Workplace Demands, Economic Reward, and Progression of Carotid Atherosclerosis

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Background Characteristics of the work environment have been associated with cardiovascular morbidity and mortality, but it is unclear whether these factors are associated with preclinical manifestations of disease.

Methods and Results We investigated the association between job demands, economic reward, and the 4-year progression of carotid atherosclerosis in a population-based sample of 940 Finnish men. Data from the Kuopio Ischemic Heart Disease Risk Factor Study were used to estimate changes in plaque height, maximum and mean intima-media thicknesses across combinations of job demands, and income. Associations were examined in relation to atherosclerotic risk factors and were stratified by baseline levels of atherosclerosis and prevalent ischemic heart disease. Men who had jobs with high demands and low economic rewards had significantly greater 4-year progression of plaque height (0.33 mm, $P=.008$) and maximum intima-media thickness (0.32, $P=.03$) than men with low-demand, high-income jobs. The magnitude of these differences was not greatly attenuated by risk factor adjustment and did not differ when examined by the level of workplace resources, social support, or employment status. Larger differences were observed in a subsample of men who had more advanced atherosclerosis at baseline.

Conclusions These results show that men with demanding work that produces little economic reward have significantly greater progression of carotid atherosclerosis than more advantaged men. The relationship between job demands and health should be understood in a broad framework of interacting economic conditions, social circumstances, and behaviors that cascade over the life course and may ultimately contribute to socioeconomic inequalities in morbidity and mortality. (***Circulation*. 1997;96:302-307.**)

Key Words • work • income • stress • atherosclerosis • ultrasound

Reports about the effects of poor working conditions on health date back to at least the 12th century, when in medieval Europe, Paracelsus noted unusually high rates of disease in miners.[1] In England, Engels[2] argued that employment in the factories of the industrial revolution had deleterious health consequences for the working class. More recently, studies have shown that organizational, psychological, and social features of the work environment are associated with cardiovascular morbidity and mortality.[3,4] However, it is unclear whether this association is apparent only late in the natural history of cardiovascular disease, is caused by selection factors, or is the result of precipitation of clinical manifestations. Currently, few data address whether workplace factors are important in the underlying process of atherosclerosis itself.[5] Ultrasound scanning of the carotid arteries has enabled the noninvasive study of the prevalence and development of atherosclerosis in population groups.[6,7] B-mode imaging of intima-media thickness (IMT) in the carotid arteries is a valid indicator of generalized atherosclerosis, is a reliable method that correlates well with the extent of disease in the coronary arteries, and has predictive validity with regard to risk of coronary and cerebrovascular events.[7-13]

This study is, to the best of our knowledge, the first to examine the association between workplace factors and the 4-year progression of carotid atherosclerosis using three indicators of the atherosclerotic process: maximum IMT, plaque height, and mean IMT. In earlier studies, we reported both cross-sectional[14] and prospective[15] inverse associations between measures of socioeconomic status and IMT. It is also possible that the level of economic rewards potentiates the impact of work demands. In light of these findings and the fact that low socioeconomic status groups are likely to have higher lifetime exposure to poor working conditions, we assessed the interaction of job conditions and socioeconomic status (measured by income) on IMT progression. Extensive information on baseline levels of atherosclerotic risk factors and prevalent disease enabled the association between workplace demands, economic reward, and atheroslerotic progression to be examined with adjustment for known risk factors and stratified by both prevalent ischemic heart disease (IHD) and the extent of atherosclerosis at baseline.

Methods

Subjects were participants in the Kuopio Ischemic Heart Disease Risk Factor Study, which was designed to investigate previously unestablished risk factors for IHD, carotid atherosclerosis, and related outcomes in a population-based sample of eastern Finnish men.[16] Of the 3433 eligible men who were 42, 48, 54, or 60 years of age and resided in the town of Kuopio or its surrounding communities, 198 were excluded because of

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death, serious disease, or migration from the area. Of the remainder, 2682 (82.9%) agreed to participate in the study. Baseline examinations were conducted between March 1984 and December 1989. No marked sociodemographic differences have been found between participants and nonparticipants.[17]

At baseline, subjects were recruited in two waves. The first group comprised 1516 men 54 years of age; the second comprised an age-stratified sample of 1116 men who were 42, 48, 54, or 60 years of age. Ultrasonographic assessment of carotid atherosclerosis at baseline was conducted between February 1987 and December 1989 on the second wave of participants only. There were no systematic differences between the two waves of recruitment for the study other than the differences in age distribution. A 4-year follow-up examination was conducted between March 1991 and December 1993 on those men who had previously undergone ultrasonographic examination of the carotid arteries at baseline.

