J Occup Health 2010; 52: 257–262

Journal of Occupational Health

Job Stress and Carotid Intima-media Thickness in Chinese Workers

Weixian Xu[1], Juan Hang[1], Tingting Cao[2], Rong Shi[2], Wenshuang Zeng[2], Yuanfei Deng[2], Wei Gao[1], Yiming Zhao[3] and Lijun Guo[1]

[1]Department of Cardiology, Peking University Third Hospital and Key Laboratory of Molecular Cardiovascular Science, Ministry of Education, [2]Department of Priority, Peking University Shenzhen Hospital and [3]Research Center of Occupational Medicine, Peking University Third Hospital, China

Abstract: Job Stress and Carotid Intima-media Thickness in Chinese Workers: Weixian Xu, *et al*. Department of Cardiology, Peking University Third Hospital and Key Laboratory of Molecular Cardiovascular Science, Ministry of Education, China—**Objectives:** Carotid intima-media thickness (CIMT) can be used as a surrogate marker for cardiovascular health, and job stress is a risk factor for cardiovascular disease. However, there have been a limited number of studies focusing on the association between job stress and CIMT. The goal of this study was to explore the association between job stress and CIMT in a Chinese working population. **Methods:** The study included 734 participants (508 males and 226 females) without coronary heart disease. Job stress was evaluated using the effort-reward imbalance (ERI) questionnaire at work. ERI is the ratio between efforts and rewards (weighted by number of items). High resolution carotid ultrasonographic studies were performed using a Sequoia 512 ultrasound system with an 8–13 MHz linear array transducer to assess CIMT. **Results:** This study detected gender-specific associations between the indictors of the ERI model and increased CIMT among the study participants in China. This study demonstrated a robust association in women between the key indicators of ERI, effort, overcommitment and ERI, and increased CIMT (adjusted $r^2=0.258$, $p=0.001$; adjusted $r^2=0.261$; $p<0.001$; adjusted $r^2=0.274$; $p<0.001$, respectively). Reward was inversely correlated with CIMT (adjusted $r^2=0.282$, $p<0.001$), controlling for age, hypertension, diabetes mellitus, hyperlipidaemia and body mass index. For men, a similar pattern of associations was observed, but the associations were lost after adjustment for confounders. **Conclusions:** Our results show that effort, overcommitment and ERI may be associated with early atherosclerosis predicted by CIMT in women, and reward is inversely related to CIMT.
(J Occup Health 2010; 52: 257–262)

Key words: Carotid intima-media thickness, Coronary artery disease, Effort-reward imbalance, Job stress, Risk factors

Coronary heart disease (CHD) is the leading cause of death in developed countries. It is also emerging as a primary cause of death in developing countries such as China. Although job strain has frequently been identified as a risk factor for CHD[1–4], many studies have reported contradictory findings[5, 6]. There is limited direct evidence indicating that job stress is a risk factor for CHD. Hlatky *et al*.[7] and Yoshimasu *et al*.[8] found no association between job strain and CHD using coronary angiography. Recently, carotid intima-media thickness (CIMT), which is related to the extent and severity of CHD[9, 10] has been used as a surrogate marker of cardiovascular health. Several studies that examined the association between job strain or its components and CIMT reported conflicting findings. According to Hintsanen *et al*.[11] and Kivimäki *et al*.[12], job strain was associated with increased CIMT in men, but no association was observed between job strain and CIMT in women (the Cardiovascular Risk in Young Finns Study). Nordstrom *et al*.[13] found job demands and intrusion of work concerns into home life to be associated with increased CIMT in men, but there was no association in women. Rosvall *et al*.[14] reported that women who experienced job strain had increased CIMT compared with women who experienced no job strain; however, these findings were not observed in men. A study by Muntaner *et al*.[15] reported no association between job strain and CIMT. The Kuopio Ischemic Heart Disease Risk Factor Study of Finnish Men showed that high job demands combined with low economic rewards[16] or high stress-induced reactivity[17] predicted

Received Dec 14, 2009; Accepted May 26, 2010
Published online in J-STAGE Jul 2, 2010
Correspondence to: L. Guo, Department of Cardiology, Peking University Third Hospital and Key Laboratory of Molecular Cardiovascular Science, Ministry of Education, Beijing, 100191, China (e-mail: gljxwx2009@163.com)

increased CIMT. This is the only longitudinal study that has used CIMT as a health outcome measure.

