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# PAPER

# The body mass index is a less-sensitive tool for detecting cases with obesity-associated co-morbidities in short stature subjects

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**OBJECTIVE:** To assess the ability of the body mass index (BMI) to detect obesity-associated morbidity in subjects with a normal or short stature.
**METHODS:** Information was obtained on 119 975 subjects from a cardiovascular risk factors detection program. Standardized questionnaires were used. Capillary glucose and cholesterol concentrations were measured. Diabetes, arterial hypertension and hypercholesterolemia were selected as end points. Sensitivity, specificity and the likelihood ratio for several BMI thresholds were calculated. ROC curves were constructed to identify the BMI cutoff points with best diagnostic performance. The area under the curve (AUC) was used to assess the proficiency of BMI.
**RESULTS:** Short stature (height ≤150 cm for women or ≤160 cm for men) was found in 24 854 subjects (20.7%). These cases had a higher prevalence of type II diabetes and arterial hypertension even after adjusting for confounding variables. In addition, the frequency of the abnormalities was higher even at the lowest BMI values; the prevalence increased in direct proportion with the BMI, but at a lower rate compared to cases with normal stature. The AUC for every co-morbidity was smaller in short stature subjects. The likelihood ratio for detecting co-morbidities increased at the same BMI value in subjects with or without short stature.
**CONCLUSIONS:** The prevalence of obesity-associated co-morbidities is higher in subjects with short stature compared to those without it. The proficiency of BMI as a diagnostic tool is poor in short stature subjects. This problem is not resolved by decreasing BMI thresholds used to define overweight.
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## Introduction

The ability of body mass index (BMI) to predict excess morbidity and mortality differ between ethnic groups.[1] In Caucasians, the risk of having obesity-related complications increase after a BMI of 25 kg/m[2].[2] In contrast, in Asian-Pacific populations, the risk increases with a BMI greater than 23 kg/m[2].[3] There are various reasons for this discrepancy. Firstly, Asian subjects have a higher fat mass for a given BMI compared to Caucasians. On average, an Asian subject with a similar body fat composition to a Caucasian

would have a BMI three units higher.[4] Secondly, the exponential relationship between weight and height of the BMI may result in discrepancies at the extremes of these variables. Differences in height may contribute to the discordant behavior of BMI between ethnic groups. The percentage of individuals with short stature is much higher in Asians compared to Caucasians. In México, short stature (≤150 cm for women or ≤160 cm for men) is found in 29% of the population.[5] Thirdly, short stature could result from malnourishment early in life. Such malnourishment is a risk factor for the development of the metabolic syndrome and abdominal adiposity.[6–8] Thus, additional studies are needed to assess the influence of these factors on the accuracy of BMI thresholds to predict obesity-related complications. The purpose of this report is to assess the ability of BMI to detect obesity-related morbidity in subjects with normal or short

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stature. Our data were obtained from a large survey based in six Mexican cities designed to detect cardiovascular risk factors.

## Material and methods

In this large cross-sectional study, individuals from six Mexican cities were included ($n = 120\,001$). Four of these cities are located in central Mexico (Mexico City ($n = 37\,457$), Guadalajara ($n = 25\,514$), Leon (13 022) and Puebla ($n = 14\,055$)), and the two remaining are in the north of the country (Monterrey ($n = 15\,614$) and Tijuana ($n = 14\,339$)). Subjects were sampled during 2001 and 2002.

The purpose of the survey was the detection of cases with modifiable cardiovascular risk factors. The Health Ministry and a private sponsor provided seven mobile units. In each one, up to 150 people could be evaluated per day. The units were located in commercial malls or prescheduled visits were made to factories or companies with large number of employees. Every adult aged 30 y or older was invited to participate. The study was done in accordance with the Helsinki Declaration of Human Studies.

Each unit had a trained team composed of a general practitioner, a nurse, a dietitian and paramedic personnel. The demographic data and medical history were recorded using a standardized questionnaire. Blood pressure was measured with the subject in the supine position after a 5-min rest. Height and body weight were measured on a daily-calibrated scale. BMI was calculated as weight (kg) divided by height ($m^2$). A blood sample for the measurement of capillary glucose and cholesterol concentrations was requested from all cases. Data are presented from the 119 975 cases in which valid capillary glucose and cholesterol results were collected (81.65% of the total). Samples were obtained after a fasting period of at least 2 h. The sampling procedure was standardized during a training course. Accutrend sensor monitors (Roche diagnostics) were used in this study for the measurement of capillary glucose levels. Reflotron plus monitors (Roche diagnostics) were used for the measurement of cholesterol concentrations.

