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Left Ventricular Mass and Arterial Compliance: Relation to Coronary Heart Disease and its Risk Factors in South Indian Adults

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Structured Abstract

Background—Rates of coronary heart disease (CHD) in India are rising, and are now similar to those in Western countries. The prevalence of conventional CHD risk factors such as hypercholesterolaemia, hypertension, smoking and obesity, tend to be lower in Indian than Western populations, and fail to explain these high rates of disease. Increased left ventricular mass (LV mass) and decreased arterial compliance predict a higher risk of CHD in Western populations, but there are no published data from India. We have measured LV mass and arterial compliance, and examined their relation to CHD and other known risk factors, in men and women living in Mysore, South India.

Methods—We examined 435 men and women born in Mysore during 1934-1953. LV mass was measured by echocardiography and arterial compliance (derived from pulse wave velocity {PWV}) was measured by a non-invasive optical method in three arterial segments.

Results—The mean LV mass was 149g (SD 37) in men and 125g (SD 32) in women. The mean PWV was 4.14m/s in the aorto-radial, 3.28m/s in the aorto-femoral and 13.59m/s in the femoro-popliteal-posterior tibial segments. LV mass and PWV were positively correlated with each other and with systolic and diastolic blood pressures, non-insulin dependant diabetes mellitus, fasting plasma glucose, insulin, proinsulin concentrations and serum triglyceride concentrations ($p<0.05$ for all), independently of age, sex and body size. In addition, LV mass correlated negatively with fasting serum HDL-cholesterol ($p=0.02$). Higher LV mass was associated with an increased risk of CHD ($p=0.05$).

Conclusions—The mean LV mass in this Indian population is low compared with Western populations, though as in the West, increased LV mass is associated with an increased risk of

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CHD. Greater LV mass and reduced arterial compliance are associated with higher levels of many known CHD risk factors especially with those which form the Insulin Resistance Syndrome.

Keywords

left ventricular mass; arterial compliance; coronary heart disease; India

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Introduction

Coronary heart disease (CHD) rates in India are rising (1) and projected statistics show that CHD will be the leading cause of mortality by 2015 (2). People of Indian origin living outside India have higher rates than indigenous populations (3). These findings are not completely explained by the prevalence of classical CHD risk factors such as high total cholesterol concentrations, hypertension and obesity, or lifestyle factors such as high saturated fat intakes and smoking (4), all of which tend to be lower in South Asian Indian than Western populations (5). However, CHD in Indians has been associated with features of the Insulin Resistance Syndrome (4). This still does not account for all of the elevated risk of CHD in Indians compared to Western populations. This prompted us to examine the role of other known risk factors that have not previously been assessed in India. Studies in the West have shown that increased left ventricular (LV) mass and reduced arterial compliance predict an increased risk of CHD (6,7). We have measured these two risk markers, and examined their relation to CHD, and other risk factors for the disease, in men and women living in Mysore, South India.

Methods

In an earlier study of the relationship between fetal growth and adult CHD, we traced 517 men and women who were born in Holdsworth Memorial Hospital (HMH), Mysore between 1934 and 1953, still lived in Mysore, and could be matched to their birth records (8). Cases of CHD were defined by the presence of one or more of the following: typical angina according to the Rose/WHO chest pain questionnaire (9); ECG Minnesota codes (10) 1-1 or 1-2 (Q and QS codes) in a standard 12-lead ECG; or a history of coronary artery angioplasty or bypass graft surgery. Waist and hip circumferences and triceps and subscapular skinfolds were measured. A fasting blood sample was taken for measurement of plasma glucose, insulin, proinsulin, 32-33 split proinsulin, fibrinogen, factor VII and serum lipid concentrations. Subjects underwent a standard WHO 75 g oral glucose tolerance test (11) with blood drawn at 30 and 120 minutes for measurement of plasma glucose and insulin concentrations. Information on medication, smoking habits, alcohol consumption and socio-economic status were obtained by questionnaire. As previously described (12), plasma glucose concentrations were measured in Mysore using standard enzymatic methods. Plasma insulin, and fasting proinsulin, 32-33 split proinsulin, fibrinogen and factor VII concentrations, and serum total cholesterol, HDL-cholesterol and triglyceride concentrations were measured in the UK8,12. Serum LDL-cholesterol concentrations were calculated8.