Follow-up examinations were conducted during the same month as the baseline and at the same time of day. Mean follow-up was 4.2 years (range, 3.9 to 4.7 years). Of the 1229 participants who were eligible for the follow-up exams, 52 had died, were suffering from severe illness, or had migrated from the area. Of the remainder, 139 could not be contacted or refused to participate. Information on workplace demands, economic reward, progression of carotid atherosclerosis, and covariates was available for 940 men. There were 221, 228, 258, and 233 men in the 42-, 48-, 54-, and 60-year-old age groups, respectively. In this sample, 71 men acted as control subjects, and 68 men were participants in the treatment group of an unrelated clinical trial of pravastatin.[18]

Assessment of Carotid Atherosclerotic Progression

Atherosclerotic progression was assessed with high-resolution B-mode ultrasonographic scanning of a 1.0- to 1.5-cm section of the left and right common carotid arteries (CCAs) below the carotid bulbs. Images were focused on the posterior (far) wall with the subject supine. At baseline, ultrasonographic scanning was conducted by use of the ATL UM4 duplex ultrasound system with 10-MHz sector transducer (Advanced Technology Laboratories). The Biosound Phase 2 equipped with an 8- or 10-MHz annular array probe was used at the 4-year follow-up examinations. Wedge phantom studies of this system, calibrated against an RMI 414B tissue phantom, have demonstrated measurement precision of ± 0.03 mm.[8] A total of five technicians, each trained for at least 6 months, conducted both the baseline and the 4-year follow-up scannings, which were also recorded on videocassettes.

Video frames of the B-mode scanning were digitized with the Data Translation DT2861 video frame grabber installed on a 80486 PC and a Panasonic AG7355 VCR. IMT was assessed with Prosound software, which incorporates an edge-detection algorithm specifically designed for use with ultrasound scanning and enables automatic detection, tracking, and recording of the lumen/intima and media/adventitia interfaces (University of Southern California).[19] On average, 100 estimates of the distance between these interfaces were recorded over the 1.0- to 1.5-cm section of each CCA. The IMT of the posterior wall was measured as the distance from the leading edge of the first echogenic line to the leading edge of the second echogenic line, as explained earlier in detail.[7] Measurements of the near wall were not conducted because of greater measurement variability.[20]

The present study uses three measures of IMT. Maximum IMT was defined as the average of the maximum IMT in the right and left CCAs. Plaque height was defined as the difference between the maximum and minimum IMT recordings averaged over the right and left CCAs, and mean IMT was defined as the mean of the ≈100 IMT readings from each CCA. These measures were conceptualized to represent potentially different aspects or stages of atherosclerotic progression. Maximum IMT was thought to provide an assessment of how deeply the greatest lesion intruded into the lumen in this segment of the CCA. The measurement of plaque height was conceptualized to be sensitive to the roughness of the arterial wall by representing the range of IMT and thus assessing how steeply atherosclerotic lesions protruded into the lumen. Mean IMT was seen as an overall measure of the process of atherosclerosis. Progression of carotid atherosclerosis was calculated as the arithmetic difference between the baseline and 4-year follow-up values for each of the three measures: maximum IMT, plaque height, and mean IMT.

Baseline IMT recordings were also classified by one physician into four categories: (1) no atherosclerotic lesion, (2) intima-media thickening, (3) nonstenotic plaque, and (4) large, stenotic plaque. "Intima-media thickening" was defined as >1.0 mm between the lumen-intima interface and the media-adventitia interface in the CCAs below the carotid bulbs. Nonstenotic plaque was defined as a distinct area of mineralization or focal protrusion into the lumen. A plaque was defined as stenotic if it obstructed >20% of the lumen diameter.[7]