The majority of research assessing the association between job stress and CHD has been performed by using the demand-control model, while studies using the effort-reward imbalance (ERI) model are relatively limited in number. Moreover, almost all of the studies assessing the association between job stress and CHD were conducted on European and North American populations, while research using the ERI model on Asian populations is rather rare.

The ERI model[18, 19] is an alternative leading psychosocial work model and includes three scales: extrinsic efforts, rewards and overcommitment. According to this model, an imbalance between high efforts and low rewards, and/or high levels of overcommitment increases the risk of CHD. The Chinese version of the ERI questionnaire has been evaluated and is considered a reliable and valid instrument for measuring psychosocial stress at work in the Chinese working population[20, 21].We have examined the association between the job stressors evaluated by the ERI model and CHD assessed by coronary angiography, and found that ERI may act as a CHD risk factor in the Chinese population[22, 23]. However, little is known about the effect of ERI on early atherosclerosis. The goal of this present study was to examine the association between ERI and early atherosclerosis indicated by CIMT in a Chinese population sample.

Methods

Subjects

Eight hundred eighty-one persons out of 900 working people from different occupations, including civil servants, managers, teachers, policeman, and other white-collar workers, undergoing physical checkups at the Peking University Shenzhen Hospital from June 2008 to February 2009, took part in this study (a participation rate of 97.9%). Participants with incomplete data (62), participants who had a history of CHD or whose symptoms or investigations suggested CHD (64) and participants who had peripheral arterial or cerebrovascular disease (21) were excluded. The final study population consisted of 734 participants (508 men and 226 women). This study was performed in accordance with the Declaration of Helsinki and all participants provided their written informed consent.

Job stress

Job stressors were assessed using the Chinese version of the 23-item effort-reward imbalance questionnaire (ERI-Q) developed by Li Jian *et al.*[20], in accordance with Siegrist's ERI-Q. The ERI-Q consists of three scales termed extrinsic effort representing job demands imposed on the employee (6 items), reward which consists of income, respect, job security and career opportunities (11 items), and overcommitment which defines a set of attitudes, behaviors, and emotions reflecting excessive striving in combination with a strong desire for approval and esteem (6 items). The responses to each item of effort is scored on a 5-point scale, on which a higher value means a higher stressful effort (1=no stressful experience to 5=very stressful experience). Each item of reward is also scored on a 5-point scale, on which a higher value implies a higher level of reward, i.e. a value of 1 indicates a very low level of reward and 5 indicates a very high level of reward. Each item of overcommitment is scored on a 4-point scale (1=full disagreement to 4=full agreement). The ERI index is calculated using the following formula: ERI=11 × effort / (6 × reward). The ERI-Q is self-reported. Cronbach's coefficients of effort, reward and overcommitment were 0.78, 0.78 and 0.85 for men and 0.80, 0.86 and 0.83 for women, respectively.

Carotid atherosclerosis

To assess CIMT, high resolution carotid ultrasonographic studies were performed using the Sequoia 512 ultrasound system with an 8–13 MHz linear array transducer. The common carotid artery was scanned by ultrasound technicians according to a standard protocol[11]. In each examination the image was recorded from the scanning angles (anterior, later-posterior) that showed the greatest distance between the lumen-intima interface and the media-adventitia interface. The digitally stored scans were analyzed by a single reader who was blind to the participants' individual characteristics. The analyses were performed using ultrasonic calipers. At least 5 measurements of the common carotid far wall were taken approximately 10 mm proximal to the bifurcation. The average of these measurements was used to determine CIMT.

CHD risk factors

Conventional CHD risk factors were collected. Laboratory data and medical treatments were extracted from the patients' medical records. The key indicators included: hypertension (blood pressure ≥140/90 mmHg and/or a history of hypertension), diabetes mellitus (fasting glucose ≥126 mg/d*l* or occasional glucose ≥200 mg/d*l* and/or diabetes mellitus history), hyperlipidaemia (total cholesterol ≥220 mg/d*l*, and/or total triglyceride ≥150 mg/d*l*, and/or low-density lipoprotein ≥140 mg/d*l* and/or a history of hyperlipidaemia) and body mass index (BMI, kg/m^2).