### Definitions

Short stature was defined as a height $\leq 150$ cm for women or $\leq 160$ cm for men.[9] Diabetes was diagnosed in known cases or if the random plasma glucose concentration was above 200 mg/dl or the fasting capillary glucose $\geq 126$ mg/dl.[10] Type II diabetes was diagnosed using the definition proposed by the American Diabetes Association.[11] BMI's 25–30 kg/m$^2$ and $\geq 30$ kg/m$^2$ were defined as overweight and obesity, respectively. Hypertension was diagnosed when the blood pressure was $\geq 140/90$ mmHg and/or with current use of antihypertensives. The National Cholesterol Education Program-III guidelines were used to identify and define independent cardiovascular risk factors.[12] Hypercholestero-

lemia was considered present if blood cholesterol was equal or greater than 200 mg/dl or the patient was on lipid-lowering medication. Tobacco smoking was considered present if the patient referred the consumption of at least one cigarette during the previous month.

### Statistical analysis

Continuous variables were described using means and standard deviations. Data from patients sampled in Mexico City, Leon, Puebla and Guadalajara were analyzed together based on the similar characteristics of the study subjects; this group was labeled Central Mexico. The same was done for the results obtained in Tijuana and Monterrey; this subset was labeled as Northern Mexico. The one-way analysis of variance test was applied to compare differences between groups. Categorical variables were compared with the $\chi^2$ statistic. The sensitivity and specificity of several BMI thresholds to predict diabetes, hypertension and hypercholesterolemia were compared in subjects of normal and short stature. The likelihood ratio was calculated to show the odds of having the above-mentioned obesity-related co-morbidities at differing BMI thresholds.[13] This ratio gives the probability of finding a case with the specified condition. It is defined as sensitivity/($1 -$ specificity). ROC curves were constructed by plotting sensitivity $vs$ $1 -$ specificity. The BMI value with the best diagnostic performance was that closest to the left corner of the graph. The overall performance of the ROC curve was quantified by estimating the area under the curve. The estimates were calculated after stratifying for gender and age groups (above or below age 40 y). Differences between areas under the ROC curves were compared using the one-way analysis of variance test. Multiple logistic regression models were used to determine the ability of obesity and overweight to predict co-morbidities; short stature, age, region and smoking were included as covariates. The statistical analysis was conducted in SPSS 10.0 for windows.

## Results

The population ($n = 119\,975$) was composed predominantly of women (58.3%). Nearly half of the study subjects were aged 40 y or younger ($n = 49\,989$ (42.5%)); subjects older than age 70 y represented a small fraction of the sample ($n = 5278$ (4.4%)). The majority of the men were blue-collar workers ($n = 37\,618$ (75.3%)); nearly half of the women worked at home (38 985 (55.7%)). The prevalence of obesity-related co-morbidities selected as end points in this report were high: hypertension 30.2% ($n = 36\,251$), type II diabetes 10.7% ($n = 12\,804$) and hypercholesterolemia 43.3% ($n = 51\,928$). Past medical history of a coronary event was reported by 0.9% ($n = 1029$). Other cardiovascular risk factors were also common: tobacco smoking 25.4% ($n = 30\,476$), family history of cardiovascular death 11.5% ($n = 13\,738$)

**Table 1** Characteristics of men and women with short stature compared with normal stature cases

|  | Women Height $\leq 150$ cm | Women Height $> 150$ cm | P-value | Men Height $\leq 160$ cm | Men Height $> 160$ cm | P-value |
|---|---|---|---|---|---|---|
| N (n(%)) | 18068 (25.8) | 51928 (74.2) |  | 6786 (13.6) | 43193 (86.4) |  |
| Age (y) | $50.9 \pm 14.5$ | $43.9 \pm 12.3$ | <0.001 | $47.7 \pm 14.5$ | $41.9 \pm 11.9$ | <0.001 |
| Living in central Mexico (n(%)) | 14345 (79.4) | 38542 (74.2) | <0.001 | 5254 (77.4) | 31894 (73.9) | <0.001 |
| Living in northern Mexico (n(%)) | 3723 (20.6) | 13379 (25.8) | <0.001 | 1532 (22.6) | 11289 (26.1) | <0.001 |
| High school education or above (n(%)) | 3599 (19.9) | 21215 (40.8) | <0.001 | 1930 (28.4) | 23715 (54.9) | <0.001 |
| Weight (kg) | $62.9 \pm 11.4$ | $69.8 \pm 12.9$ | <0.001 | $68.9 \pm 10.3$ | $80.4 \pm 12.8$ | <0.001 |
| Height (cm) | $146.8 \pm 3.5$ | $158.1 \pm 5.2$ | <0.001 | $156.8 \pm 4.7$ | $170.1 \pm 5.8$ | <0.001 |
| BMI (kg/m$^2$) | $29.1 \pm 5.3$ | $27.9 \pm 5.1$ | <0.001 | $28.1 \pm 4.9$ | $27.7 \pm 4$ | <0.001 |
| BMI 25–29.9 kg/m$^2$ (n(%)) | 7064 (39.1) | 19885 (38.3) | <0.001 | 3183 (46.9) | 21332 (49.4) | <0.001 |
| BMI> 30 kg/m$^2$ (n(%)) | 7087 (39.3) | 15892 (30.6) | <0.001 | 1887 (27.9) | 11096 (25.7) | <0.001 |
| Tobacco smoking (n(%)) | 2295 (12.7) | 10418 (20.1) | <0.001 | 2122 (31.3) | 15634 (36.2) | <0.001 |
| Type II diabetes[a] (95% CI)) | 13.8 (13.3–14.3) | 11.2 (10.9–11.5) | <0.001 | 10.8 (10.2–11.5) | 9.1 (8.8–9.3) | <0.001 |
| High blood pressure[a] (95% CI) | 32.5 (31.9–33.2) | 31.3 (30.9–31.7) | <0.001 | 27.0 (25.9–28) | 29.3 (28.9–29.7) | <0.001 |
| Hypercholesterolemia[a] (95% CI)) | 44.0 (43.3–44.8) | 44.6 (44.1–45.0) | 0.22 | 40.0 (38.9–41.3) | 43.0 (42.3–43.4) | <0.001 |
| Myocardial infarction[a] (95% CI)) | 0.81 (0.68–0.95) | 0.79 (0.71–0.87) | 0.74 | 0.75 (0.52–0.98) | 0.98 (0.89–1.08) | 0.07 |
| Stroke[a] (95% CI) | 1.1 (0.99–1.26) | 0.74 (0.66–0.82) | <0.001 | 0.78 (0.59–0.97) | 0.59 (0.52–0.67) | 0.07 |

