Work-Related Stress and Early Atherosclerosis

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The purpose of this study was to examine the link between work-related stress and early atherosclerosis as measured by common carotid artery intima-media thickness and focal lesions in the common carotid artery and bifurcation. Four hundred sixty-seven members of an occupational cohort (total N = 573) were examined via questionnaires and B-mode ultrasound. We used multiple linear and logistic models to regress lesion risk and intima-media thickness on work-related stress scores from a questionnaire administered at an 18-month follow-up examination. In an age-adjusted model, the prevalence of carotid lesions among men scoring in the highest stress quintile was 36% compared with 21% among men in the lowest quintile. We also observed an increase in intima-media thickness in the highest quintile relative to the lowest (difference = 0.048 ± 0.025 mm) among men. Among women, stress was not related to the prevalence of lesions or intima-media thickness. These findings suggest that men with greater work-related stress are at increased risk for atherosclerotic disease. Women in this age group may be protected from such effects, or current work-place questionnaires may not accurately assess stress in women. (Epidemiology 2001;12:180–185)

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A link between psychosocial stress and the etiology of atherosclerotic cardiovascular disease remains controversial.[1] Prospective cohort studies relating occupational stress to coronary heart disease provide mixed results.[2,3] Few studies have addressed the relation between subclinical atherosclerosis and work-place stress. One such study reported cross-sectional data from the Atherosclerosis Risk in Communities (ARIC) study,[4] and another reported longitudinal evidence from a cohort of Finnish men.[5]

Recent evidence suggests that the incidence of coronary heart disease (CHD) may not be declining in the United States,[6] even as major risk factors such as smoking and serum cholesterol have been declining.[7] The lack of overall decline may reflect concurrent increases in other causal factors, such as work-related stress, especially in an economy undergoing rapid transition and increased workloads. The present study examines the association between work-related stress and subclinical carotid atherosclerosis in an occupational cohort of men and women.

Subjects and Methods

Cohort

Participants were from a cohort of 573 employees of a utility company who were asymptomatic for cardiovascular disease and age 40–60 years at entry. Employees were randomly sampled within strata of sex, age, ethnicity, and smoking status; Hispanics and smokers were oversampled. The purpose of the oversampling was to improve precision assessing differences between ethnic groups and smoking-status groups. Measurement of stress was introduced at the 18-month follow-up examination. All measures used in the cross-sectional analyses presented here are from that examination. The participation rate at baseline was 85%, and the follow-up rate at 18 months was 84%. The resulting study population for the present analyses comprised 218 women and 249 men with complete data for the stress measure and covariates.

Measurement of Work-Related Stress

Stress was measured using a self-administered, six-item questionnaire concerning work-place demands and intrusion of work concerns into home life. The items were scored using a 5-point scale (from "rarely" or "never" to "very often" or "all of the time, every day"). The average of items answered was computed. The items were the following. (1) "How often, in the last year, has there been a great deal to do on your job?" (2) "How often is there a marked increase in your work load?" (3) "How often is there a marked increase in the amount of

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concentration required on your job?" (4) "Do you leave your office to eat lunch?" (5) "Do you ever have difficulty sleeping because you are thinking about work-related concerns?" (6) "Do work concerns weigh on your mind at home?" The first three questions are intended to measure the perceived "demand" aspect of work situations. These items are similar to those in the "job strain" model proposed by Karasek *et al*.[8] The remaining three questions reflect an employee's inability to separate the work-life sphere from that of home life, similar to the "intrinsic effort" construct proposed by Siegrist[9] as part of the "effort-reward imbalance model."[10] As a whole, the resulting index assesses the psychological burden that occupational demands place on workers.