In 1996-7, we re-contacted the same men and women, and invited them for further cardiovascular studies (13). All measurements were carried out by one observer (KK). A

questionnaire was administered to obtain information on current medications. Weight and height were measured and body surface area was calculated using a standard formula (14). Systolic and diastolic blood pressures were recorded using an automated device (Dinamap 8100), with the subject supine and rested for ten minutes. Subjects were defined as hypertensive if they had a systolic blood pressure >140 mmHg, or a diastolic pressure >90 mmHg (15), or were taking anti-hypertensive medication.

Left ventricular mass

LV mass was measured using 2D and M-mode echocardiography (Larsen and Toubro Sigma 1AC machine with a 3.5 Hz transducer), according to the recommendations of the American Society of Echocardiography (16). Interventricular septal thickness at end diastole, posterior cardiac wall thickness at end diastole, left ventricular internal diameter at end diastole and systole were measured from M-mode prints using a digitiser (Genitiser GT-1212B). Measurements were made in five cardiac cycles and averaged. LV mass was calculated using a standard formula (17).

Analyses were carried out using LV mass as a continuous variable, and also as a dichotomous variable: the presence or absence of LV hypertrophy (LVH). To define LVH for this population, we considered a ‘normal’ sub-group of people with a body mass index of less than 30 kg/m2, without hypertension, CHD or diabetes and not taking medications for hypertension or angina. Their mean LV mass, indexed by body surface area (BSA), was calculated (men and women separately). LVH was defined as a LV mass/BSA greater than 2 standard deviations above this mean (18,19).

Arterial compliance

Compliance was measured in 3 arterial segments (right aorto-radial, aorto-femoral and femoro-popliteal-posterior tibial) using the non-invasive optical method of photoplesythmography (20). This is based on the principle that pulse wave velocity (PWV) is increased in stiffer (less compliant) arteries. Customised software measures the time delay between the left ventricular contraction, recorded by ECG, and the arrival of the wave at the peripheral artery, detected by an infra-red probe. Measurement of the distance from the heart (sternal notch) to the probe allows PWV to be calculated. PWV in the femoro-popliteal-posterior tibial segment was obtained by calculating the time delay between the femoral and posterior tibial arteries. This method has been validated against intra-arterial measurements of pressure wave velocity, and gives similar estimates of arterial compliance to those obtained by Doppler ultrasound (20). Ethical permission for the study was obtained from the Ethics Committee of the Holdsworth Memorial Hospital.

Statistical methods

All outcome variables were normally distributed, except for PWV in the femoro-popliteal-posterior tibial segment. This was logarithmically transformed to obtain a normal distribution curve. Relationships of LV mass and PWV to age, body size and CHD risk factors were examined using Pearson correlation coefficients and multiple linear regression with all variables as continuous. Multiple logistic regression was used to examine

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relationships of dichotomous variables, including LVH with risk factors, and CHD and hypertension with LV mass and PWV. Data analysis was performed using SPSS/PC 5.1.

Results

Eight of the original 517 subjects had died. Of the remaining 509, 435 (85%; 237 men and 198 women) agreed to take part in the study. Their characteristics are shown in Table 1. 56 (12%) subjects were excluded from the analysis of LV mass; 54 due to an inadequate echocardiographic window or incorrect angle, and 2 who had hypertrophic cardiomyopathy. Inadequate echocardiographic window/angle is a well recognised and unavoidable problem in echocardiographic studies and this percentage is comparable to other studies (18,19). The LV mass analysis therefore included 379 people. One woman refused the arterial compliance measurement and pulse traces of adequate quality could not be obtained from the femoral artery for a further six subjects. The arterial compliance analysis therefore included 434 subjects for the aorto-radial segment and 428 for the aorto-femoral and femoro-popliteal-posterior tibial segments.

CHD risk factors

Subjects with CHD were older and shorter than those without the disease and had higher systolic blood pressures, a more adverse lipid profile and higher concentrations of plasma glucose and insulin (Table 2). After adjusting for sex these relationships were statistically significant for fasting plasma glucose and serum HDL-cholesterol concentrations.