Data are presented as mean $\pm$ standard deviation otherwise it is specified. [a]Adjusted for age, tobacco smoking and region.

and history of stroke 0.7% ($n = 892$). The characteristics of the participants were similar in each region (Central and Northern Mexico). However, the prevalence of obesity, hypertension, type II diabetes and hypercholesterolemia was higher in the Northern region in spite of younger age and lower prevalence of tobacco smoking.

The comparison between cases with short or normal stature is shown in Table 1. Almost a fifth of the study subjects had short stature ($n = 24\,854$ (20.7%)). This was most prevalent in women (25.8 *vs* 13.6%, $P < 0.001$) and in subjects sampled in Central México. Short stature subjects were older, had a lower level of education and had a lower prevalence of tobacco smoking. As expected, obesity was more prevalent in this group, despite a lower body weight. Short stature subjects had a higher prevalence of type 2 diabetes and arterial hypertension even after adjusting for confounding factors (age, region and tobacco smoking). Additionally, there was a higher prevalence of stroke in short stature women and of hypercholesterolemia in short stature men. Thus, these data strongly suggest that short stature is independently associated with several cardiovascular risk factors.

To explore the relationship between BMI and obesity-associated co-morbidities in subjects with short and 'normal' stature, we calculated the adjusted prevalence of high blood pressure, diabetes and hypercholesterolemia for every unit of BMI (Figure 1). In both genders, the prevalence of every co-morbidity increased along with the BMI. However, the relationship between BMI and these co-morbidities differed according to the co-morbidity. For example, a near linear relation was evident for hypertension. The diabetes curve had a linear shape, but different from the hypertension curve; in women, the highest prevalence was found at the lower BMI's. In contrast, the hypercholesterolemia curve showed a rapid rise between BMI's 20 and 25 kg/m$^2$, followed

by a plateau. These curves were different in the short stature subjects. As shown in Figure 1, short stature subjects had a significantly higher prevalence of hypertension and diabetes at the majority of the BMI thresholds. For hypertension, even though the curves are parallel, the prevalence was always higher in the short stature subjects. In contrast, for diabetes, the pattern of the curve was completely different in the short stature subjects compared to either the normal-sized subjects or the population as a whole. Thus, the presence of short stature modifies the relation between BMI and obesity-related co-morbidities.

