Interlibrary Loans and Journal Article Requests

Notice Warning Concerning Copyright Restrictions:

The copyright law of the United States (Title 17, United States Code) governs the making of photocopies or other reproductions of copyrighted materials.

Under certain conditions specified in the law, libraries and archives are authorized to furnish a photocopy or other reproduction. One specified condition is that the photocopy or reproduction is not to be *"used for any purpose other than private study, scholarship, or research."* If a user makes a request for, or later uses, a photocopy or reproduction for purposes in excess of "fair use," that user may be liable for copyright infringement.

Upon receipt of this reproduction of the publication you have requested, you understand that the publication may be protected by copyright law. You also understand that you are expected to comply with copyright law and to limit your use to one for private study, scholarship, or research and not to systematically reproduce or in any way make available multiple copies of the publication.

The Stephen B. Thacker CDC Library reserves the right to refuse to accept a copying order if, in its judgment, fulfillment of the order would involve violation of copyright law.

Terms and Conditions for items sent by e-mail:

The contents of the attached document may be protected by copyright law. The CDC copyright policy outlines the responsibilities and guidance related to the reproduction of copyrighted materials at CDC. If the document is protected by copyright law, the following restrictions apply:

- You may print only one paper copy, from which you may not make further copies, except as maybe allowed by law.
- You may not make further electronic copies or convert the file into any other format.
- You may not cut and paste or otherwise alter the text.

European Journal of Cardiovascular Nursing

EUROPEAN SOCIETY OF CARDIOLOGY®

Review

Work-related cardiovascular disease risk factors using a socioecological approach: implications for practice and research

European Journal of Cardiovascular Nursing
11(1) 114–126

DOI: 10.1177/1474515111430890
cnu.sagepub.com

SAGE

Won Ju Hwang[1] and Oisaeng Hong[2]

Abstract

Background: Cardiovascular disease (CVD) is a leading cause of mortality. Numerous investigations have linked occupational factors and CVD. Occupational factors such as overtime work have an enormous effect on the CVD risk of industrial workers. However, risk factors for CVD are not systematically reviewed in the workplace. The purpose of the paper is to review work-related risk factors for CVD.

Methods: A systematic review of work-related CVD risk factors was performed, yielding 180 articles. All articles were assessed in relation to inclusion and exclusion criteria, resulting in 44 articles being reviewed. The sole inclusion criteria was work-related environmental factors and intra/inter-personal factors (psychosocial factors), which is based on the socioecological perspective. The articles were also assessed regarding the quality of each study using the scoring methods developed by Cesario et al. and Brown et al.

Conclusion: The literature review demonstrated that work environment factors such as shift work, overtime work, and noise and chemical exposures; and psychosocial factors such as job stress, social support, and socioeconomic status cannot be explained or intervened by one single risk factor. Furthermore, certain occupational factors were shown to aggravate or attenuate other risk factors. The implication of these findings is to incorporate work-related environmental and psychosocial factors into assessment of the patient's CVD risks and intervention plan. Future research should also incorporate a well-defined conceptual framework to address the effects of work-related environmental and psychosocial factors on CVD among CVD patients.

Keywords

Cardiovascular disease, risk factors, workers

Received 10 May 2011; revised 30 June 2011, 8 August 2011; revised manuscript accepted 16 August 2011

Introduction

Cardiovascular disease (CVD) is a leading cause of death,[1,2] It has been defined as coronary heart disease consisting of angina pectoris, ischemic heart disease and myocardial infarction plus coronary heart disease death, and CVD consisting of coronary heart disease and heart failure plus stroke, peripheral artery disease, and hypertension.[3] The growing body of literature suggests that environmental, psychosocial and organizational factors have an influence on CVD in the workplace. Numerous investigations have linked occupational factors and CVD, including specific chemical hazards and non-specific job stressors such as work overload, shift work and poor nutritional habits.[4,5] These occupational factors have an enormous negative effect on workers' health and wellness, particularly blue collar workers. While white collar or professional workers are vulnerable to different occupational factors such as job stress and sedentary work, blue collar workers are more likely to be exposed to high stress and hazardous working environments, including unhealthy lifestyles.[6] Blue collar workers are also more frequently exposed to irregular shift work and heavy workloads.[7] Workers in these occupations also tend to have high rates of cigarette smoking and exposure to carbon monoxide, both of which are associated with increased risk of CVD.[8]

[1]College of Nursing Science, Kyung Hee University, Korea
[2]University of California San Francisco, USA

Corresponding author:
Dr Won Ju Hwang, College of Nursing Science, Kyung Hee University
26 Kyunghee-daero, Dongdaemun-gu, Seoul, 130-701, Korea
Email: hwangwj@hotmail.com