LV mass

Mean LV mass was greater in men than women ($p<0.001$) (Table 1). It rose with increasing body size, the strongest correlation being with BSA ($r=0.64$; $p<0.001$). We therefore adjusted for body size in the traditional way, by indexing LV mass with BSA (LV mass/BSA)18. After adjustment, the difference in mean LV mass between the sexes diminished but remained statistically significant ($p=0.001$). LV mass/BSA tended to rise with increasing age ($p=0.09$ in men and 0.03 in women).

Relationships of LV mass to blood pressure and CHD risk factors were similar in men and women. LV mass rose with increasing systolic ($r=0.41$) (Table 3) and diastolic ($r=0.36$) blood pressure, independently of age and body size. The effect of age on LV mass diminished when systolic blood pressure was added to the regression equation ($p=0.4$ and 0.5 in men and women respectively). LV mass/BSA was significantly greater in the 112 subjects with hypertension (95g/m2) than in the remainder (80g/m2) ($p<0.001$). The 55 people with LVH had higher systolic (146mmHg vs 123mmHg) and diastolic (84mmHg vs 75mmHg) blood pressures than those without ($p<0.001$ for both).

LV mass and LV mass/BSA were higher in subjects with CHD compared with those without the disease (149.1g vs 137.3g and 91.4g/m2 vs 84.3g/m2; $p=0.07$ and 0.05 respectively), independently of age, sex, and systolic blood pressure. This association was strongest in those with significant Q waves on ECG (n=10) who had a higher LV mass/BSA than those without (101g/m2 vs 85g/m2). In a multiple regression model to examine predictors of CHD, where age, sex, systolic blood pressure, fasting plasma insulin and glucose, fasting

serum cholesterol and triglyceride concentrations, waist-hip ratio, LV mass and pulse wave velocity were included as independent variables, age was the strongest predictor (r=0.05; p=0.08) of CHD followed by LV mass (r<0.01; p=0.1). It is, however, interesting to note that none of the independent variables were statistically significant in this model. No single variable explained even 1% of the variation in prevalence of CHD.

In both sexes, LV mass and LV mass/BSA were greater in the 55 subjects with non-insulin dependent diabetes mellitus (NIDDM; 150.5g and 90.3g/m2) and in the 68 with impaired glucose tolerance (IGT; 146.8g and 86.7g/m2) than in those with normal glucose tolerance (133.8g and 83.8g/m2), independently of age, sex and body size (p <0.001 and 0.01 respectively). Their prevalence of LVH was also higher, 27% in subjects with NIDDM and 20% in subjects with IGT compared with 10% in those with normal glucose tolerance (p=0.002 for those with NIDDM and IGT combined). Both LV mass and LV mass/BSA rose with increasing fasting and 120 minute glucose, fasting insulin, proinsulin, and triglyceride and decreasing HDL-cholesterol concentrations (Table 4) (p<0.05 for all). LV mass and LV mass/BSA were also greater in those with a higher waist/hip ratio (Table 4). There were no relationships with plasma fibrinogen or factor VII concentrations (Table 4). In a multiple regression model to examine predictors of LV mass with age, sex, systolic blood pressure, PWV, fasting plasma insulin and glucose, fasting serum cholesterol and triglyceride concentrations and waist-hip ratio included as independent variables, fasting plasma insulin, systolic blood pressure, PWV in the aorto-posterior segment, sex and waist-hip ratio were independently associated with LV mass (p<0.05 for all).

Arterial compliance

Mean PWV rose with age in the aorto-radial and aorto-femoral segments (p=0.09 and <0.001 respectively) but not in the femoro-popliteal-posterior tibial segment. It was also higher in men than in women in those two segments (p for difference between sexes <0.001 in both segments) (Table 1), but similar in both sexes in the femoro-popliteal-posterior tibial segment. Mean PWV rose with increasing body size; the strongest correlations were with BSA and this was used to adjust for current body size. After adjustment, the difference in PWV between the two sexes diminished but remained statistically significant in the aorto-radial segment (p<0.001).

PWV was strongly related to blood pressure, rising with increasing systolic and diastolic blood pressures (p<0.01 for all) (Table 3). On including systolic blood pressure in the regression model, the relationship between PWV and age remained significant in the aorto-femoral segment (p<0.001). Mean PWV in the aorto-radial and aorto-femoral segments rose with increasing LV mass (p<0.001 for both); on adjusting for BSA, this relationship remained statistically significant in the aorto-femoral segment (p=0.02).