The usefulness of BMI as a screening tool for the detection of obesity-associated co-morbidities among cases with short or normal stature was assessed. First, multiple logistic models were constructed to estimate the odds ratio for having each one of the studied co-morbidities associated with overweight and obesity in both groups, taking into account possible confounders (ie age, region and smoking). As shown in Table 2, both overweight and obesity had lower odds ratios for the presence of each co-morbidity in the short stature group. Thus, there is a lower likelihood of detecting obesity-related co-morbidities using BMI as a screening tool in short stature subjects. Secondly, we calculate the sensitivity, the specificity and the likelihood ratios for several BMI thresholds in subjects with short and normal stature. ROC curves were constructed to identify the BMI threshold, which diagnosed every co-morbidity. The area under the curve was used as an index of the proficiency of the BMI as a diagnostic tool. As shown in Table 3, the BMI value with the best diagnostic proficiency ranged from 27 to 29 kg/m$^2$; it varied depending on the co-morbidity being studied and the gender. The sensitivity and specificity of the BMI values used in clinical practice (23, 25 and 30 kg/m$^2$) are also shown. The sensitivity and the specificity were similar in subjects with or without short stature at BMI values of 23 and 25 kg/m$^2$; this
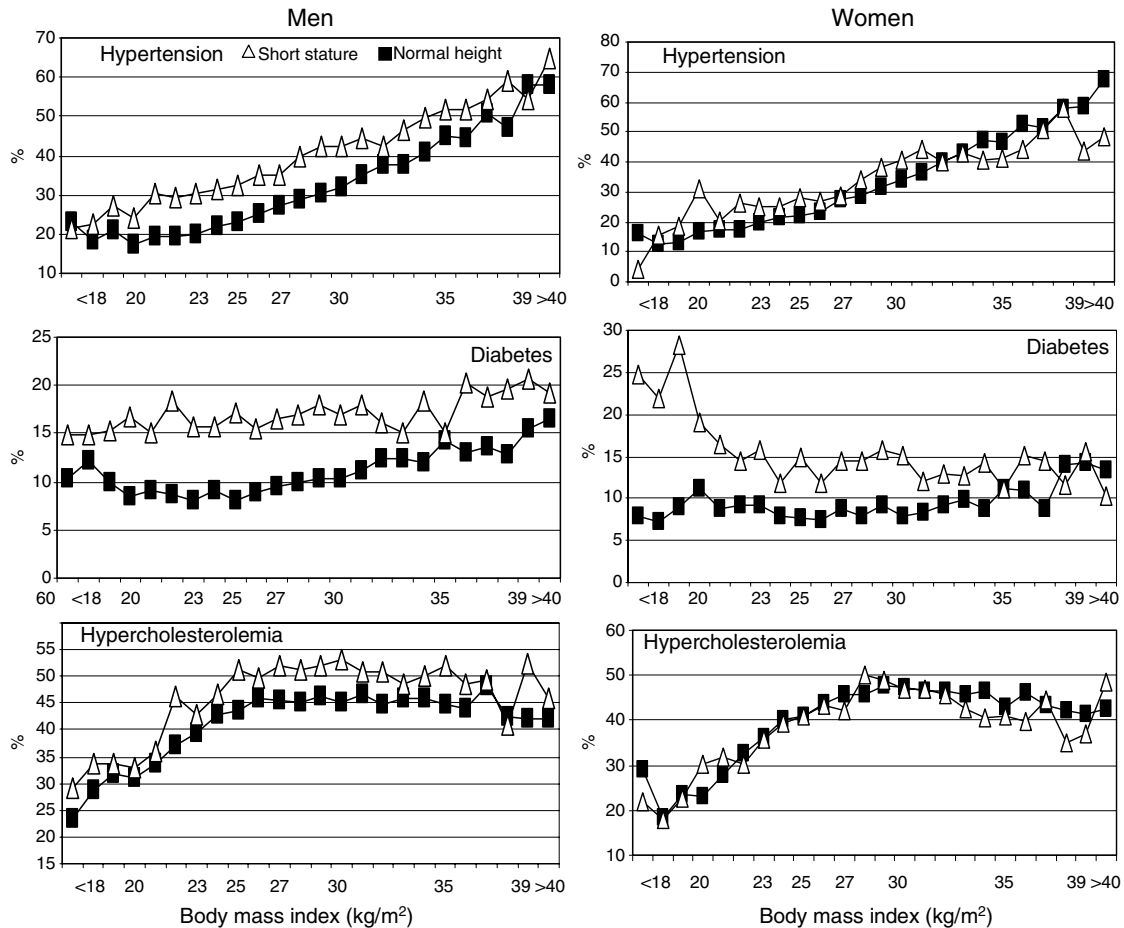
**Figure 1** Prevalence of arterial hypertension, type II diabetes and hypercholesterolemia in men and women with short stature. Data of the whole population, subjects with normal height or short stature, are presented separately. Prevalence is adjusted for age, region (north or center) and smoking. Every point represents a BMI unit (20–20.99, 21.0–21.99 kg/m$^2$, etc). Every point includes the results of 150 or more subjects.

observation suggests that lowering the BMI threshold does not increase the likelihood of detecting co-morbidities. This statement is further supported by the results of the likelihood ratio, an index less likely to be influenced by the prevalence of the disorder. As shown in Figure 2, the likelihood ratio of having diabetes or hypertension increased at the same BMI values in the two groups. Thus, decreasing the BMI thresholds in short stature subjects will not increase the probability of detecting cases with obesity-related co-morbidities in this group. Finally, the BMI is a less accurate screening tool in subjects with short stature. The likelihood ratios for finding cases with either diabetes or arterial hypertension were significantly lower in both men and women with short stature (Figure 2). In accordance, the area under the ROC curve was significantly smaller in this subset of the population. As shown in Table 4, the areas under the ROC curves constructed for every co-morbidity were consistently smaller in men and women with short stature. The same trend was observed when the population was stratified by age groups (above or below age 40 y).

