Short stature and hypertension in the city of Rio de Janeiro, Brazil

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Abstract

Objective: Stature is a powerful indicator of poor nutrition early in life in nations where undernutrition is a public health problem. Hypertension in adults has been associated with factors present early in life such as low birth weight. We tested the hypothesis that short stature is associated with hypertension among adults.
Design and setting: A household survey of representative adults in Rio de Janeiro city, Brazil was carried out in 1996.
Subjects: Blood pressure and anthropometric measures were collected from 2802 adults in their own households. Prevalence estimates and modelling incorporated the sample design and weights.
Results: Age-adjusted prevalence of hypertension for both sexes was lower in the third quartile of stature distribution. In women, but not in men, the odds ratio comparing the first quartile of stature with the fourth quartile was statistically significant with an odds ratio of 1.68 (95% CI 1.02–2.76). Adjusting for known risk factors for hypertension such as age, income, smoking, sodium and alcohol intake and race, the association among women, comparing the first with the fourth quartile for stature, was 1.84 (95% CI 1.03–3.30). With further adjustment for residual of weight on height the ratio reduced to 1.76 (95% CI 0.97–3.19, P value of trend = 0.03). Systolic blood pressure showed a U-shaped association with quartiles of stature, mainly among women, with a β-coefficient significantly lower at the third quartile.
Conclusions: This association of stature with hypertension supports the theory of an important ontogenetic dependence of adult blood pressure, at least among women.

Keywords
Stature
Race
Brazilian adults
Hypertension
Obesity
Population
Prevalence

Prenatal and postnatal factors have been implicated in the development of hypertension among adolescents[1] and adults[2]. A negative correlation between birth weight and subsequent blood pressure has been reported in most studies[1–4], suggesting that intrauterine growth retardation increases the risk of hypertension; however, other studies have reported no association[5,6].

Height is an important indicator of childhood undernutrition, mainly during the first 2 years of life[7–9]. Low maternal stature, poor socioeconomic status, inadequate diet, high diarrhoea rates and low anthropometric scores at 3 and 6 months of age were all determinants of poor growth among children from Guatemala[10].

Short stature has been associated with cardiovascular disease in well-controlled studies such as the Physicians' Health Study[11], the Nurses' Health Study[12], among women in the Framingham study[13], and among men in two New England communities[14]. In these populations height was found to be a significant predictor of cardiovascular disease even after adjustment for important risk factors. On the other hand, a study based on a sample of the US population found no association between stature and cardiovascular disease[15].

A negative association between stature and hypertension was found among 40–64-year-old British men[16], whereas positive associations have been reported in most other studies[17,18]. Theoretically, a positive association between height and blood pressure could be expected from the Poiseuille's law that states that resistance to flow in a vessel is directly proportional to the length of the vessel. In the INTERSALT study height was an independent contributor to blood pressure[19] and corrections for height in measured blood pressure have been suggested[20].

A possible explanation for these discrepancies is that an inverse association between stature and blood pressure may be evident only in populations where shortness is an important marker for early undernutrition.

Since national trends suggest that nutrition is a major determinant of stature in Brazil[21] we examined the association between stature and prevalence of hypertension among adult Brazilians.

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Methods

A survey – Pesquisa Nutrição e Saúde (PNS) – of a representative sample of adults in Rio de Janeiro was carried out in 1996, using two-stage probability sampling. In the first stage 60 primary sampling units were selected from all regions of the city. In the second stage, 34 households were sampled from the primary sampling units, totalling 2040 households. The probability of selection of primary sampling units and households was proportional to the population size. The non-response rate was 11.2%, and pregnant women were excluded. Of the 3224 adults aged 20–65 years interviewed, 422 (13%) had no blood pressure measurements (198 because of problems with the electronic device and 224 were not available for anthropometric and blood pressure measurements after three visits to the household).

Height, weight and blood pressure were collected in the households, with the participants wearing light clothes and no shoes. Stature was measured using a platform with an attached measuring bar with a precision to 0.1 cm. Three sitting blood pressures were taken on the right arm, with an electronic device after 10 min of rest. Blood pressure measurements and cut-offs were based on the sixth report of the USA Joint National Committee[22]. Blood pressure was estimated by averaging the second and third readings. Hypertension was defined as a systolic blood pressure equal to or greater than 140 mmHg, a diastolic pressure equal to or greater than 90 mmHg, or if the subject was taking antihypertensive medication.

Per capita income was calculated as the preceding month's family income divided by the number of those living on that budget. Race was classified based on skin colour and divided into white, mulatto and black. Smoking was recorded as previous smoker, current smoker and no smoker. Alcohol intakes were measured based on a food frequency questionnaire, developed for the Brazilian population, with 78 items and usual portions, which included beer, wine and spirits[23]. Salt intake was based on the family amount of salt used per month divided by the number of family members.

