Job Strain-Associated Inflammatory Burden and Long-Term Risk of Coronary Events: Findings from the MONICA/KORA Augsburg Case-Control Study.

Abstract:

Background We examined the association between job strain and coronary heart disease (CHD) and investigated the role of markers of inflammation and endothelial dysfunction as possible mediators of job strain-associated CHD risk.

Methods The sample (n = 1027) included employed participants (35-64 years old, 68% male) from the population-based MONICA/KORA (Monitoring of Trends and Determinants in Cardiovascular Disease/Kooperative Gesundheitsforschung in der Region Augsburg) studies. At baseline Karasek’s Job Strain Index was assessed during standardized personal interviews, and nine biological markers were measured (1984-1995). Participants were followed (average, 12 years) to assess incident events (sudden cardiac death or fatal and nonfatal myocardial infarction). In this case-cohort design, the final sample contained 114 cases and 913 noncases.

Results Baseline distributions of cardiometabolic risk factors were significantly different between cases and noncases, with no detectable job strain-specific differences. However, cases with high job strain had higher monocyte chemoattractant protein-1, interleukin (IL)-8, and IL-18 compared with noncases with high job strain. High-sensitivity C-reactive protein,
IL-6, and soluble intercellular adhesion molecule-1 were increased in cases versus noncases, regardless of work stress. Job strain was associated with incident coronary events in Cox proportional hazards models adjusted for age, sex, and survey (hazard ratio = 2.57, 95% confidence interval = 1.09-6.07) and after adjustment for CHD risk factors (2.35, 1.003-5.49). Adjustment for monocyte chemoattractant protein-1 or IL-8 increased this risk estimate by 14.5% or 9.4%, respectively, whereas adjustment for C-reactive protein and soluble intercellular adhesion molecule-1 led to decreased hazard ratios (-9.9% and -5.5%, respectively). Conclusions: Job strain increased CHD risk in healthy workers; the associated inflammatory burden may contribute to stress-related coronary pathogenesis.