There were no statistically significant differences between pulse wave velocity in any of the arterial segments in subjects with and without CHD (aorto-right radial 4.15 vs 4.06; aorto-femoral 3.27 vs 3.33; femoro-posterior tibial 13.60 vs 12.93 m/s in those without and with CHD respectively; p>0.05 for all). PWV in the aorto-femoral and femoro-popliteal-posterior tibial segments was higher in people with NIDDM (3.42m/s and 15.33m/s) or IGT (3.32m/s and 15.18m/s) than in those with normal glucose tolerance (3.25m/s and 12.67m/s; p<0.05

for both segments). PWV tended to rise with increasing plasma glucose, insulin, proinsulin and 32-33 split proinsulin concentrations, serum triglyceride concentrations and waist/hip ratios (Table 4). There were no relationships with plasma fibrinogen or factor VII concentrations (Table 4). In a multiple regression model to examine predictors of PWV where age, sex, systolic blood pressure, fasting plasma insulin and glucose, fasting serum cholesterol and triglyceride concentrations and waist-hip ratio were included as independent variables, fasting plasma glucose and insulin, systolic blood pressure, LV mass, age and sex were independently associated with PWV; however, these relationships were not statistically significant in all segments.

Discussion

We have measured left ventricular mass and arterial pulse wave velocity in a population of South Indian adults. Higher LV mass was associated with an increased prevalence of CHD and many of its known risk factors. Although PWV was unrelated to CHD, higher PWV was also associated with an adverse CHD risk factor profile.

This study was performed as part of an epidemiological study of the relationship between fetal growth and adult CHD and its risk factors. The relationships between fetal growth and adult LV mass and arterial compliance have been described earlier (13). This study was restricted to a group of men and women who were born in a single hospital in Mysore during 1934-53 (when the majority of births would have taken place at home), had survived childhood, still lived in Mysore and gave sufficient information to enable us to match them to their birth records. This cohort was therefore not a true population sample. It was, however, a large sample of normal men and women living in a defined geographical area, and mean height and weight, and rates of CHD and diabetes were similar to those reported in other urban South Indian populations (1,21,22).

Mean LV mass was lower and arterial compliance was increased in this Indian population compared with Western populations, even after allowing for their smaller mean body size (18,19 and Phillips N, unpublished data). These findings may be due to lower blood pressures in our population, or genuine racial differences (23). In Mysore, higher LV mass and reduced arterial compliance were associated with increasing age, greater body size and obesity, and higher blood pressure. Similar relations have been found elsewhere (24–27). As in other studies, reduced compliance was associated with an increased LV mass (27). Reduced compliance may promote LVH through its effects on systolic pressure and increased LV afterload.

As has been shown in Western studies, men and women with CHD had an increased LV mass (6). The mechanisms underlying this association are not clear. It has been suggested that LVH increases myocardial oxygen demand and coronary vascular resistance (28), decreases coronary blood flow reserve (29) and increases the incidence of ventricular arrhythmias (30). Alternatively they may be associated because they share common risk factors including obesity, raised blood pressure, diabetes mellitus, hyperinsulinaemia and an adverse lipid profile. PWV was not related to CHD, a finding which differs from most, though not all (31), studies in western populations (7). This was interesting particularly in

view of the fact that arterial compliance was related to other CHD risk factors. Approximately 13% of our subjects were on anti-hypertensive medication, particularly ACE inhibitors and beta-blockers which not only cause a reduction in blood pressure and LV mass but also improve compliance. However, re-analysis of the subjects excluding those on treatment showed similar results. It is possible that the direct link between PWV and CHD is not strong enough to be apparent in this study.

In our study, higher LV mass and PWV were associated with NIDDM and IGT, higher insulin and triglyceride concentrations and a higher waist/hip ratio. Greater LV mass was associated with lower HDL-cholesterol concentrations. These are all known to be associated with insulin resistance and form the features of Reaven's Syndrome or the Insulin Resistance Syndrome (IRS) (32,33). Total cholesterol, fibrinogen and factor VII concentrations, CHD risk factors which are not classically part of the IRS, were unrelated to either LV mass or PWV in this Indian population. One of the reasons suggested for the high rates of CHD in South Asian Indians is increased insulin resistance. The Insulin Resistance Syndrome is expressed in an exaggerated manner in this ethnic group (4,34).