Prevalence estimates incorporated the sample design and weights using the statistical software SUDAAN[24]. Multivariate analysis, through logistic regression and linear regression, included variables considered to be potential confounders: age, race, per capita income, smoking, consumption of alcohol and salt, and body weight.

Adjustment for weight used the residual of weight on height, instead of weight, because in a regression model the interpretation of weight adjusted for height is conceptually unclear[25]. A residual of weight – which is the observed value minus the expected value based on age, sex and height – was calculated from a linear regression that included height and height squared.

Stratified analysis used as a cut-off for short stature of the 25th percentile for both sexes according to the distribution of the study population or quartiles. In multivariate analyses stature was classified in quartiles and treated as indicator variables and also analysed as a continuous variable with a quadratic association tested for significance. In order to completely account for the confounding effect of age, this variable was entered in the logistic model as a continuous variable. Linear regression with both systolic and diastolic blood pressures as dependent variables excluded those individuals on medication for hypertension.

Results

Age-adjusted prevalence of hypertension for both sexes was lower in the third quartile of stature distribution. In women, but not in men, the odds ratio comparing the first quartile of stature with the fourth quartile was statistically significant with an odds ratio of 1.68 (95% CI 1.02–2.76) (Table 1).

Systolic and diastolic blood pressure increased with age, and was higher among men than among women (data not shown). An important cohort effect on stature

Table 1 Age-adjusted* prevalence (%) and odds ratios of hypertension according to quartiles of stature, in men and women. Data from the Health and Nutrition Survey, Rio de Janeiro, 1996

	Men				Women			
Stature quartiles	Median height (cm)	Sample size	Prevalence of hypertension (%)	Odds ratio (95% CI)	Median height (cm)	Sample size	Prevalence of hypertension (%)	Odds ratio (95% CI)
1st	162	256	10.7	1.05 (0.58–1.88)	151	336	14.1	1.68 (1.02–2.76)
2nd	168	260	10.3	0.92 (0.50–1.68)	156	328	12.5	1.50 (0.89–2.52)
3rd	172	229	8.1	0.67 (0.33–1.37)	160	330	7.9	0.82 (0.45–1.47)
4th	178	316	10.6	1.0	164	407	8.6	1.0

* Age adjustment through direct method by nine 5-year categories.

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Table 2 Age, age-adjusted* mean of BMI, and age-adjusted risk factors for hypertension according to stature, in men and women

	Men						Women					
	Short†			Normal			Short†			Normal		
Variables	Mean	%	SE	Mean	%	SE	Mean	%	SE	Mean	%	SE
Age (years)	43.0		0.72	38.1		0.40	44.1		0.67	37.9		0.34
BMI (kg m^{-2})	24.9		0.28	24.4		0.14	25.7		0.35	24.4		0.14
Alcohol (g day^{-1})	15.5		2.04	15.8		1.11	4.6		0.98	6.1		0.69
Salt (g day^{-1})‡	12.5		0.56	11.6		0.26	11.5		0.39	12.0		0.24
Income (US$)	265		20	459		23	281		21	390		13
Race (%)												
White		63.4	2.9		67.6	1.6		65.9	2.8		63.6	1.4
Mulatto		24.5	2.8		18.8	1.3		22.6	2.4		22.6	1.2
Black		12.1	1.9		13.6	1.2		11.5	1.9		13.8	0.98
Current smokers (%)		32.9	3.3		32.1	1.8		29.3	2.9		26.2	1.4

* Age adjustment through direct method by nine 5-year categories.
†Short stature (< 25th percentile) for men is <165.5 cm and for women is <154.5 cm.
‡ Family amount of salt used per month divided by the number of family members.

in Brazil was also observed, with the younger cohort (20–30 years old in 1996) being approximately 5 cm taller than the older cohort (50–60 years).

Short individuals were usually older, had a higher body mass index (BMI) and, among men, were more likely to be mulatto (Table 2). Thus, complete adjustment for age and ethnicity was a fundamental step in the analysis. Per capita income was much lower among short individuals and was also treated as a confounding variable rather than as an indicator of early nutritional status; however, multivariate analyses were performed adjusted and unadjusted for this variable.

The prevalence of hypertension was greater among short women in most of the stratified analyses, however stratification by BMI revealed that the association between stature and hypertension was present only among lean women (BMI $\leqslant 25\,kg\,m^{-2}$) (Table 3). A possible modification effect of BMI was tested by including a multiplicative term (stature × BMI) in multivariate analysis. This interaction was not statistically significant ($P > 0.05$), neither were interactions for salt intake and smoking.

