare neither competing nor conflicts of interests.

Authorship

JJM, AB-O, LS and RHG conceived the original idea of the PERU MIGRANT Cohort Study including the further studies obtained with the follow-ups. The authors were part of the design and supervision. AR-A coordinated and supervised fieldwork activities during the second follow-up conducted between 2015 and 2016. AB-O, AR-A and JJM developed the idea of this manuscript. AB-O led the statistical analysis supported by RMC-L and AR-A. AR-A drafted the first version of the article with great input of AB-O, RMC-L and JJM. All authors participated in writing the manuscript, provided key intellectual content and gave their final approval of the version submitted for publication.

Ethical Standards Disclosure

This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving human subjects/ patients were approved by the Ethics Committees of Universidad Peruana Cayetano Heredia (UPCH) in Peru and the London School of Hygiene and Tropical Medicine in the United Kingdom at baseline. Further follow-up assessments were approved by the Ethics Committee at UPCH. Written or verbal informed consent were obtained from all subjects. When verbal consent was obtained it was witnessed and formally recorded.

Subjects—988 adults aged 30 years (rural, rural-to-urban migrants, urban) completed the baseline study. A total of 785 and 690 were included in T2DM and HT incidence analysis, respectively.

Results—At baseline, the age mean was 48.0 years (SD: 12.0) and 47% were males. For T2 DM, in 7.6 years (SD: 1.3), 61 new cases were identified, overall incidence of 1.0 per 100 person-years; 95% confidence interval (95%CI): 0.8-1.3. Bicipital and sub-scapular skinfolds were associated with 2.8-fold and 6.4-fold risk of developing T2DM. On the other hand, in 6.5 years (SD: 2.5), overall incidence of HT was 2.6 per 100 person-years (95%CI 2.2-3.1). Sub-scapular and overall fat obesity were associated with 2.4 and 2.9-fold risk for developing HT. The PAF for sub-scapular skinfold was 73.6% and 39.2% for T2DM and HT, respectively.

Conclusions—We found a strong association between sub-scapular skinfold thickness and developing T2DM and HT. Skinfolds assessment can be a laboratory-free strategy to identify high-risk HT and T2DM cases.

Keywords

Type 2 diabetes mellitus; hypertension; skinfold-thickness; obesity

Introduction

In 2016, nearly 18 million deaths were caused by cardiovascular diseases (CVDs) accounting for one third of the global deaths. In the last decade, the impact of the increasing prevalence of non-communicable diseases has been observed in low and middle income countries, where approximately 75% of the global deaths related to CVDs occurred (1,2). Hypertension (HT) and type 2 diabetes mellitus (T2DM) are the strongest modifiable risk factors for cardiovascular disease and the subsequent mortality (3). HT is the main risk factor for cardiovascular diseases (4), which accounts for more than two thirds of the total deaths associated with non-communicable diseases. On the other hand, USD \$827 billion are expended annually in patients with T2DM worldwide (5).

A common major risk factor for CVDs is obesity with a global prevalence among adults of 13% in 2016 (6). The World Health Organization defines obesity as an excess of body fat and recommends using body mass index (BMI) to assess it, as BMI is a simple tool to use in routine clinical examinations (7). However, BMI does not differentiate fat percentage from lean mass. Therefore, the obesity diagnosis obtained by it is not necessarily correlated with body fat percentage, which is associated with cardiovascular disease or metabolic disturbances (8,9). Waist circumference is another anthropometric marker with a demonstrated association with T2DM and HT and represents the abdominal fat and android distribution of fat (10). The gold standards to quantify body fat percentage (BF%) as well as describe its distribution are the dual-energy X-ray absorptiometry (DXA) and skinfolds thickness assessment (11). Skinfold thickness, being one of the recommended standards for excess of BF%, remains understudied in its relationship with major cardiometabolic risk factors such as T2DM and HT. In terms of longitudinal evaluations, only two studies from developing countries have explored the association between the skinfolds-thickness results

and developing T2DM, and only one of these considered HT as an additional outcome(12,13).

Accordingly, we aimed to determine the association between excess of body fat, estimated by skinfold thickness using four different regions (i.e. bicipital, tricipital, subscapular, suprailiac), and the risk of developing T2DM and HT using information of the PERU MIGRANT Cohort Study. We hypothesized excess of BF% according to skinfold thickness would demonstrate a greater association with T2DM and HT than those observed with traditional anthropometric markers.