The reproducibility of the stress index was assessed in a subsample of the cohort (N = 117) with repeated measurement over an 18-month interval. The Spearman autocorrelations were 0.57 for women and 0.41 for men. These results indicate that the index has some stability over time. To the extent that change in the index over this 18-month interval is due to measurement error rather then actual change in work-related stress, there will be regression dilution bias.[11]

Carotid Lesions

The presence of raised lesions in the bifurcation and distal common segments of the left and right carotid arteries was determined from high-resolution B-mode ultrasound images. A suspected lesion was identified by a first reader. A cardiologist (CNBM) reviewed these suspected lesions, using methods similar to those of the ARIC investigators,[12] and a final decision was based on consensus between the reader and the cardiologist.

The visual criteria for determining a lesion were any one of the following: (1) wall irregularity (luminal protrusion, irregularity of the border, or loss of alignment), (2) wall heterogeneity (brighter or less bright echoes compared with surrounding walls, or acoustic shadowing), or (3) wall slope (greater than 30-degree increase from intima wall). Most identified plaques met multiple criteria.

Intima-Media Thickness

Image-acquisition and image-processing procedures for measurement of intima-media thickness (IMT) in this cohort have been described previously.[13] Briefly, IMT was ascertained from videotaped B-mode ultrasound scans. IMT was estimated as an average over 1-cm segments of the posterior (far) wall of the left and right common carotid arteries. The Prosound software developed by Robert Selzer (Jet Propulsion Laboratory, Pasadena, CA) was used to measure IMT with an automated edge-tracking algorithm.[13]

For comparison with results from a Finnish cohort that focused on relations between stress and atherosclerosis,[5] we also conducted analyses on the variable those researchers labeled "plaque height." Measurements of IMT in the Finnish study also used the Prosound software, and the output from this program includes the maximum and minimum IMT along a 1-cm segment. Lynch *et al*[5] refer to the difference between the maximum and minimum IMT as plaque height.

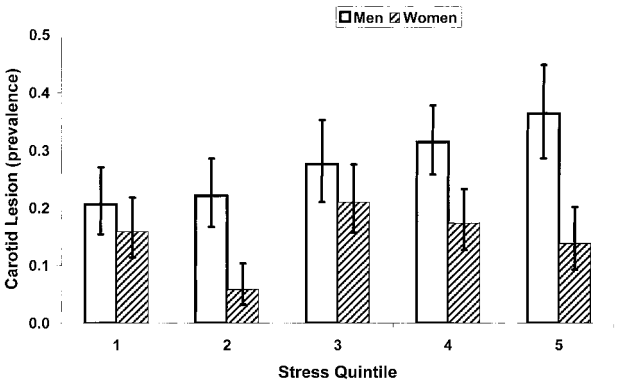

FIGURE 1. Age-adjusted prevalence of carotid lesions by quintile of work-related stress (The Los Angeles Atherosclerosis Study).

Statistical Procedures

We measured the prevalence of a lesion in either the left or right carotid artery in relation to the continuous stress index using an age-adjusted logistic regression model within sexes (Figure 1). For graphic presentation, the prevalence of lesions was estimated within sex-specific quintiles of the stress index from the logistic regression results.

We evaluated the association between IMT and work-related stress using multiple linear regression and adjusting for age and body height.

Results

Characteristics of the study group are presented in Table 1. Note that the mean and standard deviation of the stress index are comparable for women and men. Owing to oversampling, nearly a third of the sample are Hispanic, and more than 60% of men and nearly half of women are either current or former smokers. Relations between the continuous index of work-related stress and other study variables are given by sex in Table 2. Pearson correlations and unstandardized regression coefficients are given for continuous variables, and group means (and adjusted group means) are presented for categorical variables. In women, standard risk factors for cardiovascular disease tend not to show a positive correlation with the stress index. Age, body mass index, and systolic blood pressure show an inverse relation among women, whereas the protective high-density lipoprotein cholesterol is positively correlated with stress. Female smokers have slightly elevated stress levels, but these differences are largely explained by other covariates in the multivariate analysis.