The majority of risk factors associated with CVD are either preventable or modifiable through lifestyle changes.[9] However, mortality due to CVD among blue collar workers in smaller companies has been shown to be higher than that of white collar workers (192 per 100,000 person-years vs 117 per 100,000 person-years).[10] Blue collar workers in small companies tend to have a limited or no access to health screening or preventive health education programmes. These workers may be unaware of their risk factors and thus may be at greater risk for CVD.[6] The socioecological perspective is a framework to examine the occupational effects on CVD and interrelatedness of social elements in a work environment. The model can provide a conceptual framework to analyse various occupational contexts in multiple types of research.[11]

There is little doubt that understanding the CVD risk factors of workers is of great value to healthcare practitioners and researchers who serve the working population. Furthermore, within the National Institutes for Occupational Safety and Health (NIOSH) in the USA, this research is being coordinated by the NIOSH Cancer, Reproductive, and Cardiovascular Research Program.[12] More knowledge is needed regarding work-related CVD risk factors for healthcare professionals to help prevent the development and progression of CVD in the workplace. In particular, this review contributes to the developing knowledge and understanding about the world of work and its relevance to the development of CVD from a socioecological perspective. The purpose of this paper is to review CVD risk factors, specifically occupational and environmental and psychosocial factors at work.

Methods

Literature search

The PsycINFO, Educational Resource Information Center, PubMed of the National Library of Medicine, and the Cumulative Index to Nursing and Allied Health Literature (CINAHL) databases were searched. The literature was searched using various combinations of the following keywords and medical subject headings: *Cardiovascular disease*, *CVD risk factors*, *work*, *occupation*, *workers*, *employment*, and *workplace*, covering the period 1985 to January 2010. All of the studies that met the review criteria were published between 1989 and 2010. Reference lists from every relevant paper were examined to determine whether pertinent studies had been missed during the database searches.

Exclusion and inclusion criteria

A total of 1,353 articles were retrieved from the above databases using the various combinations of the keywords mentioned above. Of these, studies were excluded according to the following criteria: prevalence (30), metabolism (120), bio-physiology (80), surveillance (84), rehabilitation (12), radiation (64), genetic factors (90), respiratory (60), cancer (74), ergonomics (26), return to work (76), and youth and congenital (457). The net result was 180 articles regarding CVD risk. Additional inclusion criteria were applied to further focus the remaining citations on CVD risk in the workplace. The main inclusion criteria were work-related environmental factors (shift and overtime work, chemical and noise exposure, passive smoking and physical exertion), and intra/inter-personal factors (psychosocial factors, job stress, social support and social class), which is based on the socioecological model. The articles were also assessed with respect to the quality of each study, using the scoring methods developed by Cesario et al.[13] and Brown et al.[14] All abstracts were evaluated for inclusion criteria. Of these, 23 were redundant or low quality according to the scoring method, leaving 157 articles to be examined. Studies were excluded that focused on risk perception (15), or were letters (8), dissertations (10), non-English (15), no workers (52), non-peer reviewed (2), had no published results (1). Additionally, ten studies that measure individual risk factors only were eliminated, yielding 44 studies to be included in this review (Figure 1).

Results

The primary focus of this review was CVD risk factors in the workplace, including work-related environmental factors and psychosocial factors. Environmental factors include work-related environmental factors and organizational factors.[15] Because individual psychosocial factors such as personality traits and cognitive and behavioural variables are also referred to as psychosocial factors, it is important to emphasize that this review concentrates only on psychosocial factors at work.[16] For this review, psychosocial factors at work are grouped into categories based on social support, job stress, and social class. Individual risk factors will not be discussed in this review. Table 1 provides a brief description of the samples, country of origin, designs, theoretical model, measurement, and quality of study, of the 44 articles on work-related risk factors for CVD. The major findings by the significant work-related CVD risk factors are summarized in Table 2. Of the studies reviewed, only one involved a clinical trial, six were case-control studies, and three were review papers. The majority used longitudinal or cohort studies (22 studies) and cross-sectional design (12 studies; Table 1). There was no consistency among the 38 studies in the choice of CVD risk factor instrument, except for six studies that used the Job Content Questionnaire.[17]

A wide variety of worker populations were encountered in the review. Most studies included industrial workers from different work sites while two studies included healthcare providers or nurses.[18,19] Statistically significant differences in the relative risk for CVD outcome were reported in multiple studies, regardless of the kind of risk factors studied.[20–25] This review highlights the ten categories of CVD

