Interaction of workplace demands and cardiovascular reactivity in progression of carotid atherosclerosis: population based study

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Abstract

Objective: To examine the combined influence of workplace demands and changes in blood pressure induced by stress on the progression of carotid atherosclerosis.

Design: Population based follow up study of unestablished as well as traditional risk factors for carotid atherosclerosis, ischaemic heart disease, and other outcomes.

Setting: Eastern Finland.

Subjects: 591 men aged 42-60 who were fully employed at baseline and had complete data on the measures of carotid atherosclerosis, job demands, blood pressure reactivity, and covariates.

Main outcome measures: Change in ultrasonographically assessed intima-media thickness of the right and left common carotid arteries from baseline to 4 year follow up.

Results: Significant interactions between workplace demands and stress induced reactivity were observed for all measures of progression ($P<0.04$). Men with large changes in systolic blood pressure (20 mm Hg or greater) in anticipation of a maximal exercise test and with high job demands had 10-40% greater progression of mean (0.138 *v* 0.123 mm) and maximum (0.320 *v* 0.261 mm) intima-media thickness and plaque height (0.347 *v* 0.264) than men who were less reactive and had fewer job demands. Similar results were obtained after excluding men with prevalent ischaemic heart disease at baseline. Findings were strongest among men with at least 20% stenosis or non-stenotic plaque at baseline. In this subgroup reactive men with high job demands had more than 46% greater atherosclerotic progression than the others. Adjustment for atherosclerotic risk factors did not alter the results.

Conclusions: Men who showed stress induced blood pressure reactivity and who reported high job demands experienced the greatest atherosclerotic progression, showing the association between dispositional risk characteristics and contextual determinants of disease and suggesting that behaviourally evoked cardiovascular reactivity may have a role in atherogenesis.

Introduction

People's susceptibility to disease varies widely and may be a reflection of differences in biological predispositions, personality, behaviour, and environmental exposures. In addition, psychological stress is commonly believed to play an important part in illness and premature death, particularly with respect to cardiovascular diseases.

One model that may explain why some people under great stress develop cardiovascular diseases or other illnesses and others do not is the diathesis-stress model of disease susceptibility. This model assumes that a biological predisposition to disease will be expressed only if a predisposed individual is exposed to unusual or prolonged stress.[1] One mechanism by which stress may be associated with cardiovascular diseases is hyperreactivity of the sympathetic nervous system, which manifests as exaggerated increases in blood pressure or heart rate in response to psychological or behavioural stressors.[2,3] In the diathesis-stress framework behaviourally evoked reactivity is considered to be a biological predisposition of an individual that is expressed and consequently leads to cardiovascular diseases only under high stress conditions.[1]

Situations in which stress induced reactivity may be elicited differ between people. The work environment is probably an important source of stress for most adults.[4] Furthermore, previous studies indicate that stressful jobs have a role in the illness and death caused by cardiovascular disease.[4-6] We used the diathesis-stress model to examine the interactive effects of blood pressure reactivity to stress and high workplace demands on the progression of carotid atherosclerosis. This report is from the Kuopio ischaemic heart disease risk factor study, an ongoing, population based study of previously unestablished risk factors for carotid atherosclerosis, ischaemic heart disease, mortality, and other outcomes among middle aged men from the Kuopio region in Eastern Finland, an area of high coronary morbidity and mortality.[7,8]

Subjects and methods

A total of 2682 men aged 42, 48, 54, and 60 (82.9% of those eligible) were recruited for the baseline examination, which occurred between March 1984 and December 1989. Follow up examinations were conducted between March 1991 and December 1993 in men who had undergone ultrasound examination of the right and left carotid arteries at baseline. Of the 1229 men who were eligible for the follow up examination, 1038 (88.2%) participated, 107 refused, 52 could not participate because they had died, had severe illness, or had moved, and 32 could not be contacted. Average time to follow up was 4.2 years (range 3.9-5.1 years).

We excluded subjects if they were not fully employed at the time of the baseline examination (348 men), did not participate in the bicycle ergometer test because of scheduling difficulties (72), or had missing data on the covariates at baseline (27). Therefore, the results reported here are based on 591 men who were fully employed and had complete data on the job

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demands scale, the measure of anticipatory blood pressure reactivity, all covariates at baseline, and measurements of intima-media thickness at baseline and follow up. A comparison of the 447 subjects who were excluded with the 591 subjects with complete data found no significant differences in resting systolic blood pressure, body mass index, or mean intima-media thickness at baseline, although the subjects who were excluded were older (55.1 *v* 48.6 years, $P<0.0001$).