Methods

Study design

We analyzed data from the PERU MIGRANT Study, a prospective ongoing cohort whose baseline assessment was conducted in 2007-2008. Two follow-ups were carried out, the first one in 2012-2013 and the other during 2015-2016 (14,15).

Settings and Participants

At baseline, individuals aged ≥ 30 years old who reported living permanently in the selected rural and urban areas were invited to participate in the study after a single-stage random sampling method. Rural dwellers were recruited from the district of San Jose de Secce in Huanta, Ayacucho. Rural-to-urban migrants and urban dwellers were selected from Las Pampas de San Juan de Miraflores, Lima. Participants unable to consent and pregnant women were excluded(14).

Enrollment at baseline was made through a single-stage random sampling technique. In San Jose de Secce, the rural site, a census was conducted in 2007. In the case of Lima, the urban area, a local census was updated in that year. Those that were born and lived permanently in Las Pampas de San Juan de Miraflores, Lima, were included in the urban group, whereas those who reported to permanently live in Las Pampas de San Juan de Miraflores at the time of the baseline assessment but were born in Ayacucho were considered in the migrant group. A face-to-face administered questionnaire as well as clinical and laboratory assessments were conducted to obtain data related to CVD risk profile and sociodemographic characteristics. Only those with completed information were included in the cohort baseline, resulting in an overall response rate of 61.6% (989/1606) (14). Participants were re-evaluated during follow-up assessments by contacting them in the settings where they were originally enrolled in the study.

Definition of Variables

Outcome variables—The two outcomes of interest were T2DM and HT. T2DM cases were defined as: a fasting glucose level ≥ 126 mg/dL obtained within 8 and 12 hours of fasting, self-reported T2DM diagnosis made by a physician, or a current record that the participant was on anti-diabetic medication. In the follow-up assessment, new cases of T2DM were considered only when the participant accomplished the first two diagnostic criteria we used at baseline. Laboratory method used for plasma glucose measurement was

the enzymatic colorimetric (GOD-PAP, Modular P-E/Roche-Cobas, Grenzach-Whylen, Germany) for 5ml of whole blood.

At baseline, all cases of hypertension were defined by: the average of the last two of three measurements of SPB (≥ 140 mmHg) or DBP (≥ 90 mmHg) in the clinical assessment, the participant's self-report of HT diagnosis done by a physician, or currently receiving antihypertensive medication. New cases of hypertension identified in the first and second follow-up were determined by using only the first two criteria, as the antihypertensive medication data did not provide more information.

Exposure variables—Excess body fat, only measured at baseline, was defined as the exposure variable according to each skinfold measurement. Four different areas of the body (i.e. bicipital, tricipital, subscapular, suprailiac) were measured in triplicate and assessed by the same fieldworker, using a Holtain Tanner/Whitehouse Skinfold Caliper calibrated to the nearest 0.2 mm. The average of the three measures for each skinfold was calculated and utilized for further estimations. The Durnin-Womersley density formula was used to estimate the body density according to each skinfold measurement as well as the overall fat obesity, defined as the total sum of all the four skinfolds (16). Then, the SIRI equation was used to calculate body fat percentage for each skinfold parameter (17). Sex-specific cut-off points ($>25\%$ for males and 33% for females) established by the Spanish Society for Obesity Studies were used (18). For comparison, obesity, defined by BMI (≥ 30 kg.m⁻²), waist circumference (WC ≥ 80 cm for women and ≥ 90 cm for male), waist to hip ratio (WHR, cut-off point ≥ 0.85 for women and ≥ 0.95 for male) and waist-to-height ratio (WHtR, cut-off point ≥ 0.5) were considered as other exposure variables of interest (18,19). Anthropometric assessments were conducted following standardized procedures by trained fieldworkers.

Other variables—The following sociodemographic variables, assessed at baseline, were also included in the analysis as potential confounders: sex (female vs. male), age (<50 vs. ≥ 50 years), education level (non/some primary, complete primary, and secondary or more), and socioeconomic status based on a wealth index and split in tertiles. Additionally, non-healthy behavioral risk factors were also assessed. Physical activity was assessed through the International Physical Activity Questionnaire (IPAQ). Levels in MET minutes/week were categorized into low and medium/high). Heavy drinking was defined as either the self-report of having a hangover or having ≥ 6 drinks on the same occasion at least once per month. Finally, tobacco consumption was defined as current daily smoking.