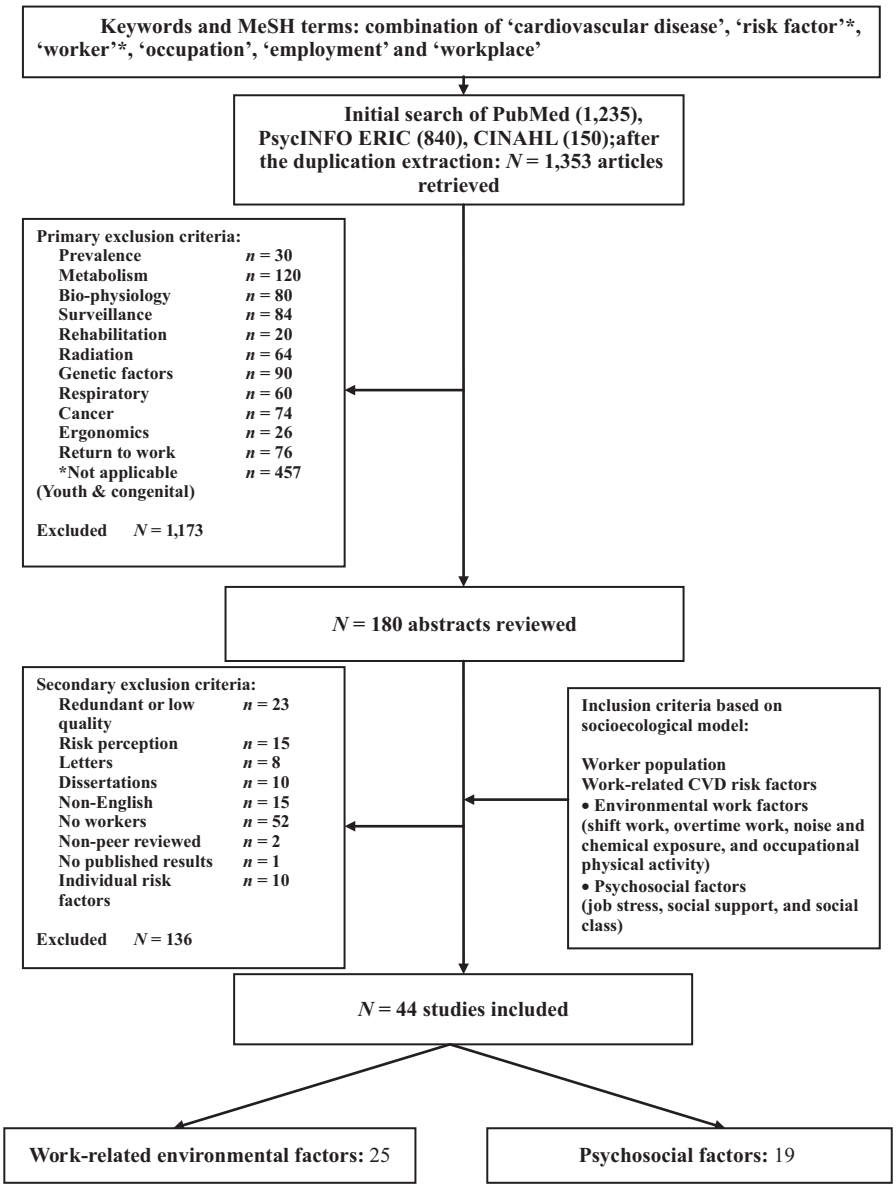

Figure 1. Flow chart showing the number of studies identified included in the literature review.

risk factors in the workplace and focuses on environmental work factors and psychosocial factors.

Work environmental factors

Work environmental factors are presumably important in the causation of prevailing social class differences as a risk factor for CVD.[15] Job characteristics have also been shown to influence CVD risk. For example shift work, in particular night work, has been associated with an increased risk of CVD.[26,27] Factors that were investigated in this review included shift and overtime work,[18–21,23,24,28-30] noise and chemical exposures,[25,31] passive smoking[32,33] and occupational physical activity.[34,35]

Shift work and overtime work. Shift work has been associated with increased risk of CVD. Nurses who worked the night shift for 6 years or more have been shown to have a higher risk of coronary heart disease (relative risk 1.51, 95% CI 1.12 to 2.03).[20] Munakata and colleagues[19] examined 18 healthy nurses in a Japanese hospital to determine whether psychological states following night work were related to alterations in CVD. The results showed that night shift work was associated with altered cardiovascular responses in healthy nurses. However, the study did not examine environmental factors such as increased workload due to housework and child care. Degrees of psychological stress and physical load related to the condition of hospital inpatients may also be involved, but were not examined.