Statistical analysis

The differences in the conventional CHD risk factors and the mean scores of each component of the ERI model were statistically tested using the chi-square test or the

Table 1. Differences of the traditional CHD risk factors, mean scores of each component of ERI model, and IMT between men and women

	men (N=508)	women (N=226)	*p* value
Mean age (yr)	40.7 ± 11.8	40.6 ± 13.7	0.893
Mean length of service	16.6 ± 10.0	15.9 ± 11.7	0.435
Primary hypertension	140 (27.6%)	47 (20.8%)	0.052
Hyperlipidaemia	162 (31.9%)	21 (9.3%)	<0.001
Diabetes mellitus	36 (7.1%)	10 (4.4%)	0.227
BMI (kg/m^2)	23.9 ± 2.3	22.8 ± 3.3	<0.001
Effort	14.9 ± 4.1	13.7 ± 4.0	<0.001
Reward	43.1 ± 5.7	44.9 ± 7.1	0.001
Overcommitment	13.9 ± 3.9	12.2 ± 3.6	<0.001
ERI	0.66 ± 0.23	0.59 ± 0.26	0.001
IMT	0.84 ± 0.38	0.81 ± 0.09	0.141

ERI: effort-reward imbalance.

unpaired *t*-test. The association between each component of the ERI model and CIMT was tested using multiple linear regression analysis. Three different regression models were formed: model 1 did not include an adjustment for confounders, model 2 included an adjustment for age, and model 3 was adjusted for all confounders including age, hypertension, diabetes mellitus, hyperlipidaemia and BMI. A $p<0.05$ was considered statistically significant. All computations were performed using the SPSS software package version 13.0.

Results

The mean age was 40.7 yr for men and 40.6 yr for women. The average total number of working years was 16.6 yr for men and 15.9 yr for women. Table 1 presents the conventional CHD risk factors, the mean scores of each component of the ERI model and CIMT for men and women. There were no differences between men and women in age, average work years or the prevalence rate of diabetes mellitus. The men had more CHD conventional risk factors, such as a higher prevalence rate of hypertension (27.6% vs. 20.8%, $p=0.052$), hyperlipidaemia (31.9% vs. 9.3%, $p<0.001$) and higher BMI (23.9 vs. 22.8, $p<0.001$). There was no difference between men and women in CIMT (0.84 mm vs. 0.81 mm, $p=0.141$). In men, the scores were higher for effort (14.9 vs. 13.7, $p<0.001$), overcommitment (13.9 vs. 12.2, $p<0.001$) and ERI (0.66 vs. 0.59, $p=0.001$), but the scores were lower for reward (43.1 vs. 44.9, $p=0.001$) compared with women.

In men, effort, overcommitment and ERI scores were positively correlated with CIMT ($r=0.104$, $p<0.05$; r=0.097, $p<0.05$; $r=0.120$, $p<0.001$), in contrast, reward was inversely correlated with CIMT ($r=–0.107$, $p<0.05$). In women, we found similar patterns, the correlation coefficients were 0.297, 0.219, 0.307 and –0.240 for effort, overcommitment, ERI and reward, respectively (all $p<0.001$).

The results of the multiple linear regression analyses between each component of the ERI model and CIMT in men and women are presented in Tables 2 and 3, respectively. For men, in the unadjusted model 1, effort ($p=0.019$), overcommitment ($p=0.029$) and ERI ($p=0.007$) were positively correlated with CIMT, while reward was inversely related to CIMT ($p=0.016$). However, these associations were attenuated after adjustment for age in model 2. After a full adjustment in model 3, the significant associations disappeared. For women, in the unadjusted model 1, effort ($p<0.001$), overcommitment ($p=0.001$) and ERI ($p<0.001$) were positively correlated with CIMT, while reward was inversely correlated with CIMT ($p<0.001$). Furthermore, these associations were not attenuated after adjustment for age, hypertension, hyperlipidaemia, diabetes mellitus or BMI.