Statistical analysis

The statistical package STATA 13 for Windows (StataCorp, College Station, TX, US) was used for analyses. Initially, the description of the study population by study groups (rural, rural-to-urban migrant and urban) defined at baseline was conducted. Means and standard deviations, as well as proportions were calculated and presented according to the population subgroups. Comparisons were performed using the one-way ANOVA and χ^2 tests as required.

For the incidence analysis, we excluded those participants who were diagnosed with HT or T2DM at baseline accordingly. Incidence rates and associated 95% confidence intervals

(95% CI) were estimated in person-years by adding the follow-up times of re-contacted participants. However, for those with a new diagnosis of T2DM or HT, only half of the time between their baseline and follow-up assessment was used in this sum, since the actual date of diagnosis is unknown (actuarial method). Since T2DM and HT diagnosis status were not available for those dead and lost to follow-up, information of the last follow-up was used if possible.

To determine the associations of interest, Poisson regression models, using link log and robust standard errors to account for the cluster effect were utilized to report risk ratios (RR) and 95% CI as the risk of death was assumed constant over time. Two regression models adjusted by different confounders were developed. Model 1 adjusted for sex, age, education, assets index, population group, heavy drinking, currently daily smoking and physical activity. Model 2 adjusted for everything in Model 1 plus BMI. In addition, population attributable fraction (PAF) was also calculated using the “*punaf*” command in STATA (20,21) and compared between models.

Results

Characteristics of the study population at baseline

At the baseline enrollment, 988 participants completed the study. Overall age was 48.0 years (SD: 12.0) and 47% were males. Baseline prevalence of T2DM and hypertension was 4.1% and 16.1% respectively, being higher in the urban group (Table 1). The mean of all skinfold measurements, as well as the sum of them, was significantly higher in the urban and migrant group, both compared to those who were rural dwellers ($p<0.001$, Table 1).

Skinfolds measurement and incidence and risk of T2DM

A total of 106 (10.7%) participants were lost-to-follow up, and 57 (5.8%) died. For the T2DM incidence, we excluded those who had T2DM diagnosis at baseline ($n=40$) (Online Figure 1). The mean follow-up was 7.6 years (SD: 1.3), leading to a 6,068 person-years of follow-up. We identified 61 new T2DM cases, resulting in overall incidence of 1.0 (95% CI 0.8-1.3) per 100 person-years. T2DM incidences according to the different obesity approaches are described in Table 2.

Overall fat obesity was associated with a 2.3-fold risk for T2DM. Bicipital and subscapular excess of BF% were the measurements strongly associated with higher risk of T2DM, with almost 3-fold and 6-fold risk, respectively. Population attributable fraction due to subscapular fat was greater than other common anthropometrics measurements such as BMI, WC and WHtR (Table 2). After controlling for BMI, results showed a strong association between subscapular skinfold thickness and the risk of T2DM with a small variation in PAF.

Skinfolds measurement and incidence and risk of hypertension

In the case of hypertension, we excluded 160 participants diagnosed at baseline (Online Figure 2). The mean follow-up was 6.5 years (SD: 2.5) leading to a 4,850 person-years of follow-up. A total of 128 new cases of hypertension were identified, which resulted in a

cumulative incidence of 2.6 (95% CI 2.2–3.1) per 100 person-years. HT incidences according to the different obesity approaches are described in Table 3.

Obesity-defined subscapular and supra-iliac fat, as well as the sum of all measurements, were significantly associated with higher risk of developing hypertension. Although almost all the obesity anthropometric markers were associated with the risk of developing HT, the sum of all skinfolds measurements, supra-iliac fat, WHtR, and subscapular fat showed higher population attributable fractions (52.2%, 47.7%, 46.5% and 42.1%, respectively) (Table 3). After controlling for BMI, associations and their respective PAF were similar to those obtained by models without that adjustment.

Discussion

We found a strong association between body fat percentage, assessed by skinfold thickness, and the risk of developing T2DM and HT even after adjusting for several confounders, including BMI. Obesity defined by body fat percentage in the subscapular area was the strongest anthropometric marker associated with the risk of T2DM, whereas overall fat obesity was most greatly associated with developing HT.