Baseline and follow up examinations

Examinations were carried out over two days, one week apart, at both baseline and follow up and consisted of a wide variety of biochemical, physiological, anthropometric, and psychosocial tests, including a questionnaire on workplace demands and characteristics (see job demands scale). In addition, a maximal exercise tolerance test on an upright bicycle ergometer was administered at baseline. Medical history and drug treatment were checked during a medical examination at both baseline and follow up.

Blood pressure measurement

Blood pressure readings were obtained on two occasions by a trained observer using a random zero muddler sphygmomanometer (Hawksley). The protocol on the first examination day was as follows. Subjects rested supine for 15 minutes and blood pressure was measured at 5, 10, and 15 minutes; they then stood at rest and blood pressure was taken once after 1 minute; they finally sat at rest for 10 minutes, and blood pressure was measured at 5 and 10 minutes. Blood pressure while subjects were seated was also measured on the second examination day, one week later, after the subject had been seated on the bicycle ergometer for 5 minutes but before the exercise test protocol was begun. Measurements on both examination days were taken in the mornings.

Cardiovascular reactivity

The measure of reactivity used in this study was change in systolic blood pressure in anticipation of the exercise test (blood pressure after five minutes of seated rest on the bicycle ergometer before the start of the exercise test), which was calculated as the difference between the blood pressure reading while seated before exercise and the average of the two resting blood pressure readings while seated on the first examination day. The anticipation of exercise is characterised by emotional, behavioural, and physiological arousal attendant to the impending challenge. Blood pressure increases during this anticipatory period thus reflect cardiovascular adjustments in response to psychological and behavioural stress. In this study men with a systolic response of 20 mm Hg or greater were considered to be high reactors (45.7%). This measure of reactivity is a significant, independent predictor of incident hypertension in this population.[9]

Job demands scale

As part of the baseline examination participants completed a detailed questionnaire that included items about their work environment. The measure of job demands used in this study was an 11 item scale developed by Lynch *et al*[10] that conformed to important theoretical dimensions of the work environment.[4 5 11] Using a four point Likert response, respondents indicated how much stress they experienced from excessive supervision of time schedules, troublesome supervisors, troublesome coworkers, job responsibilities, poorly defined tasks and responsibilities, risk of accidents, risk of unemployment, and irregular work schedules. They were also asked to indicate how often they had work deadlines and how much stress this caused them and to rate both the mental and physical strenuousness of their work. Individual items were dichotomised such that only men reporting more than average strain or stress for any given item were considered positive for that item. The 11 dichotomised items were then summed to create the job demands scale score, which had a mean of 3.0 and range of 0-11 in the full sample, with Cronbach's α of 0.78. We considered men with a score of 4 or greater on the job demands scale to have high job demands. This cut off point was selected because it represented the upper quartile (26%) of the distribution of job demands scores in this population. Scores on the job demands scale were imputed based on non-missing items for men who had two or fewer items with missing responses (5%); those with more than two missing items were excluded from analyses. This scale predicts mortality and acute myocardial infarction in this population.[10]

Measurement of carotid atherosclerosis

The extent of carotid atherosclerosis was assessed by high resolution B mode ultrasonography of the right and left common carotid arteries in a 1.0-1.5 cm section at the distal end, proximal to the carotid bulb. Images were focused on the posterior wall of the right and left common carotid arteries and recorded on videotape for later analysis. Near wall images were not obtained because of their greater variability in measurement.[12] Ultrasound examinations at baseline and follow up were conducted by one of four sonographers who had been trained for a minimum of six months, and they were performed with the subject lying supine after a 15 minute rest. Details of the scanning protocol, technical aspects of measurement, and their reliability have been described elsewhere.[13]

At baseline arterial images were obtained using the ATL UM4 duplex ultrasound system with a 10 MHz sector transducer (Advanced Technology Laboratories, Bothell, WA). At follow up images were obtained with a Biosound Phase 2 scanner equipped with a 10 MHz annular array probe.[13] Wedge phantom studies of this system, calibrated against a 414B tissue phantom (Radiation Measurement, Middleton, WI), have shown measurement precision of 0.03 mm.[14 15]

Intima-media thicknesses were measured at baseline and follow up by computerised analysis of the videotaped ultrasound images using Prosound software (University of Southern California, Los Angeles, CA). This software uses an edge detection algorithm specifically designed for use with ultrasound imaging and permits automatic detection, tracking, and recording of the interfaces between intima and lumen and media and adventitia.[16] Intima-media thickness, calculated as the mean distance between these interfaces, was estimated at around 100 points in both the right and left common carotid arteries.