Table 1. Summary of work-related environmental and psychosocial factors (n = 44)

Investigator year	Country of study	Design	Subjects	Theoretical model	Purpose	Measurement	[1]Quality score
Work-related environmental factors							
Shift work							
Kawachi et al. 1995	Japan	Prospective cohort	Night shift workers (79,109)	N/A	To assess shift work and the risk of CHD	CHD incidence, shift work	[2]QI
Munakata et al. 2001	Japan	Clinical trial	Healthy nurses (18)	N/A	To examine night work and CVD	BP, HR, RR interval variability	QI
Copertaro et al. 2008	Italy	Prospective cohort	Healthcare providers (552)	N/A	To explore metabolic risk factors for CVD	HDL-cholesterol, triglycerides, BP	QI
Su et al. 2008	Taiwan	Longitudinal	Shift workers (15)	Mixed model	To determine the haemodynamic effects	ABP monitoring, BP, HR and HRV	QI
Overtime work							
Park et al. 2001	Korea	Cross-sectional	Male engineers (238)	N/A	To evaluate overtime work and CVD	BP and HRV	QII
Nakanishi et al. 2001	Japan	Prospective cohort	Male workers (941)	N/A	To evaluate the long working and hypertension	BP and working hours, 5-year follow-up	QII
Liu et al. 2002	Japan	Case-control	Cases: 260 Control: 445 (705)	N/A	To examine the working hours and MI	Working hours per week Daily hours of sleep	QII
Yang et al. 2006	USA	Cross-sectional	Workers (24,205)	N/A	To analyse work hours and hypertension	Self-reported hypertension	QII
Noise exposure							
Fujino et al. 2007	Japan	Prospective cohort	Workers without CVD(14,568)	N/A	To assess perceived noise exposure and CVD	Self-report about noise exposure	QII
Lee et al. 2009	Korea	Retrospective cohort	Male metal workers (530)	N/A	To assess noise exposure and CVD	Noise level categories (NLCs) SBP, DBP, BMI	QI
Davies et al. 2009	Canada	Retrospective cohort	Sawmill workers (27,499)	N/A	To estimate historical exposures to noise	Noise exposure data	QI
Tomei et al. 2010	Italy	Meta-analysis	Workers 15 papers (18,658)	N/A	To evaluate the cardiovascular system and noise	High, intermediate and low exposure	QI
Chemical exposure							
Kristensen 1989	Denmark	Review	Chemical factors (lead, cadmium, cobalt, etc.)	N/A	To review the epidemiological research on CVD	The epidemiological literature	QII
Axelson et al. 1994	Sweden	Cross-sectional	Cohort exposed to TRI	N/A	To evaluate trichloroethylene (TRI)	SMR	QII
Persson et al. 2007	Sweden	Longitudinal cohort	Pulp and paper mill workers (7107)	N/A	Focused on CVD mortality in relation to various exposures	The causes of death	QI

(Continued)

Table 1. (Continued)

Investigator year	Country of study	Design	Subjects	Theoretical model	Purpose	Measurement	[1]Quality score
Laden et al. 2007	USA	Retrospective cohort	Male employees (54,319)	N/A	To examine rates of cause-specific mortality	SMR	QI
Humblet et al. 2008	USA	Systematic review	PubMed search 12 courts	N/A	To evaluate the dioxin exposure and CVD	Internal comparisons of the high quality	QII
Passive smoking							
Lopez et al. 2007	Spain	Retrospective cohort	Non-smokers (6,492)	N/A	To estimate the deaths by exposure to ETS	ETS exposure	QII
Felber et al. 2007	Switzerland	Cross-sectional	Non-smokers (1,218)	N/A	To test ETS exposure and HRV	24 hour ECG recordings – BP	QI
He et al. 2008	China	Cross-sectional	943 men, 1,391 women (2,334)	N/A	To examine the ETS and CVD	ETS exposure – response rate: 87.1%	QI
Venn et al. 2007	UK	Cross-sectional	Never smokers (7,599)	N/A	To investigate the ETS and CHD	Cotinine, biomarkers of heart disease	QI
Occupational physical activity							
Fransson et al. 2004	Sweden	Case-control	Cases: 1754 Controls: 1311 (3065)	N/A	To estimate physical activity and acute MI	A questionnaire on lifestyle factors	QI
Krause et al. 2007	Finland	Prospective cohort	Finnish men (612)	Hemodynamic Theory	To assess the effects of physical activity on atherosclerosis	Intima media thickness (IMT)	QI
Hu et al. 2007	Finland	Prospective cohort	Finnish men (4,660)	N/A	To examine the physical activity and CHD	CHD event	QII
Kristal et al. 2000	Israel	Prospective cohort	Industrial workers (3,488)	N/A	To evaluate physical activity and the CVD	CVD mortality	QII
Psychosocial factors:							
Social support							
Steptoe et al. 2000	UK	Longitudinal study	School teachers (81)	DCM	To explore the stress and CVD risk	Job strain – BP	QI
Guimont et al. 2006	Canada	Prospective cohort	White collar workers (8,395)	N/A	To evaluate the job strain and BP	Cumulative exposure to job strain	QI
Hughes et al. 2009	Ireland	Cross-sectional	Non-smoking women (211)	N/A	To investigate the support and CVD	Psychometric indices of social support	QII
Job stress							
Niedhammer et al. 1998	France	Cohort study	French workers (12,221)	DCM	To examine the job stress and CVD	Psychosocial work indices, CVD risk	QI
Bosma et al. 1998	UK	Cohort study	Civil servants (1,318)	DCM, ERI	To examine the CHD risk and job stress models	ERI – job strain: CHD report	QI
Peter et al. 2002	Sweden	Case-control	(951 MI cases/1,147 referents)	DCM, ERI	To study 2 alternative job stress	Job strain – CVD outcome: MI	QI