Discussion

Our study showed a gender-specific association between ERI and increased CIMT. For women, we saw a robust association between effort, overcommitment, ERI and increased CIMT; reward was inversely correlated with CIMT, controlling for age, hypertension, diabetes mellitus, hyperlipidaemia and BMI. For men, similar results were observed, but the association was lost after adjustment for confounders.

A major strength of this study is that the exposure variable, job stress, was assessed using the ERI model and the health outcome variable, CHD, was assessed by a noninvasive CIMT ultrasound method. The ERI model is an alternative method for evaluating job stress. It has been shown that the ERI model has relatively more explanatory power than the job demand-control model[24, 25]. This result was also observed in a Chinese population in our previous

Table 2. Multiple linear regression analysis of each component of ERI model and IMT in men

Predictor	Model 1			Model 2			Model 3		
	Adjusted R^2	β	p	Adjusted R^2	β	p	Adjusted R^2	β	p
Effort	0.009	0.010	0.019	0.021	0.006	0.176	0.042	0.004	0.319
Reward	0.009	–0.007	0.016	0.030	–0.007	0.011	0.045	–0.005	0.109
Overcommitment	0.007	0.009	0.029	0.022	0.006	0.143	0.043	0.005	0.281
ERI	0.013	0.193	0.007	0.026	0.151	0.036	0.044	0.108	0.149

Model 1: unadjusted confounders; Model 2: adjusted for age; Model 3: adjusted for age, primary hypertension, diabetes mellitus, hyperlipidaemia and BMI; ERI: effort-reward imbalance.

Table 3. Multiple linear regression analysis of each component of ERI model and IMT in women

Predictor	Model 1			Model 2			Model 3		
	Adjusted R^2	β	p	Adjusted R^2	β	p	Adjusted R^2	β	p
Effort	0.084	0.006	<0.001	0.139	0.006	<0.001	0.258	0.004	0.001
Reward	0.052	–0.003	<0.001	0.217	–0.005	<0.001	0.282	–0.004	<0.001
Overcommitment	0.044	0.005	0.001	0.129	0.006	<0.001	0.261	0.001	<0.001
ERI	0.090	0.101	<0.001	0.182	0.117	<0.001	0.274	0.082	<0.001

ERI: effort-reward imbalance; Model 1: unadjusted confounders; Model 2: adjusted for age; Model 3: adjusted for age, primary hypertension, diabetes mellitus, hyperlipidaemia and BMI.

study[21]. Several studies have reported that ERI increases the risk of CHD[26–28]. CHD is a multifactorial disease that produces clinically significant changes at a relatively late stage in the prognosis. The presence of CHD symptoms has been observed to bias the perception of ERI and lead to a spurious association between ERI and CHD[29, 30]. As a surrogate marker of CHD, CIMT makes it possible to study the early, non-symptomatic phase of atherosclerosis among adults without manifest symptoms of CHD. This study fills a gap in the research concerning the association between ERI and early atherosclerosis as predicted using CIMT.

In our study, increased effort, overcommitment and ERI correlated with increased CIMT, and reward was inversely correlated with CIMT in women. This supports the theory of the ERI model. The results were also in agreement with previous studies in which stressors were evaluated using the demand-control model; e.g. Rosvall *et al*.[14] reported that job strain increased CIMT in women and a cross-sectional study of Japanese workers showed that high job strain (high demands and low control) was associated with increased arterial stiffness[31]. The results reported by Wang *et al*.[32] also support these findings; i.e. the mean coronary luminal diameter of women with job strain was decreased by 0.20 mm when compared to women with no job strain, whose luminal diameter was increased by 0.22 mm.