Assessment of Workplace Demands and Economic Reward

At the baseline examinations, participants completed detailed questionnaires that included items on aspects of their work environment and income. Items that conformed to important theoretical domains discussed in the literature were considered for inclusion in the measurement of workplace demands.[4] In accordance with suggestions made in this literature, items on risk of unemployment, accidents, and physical exertion were included to supplement the questions about psychological demands. Participants were asked to rate on a Likert-type scale (0 to 4) how much mental strain or stress each of the following things caused them at work: excessive supervision of time schedules, troublesome supervisors, troublesome fellow workers, job responsibility, poorly defined tasks and responsibilities, risk of accidents, risk of unemployment, irregular work schedules, and the mental strain of work. They also were asked how often they had work deadlines and how much stress this caused them and were requested to rate the physical strain of their work. Scores for the demands scale were imputed on the basis of an average of nonmissing values for men who had no more than two missing items (n=63). Men who had more missing information than this were excluded from the analyses (n=42). The 11 individual items were dichotomized at the midpoint of the rating scale so that only when men reported that the particular aspect of work caused them more than "average" strain or stress were their responses considered positive. The 11 dichotomized items were then summed to form the workplace demands scale, which had high internal consistency (Cronbach's α=0.78). The scale was dichotomized so that men in the top 30% of total workplace demands were considered "high." In previous analyses, this dichotomization had been shown to predict mortality and acute myocardial infarction in this population.[21] Economic reward was assessed by self-reported income, which was dichotomized so that the lowest 20% of income earners were considered "low." Previous analyses in the same population had shown that men in the bottom quintile of the income distribution had accelerated progression of atherosclerosis[15] and also were at greatly elevated risk of mortality and acute myocardial infarction.[22]

Assessment of Covariates

Lipoproteins were separated from unfrozen plasma within 3 days of sampling. HDL and LDL fractions were separated from fresh plasma by use of both ultracentrifugation and precipitation. The cholesterol content of all lipoprotein fractions was measured enzymatically (CHOD-PAP cholesterol method, Boehringer Mannheim) on the day after the last spin.[23] Blood pressure was measured with a random-zero sphygmomanometer with participants in both supine and sitting positions after 5-

TABLE 1. Workplace Demands, Economic Reward, and Mean or Prevalence of Selected Baseline Covariates in 940 Finnish Men

	High Work Demands			Low Work Demands		
	Low Income (n=87)	High Income (n=239)	P*	Low Income (n=84)	High Income (n=530)	P*
Age, y	53.5	51.1	.003	54.9	50.3	<.001
Completed high school, %	3.5	16.7	.002	3.6	27.6	<.001
LDL, mmol/L	3.87±0.92	3.88±0.90	NS	4.02±1.13	3.82±0.94	.08
HDL, mmol/L	1.28±0.28	1.30±0.29	NS	1.32±0.33	1.30±0.29	NS
Systolic blood pressure, mm Hg	133.4±16.4	132.5±16.3	NS	131.1±18.5	131.9±15.4	NS
Alcohol, g/wk	81.7±138.7	81.1±124.8	NS	69.6±128.2	77.1±121.6	NS
Serum triglyceride, mmol/L	1.58±0.91	1.44±0.79	NS	1.39±0.89	1.41±0.91	NS
Body mass index, kg/m²	26.6±3.3	26.7±3.2	NS	25.9±3.6	26.8±3.3	.03
Baseline maximum IMT, mm	1.04±0.29	0.97±0.30	.07	1.00±0.22	0.92±0.19	<.001
Baseline mean IMT, mm	0.82±0.21	0.78±0.21	.07	0.81±0.17	0.74±0.14	<.001
Baseline plaque height, mm	0.43±0.21	0.40±0.22	NS	0.41±0.16	0.36±0.14	.01
Smoking						
Nonsmokers, %	20.7	25.9	NS	22.6	28.1	NS
Former	33.3	39.9	NS	40.5	41.1	NS
Current, pack-y	577.1±295.7	561.9±345.1	NS	578.3±344.8	549.7±303.7	NS
Other cholesterol medication	3.5	1.7	NS	1.2	0.4	NS
Treatment for hypertension	33.3	20.9	.02	25.0	17.6	.10
History of ischemic heart disease	40.2	26.8	.02	32.1	15.1	<.001
Stenosis or nonstenotic plaque	49.4	28.0	<.001	45.2	24.9	<.001

IMT indicates intima-media thickness. Values are mean±SD when appropriate; prevalence is given in percentage.
*As determined by Student's t or the χ^2 test for the difference between low and high income groups in each work demand category.

minute rests in each position. Three systolic and diastolic pressures were taken and averaged. Average systolic pressure was used in this analysis. Body mass index was calculated by dividing the subject's weight by the square of his height (kilogram per square meter). Alcohol consumption was assessed by instructed dietary recording for a 4-day period and for the previous 12 months by a self-administered questionnaire.[24] Smoking was measured by questionnaire, and participants were classified for this analysis as never smoked, former smoker, and current smoker (measured in pack-years). Treatment for hypertension or hyperlipidemia was assessed by a review of medications.