(Continued)

Table 1. (Continued)

Investigator year	Country of study	Design	Subjects	Theoretical model	Purpose	Measurement	[1]Quality score
Fauvel et al. 2003	France	Cohort study	Healthy subjects (292)	DCM	To assess the job strain and BP	Job strain – SBP, DBP	QI
Ramey 2003	USA	Cross-sectional	Male officers (2,818)	N/A	To investigate CVD morbidity and job stress	Behavioural risk factors	QII
Ferris et al. 2005	Canada	Cross-sectional	Employees (428)	DCM, ERI	To examine strain and risk of CVD	JCQ, ERI – BPSS	QI
Kang et al. 2005	Korea	Cross-sectional	Workers (152)	DCM	To examine job stress and CVD risk factors.	JCQ – BP, lipid level	QI
Ducher et al. 2006	France	Case-control	Hypertensive (926)	DCM	To assess the job strain and hypertension	JCQ – ABP	QI
Rose et al. 2006	Sweden	Longitudinal study	Workers (926)	N/A	To analyse life events and CVD risk factors	Life events, social support, mental strain	QI
Tobe et al. 2007	Canada	Longitudinal study	Volunteers (229)	Combined model	To evaluate job stress and ABP	JCQ – ABP	QI
Social class, occupational status, and CVD							
Tuchsen et al. 1999	Denmark	Cohort study	Workers in 1981, 1986	N/A	To estimate relative risk of IHD morbidity	IHD morbidity	QII
Netterstrom et al. 1999	Denmark	Case-control	Workers (76)	DCM	To test socioeconomic factors and MI	Job strain, MI case	QI
Baigi et al. 2002	Sweden, Netherlands	Cross-sectional	Men (45,394) Women (43,403)	N/A	To investigate the CVD risk and SES	CVD mortality	QI
Landsbergis et al. 2003	USA	Cross-sectional	8 work sites (283)	DCM	To determine the strain and BP by SES	Job strain (QES), ABP	QI
Netterstrom et al. 2006	Denmark	Prospective cohort	Workers (659)	DCM	To test the job strain and IHD	Job strain, IHD incidence	QI
Chen et al. 2007	Taiwan	Case-control	119 cases, 238 controls (357)	N/A	To test the relationship between work and IHD	IHD incidence	QI

ABP, ambulatory blood pressure; BMI, body mass index; BP, blood pressure; CHD, coronary heart disease; CVD, cardiovascular disease; DBP, diastolic blood pressure; ETS, environmental tobacco smoke; HR, heart rate; HRV, heart rate variability; HWE, healthy worker effect; IHD, ischemic heart disease; MI, myocardial infarction; OR, odds ratio; SBP, systolic blood pressure; SES, socioeconomic status; SMR, standardised mortality ratio.
Quality scores:
[1]scored 0–3 depending upon the quality criteria:
Justification; Rigor in documentation; Design; Sample; Methods; Ethical rigor; Analytic preciseness; Theoretical model; Relevance
[2]QI = total score of 20 – 27; QII = 13–19; QIII = < 13

A recent cohort study explored how metabolic risk factors for CVD differed between shift workers and day workers in an Italian cohort of 262 healthcare providers, 204 forestry workers, and 86 factory workers.[18] Disruptions of circadian rhythms, unhealthy lifestyles and increased stress provided a worse profile of CVD risk factors among shift workers. However, the healthy worker effect, which refers to a phenomenon observed initially in occupational disease studies, might make it more difficult to observe such a profile, particularly in prevalence studies.