The association between job stress and cardiovascular disease differs between genders. In the Job stress, Absenteeism and Coronary Heart Disease in Europe (JACE) study[33], Karasek's demand-control model showed large gender and regional differences, whereas geographic regions explained only a small fraction of the total demand-control variance, notwithstanding large differences in the labour market and working conditions. Our study indicated a strong gender difference in the associations, similar to the findings of Rosvall *et al*.[14]. On the other hand, Hintsanen *et al*.[11] and Nordstrom *et al*.[13] observed a significant association in men, but not in women. These contradictory findings may be attributed to the different age structures of the study populations, the assessment of job stress, and the confounders considered in the analyses. The mean age of women in the study of Hintsanen *et al*. was 32.3 yr, while it was 40.6 yr in our study. Atherosclerosis develops relatively slowly in young women and accelerates after menopause; therefore, the association may exist in older women, but not in young women. In the study of Nordstrom *et al*[13]. the mean age of the women was 52.8 yr, but the job demand was assessed by only 3 items which may not have reflected the job stress adequately leading to null findings. Recently, some studies reported that certain genotypes moderate the association between job stress and the risk of CHD[34, 35]. It may be important to explore whether different races have genotypic variation that may moderate this association. In China, the double burden of work and family has to be taken into account in the case of working women. Xu *et al*.[36] found that the combined exposure to work and family-related stress was associated with a significant increase in systolic blood pressure. A prospective study in the USA[37] indicated that women with high levels of caregiving burden and family obligations have an increased risk of CHD. The UK Whitehall II study[38] also reported that low control at home predicted CHD in women, but not in men. This additional source of stress may confound the association between job stress and CIMT in women. Unfortunately, our study did not assess the work-family conflict while examining gender differences at work. The association between job stress and CIMT in men was lost after adjustment for all confounders in the present study. This may represent overmatching as hyperlipidaemia and hypertension could be the link between ERI and CIMT. A longitudinal study in a healthy population needs to be performed to address this issue.

Several limitations of this study need to be addressed. Firstly, as our study was cross-sectional, we could not draw a cause-effect conclusion. Secondly, our study did not include smoking, which is a risk factor for CHD, as a confounder in the analysis; however, as the smoking rate among women in China is low[39], the effect of smoking should not be powerful enough to change the results. Finally, it was reported that socioeconomic status, indicated by income, education level and occupation, affects the risk of CHD. While a higher social status in a developed country is related to a lower risk for CHD, there is a greater risk for CHD among higher socioeconomic groups in developing countries[40, 41]. Recent research conducted in Japan, an industrialised, non-Western country, did not find an association between socioeconomic status and cardiovascular disease[42, 43]; therefore, the findings for socioeconomic status and CHD are contradictory. Furthermore, the patient information was confidential in this study, and it was difficult to gain accurate information on the participants' socioeconomic status, so we did not include this factor in our analysis. This type of information should be collected in a future study to examine the effect of socioeconomic status on CHD in the Chinese population

In summary, our results show that effort, overcommitment and ERI may be associated with early atherosclerosis as predicted by CIMT in women, and reward was inversely related to CIMT. The association was not accounted for by age, hypertension, diabetes mellitus, hyperlipidaemia and body mass index. More attention should be paid to women who are under the double burden of job and family. Longitudinal research and intervention studies are needed to examine whether job stressors, evaluated using the ERI model, contribute to the development of atherosclerosis from its early, non-symptomatic stages to its later, clinically relevant phase.

Acknowledgments: The authors thank Haiou Yang, Stacey Willis, Jian Li, Paul Landsbergis and Peter Schnall for their help. This study was supported by the Major National Basic Research Program of the PR China (No. 2007CB512107 to W. G.).