We used three measures of intima-media thickness: (*a*) mean intima-media thickness, calculated as the mean of all estimates from the right and left common carotid arteries and considered an overall measure of the atherosclerotic process in the carotid arteries; (*b*) maximum intima-media thickness, the average of the points of maximum thickness from the right and left common carotid arteries and indicative of the depth of intrusion of atherosclerotic thickening into the lumen in this part of the arteries; and (*c*) plaque height, the average of plaque height in the right and left common carotid arteries, calculated as the difference between maximum and minimum thickness, and an assessment of how steeply atherosclerotic lesions protruded into the lumen. Progression of atherosclerosis was estimated as follow up minus baseline values for each of these measures.

Baseline covariates

Biological risk factors—Resting systolic blood pressure was calculated as the average of the last two supine and the two seated blood pressure measurements obtained on the first examination day. Apolipoprotein B concentration was determined by an immunoturbidimetric method (KONE, Espoo, Finland). High density lipoprotein cholesterol-2, the protective subfraction of high density lipoprotein cholesterol, was separated from fresh plasma using ultracentrifugation and precipitation and its concentration measured enzymatically (CHOD-PAP cholesterol method, Boehringer Mannheim, Mannheim, Germany). Blood glucose concentration was measured in whole blood samples after at least 12 hours of overnight fasting by the glucose dehydrogenase method after precipitation of the proteins with trichloroacetic acid (Granutest 100, Merck, Darmstadt, Germany). Body mass index was calculated as weight divided by height squared (kg/m^2).

Behavioural and educational factors—Alcohol consumption was assessed by a questionnaire on drinking behaviour over the previous 12 months and from dietary records over four days. Cigarette smoking was assessed by self report of never, former, and current smoking (measured in pack years). Education was assessed by self report of completed years of schooling.

Drug treatment—The use of drugs to treat hypertension and hyperlipidaemia was assessed at baseline by interview.

History of diabetes—A participant was considered to have a history of diabetes if he reported having taken drugs for or used diet to control diabetes or if he had a fasting blood glucose concentration of 6.7 mmol/l or greater at baseline.

Evaluation of prevalent ischaemic heart disease—Participants were considered to have prevalent ischaemic heart disease at baseline if they had a history of angina or myocardial infarction; if they currently took drugs for angina; or if they had positive findings of angina according to the Rose questionnaire.[17]

Data analyses

We examined the influence of workplace demands and cardiovascular reactivity on the progression of atherosclerosis over four years by estimating the mean change in each measure of intima-media thickness (mean, maximum, plaque height) according to low *v* high job demands and low *v* high reactors. These dichotomous variables and their product interaction term were entered as predictors into our linear regression models. Two sets of regression models were calculated. The initial model included adjustments for age, baseline intima-media thickness, zooming depth of the ultrasound scan, sonographer, and participation in the placebo or treatment arm of an unrelated clinical trial of pravastatin.[18] The second model included all variables in the initial model plus variables representing systolic blood pressure, apolipoprotein B concentration, high density lipoprotein cholesterol-2 concentration, body mass index, cigarette smoking, alcohol consumption, use of antihypertensive or antihyperlipidaemic drugs, and history of diabetes.

Analyses were performed using the general linear models procedure in SAS, version 6.09,[19] installed on a Sun Sparcstation 20. This procedure allowed least square mean values of intima-media thickness to be estimated and contrasted for each job demands/reactor group while simultaneously controlling for age and other covariates.

Table 1 Baseline covariates according to job demands and reactivity. Values are means (SD) unless stated otherwise