Assessment of Prevalent IHD

Subjects were considered to have prevalent IHD at baseline if they had any history of prior myocardial infarction or angina pectoris, currently used antianginal medication, or had positive findings of angina from the London School of Hygiene Cardiovascular Questionnaire.[25]

Statistical Methods

The association between workplace demands, economic reward, and progression of IMT was assessed by estimating the mean change in each measure of IMT (maximum thickness, mean thickness and plaque height) for each combination of demands and economic reward. The analyses were conducted by use of the GLM procedure in SAS version 6.09 on a Sun Sparc Station II.[26] This procedure allows age-adjusted, least-squares mean values of IMT to be estimated and contrasted for each combination of demands and economic reward while simultaneously controlling for baseline IMT and other covariates.

We were interested in comparing differences in mean IMT progression between the low-demand, high-income group (reference category) and the low-demand, low-income; high-demand, high-income; and high-demand, low-income groups. These three pairwise contrasts were adjusted for multiple comparisons with Holm's procedure as described by Aickin and Gensler.[27] In addition to age, baseline levels of IMT, and covariates, all estimates were adjusted for participation in the clinical trial of pravastatin, the zooming depth of the ultrasound scan, and separate indicator variables for the individual technicians who conducted the scans.[7]

Results

Table 1 provides mean±SD and prevalence (percent) for baseline levels of IMT and other covariates distributed across categories of workplace demands and economic rewards. Table 2 presents the estimated mean changes in maximum thickness, plaque height, and mean thickness of the intima-media complex by work demands and income, with adjustment for age and baseline levels of IMT (model 1) and further adjustment for covariates (HDL, LDL, triglycerides, systolic blood pressure, smoking, body mass index, alcohol consumption, and treatment for hyperlipidemia and hypertension) in model 2.

Workplace Demands, Economic Reward, and IMT Progression

The impact of workplace demands on atherosclerotic progression of maximum IMT and plaque height depended on the level of economic rewards (see the Figure). There were important interactions between workplace demands and economic rewards in relation to the progression of maximum IMT (cross-product interaction term, $P=.18$) and plaque height (cross-product interaction term, $P=.02$), but this effect was not observed for changes in mean IMT. Men with high levels of workplace demands and low economic rewards had significantly greater average progression in the maximum wall thickness of the CCA (0.32 mm, $P=.03$) compared with men with low demands and high economic rewards (0.26 mm). Men with high demands and low economic rewards also had significantly greater progression of plaque height (0.33 mm, $P=.008$) compared with men with low demands and high incomes (0.27 mm). These patterns were not evident in relation to changes in mean IMT, for which there was no interaction between workplace demands and economic

TABLE 2. Workplace Demands, Economic Reward, and Mean Changes in Three Measures of IMT in 940 Finnish Men

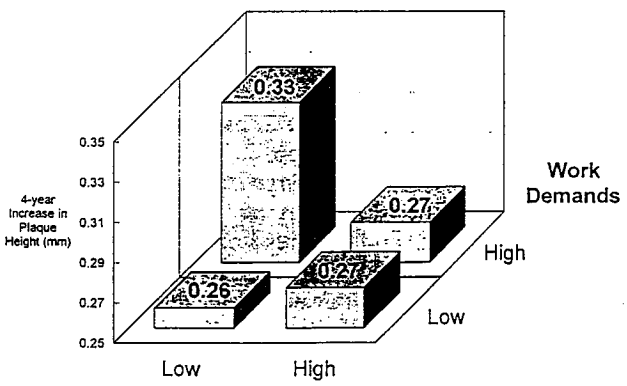
Demands	Income	n	Model 1, Adjusted for Age and Baseline IMT		Model 2, With Additional Adjustment for Covariates	
			Mean±SE	P*	Mean±SE	P*
Change in maximum IMT, mm						
High	Low	87	0.32±0.021	.03	0.31±0.021	.13
High	High	239	0.26±0.012	NS	0.26±0.013	NS
Low	Low	84	0.27±0.022	NS	0.27±0.021	NS
Low	High	530	0.26±0.009	Reference	0.26±0.009	Reference
Change in plaque height, mm						
High	Low	87	0.33±0.019	.008	0.32±0.020	.04
High	High	239	0.27±0.012	NS	0.27±0.012	NS
Low	Low	84	0.26±0.020	NS	0.25±0.020	NS
Low	High	530	0.27±0.008	Reference	0.27±0.008	Reference
Change in mean IMT, mm						
High	Low	87	0.14±0.015	.23	0.13±0.014	NS
High	High	239	0.11±0.009	NS	0.11±0.009	NS
Low	Low	84	0.13±0.015	.26	0.14±0.015	NS
Low	High	530	0.11±0.006	Reference	0.11±0.006	Reference