In men, stress tends to show a pattern of association more suggestive of an adverse impact of stress on cardiovascular risk or of confounding between stress and some of these factors. Although stress does not increase

TABLE 1. Characteristics of the Study Cohort: The Los Angeles Atherosclerosis Study

Variable	Men (N = 249)		Women (N = 218)	
	Mean	SD	Mean	SD
Carotid IMT (mm)	0.69	0.12	0.67	0.10
Stress index (1–5)	3.0	0.7	2.9	0.9
Height (m)	1.8	0.1	1.6	0.1
Age (years)	50.0	4.6	52.8	4.4
Physical activity (times per week)	2.4	2.3	1.9	2.1
Alcohol intake (gm/day)	8.6	12.6	4.3	9.1
BMI (kg/m^2)	28.8	4.6	27.5	5.8
SBP (mmHg)	128.4	13.5	126.9	15.1
Total cholesterol (mg/dl)	175.1	40.8	156.3	41.5
HDL-C (mg/dl)	43.8	13.5	58.3	14.7
Carotid lesion (%)	25.7		19.7	
Race/ethnicity (%)				
Black	4.0		6.8	
Asian	5.6		10.1	
Latino	32.5		27.1	
Other	3.6		1.4	
non-Hispanic white	54.3		54.6	
Job category (%)				
Unionized	8.8		4.1	
Management	56.2		32.1	
Administrative	9.6		28.0	
Other	25.3		35.8	
Smoking status (%)				
Current	26.9		20.2	
Former	34.5		28.0	
Fat intake (% energy)	31.8		31.4	
Users of BP meds (%)	8.4		8.7	
Users of chol meds (%)	6.0		3.7	
Diabetes mellitus (%)	1.6		2.8	

SD = standard deviation; IMT = intima-media thickness; BMI = body mass index; SBP = systolic blood pressure; HDL-C = high-density lipoprotein cholesterol; BP meds = hypotensive medications; chol meds = cholesterol-lowering medications.

with age, it does show a positive association with body mass index and an inverse association with two protective factors among men (physical activity and high-density lipoprotein cholesterol).

The strongest association with the stress index is found for job category. Those persons in the "other" category reported lower stress levels, but job category was unrelated to lesion risk or IMT.

Carotid Lesions

Carotid lesions were detected in 25.7% of men and 19.7% of women. Among men in the lowest quintile of the stress measure, 20.7% had carotid lesions, compared with 36.4% of men in the highest quintile of stress, using an age-adjusted model. There was a strong linear trend across quintiles of the stress measure (β = 0.829 ± 0.425).

Among women, there was no pattern of association between work-place stress and the prevalence of carotid lesions. Fourteen per cent of women in the lowest quintile of stress had lesions compared with 10.4% of women in the highest quintile. There was no evidence of any important linear trend across quintiles of the stress measure (β = 0.223 ± 0.516).

To investigate the potential role of hormones in the response of women to work-related stress, a subgroup analysis was performed among women with a history of hysterectomy and bilateral oophorectomy (N = 55). After adjusting for age and height, lesion risk showed some relation to work-related stress (odds ratio = 2.11; 95% confidence interval = 0.7–6.6). Among women who had not undergone this surgery, the relation between lesion risk and stress remained close to the null (odds ratio = 0.9; 95% confidence interval = 0.6–1.6).

Carotid IMT

A model regressing IMT on work-related stress and controlling for age and body height showed a trend toward a relation between work-place stress and IMT for men in the 90th percentile of the stress measure compared with those scoring at the 10th percentile (β = 0.043 ± 0.023 mm).

Among women, there were no apparent relations between work-place stress and IMT in age-adjusted or fully adjusted models. In a subgroup analysis of women with hysterectomy and bilateral oophorectomy, we found no association between IMT and stress in a regression model controlling for age and height (β = 0.002 ± 0.021 mm). Women who had not undergone this surgery evinced a slightly negative association between work-place stress and IMT (β = −0.010 ± 0.009 mm). The analysis of maximum minus minimum IMT ("plaque height") yielded no apparent association in women or men. For women, a regression model adjusted for age and height yielded a coefficient of 0.0001 ± 0.017 mm for a comparison of the 90th with the 10th stress percentiles. The findings for men were also close to the null (β = 0.001 ± 0.021 mm).