## Discussion

The definition of obesity is proposed to be population specific. In countries with non-Caucasian populations, a higher than expected prevalence of co-morbidities is observed at BMI values considered normal for Caucasians.[14] This discrepancy has been described in populations with a high prevalence of short stature. Our results show that short stature modifies the relationship between BMI and the prevalence of obesity-associated co-morbidities. The frequency of the abnormalities is higher at all BMI thresholds and the prevalence increases in direct relation with the BMI, but at a lower rate than cases of normal stature. As a consequence, the use of BMI, as a tool for finding cases with obesity-associated co-morbidities, is not as accurate in the presence of short stature. This problem is not solved by decreasing the BMI threshold used to define overweight, as has been proposed for Asian populations. Thus, between ethnic groups, short stature could explain, to some extent, the variation in the ability of BMI to detect the obesity-related complications.

**Table 2** Logistic regression models to determine the ability of overweight and obesity to detect obesity related co-morbidities in subjects with short and normal stature

| | Men | | | Women | | |
|---|---|---|---|---|---|---|
| | Total | Short stature | Normal stature | Total | Short stature | Normal stature |
| *Diabetes* | | | | | | |
| P-value of the model | <0.001 | | | <0.001 | | |
| $R^2$ (Cox and Snell) | 0.075 | | | 0.075 | | |
| $\chi^2$ for short stature | 10.445 | | | 46.55 | | |
| (P-value) | (<0.001) | | | (<0.001) | | |
| Odds ratio | 1.8 | 0.95 | 1.2 | 1.552 | 1.2 | 1.72 |
| BMI 25–29.9 kg/m$^2$ (95% CI) | (1.07–1.29) | (0.782–1.16) | (1.11–1.36) | (1.45–1.65) | (1.07–1.3) | (1.58–1.86) |
| Odds ratio | 1.6 | 0.96 | 1.21 | 1.228 | 1.08 | 1.41 |
| BMI ≥ 30 kg/m$^2$ (95% CI) | (1.07–1.25) | (0.812–1.14) | (1.11-.31) | (1.22–1.36) | (0.99–1.19) | (1.31–1.57) |
| | | | | | | |
| *Hypertension* | | | | | | |
| P-value of the model | <0.001 | | | <0.001 | | |
| $R^2$ (Cox and Snell) | 0.117 | | | 0.179 | | |
| $\chi^2$ for short stature | 19.626 | | | 6.4 | | |
| (P-value) | (<0.001) | | | (<0.001) | | |
| Odds ratio | 3.22 | 2.6 | 3.21 | 3.42 | 2.53 | 3.83 |
| BMI 25–29.9 kg/m$^2$ (95% CI) | (3.03–3.42) | (2.2–3.03) | (2.92–3.51) | (3.26–3.57) | (2.33–2.76) | (3.6–4.08) |
| Odds ratio | 1.97 | 1.75 | 1.93 | 1.83 | 1.65 | 1.93 |
| BMI ≥ 30 kg/m$^2$ (95% CI) | (1.88–2.07) | (1.545–1.986) | (1.81–2.06) | (1.76–1.91) | (1.54–1.76) | (1.84–2.01) |
| | | | | | | |
| *Hypercholesterolemia* | | | | | | |
| P-value of the model | <0.001 | | | <0.001 | | |
| $R^2$ (Cox and Snell) | 0.022 | | | 0.066 | | |
| $\chi^2$ for short stature | 16.56 | | | 4.837 | | |
| (P-value) | (<0.001) | | | (0.028) | | |
| Odds ratio | 1.64 | 1.58 | 1.64 | 1.42 | 1.39 | 1.4 |
| BMI 25–29.9 kg/m$^2$ (95% CI) | (1.55–1.72) | (1.38–1.81) | (1.55–1.74) | (1.36–1.48) | (1.29–1.51) | (1.34–1.47) |
| Odds ratio | 1.06 | 1.02 | 1.07 | 0.98 | 0.957 | 0.99 |
| BMI ≥ 30 kg/m$^2$ (95% CI) | (1.02–1.11) | (0.91–1.14) | (1.02–1.112) | (0.95–1.02) | (0.89–1.02) | (0.95–1.04) |

The obesity-related co-morbidity was included as dependent variable, BMI strata (<25, 25–29.9 and ≥30 kg/m$^2$) as factors and short stature, age, region and smoking as covariates.

Our data show that the power of BMI to detect subjects with obesity-associated co-morbidities is significantly lower in cases with short stature compared to those without it.