Skinfolds thickness measurements as predictor of T2DM

The impact of obesity, assessed by BMI, waist circumference and waist-to-height ratio, on the development of T2DM has been largely described in large cohort prospective studies (22,23). However, there is still very limited prospective information assessing the association of skinfold thickness with incident T2DM. To our knowledge, there is no specific recommendation about the use of an anthropometric measurement as a marker for T2DM risk prediction besides BMI and waist circumference (24,25). Our findings provide evidence favoring the utility of measuring skinfolds as a laboratory-free marker for the risk of T2DM in non-specialist and low-resource settings.

Upper body fat, known as truncal fat, has been associated with an increased insulin resistance among men (26). Many markers of insulin secretion and its metabolism have been studied according to fat distribution. These studies have indicated that visceral and upper body fat are more related to the release of free fatty acids and their accumulation in the liver, which subsequently leads to glucose metabolism impairment and even cardiovascular mortality (27–29). Few observational studies have approached the relationship between subscapular skinfold-thickness and the risk of developing T2DM. Of these studies, only three were prospective in nature (12,13,30). Mensik et al. reported that a greater sub-scapular skinfold thickness was strongly associated with the risk of transient and persistent impaired glucose tolerance after 4 months of follow-up compared to BMI, WC, and the sum of all skinfold measurements (12). However, Chei et al. questioned this association, as only a marginal significant risk for T2DM and HT was found (13). Data regarding physical activity and sociodemographic characteristics were not available in this latter report, leaving room to better assess the association including further confounders as did herein. Sosenko et al. noted a strong association between subscapular skinfold-thickness and non-insulin dependent diabetes. Compared to BMI and WHtR, subscapular skinfold-thickness was superiorly associated with T2DM, even after controlling for sex (30).

Some studies suggest that the relationship between insulin secretion and high carbohydrate diets, which are particularly common in Peru and other Latin American countries (31), varies according to the phenotype and body fat distribution. The positive association between abdominal obesity and T2DM is clear and seems to be stronger when other criteria of metabolic syndrome (e.g., high cholesterol) are present (32–34). Moreover, in patients with a history of vascular disease, waist circumference has a positive association with T2DM risk (35). A diet rich in carbohydrates would increase the abdominal perimeter, leading to high levels of inflammation markers related to T2DM, e.g. IL-6, complement factor C3 and C reactive protein (36). In Asian populations waist circumference has been associated not only with T2DM but also with diabetic retinopathy (37). Among Hispanics, data is limited. Mexican populations had greater levels of insulin secretion after oral glucose intake unlike to white counterparts (38). Finally, truncal fat is linked to parental history of T2DM, which is also an established risk factor for T2DM (39). Overall, the assessment of body fat distribution could play an important role in the T2DM screening and follow-up.

Skinfolds thickness measurements as predictor of hypertension

Data regarding the association between skinfold thickness and HT is even more limited. To our knowledge, there is only one study addressing this association prospectively. The Kaunas Cardiovascular Cohort Risk Study conducted in Lithuania showed that an increased skinfold thickness measured in childhood was not associated with hypertension in adulthood, after 35 years of follow-up (40). There are chemical changes during adolescence that may explain why skinfold thickness assessed during childhood might not reflect final fat distribution in adulthood (41). In addition, studies in twins have reported that BMI and skinfolds vary with age and sex independently of the heritability, and that skinfold thickness measurements performed in childhood actually overestimate body fat percentage (42). Our study focused only on adults and demonstrated the long-term effect of body fat distribution on blood pressure outcomes.

A few cross-sectional studies, mainly in children, considered skinfold thickness measurements as exposure variables while assessing HT or blood pressure as outcomes. Khadilkar et al. found a relevant association between higher blood pressure and tricipital skinfold thickness in children aged 5-17 years (43). Ying-Xiu et al. assessed upper body fat as the sum of tricipital and sub-scapular skinfold thickness, obtaining similar results in 12-year-old children (44). Okosun et al. found that truncal obesity, abdominal obesity and their co-occurrence had higher odds of HT, especially in adult women (45). Our longitudinal study confirms these associations. A cross-sectional analysis in young Mexican women (mean age: 29.6 SD 5.4 years), reported that the sum of all skinfold thickness was almost equally efficient in predicting HT compared to BMI and WC (46); however, in our prospective analysis, BMI had the weakest association and lowest PAF. In that sense, our results are physiologically compatible with previous studies where sub-scapular, supra-iliac and tricipital skinfold thickness had association to standard indicators of organ damage due to HT (47).