Insulin resistance may link CHD, increased LV mass, reduced arterial compliance and other CHD risk factors. Our earlier study on the Mysore cohort showed that the subjects were markedly insulin resistant, indicated by higher fasting insulin, proinsulin and 32-33 split proinsulin concentrations than in a UK population of similar age, despite being considerably less obese (12). Hyperinsulinaemia promotes vascular hypertrophy either by a direct action or through increased sympathetic activity (35). This may lead to hypertension, reduced compliance and increased LV mass. It has also been suggested that the so-called 'inactive' precursors of insulin, such as proinsulin and 32-33 split proinsulin, cause vascular disease, rather than insulin itself (36) and in our study proinsulin had the strongest correlations with LV mass and PWV. Components of the IRS are associated with CHD in the Mysore men and women and we speculate that these associations may be partly mediated by increased LV mass and reduced arterial compliance.

Public health interventions for prevention of CHD in Western countries have traditionally focussed on reduction of blood pressure and serum cholesterol concentrations. Our findings suggest that, in urban South Asian Indians, it may also be effective to try to reduce insulin resistance by encouraging avoidance of obesity and promoting physical activity.

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Table 1
Mean (SD) characteristics of the Mysore men and women

	Men n=237	Women n=198
General characteristics		
Age (years)	**49.5**(4.8)	**49.5**(4.8)
Height (cm)	**165.5**(6.1)	**151.6**(6.5)
Body mass index (kg/m2)	**23.1**(4.0)	**25.4**(5.1)
Cardiac dimensions		
Interventricular septal thickness (mm)	**10.6**(1.7)	**9.9**(1.6)
Posterior wall thickness (mm)	**8.8**(1.1)	**8.5**(0.9)
Left ventricular end-diastolic diameter (mm)	**45.1**(4.1)	**42.5**(3.5)
Left ventricular end-systolic diameter (mm)	**27.3**(3.6)	**24.8**(2.8)
Left ventricular mass (g)	**149.1**(36.6)	**125.2**(31.9)
Left ventricular mass indexed by body surface area (g/m2)	**87.7**(18.3)	**81.9**(16.9)
Relative wall thickness	**0.4**(0.05)	**0.4**(0.05)
Pulse wave velocity (m/s)		
Aorto-radial segment	**4.32**(0.45)	**3.92**(0.40)
Aorto-femoral segment	**3.35**(0.51)	**3.19**(0.50)
Femoro-popliteal-posterior tibial segment *	**13.46**(1.54)	**13.60**(1.58)

* Geometric mean

Table 2
Risk profile of men and women with and without CHD (Mean (SD))

	MEN		WOMEN	
	With CHD (n=22)	**Without CHD (n=215)**	**With CHD (n=23)**	**Without CHD (n=175)**
General characteristics				
Age (years)	**51.5**(5.5)	**49.3**(4.7)	**51.2**(5.0)	**49.2**(4.7)
Height (cm)	**164.7**(6.3)	**165.6**(6.0)	**150.9**(7.7)	**151.7**(6.4)
Body mass index (kg/m2)	**23.2**(3.3)	**23.0**(4.0)	**25.0**(4.6)	**25.4**(5.2)
CHD risk factors				
Systolic blood pressure (mmHg)	**136**(20)	**126**(22)	**129**(20)	**127**(24)
Diastolic blood pressure (mmHg)	**81**(7)	**78**(12)	**76**(9)	**74**(12)
Serum triglycerides (mmol/L) *	**1.9**(1.8)	**1.7**(1.7)	**1.8**(1.5)	**1.4**(1.7)
HDL-cholesterol (mmol/L)	**0.9**(0.2)	**0.9**(0.2)	**0.9**(0.2)	**1.0**(0.2)
LDL-cholesterol (mmol/L)	**3.7**(1.0)	**3.2**(0.8)	**3.0**(0.6)	**3.2**(0.8)
Total cholesterol (mmol/L)	**5.4**(1.2)	**5.0**(1.0)	**4.8**(0.7)	**4.9**(0.9)
Fasting insulin (pmol/L) *	**43**(3)	**47**(2)	**75**(2)	**54**(2)
30-min insulin (pmol/L) *	**344**(2)	**376**(2)	**459**(2)	**369**(2)
120-min insulin (pmol/L) *	**365**(3)	**321**(2)	**446**(2)	**376**(2)
Proinsulin (pmol/L) *	**9.6**(2.5)	**7.7**(2.4)	**7.9**(1.93)	**6.2**(2.13)
32-33 split proinsulin (pmol/L) *	**9.8**(2.6)	**8.4**(2.9)	**9.2**(2.0)	**8.0**(2.4)
Fasting glucose (mmol/L) *	**5.5**(1.5)	**5.1**(1.3)	**5.9**(1.5)	**5.3**(1.3)
30-min glucose (mmol/L) *	**8.3**(1.4)	**8.3**(1.3)	**8.7**(1.4)	**7.9**(1.3)
120-min glucose (mmol/L) *	**6.9**(1.4)	**6.4**(1.4)	**7.5**(1.5)	**6.7**(1.3)
Waist/hip ratio	**0.92**(0.05)	**0.91**(0.06)	**0.85**(0.06)	**0.83**(0.06)
Subscapular/triceps skinfold ratio	**1.84**(0.46)	**1.83**(0.49)	**1.40**(0.30)	**1.27**(0.32)
Plasma fibrinogen (g/L)	**291**(49)	**306**(67)	**344**(56)	**338**(61)
Factor VII (g/L)	**113**(30)	**113**(37)	**139**(38)	**122**(41)