Among short stature subjects, the power of BMI to detect some conditions (ie diabetes) was low enough to doubt its clinical usefulness (Table 4). As shown in Figure 2, the likelihood ratio (LLR), an index that measures the probability of finding abnormal cases using the test under study, was always lower in short stature subjects. We assessed whether lowering the BMI thresholds for the definition of either overweight or obesity could overcome this limitation; selecting a lower cutoff value (ie 23 kg/m$^2$) did not increase the probability of finding abnormal cases because the likelihood ratio increased at the same BMI values in cases with or without short stature (Figure 2). Thus, our data clearly show that the risk of diabetes and high blood pressure in short stature subjects could not be adequately assessed using BMI. Future studies should be designed to find new indicators of increased metabolic risk in short stature subjects. Meanwhile, the limitations of BMI in these cases should be recognized; conclusions based on BMI must be interpreted with caution.

Short stature may be an independent risk factor for fatal and nonfatal coronary heart disease (CHD), even in Caucasian groups.[15] Forsén[16] demonstrated, in the Finish component of the Seven Countries Study, that, even after adjusting for confounding variables, there is a 19% increased risk of coronary events for a 10 cm decrease in height. In accordance, our results show that short stature subjects have an increased prevalence of diabetes and high blood pressure compared to women and men with a 'normal' height, even after adjusting for confounding variables. Possible reasons for the increased prevalence of diabetes and high blood pressure in short stature individuals include malnourishment early in life and genetic factors. Many authors have shown that exposure to malnutrition *in utero* or during childhood is associated with short stature and increased risk of suffering CHD, type II diabetes and the metabolic syndrome later in adult life.[17–22] These cases have decreased insulin sensitivity and an abnormal body fat distribution.[23] Hence, higher levels of abdominal adipose tissue are more detrimental in this group than in BMI-paired subjects without excessive abdominal adiposity.[24] This phenomenon can be demonstrated even in subjects with BMI lower than 25 kg/m$^2$. Thus, short stature due to malnourishment early on in life may lead to significant changes in fat metabolism that are impossible to evaluate with BMI. Additionally, short stature subjects have a significantly higher amount of body

**Table 3** Diagnostic performance of several BMI cutoff points for the detection of several obesity-related co-morbidities

| Women | Normal stature | | | | Short stature | | | |
|---|---|---|---|---|---|---|---|---|
| BMI (kg/m$^2$) | 23 | 25 | 30 | Optimal BMI$^a$ (BMI value) | 23 | 25 | 30 | Optimal BMI$^a$ (BMI value) |
| **High blood pressure** | | | | | | | | |
| Sensitivity (%) | 92.8 | 82.8 | 44.4 | 53.3 (29) | 93.5 | 85.1 | 46.8 | 55.4 (29) |
| Specificity (%) | 18.6 | 36.4 | 76.2 | 67.9 (29) | 12.2 | 25.8 | 68.1 | 60.7 (29) |
| Likelihood ratio (%, 95% CI) | 1.12 | 1.30 | 1.86 | 1.66 | 1.06 | 1.14 | 1.46 | 1.41 (29) |
| | (1.02–1.21) | (1.2–1.39) | (1.74–1.97) | (29) (1.55–1.76) | (0.91–1.20) | (0.98–1.29) | (1.28–1.63) | (1.23–1.58) |
| **Diabetes** | | | | | | | | |
| Sensitivity (%) | 90.5 | 79.4 | 40.7 | 56.8 (28) | 90.9 | 81.2 | 40.6 | 49.2 (29) |
| Specificity (%) | 16.0 | 32.0 | 71.5 | 57.5 (28) | 11.1 | 22.0 | 62.8 | 55.0 (29) |
| Likelihood ratio (%, 95% CI) | 1.08 | 1.16 | 1.42 | 1.33 (28) | 1.01 | 1.04 | 1.09 | 1.09 (29) |
| | (0.99–1.16) | (1.06–1.25) | (1.31–1.52) | (1.23–1.42) | (0.86–1.15) | (0.89–1.18) | (0.93–1.24) | (0.93–1.24) |
| **Hypercholesterolemia** | | | | | | | | |
| Sensitivity (%) | 89.6 | 75.3 | 32.7 | 59.7 (27) | 92.3 | 82.1 | 39.2 | 56.5 (28) |
| Specificity (%) | 19.0 | 35.5 | 72.6 | 49.3 (27) | 12.1 | 24.8 | 63.5 | 48.3 (28) |
| Likelihood ratio (%, 95% CI) | 1.10 | 1.16 | 1.19 | 1.18 (27) | 1.05 | 1.09 | 1.07 | 1.09 (28) |
| | (1.01–1.18) | (1.06–1.25) | (1.09–1.28) | (1.08–1.27) | (0.9–1.19) | (0.93–1.24) | (0.91–1.22) | (0.93–1.24) |
| **Men** | Normal stature | | | | Short stature | | | |
| BMI (kg/m$^2$) | 23 | 25 | 30 | Optimal BMI$^a$ (BMI value) | 23 | 25 | 30 | Optimal BMI$^a$ (BMI value) |
| **High blood pressure** | | | | | | | | |
| Sensitivity (%) | 95.2 | 85.0 | 37.5 | 57.4 (28) | 93.1 | 81.3 | 34.7 | 56.0 (28) |
| Specificity (%) | 11.7 | 28.2 | 79.9 | 61.9 (28) | 11.5 | 28.2 | 77.9 | 60.1 (28) |
| Likelihood ratio (%95% CI) | 1.07 | 1.18 | 1.86 | 1.51 (28) | 1.05 | 1.13 | 1.57 | 1.40 (28) |
| | (0.97–1.16) | (1.07–1.28) | (1.73–1.98) | (1.39–1.62) | (0.8–1.29) | (0.87–1.38) | (1.27–1.86) | (1.12–1.67) |
| **Diabetes** | | | | | | | | |
| Sensitivity (%) | 91.2 | 78.1 | 29.5 | 48.3 (28) | 88.3 | 74.0 | 25.4 | 56.9 (27) |
| Specificity (%) | 10.0 | 24.7 | 75.4 | 57.1 (28) | 9.7 | 25.0 | 73.7 | 44.9 (27) |
| Likelihood ratio (%, 95% CI) | 1.01 | 1.03 | 1.19 | 1.13 (28) | 0.97 | 0.99 | 0.96 | 1.03 (27) |
| | (0.91–1.1) | (0.93–1.12) | (1.08–1.29) | (1.03–1.22) | (0.73–1.2) | (0.75–1.22) | (0.72–1.19) | (0.78–1.27) |
| **Hypercholesterolemia** | | | | | | | | |
| Sensitivity (%) | 93.8 | 80.7 | 27.4 | 48.2 (28) | 93.2 | 79.7 | 27.8 | 50.1 (28) |
| Specificity (%) | 12.4 | 28.3 | 76.8 | 59.7 (28) | 12.3 | 28.7 | 75.0 | 58.5 (28) |
| Likelihood ratio (%, 95% CI) | 1.07 | 1.12 | 1.18 | 1.19 (28) | 1.06 | 1.12 | 1.11 | 1.21 (28) |
| | (0.97–1.16) | (1.02–1.21) | (1.07–1.28) | (1.08–1.29) | (0.81–1.3) | (0.86–1.37) | (0.86–1.35) | (0.94–1.47) |