References

1) Eller NH, Netterstrøm B, Gyntelberg F, et al. Work-related psychosocial factors and the development of ischemic heart disease: a systematic review. Cardiol Rev 2009; 17: 83–97.
2) Kivimäki M, Virtanen M, Elovainio M, Kouvonen A, Väänänen A, Vahtera J. Work stress in the etiology of coronary heart disease—a meta-analysis. Scand J Work Environ Health 2006; 32: 431–42.
3) Aboa-Eboulé C, Brisson C, Maunsell E, et al. Job strain and risk of acute recurrent coronary heart disease events. JAMA 2007; 298: 1652–60.
4) Peter R, Siegrist J, Hallqvist J, Reuterwall C, Theorell T, SHEEP Study Group. Psychosocial work environment and myocardial infarction: improving risk estimation by combining two complementary job stress models in the SHEEP Study. J Epidemiol Community Health 2002; 56: 294–300.
5) Eaker ED, Sullivan LM, Kelly-Hayes M, D'Agostino RB Sr, Benjamin EJ. Does job strain increase the risk for coronary heart disease or death in men and women? The framingham offspring study. Am J Epidemiol 2004; 159: 950–8.
6) Lee S, Colditz G, Berkman L, Kawachi I. A prospective study of job strain and coronary heart disease in US women. Int J Epidemiol 2002; 31: 1147–53.
7) Hlatky MA, Lam LC, Lee KL, et al. Job strain and the prevalence and outcome of coronary artery disease. Circulation 1995; 92: 327–33.
8) Yoshimasu K, Liu Y, Kodama H, et al. Job strain, Type A behavior pattern, and the prevalence of coronary atherosclerosis in Japanese working men. J Psychosom Res 2000; 49: 77–83.
9) O'leary DH, Polak JF. Intima-media thickness: a tool for atherosclerosis imaging and event prediction. Am J Cardio 2002; 90: 18L–21L.
10) Lorenz MW, Markus HS, Bots ML, Rosvall M, Sitzer M. Prediction of clinical cardiovascular events with carotid intima-media thickness: a systematic review and meta-analysis. Circulation 2007; 115: 459–67.
11) Hintsanen M, Kivimäki M, Elovainio M, et al. Job strain and early atherosclerosis: the Cardiovascular Risk in Young Finns Study. Psychosom Med 2005; 67: 740–7.
12) Kivimäki M, Hintsanen M, Keltikangas-Järvinen L, et al. Early risk factors, job strain, and atherosclerosis among men in their 30s: the Cardiovascular Risk in Young Finns Study. Am J Public Health 2007; 97: 450–2.
13) Nordstrom CK, Dwyer KM, Merz CN, Shircore A, Dwyer JH. Work-related stress and early atherosclerosis. Epidemiology 2001; 12: 180–5.
14) Rosvall M, Ostergren PO, Hedblad B, Isacsson SO, Janzon L, Berglund G. Work-related psychosocial factors and carotid atherosclerosis. Int J Epidemiol