	Low job demands		High job demands	
	Low reactors (n=233)	High reactors (n=192)	Low reactors (n=88)	High reactors (n=78)
Job demands scale	1.7 (0.8)	1.8 (0.8)	5.0 (1.3)	5.0 (1.1)
Systolic pressure response (mm Hg)	8.5 (7.2)	31.6 (9.5)	9.3 (7.3)	33.0 (10.5)
Age (years)	47.6 (5.4)	50.7 (5.9)	47.8 (5.4)	51.6 (5.6)
Education (years)	10.9 (3.9)	9.5 (3.4)	9.4 (3.6)	9.3 (4.0)
Intima-media thickness (mm):				
Mean	0.70 (0.13)	0.76 (0.15)	0.73 (0.20)	0.77 (0.21)
Maximum	0.87 (0.17)	0.94 (0.20)	0.91 (0.27)	0.97 (0.29)
Plaque height (mm)	0.35 (0.13)	0.37 (0.14)	0.37 (0.17)	0.41 (0.22)
Resting systolic pressure (mm Hg)	131.2 (16.2)	131.9 (13.5)	132.2 (15.6)	130.8 (12.8)
Apolipoprotein B (mmol/l)	2.58 (0.60)	2.63 (0.53)	2.57 (0.54)	2.62 (0.51)
High density lipoprotein cholesterol 2 (mmol/l)	0.89 (0.30)	0.88 (0.27)	0.87 (0.28)	0.87 (0.28)
Blood glucose (mmol/l)	4.70 (1.08)	4.72 (1.21)	4.57 (0.54)	4.51 (0.46)
Body mass index (kg/m^2)	26.9 (3.6)	26.7 (3.2)	26.2 (2.9)	26.6 (3.1)
No (%) of subjects:				
Drinking alcohol (drinks/day):				
None	21 (9)	22 (12)	8 (9)	8 (10)
>2	61 (26)	37 (19)	18 (21)	15 (19)
Smoking:				
Never	76 (33)	52 (27)	29 (33)	23 (30)
Formerly	80 (34)	70 (37)	31 (35)	32 (41)
Currently	77 (33)	70 (36)	28 (32)	23 (30)
Receiving drug treatment:				
Antihyperlipidaemic agent	0	2 (1)	1 (10)	1 (1)
Antihypertensive agent	24 (10)	25 (13)	10 (11)	13 (17)
With diabetes	10 (4)	5 (3)	1 (1)	2 (3)

Results

Table 1 shows scores on the job demands scale, change in systolic blood pressure in anticipation of exercise, age, intima-media thicknesses at baseline, and other covariates by job demands and reactivity.

Demands, reactivity, and atherosclerotic progression

Table 2 shows age adjusted scores for changes in mean and maximum intima-media thickness and plaque

Table 2 Progression of carotid atherosclerosis over four years according to job demands and reactivity in 591 Finnish men. Values are mean (SE) increases in millimetres

	Low job demands		High job demands		
	Low reactors (n=233)	High reactors (n=192)	Low reactors (n=88)	High reactors (n=78)	P value for interaction
Model 1*					
Intima-media thickness:					
Mean	0.123 (0.008)	0.109 (0.009)	0.099 (0.013)	0.138 (0.013)	<0.014
Maximum	0.261 (0.013)	0.267 (0.013)	0.238 (0.020)	0.320 (0.021)	<0.021
Plaque height	0.264 (0.013)	0.289 (0.013)	0.255 (0.020)	0.347 (0.021)	<0.04
Model 2†					
Intima-media thickness:					
Mean	0.122 (0.008)	0.106 (0.009)	0.093 (0.013)	0.134 (0.013)	<0.008
Maximum	0.261 (0.013)	0.262 (0.013)	0.228 (0.020)	0.312 (0.021)	<0.013
Plaque height	0.264 (0.013)	0.282 (0.013)	0.246 (0.020)	0.340 (0.020)	<0.022

*Adjusted for age, baseline intima-media thickness, ultrasound zooming depth, sonographer, and participation in placebo or treatment arms of Kuopio artherosclerosis prevention study.[18]
†Includes adjustments from model 1 plus covariates representing education, resting systolic blood pressure, apolipoprotein B concentration, high density lipoprotein cholesterol-2 concentration, fasting blood glucose concentration, former and current smoking, body mass index, alcohol consumption, use of antihyperlipidaemic or antihypertensive drugs, and history of diabetes.

Table 3 Progression of carotid atherosclerosis over four years according to job demands and reactivity in 518 men without prevalent ischaemic heart disease. Values are mean (SE) increases in millimetres*

	Low job demands		High job demands		
	Low reactors (n=216)	High reactors (n=168)	Low reactors (n=77)	High reactors (n=57)	P value for interaction
Intima-media thickness:					
Mean	0.122 (0.008)	0.110 (0.008)	0.112 (0.012)	0.126 (0.014)	<0.15
Maximum	0.256 (0.012)	0.255 (0.012)	0.252 (0.019)	0.290 (0.021)	<0.20
Plaque height	0.262 (0.013)	0.276 (0.013)	0.257 (0.020)	0.324 (0.022)	<0.12