Discussion

These results indicate that middle-aged men who report more work-related stress had an increased likelihood of carotid lesions and common carotid artery (CCA) wall thickening than did men who report less stress. Previous research has considered the relation between job strain and clinical cardiovascular disease. The results are inconclusive. Johnson *et al*[2] reported that Swedish men with long-term exposure to low job control had an elevated risk for cardiovascular mortality compared with men with high-control jobs (as determined using a work-organization exposure matrix). Other measures of job strain were either not associated with mortality (physical job demands) or were unexpectedly protective (psychological job demands) during the 14-year follow-up period. Interestingly, social support at work appeared to moderate the effect of job control on cardiovascular mortality in this sample. Those workers reporting extensive periods of low control and low

TABLE 2. Magnitude of Associations between Stress Index and Each Covariate: Continuous Covariates Are Related Using Correlation and Multiple Regression; Means and Least-Square Adjusted Means of the Stress Index Are Reported for Categorical Covariates

	Women		Men	
	Correlation with Stress Measure	Regression Coefficients	Correlation with Stress Measure	Regression Coefficients
Height (m)	0.07	0.129 ± 0.917	−0.07	−0.851 ± 0.667
Age (years)	−0.30	−0.028 ± 0.015	−0.25	−0.006 ± 0.011
Physical activity (times per week)	−0.02	0.016 ± 0.029	−0.10	−0.033 ± 0.019
Fat intake (% energy)	−0.01	0.001 ± 0.01	0.00	−0.003 ± 0.006
Alcohol Intake (gm/day)	−0.02	−0.009 ± 0.006	0.06	0.005 ± 0.004
BMI (kg/m^2)	−0.13	0.007 ± 0.011	0.13	0.018 ± 0.010
SBP (mmHg)	−0.06	−0.0001 ± 0.004	−0.07	−0.003 ± 0.003
Total cholesterol (mg/dl)	0.02	0.0002 ± 0.0014	0.03	−0.0003 ± 0.0011
HDL-C (mg/dl)	0.13	0.007 ± 0.004	−0.07	−0.004 ± 0.003

	N	Means*	Adjusted Means*	N	Means	Adjusted Means
Categorical variables						
Ethnicity						
Native American	0	NA	NA	2	2.75 ± 0.52	2.65 ± 0.46
Asian/Pacific Islander	27	2.80 ± 0.19	2.76 ± 0.19	14	2.97 ± 0.20	2.91 ± 0.19
Black	18	2.78 ± 0.25	2.82 ± 0.21	13	2.77 ± 0.22	2.85 ± 0.21
White	149	2.97 ± 0.08	2.99 ± 0.08	166	3.04 ± 0.06	2.99 ± 0.06
Latino	72	2.91 ± 0.12	2.84 ± 0.11	100	3.06 ± 0.08	3.10 ± 0.08
Other	3	3.00 ± 0.53	2.95 ± 0.47	9	3.43 ± 0.28	3.34 ± 0.25
Job category						
Union	9	2.93 ± 0.26	3.10 ± 0.28	24	3.06 ± 0.14	3.13 ± 0.15
Management	72	3.41 ± 0.09	3.31 ± 0.10	145	3.28 ± 0.06	3.25 ± 0.06
Administration	64	3.16 ± 0.10	3.16 ± 0.11	24	2.99 ± 0.14	3.01 ± 0.14
Other	81	2.30 ± 0.09	2.36 ± 0.10	66	2.51 ± 0.08	2.48 ± 0.09
Education						
Some high school	0	NA	NA	3	3.22 ± 0.42	3.18 ± 0.47
Completed high school	45	2.90 ± 0.14	2.78 ± 0.14	23	2.78 ± 0.17	2.72 ± 0.16
Vocational training	12	2.02 ± 0.30	2.44 ± 0.29	6	2.56 ± 0.30	2.65 ± 0.28
Some college	135	2.94 ± 0.08	2.95 ± 0.08	142	3.12 ± 0.07	3.09 ± 0.07
Completed college	52	2.93 ± 0.13	2.91 ± 0.13	69	3.02 ± 0.09	3.01 ± 0.09
Graduate school	25	3.26 ± 0.19	3.18 ± 0.18	61	3.02 ± 0.10	3.04 ± 0.10
Smoking status						
Current	48	3.11 ± 0.13	2.98 ± 0.13	70	3.04 ± 0.09	3.12 ± 0.09
Former	61	2.81 ± 0.12	2.85 ± 0.11	91	3.06 ± 0.08	3.08 ± 0.07
Never	117	2.91 ± 0.08	2.92 ± 0.08	98	3.02 ± 0.08	2.91 ± 0.07
Blood pressure medications						
No	204	2.92 ± 0.06	2.89 ± 0.06	235	3.05 ± 0.05	3.03 ± 0.04
Yes	21	3.01 ± 0.20	3.16 ± 0.20	24	2.92 ± 0.15	2.94 ± 0.15
Cholesterol medications						
No	217	2.92 ± 0.06	2.91 ± 0.05	242	3.05 ± 0.05	3.03 ± 0.04
Yes	9	2.96 ± 0.30	2.91 ± 0.30	17	2.88 ± 0.18	2.97 ± 0.18
Diabetes mellitus						
No	263	2.93 ± 0.06	2.92 ± 0.05	300	3.04 ± 0.05	3.02 ± 0.04
Yes	6	2.78 ± 0.37	2.71 ± 0.36	4	2.75 ± 0.37	3.10 ± 0.33