Other concerns were reported regarding the impact of overtime work.[28] Overtime work as a risk factor for CVD is difficult to separate from the more general literature regarding stress, because overtime work is generally considered to be stressful. A few recent studies suggest that long work hours increase the risk of CVD.[23,36] The most important studies to date attempt to separate the independent effects of long working hours and stress by measuring hours worked.[21,22,29] In these studies overtime work was associated with CVD risk in two studies.[22,29] In a case-control

study of Japanese workers, overtime work during the previous month was shown to be associated with an increased risk for acute myocardial infarction.[22] The authors reported that 61 or more hours of work per week and fewer than 2 days off per month increased the odds of acute myocardial infarction by two times or more. Furthermore, Park et al.[29] used both a self-report questionnaire (working hours, health conditions and fatigue) and measurements of blood pressure and heart rate variability to study the association between overtime work and CVD risk. When adjusted for age and hours of sleep there was evidence of a possible link in multivariate analysis between long working hours (particularly in those who exceeded 52 hours a week) and the risk of significant health problems, including hypertension.

In contrast, Nakanishi et al.[21] found that white collar workers who reported 10 or more hours of work per day had a lower risk of developing hypertension when compared with workers reporting less than 8 hours of work per day. However, the studies used varying criteria to determine the number of hours worked. For example, the criterion used to define the group with the lowest number of hours worked ranged widely from 39 to 60 hours per week across studies[22,23] (Table 1).

Summarizing study findings from the existing literature, there is some evidence to suggest that shift work and long working hours can increase blood pressure and lead to increased CVD risk, independent of other stressful conditions at work.[19,20,22–24,29] These findings must be viewed as preliminary, but are intriguing enough to warrant further research on shift or overtime work and CVD.

Noise exposure. Occupational conditions and psychological factors are often indirect, through damage to the central nervous, respiratory and neuroendocrine systems.[37] Noise has been shown to increase catecholamine and cholesterol concentrations in the blood, have an effect on plasma lipoprotein levels, and increase heart rate, arterial blood pressure, and risk of myocardial infarction. Psychophysiological changes caused by long-term stress influence constant pathological changes in the central nervous, endocrine and cardiovascular systems.[38,39] Several epidemiological studies have reported that exposure to noise was associated with CVD, including myocardial infarction and coronary heart disease.[37,40] This association may be due to the fact that noise exposure enhances the development of hypertension.[39]

Chemical exposure. A few occupational exposures to chemicals have been conclusively related to CVD, including dioxin, carbon monoxide, carbon disulfide and nitrate esters.[31,41] Evidence for these exposures is strongest when the level of occupational exposure is high.[31] Kristensen[41] reviewed the epidemiological research on CVD and the work environment and concluded that the causal relationship between CVD and two chemicals, carbon disulfide and nitroglycerin/nitroglycol, was very well documented. However, little is known about other chemical exposures including cobalt, arsenic and antimony. More research is needed to examine these exposures because their relationship with CVD is controversial.[41]

A Swedish cohort study[42] found a slightly increased CVD risk among pulp and paper mill workers. Work with sulfate digestion, steam and power generation, and maintenance were all related to significantly increased risks of death from CVD. These risks were mainly due to deaths from ischemic heart disease, although maintenance work was associated with an increased risk of death from CVD. However, this study did not measure or account for other possible confounding risk factors, in particular smoking.

Passive smoking. There is sufficient evidence that passive smoking increases CVD risk.[43–45] Passive smoking occurs as a consequence of exposure to indoor atmospheric pollution by tobacco smoke. In France, a review underlined the importance of passive smoking at work as a risk factor for CVD.[33] Workers in bars, restaurants, aeroplanes, and nightclubs were particularly vulnerable and displayed significant increases in biological markers of exposure. The effects of passive smoking on health are now scientifically established.[45] A longitudinal cohort study by Felber Dietrich[32] in Sweden used a 24-hour electrocardiogram to test the effect of environmental tobacco smoke on heart rate variability and the role of heart rate and blood pressure in this context. Increased heart rate and blood pressure have been shown to increase the risk for CVD and death. These findings suggest that exposure to environmental tobacco smoke increases cardiac risk through disturbances in the autonomic nervous system.[46] However, so far little is known about the exact mechanism by which passive smoking causes CVD.