*Adjusted for age, baseline intima-media thickness, ultrasound zooming depth, sonographer, and participation in placebo or treatment arms of Kuopio artherosclerosis prevention study.[18]

Table 4 Progression of carotid atherosclerosis over four years according to job demands and reactivity and degree of stenosis at baseline. Values are mean (SE) increases in millimetres*

	Low job demands		High job demands		
	Low reactors	High reactors	Low reactors	High reactors	P value for interaction
Carotid stenosis ≥20% or presence of non-stenotic atherosclerotic plaque					
No of subjects	34	54	13	20	
Intima-media thickness:					
Mean	0.118 (0.026)	0.097 (0.021)	0.004 (0.044)	0.173 (0.034)	<0.003
Maximum	0.250 (0.044)	0.252 (0.037)	0.134 (0.074)	0.420 (0.059)	<0.0095
Plaque height	0.255 (0.040)	0.272 (0.035)	0.280 (0.070)	0.418 (0.056)	<0.24
Carotid stenosis <20%					
No of subjects	199	138	75	58	
Intima-media thickness:					
Mean	0.123 (0.009)	0.113 (0.010)	0.112 (0.013)	0.120 (0.014)	<0.40
Maximum	0.262 (0.013)	0.276 (0.015)	0.254 (0.020)	0.279 (0.021)	<0.71
Plaque height	0.257 (0.014)	0.296 (0.014)	0.253 (0.019)	0.310 (0.021)	<0.56

*Adjusted for age, baseline intima-media thickness, ultrasound zooming depth, sonographer, and participation in placebo or treatment arms of Kuopio artherosclerosis prevention study.[18]

height for the four groups from both the initial and fully adjusted models. Significant interactions between self reported job demands and reactivity were noted for progression of mean intima-media thickness ($\beta=0.052$, 95% confidence interval 0.011 to 0.093, SE 0.021, $P<0.014$), maximum intima-media thickness ($\beta=0.076$, 0.012 to 0.141, SE 0.033, $P<0.021$), and plaque height ($\beta=0.067$, 0.003 to 0.131, SE 0.033, $P<0.04$). Men who reported high job demands and who were high reactors had the largest increases in mean and maximum intima-media thickness and plaque height over the four years of follow up (table 2). Results were essentially unchanged after including adjustments for education, resting systolic blood pressure, apolipoprotein B concentration, high density lipoprotein cholesterol-2 concentration, body mass index, former and current smoking, alcohol consumption, use of drugs for hypertension or hyperlipidaemia, and history of diabetes. Indeed, the group with high demands and high reactivity showed 10-40% greater atherosclerotic progression than the other groups, even after adjustment for the various risk factors.

Demands, reactivity, and atherosclerotic progression in men without prevalent ischaemic heart disease

Table 3 shows mean (SE) changes in mean and maximum intima-media thickness and plaque height for the four groups, excluding 73 men with prevalent ischaemic heart disease at baseline. The pattern of findings was similar, albeit slightly weaker, to that in the full sample, with interactions between job demands and reactivity noted for each measure of atherosclerotic progression ($\beta=0.03$, -0.011 to 0.070, SE 0.021, $P<0.15$ for mean intima-media thickness; $\beta=0.04$, -0.021 to 0.100, SE 0.031, $P<0.20$ for maximum intima-media thickness; and $\beta=0.052$, -0.013 to 0.117, SE 0.033, $P<0.12$ for plaque height). Men with high job demands and high reactivity had a 13-15% greater increase in maximum intima-media thickness and a 17-26% greater increase in plaque height relative to the other groups. Adjustment for atherosclerotic risk factors in this subgroup did not alter the size of these differences (data not shown).

Demands, reactivity, and atherosclerotic progression by degree of stenosis at baseline

Table 4 shows the mean values of the three measures of atherosclerotic progression for the four groups according to the presence of carotid stenosis or non-stenotic atherosclerotic plaque at baseline.

Among the 121 men with at least 20% carotid stenosis or evidence of non-stenotic atherosclerotic plaque at baseline there were significant interactions between job demands and reactivity for progression of mean and maximum media-intima thickness ($\beta=0.189$, 0.065 to 0.313, SE 0.063, $P<0.003$ and $\beta=0.284$, 0.073 to 0.495, SE 0.108, $P<0.0095$ respectively); the interaction for plaque height was almost significant ($\beta=0.121$, -0.080 to 0.323, SE 0.103, $P<0.24$). Men with high job demands and reactivity showed the greatest increases in mean and maximum intima-media thickness and plaque height from baseline to follow up, being more than 46% larger than those in the other groups. Models that included adjustments for atherosclerotic risk factors produced essentially the same patterns of results for both strata (data not shown). No interactions between job demands and reactivity were seen in the men without advanced thickening at baseline. However, high reactors showed greater progression of plaque height than low reactors (0.303 *v* 0.255 mm, $P<0.037$). No differences were noted for mean or maximum intima-media thickness in this subgroup.