In multivariate analyses the age- and race-adjusted odds ratio of hypertension associated with stature was 1.85 (95% CI 1.10–3.11), comparing the first with the

Table 3 Age-adjusted prevalence (%) and prevalence ratio of hypertension according to stature and risk factors for hypertension among women. Data from the Health and Nutrition Survey, Rio de Janeiro, 1996

	Prevalence (%)		Prevalence ratio (short/normal) (95% CI)
	Short (n=316)*	Normal (n=1003)*	
Income (US$ year^{-1})			
< 2400	14.6	12.9	1.13 (0.40–3.13)
⩾ 2400	14.5	8.0	1.81 (0.91–3.59)
BMI (kg m^{-2})			
⩽25	11.3	5.0	2.26 (1.02–5.24)
>25	17.3	15.3	1.13 (0.41–3.05)
Race			
White	13.2	7.4	1.78 (1.04–3.03)
Mulatto	19.6	13.7	1.43 (0.72–2.71)
Black	18.7	14.4	1.30 (0.54–3.30)
Alcohol intake (g day^{-1})			
None	15.7	12.7	1.24 (0.48–3.16)
<4.0	11.7	3.9	3.00 (1.03–7.87)
⩾4.0	7.8	4.5	1.73 (0.35–15.9)
Salt intake (g day^{-1})†			
<10.0	13.2	11.8	1.12 (0.60–2.05)
⩾10.0	15.3	9.3	1.64 (1.02–2.84)
Smoking			
Never	12.7	10.7	1.19 (0.42–3.34)
Previous	14.4	11.3	1.27 (0.35–4.53)
Current	18.8	8.1	2.32 (1.01–5.29)

* Short stature (<25th percentile) for women is <154.5 cm and normal stature (⩾25th percentile) is ⩾154.5 cm.
† Family amount of salt used per month divided by the number of family members.

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Table 4 Odds ratios and 95% confidence intervals (in brackets) of multivariate logistic regression analyses for hypertension among women, aged 20–65 years. Data from the Health and Nutrition Survey, Rio de Janeiro, 1996

Adjusted for:	Stature quartiles (cm)				*P* test of trend
	<154.5	154.5–159.0	160.0–163.0	>163.0	
Age*	1.80 (1.06–3.04)	1.69 (0.91–2.76)	0.87 (0.47–1.60)	1	0.01
Age and race	1.85 (1.10–3.11)	1.79 (1.04–3.07)	0.86 (0.48–1.56)	1	0.005
Age, race, smoking, income, and salt and alcohol intake	1.84 (1.03–3.30)	1.93 (1.05–3.53)	0.85 (0.45–1.65)	1	0.009
Further adjusted for weight†, in addition to previous model	1.76 (0.97–3.19)	1.76 (0.95–3.29)	0.84 (0.43–1.66)	1	0.03

* Age adjusted by including age in years and age × age in the model.
† Residual of weight on height (kg).

Table 5 Age-adjusted* mean systolic and diastolic blood pressures (BP), and β-coefficients for stature from linear models† and associated *P* values. Data from the Health and Nutrition Survey, Rio de Janeiro, 1996

	Stature quartiles				*P* value of trend
	1st	2nd	3rd	4th	
Men					
Systolic mean BP	121.3	121.4	119.2	123.2	
β-coefficient	–2.43	–2.10	–2.92	–	
P value	0.08	0.11	0.03		0.10
Diastolic mean BP	79.0	79.2	77.4	78.1	
β-coefficient	–0.57	0.66	–1.01	–	
P value	0.62	0.54	0.34		0.50
Women					
Systolic mean BP	113.3	108.7	115.0	116.3	
β-coefficient	–0.89	–0.70	–2.47	–	
P value	0.52	0.57	0.03		0.18
Diastolic mean BP	75.8	72.1	76.2	76.3	
β-coefficient	0.43	0.23	–0.12	–	
P value	0.68	0.81	0.89		0.95

* Age adjustment through direct method by nine 5-year categories.
† Adjusted for age, race, income, smoking, salt and alcohol intake, and weight.

fourth quartile for stature. With additional adjustment for income, smoking, and salt and alcohol intake there was almost no change in the odds ratio, and further adjustment for residual of weight reduced the odds ratio to 1.76 (95% CI 0.97–3.19, *P* value of trend = 0.03) (Table 4).

Linear regression analyses for blood pressure were done for individuals not taking medication for hypertension (1006 men and 1287 women), with models adjusted for age, race, income, smoking, salt and alcohol intake, and weight. Systolic blood pressure showed a significant U-shaped association, mainly among women. No association was found with diastolic blood pressure (Table 5).