Occupational physical activity. Uncertainty exists about the potential protection provided by different levels and types of physical activity on the job. Three specific physical occupational factors such as physical exertion, lifting and vibration have been suggested as possible risk factors for CVD.[47] While increasing the level of physical activity such as exercise or household work is beneficial in preventing CVD, irregular heavy physical exertion has been shown to be associated with substantially increased CVD risk.[34,35]

Consideration for occupational physical activity requires quantification of the work-related metabolic demand. Such studies should evaluate physical activity for leisure and work separately. For example, Krause et al.[34] assessed energy expenditure at work using predicted metabolic equivalents for work tasks and leisure time physical activity by self-report of hours spent exercising. However, when occupational physical activity was considered separately the results were mixed, with some studies demonstrating reduced CVD risk[48] and some demonstrating an increase in risk.[35,49]

Table 2. Summary of major finding by work-related CVD risk factors

Key risk factors		Major findings
Shift work	Kawachi et al. 1995	Relative risks (RR) of CHD: 1.51 (95% CI, 1.12 to 2.03) reporting 6 or more years of rotating night shifts.
	Munakata et al. 2001	SBP and DBP during night shift work and the subsequent awake period correlated with vigour and with confusion.[a]
	Copertaro et al. 2008	No significant association was found between metabolic syndrome and shift work. Shift work was significantly associated with high triglycerides and abdominal obesity.
	Su et al. 2008	12 hour night shift work resulted in elevated SBP, DBP and HR, and decreased HRV.
Overtime work	Park et al. 2001	Long working hours exceeding 50 hours/week is a risk for significant negative health outcomes, including CVD.
	Nakanishi et al. 2001	RR 3.3, 95% CI, 0.11 to 0.95 for hypertension, ≥ 11 hours per day was compared with < 8 hours per day.DBP and ABP decreased as working hours per day increased; 95% CI, 0.11 to 0.95 for hypertension, ≥ 11 hours per day was compared with < 8 hours per day.
	Liu et al. 2002	Weekly working hours were related to progressively increased OR of AMI (weekly working hours ≥ 61) compared with working hours ≤ 40).
	Yang et al. 2006	Working 40 hours/week were 14% (95% CI, 1.01 to 1.28) more likely to report hypertension than were those working 11–39 hours/week.
Noise exposure	Fujino et al. 2007	Noise did not increase CVD risk[b].Perceived noise increased the risk of intra-cerebral haemorrhage disease HR: 2.38.
	Lee et al. 2009	The SBP were 3.8, 2.0 and 1.7 mmHg higher in exposure groups in comparison to that of the NLC-I group (< 60 dB).
	Davies et al. 2009	The estimates were successfully used to demonstrate positive exposure–response relationships for noise and CVD outcomes.
	Tomei et al. 2010	A statistically significant increase of SBP and DBP in high exposure workers compared to low exposure and intermediate exposure workers.
Chemical exposure	Kristensen 1989	A causal relationship between carbon disulfide and nitroglycerin/nitroglycol, and CVD. Exposure to carbon monoxide (CO) increases the risk of CVD.
	Axelson et al. 1994	Increased mortality from circulatory disorders (SMR 1.17; 95% CI, 1.00 to 1.37).
	Persson et al. 2007	Work with sulfate digestion, steam and power generation, and maintenance was associated with significantly increased CVD risks.
	Laden et al. 2007	Observed elevated rates for lung cancer, IHD, and transport-related accidents.
	Humblet et al. 2008	Dioxin exposure was associated with mortality from both IHD and all CVD. Weakness: lack of adjustment for confounding with other major risk factors for CVD.
Passive smoking	Lopez et al. 2007	Exposure to ETS at home and work could be responsible for 1228–3237 of deaths.
	Felber Dietrich et al. 2007	Exposure to ETS at home and work is associated with lower HRV and with higher heart rate in an aging population.Exposure to ETS increases cardiac risk through disturbances in the autonomic nervous system.
	He et al. 2008	Individuals exposed to ETS had a higher risk of CHD (OR, 1.69; 95% CI, 1.31 to 2.18) and ischemic stroke (OR, 1.56; 95% CI, 1.03 to 2.35) than did those never exposed to ETS after adjustment for potential risk factors.
	Venn et al. 2007	Compared with subjects with no detectable cotinine, those with detectable cotinine had significantly higher levels of both fibrinogen (8.9 mg/dL; 95% CI, 0.9 to 17.0) and homocysteine (0.8 μmol/L; 95% CI, 0.4 to 1.1).
	Fransson et al. 2004	A distinct dose–response was noted between leisure-time physical activity and the MI.
	Krause et al. 2007	Maximum strain resulted in a 90% increase in IMT among the men with IHD, compared with a 46% increase among those without IHD.
	Hu et al. 2007	The multivariable-adjusted HRs of CHD events associated with low, moderate and high leisure-time physical activity were 1.00, 0.95 and 0.84 ($p < 0.05$).Active commuting had a significant inverse association with the risk of CHD events in women but not in men.
	Kristal-Boneh et al. 2000	HR of CVD mortality in workers with a high physical workload was 1.82 (95% CI, 1.18 to 2.81) compared with workers having a low workload. A high physical workload is associated with increased mortality rates.