Our findings suggest that body fat located in sub-scapular area is a risk factor for developing T2DM and HT. Thus, sub-scapular skinfold thickness measurement could be used as an

individual potential tool or part of a risk score to identify individuals at high risk for developing T2DM and HT. In addition to BMI, WHtR and overall fat obesity could be also used as a screening tool for HT. In resource-limited settings, where laboratory analysis is not always included in the minimal health insurance package, these anthropometric parameters could provide a cost-effective and individualized approach identify and monitor patients at risk for developing either of these two conditions.

Regardless of the settings, inserting some skinfold parameters may improve the surveillance and screening of CVD risk factors by identifying subjects at high risk for developing T2DM and HT. The strong association and high PAF percentages from sub-scapular skinfold-thickness support the utility of using this anthropometric marker as part of the general clinical evaluation in adults.

Strengths and limitations

The PERU MIGRANT Cohort Study has three well-defined socio-demographic groups, controlling our models by urbanization exposure as in previous studies (48,49). In addition, our regression analysis allows us to pursue PAF of all the anthropometric measurements and compare them.

Nevertheless, our study remained with limitations that should be declared. First, potential misclassification of T2DM cases can arise, because oral glucose tolerance was not used as diagnostic method (14). However, our main findings will not change since the under-diagnosis of T2DM would have a negative impact in the strength of the associations, which implies that risk of developing T2DM would be even greater. Second, selection bias might arise due to difference in baseline response rates among population groups, which was higher among rural dwellers (14). Third, skinfold thickness measurement has its own limitations such as variance among measurers and caliper types, ethnic phenotypes, etc. In order to minimize the effect of these variations, we standardized the skinfold thickness measurement at baseline. Nevertheless, we recognized potential additional bias due to variation of body fat during the observation period, which was not assessed periodically by skinfold thickness. Finally, data regarding diet was not collected, representing a potential but minimal bias because of its relationship with body fat distribution according to the intake of simple and complex carbohydrates and saturated fat, which typically accumulates in waist circumference. Therefore, further studies are needed to address the impact of diet patterns.

Conclusions

There is evidence of a strong association between sub-scapular an overall fat obesity assessed through skinfold thickness and the risk of developing T2DM and HT. Moreover, after adjusting for obesity according to BMI, which is used the most in the daily clinical practice; subscapular obesity remained as one of the best predictors for T2DM. We remark that body mass index and waist circumference, commonly used anthropometric measurements, had lower population attributable fractions, particularly for hypertension. Our findings suggest that skinfold thickness, if used as part of routine assessments, might improve the detection process of T2DM and HT in settings without clinical specialists or accessible laboratory support. Nevertheless, considering the limitations of the skinfold

thickness measurements, implementation type studies are needed in order to disentangle the impact of subscapular skinfold thickness measurement as part of the routine clinical examination in elderly or overweight patients.

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Online Figure 1. Baseline enrollment and participants included in the T2DM incidence analysis of the PERU MIGRANT study

Online Figure 2. Baseline enrollment and participants included in the HT incidence analysis of the PERU MIGRANT study