$^a$The BMI threshold closest to the left corner of the ROC curve.
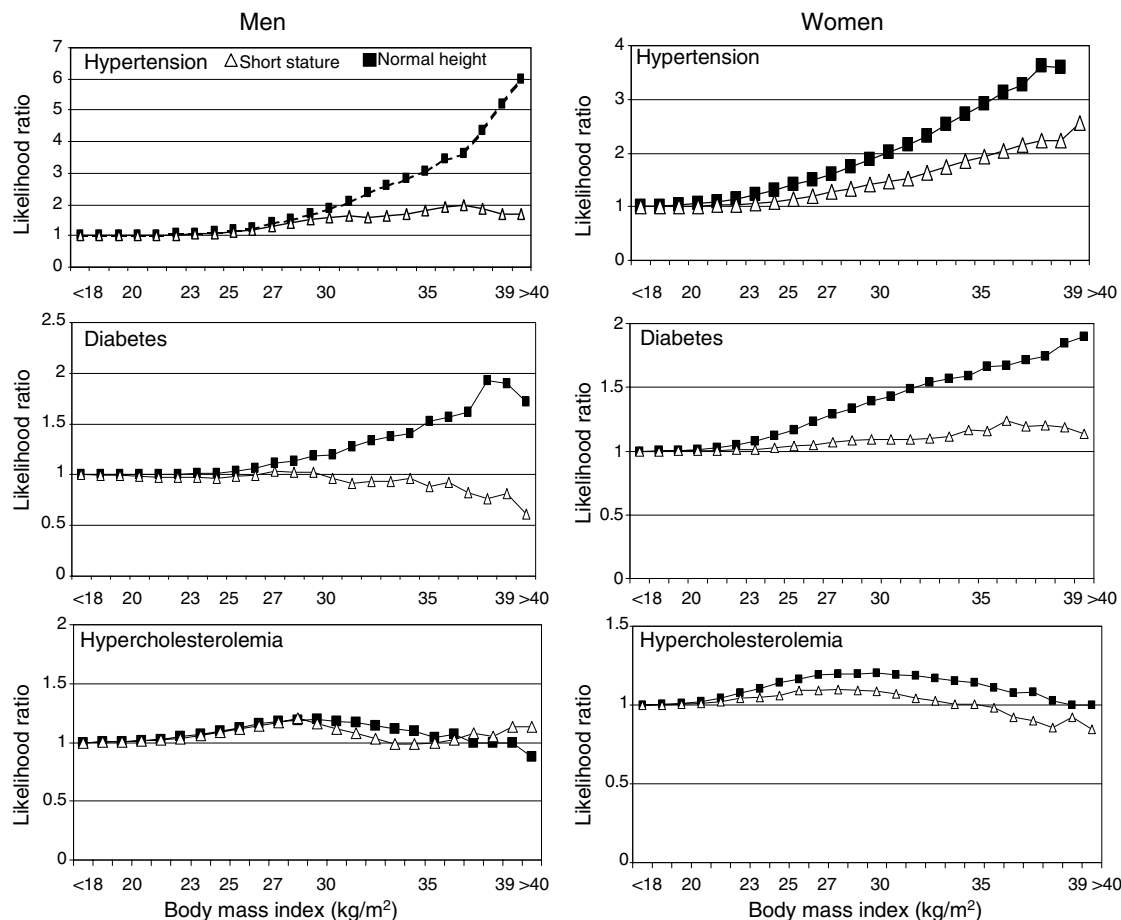
**Figure 2** Likelihood ratio of several BMI thresholds for the detection of arterial hypertension, type II diabetes and hypercholesterolemia in men and women with short stature. Data of the whole population, subjects with normal height or short stature, are presented separately. Every point represents a BMI unit (20–20.99, 21.0–21.99 kg/m², etc). Every point includes the results of 150 or more subjects.