The individuals with missing blood pressure measurements but with anthropometric measurements, tended to have almost the same age and income, but were shorter (27% were of short stature), than those without missing blood pressure measurements (23% were of short stature). A sensitivity analysis was conducted assuming that all 198 missing values were of normal blood pressure. Association of stature with blood pressure did not change in this analysis.

Discussion

Associations between early growth and cardiovascular disease, first shown by Barker, have been found in many studies[2,26,27]. Possible bias in these studies is that the adverse environment *in utero* and during infancy may persist into adult life and later this condition may produce the effects attributed to programming[26].

An advantage of using stature as a marker compared to studies that used reported birth weight is that stature can be measured more accurately. In this study, after control for biological and environmental risk factors, we found an inverse association between stature and prevalence of hypertension among women and no association among men. In the Framingham Heart Study short stature was associated with myocardial infarction in women but not in men[13]. Baseline characteristics in the Framingham study also showed

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that the age-adjusted prevalence of hypertension among women decreased from 16% in the first quartile of height to 12.7% in the third quartile, and increased to 14% in the fourth quartile of height, whereas there was not such a relationship among men[13]. In a study carried out in Paris among 18 336 men and 9351 women, short women but not short men had an increased systolic and pulse blood pressure[28].

In this study, a statistically significant U-shaped association between systolic blood pressure and quartiles of stature was found for both sexes, but the association was stronger for women. Higher operative mortality from coronary bypasses among women, compared to men, has been attributed to narrow arteries in women[29]. In addition, Safar and London[28] suggested that gender differences in myocardial infarctions may be related to a greater increase in wave reflection with age observed among women. Short stature has also been related to fasting and 120-min plasma glucose concentrations in females but not in males[30].

In a recent study, short stature was associated with raised concentrations of glucose and insulin after 2 hours of glucose load[27]. Lithell *et al.*[31] reported that thinness at birth raised the insulin concentration after a glucose challenge in middle age and increased the risk of diabetes. Hyperinsulinaemia has also been associated with hypertension[32].

In a representative sample of British adults it was found that height was independently and inversely related to the prevalence of elevated blood pressure[16], but other studies found positive associations between height and blood pressure. In a follow-up study of children in Boston, USA, childhood height had a negligible effect on future blood pressure, but adult height was positively associated with blood pressure[17] and a study of US adolescents also showed a positive association between stature and blood pressure[18].

Stature is both a marker for early nutrition as well as a marker for the mother's stature and mother's nutrition during the pregnancy. The mother's stature is a strong determinant of birth weight[10] and adult stature is related to the size of the mother's uterus. The diet of pregnant women may also affect growth, and a low intake of animal protein in late pregnancy was associated with low placental and birth weight[26]. Thus, an association between adult stature and hypertension may be dependent on the trend of increasing stature and may not be observed in more recent studies of developed countries. Short adult stature may be a marker for starvation *in utero* as well as during infant life. This study only had data on adult stature which did not allow us to identify whether the gestational or infant periods have an influence on adult blood pressure.

In contrast to Stanner *et al.*[27] who suggested that the relationship between BMI and systolic blood pressure in women was significantly stronger among those women with growth retardation, we found an association only among leaner women. However, this interaction found in the stratified analyses, as well as an interaction with age, was highly attenuated and did not reach statistical significance in models that included all variables.

The youngest individuals in our study did not show an association between stature and hypertension (data not shown). A possible explanation for the lack of association in the young group is that the effects caused by intrauterine or early infancy growth retardation will become manifest only with ageing. Supporting this argument, is the finding that an inverse relationship between systolic blood pressure and birth weight becomes amplified with increasing age[4]. The age effect in our data may also be related to a cohort improvement in early nutrition, with a reduction of stunting in recent cohorts. The stature of the adolescent population in Brazil has increased approximately 8 cm in a 15-year period[21]. Our data also show that the older age group was shorter than the younger group. Reduction of stature with ageing in this study can only partially explain the shortness of the older individuals because this reduction used to be smaller than the observed 4 cm in this age range.

Our model including weight as a measure of obesity showed a reduction of the association between hypertension and stature, indicating that the effect of height on blood pressure is partially due to obesity.

The cut-off we used to classify short-stature individuals was the 25th percentile. This corresponds approximately to the 5th percentile of heights for the middle-aged USA population. This 5th percentile is the cut-off for stunting according to Frisancho[33]. Thus, short stature in Brazil is clearly a marker for early nutritional deficiency.

In conclusion, our results showed that short stature, independent of confounding by race and environmental factors, was an important risk factor for hypertension among women. This finding may be used in future studies to identify factors related to the pathogenesis of hypertension related to developmental problems. In addition, stature – an easily collected variable – could be used as a marker for high risk of hypertension in populations of developing countries.

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