BMI = body mass index; SBP = systolic blood pressure; HDL-C = high-density lipoprotein cholesterol; NA = not applicable.
* ± standard error.

social support had worse outcomes when compared with those with long periods of high control coupled with high social support.

Hlatky *et al*[14] found that self-reported job strain was most likely to occur in those with normal coronary arteries compared with those with disease, regardless of severity (as assessed by coronary angiography). Furthermore, job strain was not associated with angina frequency and was not a predictor of either nonfatal or fatal myocardial infarctions during follow-up.

Reed *et al*[3] found little association between CHD incidence and either job demands, job control, or the interaction of these two factors (job strain) in the Honolulu Heart Program. In fact, their results show trends suggesting that high strain is inversely related to the development of CHD in men of Japanese ancestry living in Hawaii.

Lynch *et al*[15] reported that the detrimental effects of work-place conditions (job demands and available resources) on cardiovascular mortality depended on the level of economic reward in a prospective study conducted in Eastern Finland. Those in low-paying jobs experienced the greatest age-adjusted relative hazards, regardless of the level of demands and resources. Elevated risk of acute myocardial infarction, however, was found only in men with high demands, low resources, and low income. Much of the excess risk found appeared to be mediated by known risk factors.

Taken together, these studies do not show consistent findings when we examine the association between job

strain (or its components) and atherosclerosis, nonfatal CHD events, or coronary mortality. Several different approaches to assessing work-related stress were used in the different studies. These differences include the important distinction between stress as inferred from formal job characteristics, those derived from observation of the work place, and those derived from self-reports of job characteristics or perceived outcomes of stress. Thus, even if there is an effect of some of the types of work-related stress on clinical CHD risk, it is not surprising that results across existing studies are inconsistent.

The relation between occupational stress and subclinical carotid atherosclerosis has been investigated in two other cohorts: a large U.S. cohort of black and white women and men (ARIC)[4] and a Finnish cohort of men.[5] The ARIC investigators found cross-sectional associations in the expected direction between some measures of stress (assessed primarily through occupational characteristics) and carotid IMT. These associations, however, were not found in all four sex-race groups, and they were substantially diminished by adjustment for potential confounders. In terms of sex differences, the ARIC investigators found that job insecurity was positively related to IMT among white women and black men only, and decision authority was related only in men. The current study did not find the relation between work-related stress and IMT in men to be diminished when adjusted for possible confounding variables, including known atherosclerotic risk factors.