**Table 4** Area under the ROC curve constructed for the body mass index and obesity-related co-morbidities

| | Women Area under the curve (95% confidence interval) | | Men Area under the curve (95% confidence interval) | |
|---|---|---|---|---|
| | Short stature | Normal stature | Short stature | Normal stature |
| *Hypertension* | | | | |
| Age 30–80 y | 0.610* (0.601–0.618) | 0.659 (0.654–0.664) | 0.598* (0.584–0.613) | 0.637 (0.631–0.643) |
| Age ≥40 y | 0.587* (0.578–0.597) | 0.621 (0.614–0.627) | 0.577* (0.559–0.594) | 0.605 (0.597–0.613) |
| Age <40 y | 0.645 (0.622–0.668) | 0.673 (0.663–0.684) | 0.626 (0.593–0.658) | 0.661 (0.652–0.671) |
| | | | | |
| *Diabetes* | | | | |
| Age 30–80 y | 0.527* (0.516–0.538) | 0.595 (0.587–0.603) | 0.495* (0.475–0.515) | 0.539 (0.529–0.549) |
| Age ≥40 y | 0.498* (0.486–0.510) | 0.544 (0.535–0.553) | 0.465* (0.444–0.486) | 0.501 (0.490–0.512) |
| Age <40 y | 0.603 (0.559–0.646) | 0.660 (0.638–0.682) | 0.524 (0.441–0.608) | 0.582 (0.554–0.611) |
| | | | | |
| *Cholesterol >200 mg/dl* | | | | |
| Age 30–80 y | 0.532* (0.524–0.541) | 0.564 (0.559–0.569) | 0.553* (0.539–0.566) | 0.560 (0.554–0.565) |
| Age ≥40 y | 0.499* (0.489–0.509) | 0.505 (0.498–0.512) | 0.513 (0.495–0.530) | 0.510 (0.503–0.518) |
| Age <40 y | 0.569 (0.552–0.587) | 0.585 (0.577–0.593) | 0.608 (0.585–0.632) | 0.591 (0.583–0.599) |

*P<0.05 *vs* normal stature subjects.

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fat compared to controls of the same ethnic group matched for BMI, age and gender.[9] These facts may explain why the BMI is a less-sensitive tool for detecting cases with obesity-associated co-morbidities in short stature subjects.

Our results help to explain why the higher prevalence of obesity-associated complications in Oriental populations at BMI's considered normal in Caucasian groups.[25,26] Short stature subjects represent a significant proportion of the population in Oriental and Latin American countries (20% in México). We analyzed the impact of short stature on the odds ratio of suffering a co-morbidity in our study sample. For example, as shown in Figure 1, the odds ratio of having high blood pressure increases after a BMI value of $23 \, kg/m^2$ (odds ratio 1.35; CI 1.29–1.41) in the whole population. However, if short stature subjects are excluded, the increased risk disappears (odds ratio 1.09; CI 0.89–1.21). The same phenomenon was observed for the risk of having high cholesterol levels. The higher percentage of short stature individuals in Oriental and Mexican populations may be one of the reasons for the discrepancy in the performance of BMI; hence, this set of the population should be considered separately.

Strengths and limitations of our report must be recognized. The large number of study subjects provides sufficient short stature cases to enable us to evaluate differences by a single unit of BMI. In contrast, the cross-sectional nature of the survey and the absence of measurements of other relevant obesity-associated co-morbidities (ie low HDL cholesterol and hypertriglyceridemia) limit our conclusions. Also, we do not have information for potentially relevant confounders (ie the waist circumference and alcohol intake). The prevalence of diabetes may be underestimated due to limitations in the methods used for diagnosis.

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