IMT indicates intima-media thickness.
*As determined by pairwise contrast with reference category (adjusted for multiple comparisons). Adjusted for HDL, LDL, triglycerides, smoking, alcohol, body mass index, systolic blood pressure, treatment for hypertension, or hyperlipidemia.

rewards. Low-income men, regardless of their levels of work demands, had a higher mean progression of IMT. The magnitude of the associations between work demands, economic rewards, and progression of maximum thickness and plaque height was largely unaffected by adjustment for atherosclerotic risk factors. In other analyses (not shown), these associations did not differ when examined by the level of workplace resources, social support at work, or employment status.

Workplace Demands, Economic Reward, and IMT Progression Stratified by Prevalent IHD and the Degree of Atherosclerosis at Baseline

Table 3 presents the associations between work demands, economic rewards, and changes in plaque height according to prevalent IHD and the extent of atherosclerosis at baseline. In men who had more advanced atherosclerosis (ie, presence of stenosis ≥20% lumen diameter or evidence of nonstenotic plaque) and prevalent IHD at baseline, the combination of high demands and low economic rewards was associated with the largest mean increases in plaque height (0.40 mm). Similarly, men with high demands and low incomes who had advanced atherosclerotic disease but no history of prevalent IHD at baseline had the largest changes in plaque height (0.40 mm), whereas men with low demands and high incomes had a mean change of only 0.24 mm. There were no differences in progression of plaque height between the demands–economic rewards groups for men who were free of IHD and had no advanced atherosclerotic disease. Although Table 3 presents only data for changes in plaque height, the pattern of results described for the analyses stratified by prevalent IHD and extent of atherosclerosis was generally consistent across the other measures of IMT progression.



Workplace demands, economic reward, and 4-year progression of carotid atherosclerosis (plaque height) in 940 Finnish men.

Discussion

These results show that men whose work has high demands but low economic rewards have significantly greater 4-year progression of carotid atherosclerosis compared with men with low job demands and high incomes. These results are consistent with the "effort-reward imbalance" model developed by Siegrist,[28] which suggests that work that has an imbalance between high demands, high psychological "immersion" in work roles, and low economic and psychosocial rewards is associated with poor health outcomes.

The magnitude of these relationships did not differ by the level of workplace resources and social support or by employment status and was largely unaffected by adjustment for known atherosclerotic risk factors. However, workplace demands and income are associated with baseline levels of IMT, atherogenic risk factors, and changes in those risk factors over time. Analyzing the association of demands and economic reward with changes in carotid atherosclerosis with adjustment for baseline levels of IMT and atherogenic risk factors may

TABLE 3. Workplace Demands, Economic Reward, and Age-Adjusted Mean Changes in Plaque Height in Subsamples of Men With a History of IHD and Advanced Atherosclerosis* at Baseline, With Advanced Disease but No Prevalent IHD at Baseline, and With No Prevalent IHD or Advanced Disease at Baseline

Demands	Income	n (%)	Mean±SE	P†
Advanced atherosclerosis and prevalent IHD (n=109)				
High	Low	22 (20.3)	0.40±.062	NS
High	High	38 (34.9)	0.28±.049	NS
Low	Low	18 (16.5)	0.27±.056	NS
Low	High	31 (28.4)	0.32±.043	Reference
Advanced atherosclerosis and no prevalent IHD (n=171)				
High	Low	16 (9.4)	0.40±.043	.003
High	High	94 (55.0)	0.30±.037	NS
Low	Low	25 (14.6)	0.24±.054	NS
Low	High	36 (21.1)	0.24±.022	Reference
No advanced atherosclerosis and no prevalent IHD (n=563)				
High	Low	41 (7.3)	0.24±.029	NS
High	High	356 (63.2)	0.27±.012	NS
Low	Low	27 (4.8)	0.26±.023	NS
Low	High	139 (24.7)	0.26±.008	Reference