Table 1
Characteristics of the sample at baseline by population group

	Rural	Migrant	Urban	p-value*
Sex	N=200	N=589	N=199	
Female	106 (53.0%)	309 (52.5%)	107 (53.8%)	0.95
Age	N=200	N=589	N=199	
50+	84 (42.0%)	252 (42.8%)	89 (44.8%)	0.83
Asset index	N=200	N=589	N=199	
Low	123 (61.5%)	242 (41.1%)	67 (33.7%)	<0.001
Middle	14 (7.0%)	156 (26.5%)	69 (34.7%)	
High	63 (31.5%)	191 (32.4%)	63 (31.7%)	
Education	N=200	N=588	N=198	
None/Some primary	132 (66.0%)	183 (31.1%)	13 (6.6%)	<0.001
Complete primary	29 (14.5%)	99 (16.8%)	23 (11.6%)	
Some secondary or more	39 (19.5%)	306 (52.1%)	162 (81.8%)	
Current daily smoking	N=200	N=587	N=199	
Yes	1 (0.5%)	15 (2.6%)	17 (8.5%)	<0.001
Heavy drinking	N=200	N=589	N=199	
Yes	24 (12.0)	48 (8.2)	19 (9.6)	0.26
Physical activity levels	N=200	N=582	N=198	
Low	4 (2.0%)	173 (29.7%)	78 (39.4%)	<0.001
Biceps skinfold continuous	N=198	N=583	N=198	
	5.0 SD 2.4	11.1 SD 8.4	15.4 SD 9.6	<0.001
Biceps skinfold (obesity)	N=198	N=583	N=198	
	22 (11.0%)	362 (61.5%)	158 (79.4%)	<0.001
Triceps skinfold continuous	N=198	N=583	N=198	
	11.1 SD 5.2	22.7 SD 11.3	28.6 SD 12.2	<0.001
Triceps skinfold (obesity)	N=198	N=583	N=198	
	39 (19.5%)	478 (81.2%)	175 (87.9%)	<0.001
Subscapular skinfold continuous	N=198	N=583	N=198	
	12.7 SD 5.5	20.8 SD 7.7	23.8 SD 8.3	<0.001
Subscapular skinfold (obesity)	N=198	N=583	N=198	
	49 (24.5%)	389 (66.0%)	158 (79.4%)	<0.001
Supra-iliac skinfold continuous	N=197	N=581	N=197	
	13.2 SD 10.7	26.7 SD 8.8	28.4 SD 9.3	<0.001
Supra-iliac skinfold (obesity)	N=197	N=581	N=197	
	62 (31.0%)	495 (84.0%)	170 (85.4%)	<0.001
Sum of all skinfolds continuous	N=197	N=581	N=197	
	42.1 SD 20.1	81.4 SD 30.6	96.0 SD 35.1	<0.001
Sum of all skinfolds (obesity)	N=197	N=581	N=197	
	43 (21.5%)	464 (78.8%)	171 (85.9%)	<0.001
BMI continuous	N=200	N=589	N=199	

	Rural	Migrant	Urban	p-value*
	23.2 SD 2.7	27.0 SD 4.3	28.3 SD 5.4	<0.001
BMI categorized (obesity)	N=200	N=589	N=199	
	6 (3.0%)	124 (21.1%)	68 (34.2%)	<0.001
WC continuous	N=197	N=587	N=198	
	76.2 SD 8.5	88.1 SD 9.9	91.4 SD 12.1	<0.001
Waist categorized (obesity)	N=197	N=587	N=198	
	33 (16.5%)	356 (60.4%)	133 (66.8%)	<0.001
Waist to hip ratio continuous	N=196	N=586	N=198	
	0.8 SD 0.1	0.9 SD 0.1	0.92 SD 0.7	<0.001
Waist to hip ratio (obesity)	N=196	N=586	N=198	
	66 (33.0%)	424 (72.0%)	129 (64.8%)	<0.001
Waist to height ratio continuous	N=197	N=586	N=198	
	0.5 SD 0.1	0.6 SD 0.1	0.6 SD 0.1	<0.001
Waist to height ratio (obesity)	N=197	N=586	N=198	
	94 (47.7%)	511 (87.2%)	170 (85.9%)	<0.001
Hypertension	N=200	N=588	N=199	
	24 (12.0%)	76 (12.9%)	59 (29.7%)	<0.001
Type 2 Diabetes Mellitus	N=199	N=589	N=199	
	3 (1.5%)	21 (3.6%)	16 (8.0%)	0.003

* Chi square, t test and analysis of variance (ANOVA) were used to assess difference between groups accordingly to the type of variable.

Table 2
Incidence and risk ratio of T2DM according to obesity defined by skinfolds