The Finnish study by Lynch *et al*[5] related work-place demands and income to longitudinal change in IMT and plaque height in men. (Plaque height is the maximum IMT along a 1-cm segment, as opposed to the average IMT over that segment.) In contrast to the use of occupation to assign stress scores in ARIC, the stress measure in the Finnish study was self-reported (as in our study). Lynch *et al*[5] found an interaction between work-place demands and economic reward with progression of maximum IMT and plaque height, but not with progression of average IMT. Their findings were strongest for plaque height. Our cross-sectional findings were strongest for presence of lesions and average IMT. We found little relation between stress and plaque height. Furthermore, adjustment for education and job status (management, administration, union, or other) as a rough control for socioeconomic status (similar to economic reward) does not dampen the relation between stress and IMT. Our analyses, however, were cross-sectional, whereas that of the Finnish study was longitudinal.

Comparison across studies is hampered by differences in the measurement of work-place stress. Our own measure focused on discrete consequences of stress (for example, difficulty sleeping), the Finnish study relied on ratings of the stressful nature of work, and the ARIC study used characteristics of occupational categories. Differences among these measures need to be explored.

The cohort used in our analyses was different from those in other studies in that all members were employed by the same company at baseline. This design feature offers control over a number of factors (for example, health benefits, retirement plans, and vacation policies) that may vary between similar positions at different companies.

In terms of ultrasound measurement methods, the ARIC study assessed maximum IMT across the near and far walls of the CCA, bifurcation, and internal carotid artery. Our IMT measure was restricted to the far wall of the CCA, as was the measure reported by Lynch and colleagues.

One limitation of our study is the reliability of self-reported stress levels. Ideally, a combination of objective and subjective measures of stress would be used. Given that the study population was asymptomatic for cardiovascular disease, however, an effect of disease on perceptions is precluded. Although categorization by occupation may circumvent potential self-report errors, self-reports from individuals within job titles may yield better estimates of the actual psychological impact of work-place stressors on individuals.

It is possible that some factor, such as level of endogenous estrogen or other hormones, protects women from the atherogenic effects of stress that were observed in men. Analyses of data from women with bilateral oophorectomy suggested a positive association between stress and prevalence of carotid lesions in the small sample of oophorectomized women. This finding provides some weak support for a model in which premenopausal women are protected by hormonal factors against the atherogenic effects of stress.

We do not find that women and men differ in average levels of work-place stress, as assessed by our scale. Only 32% of women, however, are in management positions, compared with 56% of men. Conversely, 28% of women are in administrative positions, compared with only 10% of men. Additional factors associated with the type of position held, such as those put forth in the job strain[8] or effort-reward imbalance[10] models of job stress, may influence our results and the discrepancies between women and men in this sample.

Sex differences in other psychosocial variables not assessed in the current study, such as social support, could also explain the observed sex difference in response to stress. Increased social support among women, for example, may dampen the atherogenic response to stress.

Similarly, work-place stress may not be an independent factor among women. Psychological stress experienced by women may be a combination of stress occurring at work and stress occurring at home, whereas men may respond specifically to stress experienced at work.[16] Additional measures of home stress may be needed to better assess effects of overall stress among women. This interpretation is supported by the finding in primates that stress promotes atherosclerosis in both males and females.[17,18]

The finding that work-related stress was more strongly related to lesions than to IMT in men may be due to chance. It is also possible that IMT and intrusive lesions reflect earlier and later stages of the atherosclerotic process, respectively, and that work-related stress is more

strongly related to the later stages of atherosclerosis. If this is the case, then efforts to reduce stress levels among men with relatively thickened intima-media complexes may attenuate the later development of focal plaques. This hypothesis is consistent with the theory that factors other than low-density lipid oxidation may play a role in the later stages of atherosclerosis (that is, plaque formation).[12] Also, stress combined with elevated blood pressure reactivity may favor plaque rupture, producing more intrusive lesions rather than just increased IMT.

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