2002; 31: 1169–78.
15) Muntaner C, Nieto FJ, Cooper L, Meyer J, Szklo M, Tyroler HA. Work organization and atherosclerosis: findings from the ARIC study. Atherosclerosis risk in communities. Am J Prev Med 1998; 14: 9–18.
16) Lynch J, Krause N, Kaplan GA, Salonen R, Salonen JT. Workplace demands, economic reward, and progression of carotid atherosclerosis. Circulation 1997; 96: 302–7.
17) Everson SA, Lynch JW, Chesney MA, et al. Interaction of workplace demands and cardiovascular reactivity in progression of carotid atherosclerosis: population based study. BMJ 1997; 314: 553–8.
18) Siegrist J. Adverse health effects of high-effort/low-reward condition. J Occup Health Psychol 1996; 1: 27–41.
19) Tsutsumi A, Kawakami N. A review of empirical studies on the model of effort-reward imbalance at work: reducing occupational stress by implementing a new theory. Soc Sci Med 2004; 59: 2335–59.
20) Li J, Yang WJ, Cheng Y, Siegrist J, Cho SI. Effort-reward imbalance at work and job dissatisfaction in Chinese healthcare workers: a validation study. Int Arch Occup Environ Health 2005; 78: 198–204.
21) Li J, Yang JW, Cho S. Gender differences in job strain, effort-reward imbalance, and health functioning among Chinese physicians. Soc Sci Med 2006; 62: 1066–77.
22) Xu W, Zhao Y, Guo L, Guo Y, Gao W. Job stress and coronary heart disease: a case-control study using a Chinese population. J Occup Health 2009; 51: 107–13.
23) Xu W, Zhao Y, Guo L, Guo Y, Gao W. The association between effort-reward imbalance and coronary atherosclerosis in a Chinese sample. Am J Ind Med 2010 [Epub ahead of print]. PMID 20306511.
24) Ostry AS, Kelly S, Demers PA, Mustard C, Hertzman C. A comparison between the effort-reward imbalance and demand control models. BMC Public Health 2003; 3: 10.
25) Ostry AS, Hershler R, Chen L, Hertzman C. A longitudinal study comparing the effort-reward imbalance and demand-control models using objective measures of physician utilization. Scand J Public Health 2004; 32: 456–63.
26) Kuper H, Singh-Manoux A, Siegrist J, Marmot M. When reciprocity fails: effort-reward imbalance in relation to coronary heart disease and health functioning within the Whitehall II study. Occup Environ Med 2002; 59: 777–84.
27) Kivimäki M, Leino-Arjas P, Luukkonen R, Riihimaki H, Vahtera J, Kirjonen J. Work stress and risk of cardiovascular mortality: prospective cohort study of industrial employees. BMJ 2002; 325: 857–61.
28) van-Vegchel N, de-Jonge J, Bosma H, Schaufeli W. Reviewing the effort-reward imbalance model: drawing up the balance of 45 empirical studies. Soc Sci Med 2005; 60: 1117–31.
29) Macleod J, Davey Smith G, Heslop P, Metcalfe C, Carroll D, Hart C. Limitations of adjustment for reporting tendency in observational studies of stress and self reported coronary heart disease. J Epidemiol Community Health 2002; 56: 76–7.
30) Macleod J, Davey Smith G, Heslop P, Metcalfe C, Carroll D, Hart C. Psychological stress and cardiovascular disease: empirical demonstration of bias in a prospective observational study of Scottish men. BMJ 2002; 324: 1247–51.
31) Utsugi M, Saijo Y, Yoshioka E, et al. Relationship between two alternative occupational stress models and arterial stiffness: a cross-sectional study among Japanese workers. Int Arch Occup Environ Health 2009; 82: 175–83.
32) Wang HX, Leineweber C, Kirkeeide R, et al. Psychosocial stress and atherosclerosis: family and work stress accelerate progression of coronary disease in women. The Stockholm female coronary angiography study. J Intern Med 2007; 261: 245–54.
33) de Smet P, Sans S, Dramaix M, et al. Gender and regional differences in perceived job stress across Europe. Eur J Public Health 2005; 15: 536–45.
34) Hintsanen M, Elovainio M, Puttonen S, et al. Val/Met polymorphism of the COMT gene moderates the association between job strain and early atherosclerosis in young men. J Occup Environ Med 2008; 50: 649–57.
35) Hintsanen M, Elovainio M, Puttonen S, et al. Neuregulin-1 genotype moderates the association between job strain and early atherosclerosis in young men. Ann Behav Med 2007; 33: 148–55.
36) Xu L, Siegrist J, Cao W, Li L, Tomlinson B, Chan J. Measuring job stress and family stress in Chinese working women: a validation study focusing on blood pressure and psychosomatic symptoms. Women & Health 2004; 39: 31–46.
37) Lee S, Colditz GA, Berkman LF, Kawachi I. Caregiving to children and grandchildren and risk of coronary heart disease in women. Am J Public Health 2003; 93: 1939–44.
38) Chandola T, Kuper H, Singh-Manoux A, Bartley M, Marmot M. The effect of control at home on CHD events in the Whitehall II study: gender differences in psychosocial domestic pathways to social inequalities in CHD. Soc Sci Med 2004; 58: 1501–9.
39) Li L, Chen Q, Jia R, Cui X. The smoking pattern and disease burden of tobacco use in China. Chinese Health Economics 2008; 27: 26–30.
40) Marmot M, Bartley M. Social class and coronary heart disease. In: Stansfeld SA and Marmot MG, eds. Stress and the heart. London: BMJ, 2002.
41) Tezcan S, Altintas H, Sonmez R, et al. Cardiovascular Risk Factor Levels in a lower middle class community in Ankara, Turkey. Trop Med Int Health 2003; 8: 660–7.
42) Honjo K, Tsutsumi A, Kayaba K. The Jichi Medical School Cohort Study Group. Socioeconomic indicators and cardiovascular disease incidence among Japanese community residents: the Jichi medical school cohort study. Int J Behav Med 2009 [Epub ahead of print]. PMID: 19554455.
43) Honjo K, Iso H, Inoue M, Tsugane S. Japan public health center-based prospective study group. Education, social roles, and the risk of cardiovascular disease among middle-aged Japanese women: the JPHC Study Cohort I. Stroke 2008; 39: 2886–90.