Discussion

We found a significant interaction between cardiovascular reactivity and reported job demands such that men who showed a heightened increase in systolic blood pressure before an exercise stress test and who reported a highly demanding work environment experienced greater progression of carotid atherosclerosis than men who were less reactive or had fewer job demands, or both. This interaction was observed for the measures of mean and maximum intima-media thickness and plaque height and was largely unaffected by adjustment for known atherosclerotic risk factors. A similar, albeit slightly weaker, pattern of findings was seen in the subset of healthy men without prevalent ischaemic heart disease at baseline. However, the strongest associations were seen among men with early evidence of atherosclerosis, suggesting that the combined effects of stress induced reactivity and high job demands may be more pronounced once atherosclerotic plaque or measurable stenosis has occurred.

Support for diathesis-stress model of disease

Our data clearly show that dispositional characteristics of the individual, in combination with the work environment, are importantly related to disease progression. These findings support the diathesis-stress model of disease susceptibility, which emphasises the synergistic relation between dispositional risk characteristics and contextual determinants of diseases. Moreover, our results provide some of the clearest human evidence to date that stress induced cardiovascular reactivity may play a part in atherogenesis. Additional support for this hypothesis comes from research in cynomolgus macaques which has shown that heightened sympathetic nervous system arousal and endothelial injury induced by psychosocial stressors (threat of capture, social disruption, and reorganisation) potentiate diet induced coronary and carotid atherosclerosis.[20-22] Interestingly, these psychosocially mediated effects are abolished after administration of β adrenergic antagonists.[5 22]

Interpretation of results

Our consistent results across the measures of mean and maximum intima-media thickness and plaque height suggest that the interactive effects of heightened blood pressure responses and high job demands influence the overall atherosclerotic process and contribute to the development or progression of focal lesions of the common carotid arteries. Increased arterial wall roughness with steeply projecting lesions may lead to greater shear stress and flow turbulence on the vessel walls, thereby increasing the likelihood of plaque rupture and thrombus formation.[22] Furthermore, although the differential pathological and clinical significance of the three measures of carotid atherosclerosis used in this study remains to be determined, cross sectional data from the entire Kuopio ischaemic heart disease risk factor study sample showed that each 0.10 mm difference in maximum thickness was associated with an 11% increase in risk of acute myocardial infarction ($P<0.001$).[13]

The relations between behaviourally evoked reactivity and high job demands and atherosclerotic progression identified in our study were essentially unchanged after adjustments for known atherosclerotic risk factors, including resting blood pressure, lipoprotein concentrations, alcohol and cigarette consumption, body mass index, and education. This relative lack of confounding is somewhat surprising because of the strength of the known associations between these risk factors and atherosclerosis.[24 25] However, the cumulative effect of these variables on atherosclerosis may be accounted for by their associations with baseline intima-media thickness, which is a highly significant covariate in all models.

Key messages

- Psychological stress plays an important part in the illness and premature death associated with cardiovascular disease, but individual susceptibility to disease varies according to biological predispositions, personality, behaviour, and environmental exposures
- This study found that a demanding work environment in combination with a predisposition to exaggerated blood pressure reactivity to stress was significantly related to progression of carotid atherosclerosis over four years among employed middle aged men and was independent of known atherosclerotic risk factors
- These findings support the role of stress induced reactivity in human atherogenesis
- Future research needs to confirm these findings in other populations and to examine the influence of other risk factors and environments on the progression of disease

Conclusions

Our findings are limited to employed, middle aged white men. Additional research is needed to determine if these relations are also evident in non-white or female populations or among other age groups. Given that men and women are often employed in different occupational sectors and may perceive and experience workplace demands and job stress differently from one another, it is particularly important to examine the influence of sex differences on these associations. Furthermore, work is only one aspect of life. Therefore, it is important to consider a variety of social and interpersonal contexts that may be potential sources of stress in individuals' daily lives. The diathesis-stress model offers a valuable framework within which future research may be conducted and for examining the relations between various dispositional traits and work or other environments in relation to cardiovascular diseases and other illnesses.

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