* Geometric mean and SD

Table 3

Mean LV mass and pulse wave velocity according to quintiles of systolic blood pressure

Systolic blood pressure (mmHg)	LV mass (g)	LV mass/BSA (g/m2)	Pulse wave velocity (m/s)		
			Aorto-radial	Aorto-femoral	Femoro-popliteal-posterior tibial
<108	**118.5**(70)	**77.7**(70)	**3.97**(80)	**3.06**(80)	**11.55**(80)
108-	**130.9**(80)	**80.7**(80)	**4.04**(90)	**3.16**(90)	**13.75**(90)
116-	**133.4**(76)	**81.8**(76)	**4.16**(84)	**3.23**(81)	**14.56**(81)
131-	**146.1**(83)	**86.9**(83)	**4.19**(93)	**3.40**(90)	**13.81**(90)
145+	**162.4**(70)	**98.7**(70)	**4.30**(87)	**3.54**(87)	**14.48**(87)
All	**138.3** (379)	**85.1**(379)	**4.14**(434)	**3.28**(428)	**13.59**(428)
p value	<0.0001	<0.0001	<0.001	<0.0001	0.009
p * value			<0.001	<0.0001	0.03

Figures in parentheses indicate numbers of subjects.

p values adjusted for age and sex;

* indicates additional adjustment for body surface area

Table 4

Relation of LV mass and pulse wave velocity to risk factors for CHD: unadjusted Pearson correlation coefficients

	LV mass (g)	LV mass/BSA (g/m2)	Pulse wave velocity (m/s)		
			Aorto-radial	Aorto-femoral	Femoro-popliteal-posterior tibial
Glucose					
Fasting	0.19**	0.17**	0.04	0.15*	0.15**
30 minutes	0.17**	0.08	0.16	0.15	0.15**
120 minutes	0.23**	0.13**	0.06	0.10	0.16*
Insulin					
Fasting	0.26**	0.13*	0.10	0.16	0.12
30 minutes	0.09	-0.02	0.14*	0.12	<0.01
120 minutes	0.20**	0.07	0.12*	0.11	0.15*
Proinsulin	0.37**	0.21**	0.22*	0.26*	0.15*
32-33 split proinsulin	0.27**	0.11	0.18*	0.22*	0.13
Lipids					
Total cholesterol	0.13*	0.05	0.05	0.10	0.06
LDL cholesterol	0.12*	0.05	0.02	0.05	0.02
HDL-cholesterol	-0.22**	-0.16*	- 0.07	0.01	- 0.07
Triglycerides	0.29**	0.20**	0.16	0.16	0.16**
Central obesity					
Waist/hip ratio	0.47**	0.27**	0.38*	0.29**	0.09
SS/TR	0.21	0.13	0.23	0.10	0.06
Clotting factors					
Fibrinogen	-0.07	-0.06	- 0.05	0.06	<0.01
Factor VII	0.06	0.02	0.04	0.09	0.09

* **p <0.05**

** **p <0.01**

p values adjusted for age and sex; PWV values also adjusted for BSA.