*Advanced atherosclerosis defined as presence of stenosis (≥ 20% lumen diameter) or nonstenotic plaque at baseline ultrasound examination.
†As determined by pairwise contrast with reference category (adjusted for multiple comparisons).

be somewhat problematic. Thus, the lack of confounding effects from the atherogenic risk factors should not necessarily be interpreted as evidence that the association between high workplace demands, low income, and progression of carotid atherosclerosis is independent of these risk factors. Furthermore, we have previously reported that the cross-sectional association between income and carotid atherosclerosis in the same population was largely mediated by a similar group of atherosclerotic risk factors.[14]

Although these findings demonstrated a role for high work demands and low economic reward in the progression of IMT, we conducted stratified analyses to identify whether the associations differed by the extent of atherosclerosis and prevalent IHD at baseline. The combination of high job demands and low economic reward was strongly associated with larger changes in IMT, but only for men who had some degree of advanced atherosclerosis at baseline. In these subsamples, the magnitude of change did not differ by the presence of IHD at baseline. The lack of association between work demands, income, and changes in IMT for men who were free of IHD and had little evidence of atherosclerotic thickening at baseline suggests that the impact of the demands-income imbalance is more important in later stages of the disease process. Although the differential pathological importance of changes in measures of maximum thickness and plaque height remains to be clearly established, it seems reasonable to suggest that high work demands, combined with low economic rewards, are related to the development of focal lesions that protrude into the lumen and increase the surface roughness of the CCAs. The development of roughened arterial walls with steeply sided projections into the lumen can subject the lesion to increased shear stress and flow turbulence, raising the potential for plaque instability, fissuring, and possible rupture.

Several issues must be considered before conclusions can be drawn from these results. First, the measure of workplace demands used in these analyses may have been subject to a reporting bias because it was based on a self-assessment of the extent of stress or strain associated with psychosocial aspects of work. Although the most accurate assessment of job demands would be achieved by a combination of subjective and objective measures, high correlations between subjective assessments and expert ratings of job conditions have been demonstrated.[29] Furthermore, we do not believe that bias in the self-reporting of job demands could explain why greater changes in arterial wall thickness were observed in men who had high demand-low income jobs, even if they were free of IHD at baseline.

Second, the assessment of job demands and economic reward was based on a single measurement and does not take into account changes in job exposures and income over time. Moreover, structural alterations to the Finnish economy have seen large increases in unemployment and changes in the occupational structure of the region both before and during the period of this study.[30] However, our results were unchanged in stratified analyses (not shown) that excluded men who reported any change in job title over the last 10 years and in other analyses that excluded men who were either unemployed or retired at baseline. Third, because our findings are based on a population of men in eastern Finland, they need to be replicated in women.

This study is the first in a large unselected sample to show strong relationships between workplace factors, economic rewards, and progression of atherosclerotic vascular disease. These findings add to the evidence that factors associated with work are important in the development of atherosclerotic vascular disease before the appearance of clinically relevant and more distal manifestations of the disease process. They are entirely consistent with a previous study in this population that demonstrated that the combination of high work demands and low economic reward was prospectively

associated with a twofold increased risk of incident acute myocardial infarction.[21]

The differences in atherosclerotic progression between high-demand, low-income and low-demand, high-income men observed in these data have potentially important clinical and public health interpretations. Although there is little information on the relationship between carotid atherosclerotic progression and clinical events, Salonen and Salonen[7] have demonstrated cross-sectionally that a 0.1-mm difference in maximum IMT was associated with an 11% increased risk of acute myocardial infarction (95% confidence interval, 6% to 16%, $P<.001$).

The evidence presented here indicates that jobs with high demands and low economic rewards are associated with accelerated progression of carotid atherosclerosis. Reducing the atherosclerotic vascular disease burden associated with demanding, low-income work may require interventions that do more than target low-income workers for behavioral modification.[21] Although the intent of these programs is to improve behavioral risk factor profiles, they largely ignore the fact that demanding, low-paying work is only one, albeit important, aspect of life for individuals of low social class. In this light, employment in low-paying, high-demand jobs may well be an important link in a chain of causation between lack of education and increased cardiovascular morbidity and mortality. If poor job conditions are just one of many deleterious exposures for people who receive little education, then we need to understand the relationship between job demands and health in a broad framework of interacting economic conditions, social circumstances, and behaviors that cascade over the life course[31-33] and ultimately result in social class inequalities in morbidity and mortality.

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