	Incidence (95% CI) per 100 person-years	Risk ratio (95% CI) Crude model	Risk ratio (95% CI) Model 1*	Population Attributable Fraction	Risk ratio (95% CI) Model 2**	Population Attributable Fraction
<i>Bicep fat</i>						
Yes	1.47 (1.11 - 1.94)	3.23 (1.72 - 6.07)	2.75 (1.39 - 5.41)	51.1%	1.91 (0.90 - 4.08)	38.3%
<i>Triceps fat</i>						
Yes	1.22 (0.93 - 1.60)	2.47 (1.17 - 5.19)	1.84 (0.82 - 4.15)	39.8%	1.36 (0.58 - 3.18)	22.8%
<i>Subscapular fat</i>						
Yes	1.52 (1.17 - 1.98)	6.87 (2.75 - 17.17)	6.40 (2.52 - 16.23)	77.5%	5.04 (1.85 - 13.73)	73.6%
<i>Supra-iliac fat</i>						
Yes	1.20 (0.92 - 1.56)	2.71 (1.17 - 6.30)	2.11 (0.90 - 4.96)	47.4%	1.65 (0.66 - 4.12)	35.5%
<i>Overall fat</i>						
Yes	1.26 (0.96 - 1.64)	2.98 (1.35 - 6.54)	2.34 (1.10 - 4.99)	50.7%	1.74 (0.77 - 3.94)	37.6%
<i>Obesity (WC)</i>						
Yes	1.44 (1.08 - 1.92)	2.77 (1.52 - 5.04)	2.31 (1.15 - 4.66)	43.5%	1.45 (0.64 - 3.28)	23.7%
<i>Obesity (WHR)</i>						
Yes	1.26 (0.95 - 1.67)	2.37 (1.32 - 4.27)	2.08 (1.02 - 4.26)	41.7%	1.59 (0.76 - 3.34)	29.8%
<i>Obesity (WHrT)</i>						
Yes	1.15 (0.88 - 1.49)	2.79 (1.21 - 6.46)	2.40 (0.94 - 6.11)	53.5%	1.70 (0.63 - 4.61)	37.8%
<i>Obesity (BMI)</i>						
Yes	2.52 (1.75 - 3.62)	3.89 (2.36 - 6.42)	3.53 (2.02 - 6.16)	34.1%	--	--

In bold p<0.05, CI: confidence interval

* Model 1 adjusted by age, sex, education, assets index, current daily smoking, heavy drinking, population group, and physical activity levels.

** Model 2 adjusted by age, sex, education, assets index, current daily smoking, heavy drinking, population group, physical activity levels and BMI (in categories).

Table 3
Incidence and risk ratio of hypertension according to obesity defined by skinfolds

	Incidence (95% CI) per 100 person-years	Risk ratio (95% CI) Crude model	Risk ratio (95% CI) Model 1*	Population Attributable Fraction	Risk ratio (95% CI) Model 2**	Population Attributable Fraction
<i>Bicep fat</i>						
Yes	3.19 (2.57 - 3.97)	1.52 (1.05 - 2.21)	1.92 (1.20 - 3.08)	30.3%	1.68 (1.02 - 2.77)	25.7%
<i>Triceps fat</i>						
Yes	2.68 (2.18 - 3.29)	1.02 (0.69 - 1.51)	1.24 (0.74 - 2.09)	13.7%	1.08 (0.63 - 1.86)	5.3%
<i>Subscapular fat</i>						
Yes	3.36 (2.75 - 4.11)	1.96 (1.31 - 2.94)	2.35 (1.46 - 3.78)	42.1%	2.15 (1.30 - 3.55)	39.2%
<i>Supra-iliac fat</i>						
Yes	2.96 (2.45 - 3.58)	1.64 (1.02 - 2.64)	2.33 (1.31 - 4.13)	47.7%	2.19 (1.22 - 3.94)	45.4%
<i>Overall fat obesity</i>						
Yes	3.12 (2.57 - 3.78)	1.82 (1.17 - 2.85)	2.85 (1.64 - 4.96)	52.2%	2.64 (1.49 - 4.67)	50.0%
<i>Obesity (WC)</i>						
Yes	3.36 (2.71 - 4.17)	1.71 (1.18 - 2.48)	2.12 (1.37 - 3.29)	33.8%	1.86 (1.16 - 3.00)	29.7%
<i>Obesity (WHR)</i>						
Yes	3.20 (2.62 - 3.91)	1.87 (1.25 - 2.81)	2.12 (1.32 - 3.41)	39.2%	1.93 (1.17 - 3.20)	35.8%
<i>Obesity (WHtR)</i>						
Yes	2.89 (2.40 - 3.49)	1.64 (1.00 - 2.71)	2.21 (1.28 - 3.80)	46.5%	2.01 (1.14 - 3.52)	42.7%
<i>Obesity (BMI)</i>						
Yes	4.13 (2.97 - 5.76)	1.78 (1.18 - 2.67)	1.91 (1.21 - 3.01)	13.0%	--	--

In bold p<0.05, CI: confidence interval

* Model 1 adjusted by age, sex, education, assets index, current daily smoking, heavy drinking, population group, and physical activity levels.

** Model 2 adjusted by age, sex, education, assets index, current daily smoking, heavy drinking, population group, physical activity levels and BMI (in categories)