(Continued)

Table 1. (Continued)

Key risk factors		Major findings
Social support	Steptoe et al. 2000	SBP and DBP during the work day were greater in high job demand participants who were stress reactive than in other groups.Social support buffering the stress.
	Guimont et al. 2006	Men with cumulative exposure showed significant SBP increments of 1.8 mmHg (95% CI, 0.1 to 3.5).Effects tended to be more pronounced among workers with low levels of social support.
	Hughes et al. 2009	Social support was associated with reduced resting cardiovascular function.Social support accounted for as much variance as did gender.
Job stress	Niedhammer et al. 1998	Psychosocial work factors were associated with hypertension, hyperlipidemia and diabetes.The results underline the potential effect of psychosocial work characteristics on CVD risk and differences between the effects of job stress in men and women.
	Bosma et al. 1998	The imbalance between personal efforts and rewards was associated with a 2.15-fold higher risk of new CHD.ORs for low job control were 2.38 and 1.56 for self-reported and externally assessed job control.
	Peter et al. 2002	Improved risk estimation of acute MI by combining the two job stress models. Controlling each job stress model for the other to test the independent effect of either approach did not show systematically increased OR[b].
	Fauvel et al. 2003	High stress group did not show an increased incidence of progression to hypertension.
	Ramey 2003	Stress contributes to CVD development and several CVD risk factors.
	Ferris et al. 2005	Job demand predicted perception of lack of job resilience but not lack of personal resilience[b].
	Kang et al. 2005	Job strain was significantly related to higher levels of SBP.
	Ducher et al. 2006	Job strain was associated with BP in a predominantly male subgroup.
	Rose et al. 2006	Job stress factors were not related to coronary risk factors[b].
	Tobe et al. 2007	The interaction between job strain and marital cohesion was significantly associated with a change in ABP during one year.
Social class and occupational status	Tuchsen et al. 1999	Blue collar workers had a higher relative risk than white collar workers.
	Netterstrom et al. 1999	Job stress had a significantly increased OR 95% CI of 2.1 (1.2 to 3.8) for MI.Job category (blue collar workers vs white collar workers, OR 2.8), and employment sector (private vs public, OR 3.1).
	Baigi et al. 2002	The risk was 23% higher for male blue collar workers and 44% higher for female blue collar workers when compared to white collar.
	Landsbergis et al. 2003	An association between job strain and work ABP was found from 2.7–11.8 mmHg SBP, 1.9–6.1 mmHg DBP.These data provide evidence that the relationship between job strain and BP is greater among men with lower SES.
	Netterstrom et al. 2006	High job demands contributed significantly to IHD incidence.
	Chen et al. 2007	Blue collar workers had a 5.3-fold (95% CI 1.5 to 18.5) increased risk of acute IHD.

[a]Statistically significant; [b]nr, not related to CVD risk.

Psychosocial factors at work

Psychosocial factors at work have been examined for their relationships with CVD in many studies conducted in diverse occupational settings. The majority of studies were mediated by job stress.[50–53] The existing literature shows inconsistent results across studies,[54,55] but a review study showed that considerable evidence for significant associations between job stress and CVD risk has nonetheless accumulated.[54]

Social support. Various aspects of social support have been measured in a few studies. Social support at work might reduce the effects of job strain on CVD or blood pressure.[55,56] In a review of the effect of social support on CVD,[57] physiological evidence suggests that cardiovascular responses to stress are associated with the development of CVD, but did not indicate how potential stress responses could be reduced by social support. Experimental work on social support and CVD overcomes many of these limitations. Recently, Hughes and Howard[58] examined healthy

people in a cross-sectional psychosocial screening study ($N=211$). They used regression analysis to assess associations with psychometric indices of social support (perceived network size and perceived satisfaction with support) while controlling for a range of potential biometric and psychometric confounders. Overall, social support was found to be independently associated with reduced resting cardiovascular function, which could reduce CVD risk.

Job stress. Previous occupational stress studies provide strong evidence that job stress is a risk factor for CVD.[36,51–53,59] Direct and indirect mechanisms might explain the relationship between job stress and CVD. The direct mechanism increases left ventricular mass through physiological variables such as increased blood pressure and serum cholesterol. Suspected physiological effects of job stress that could increase blood pressure mainly include sympathetic pathways[52] and pituitary-adrenocortical hormones,[60] and a highly deleterious combination.[61] The indirect mechanism works through behavioural risk factors such as smoking and alcohol consumption to cope with job stress.

Psychosocial factors at work have been associated with an increased risk of CVD.[62] Job stress has mainly been evaluated using the Job Demand Control Model[53,63] and the Effort–Reward Imbalance Model.[53,64] There is a conceptual and methodological overlap between the two models with respect to the demand component. However, clear conceptual and methodological differences exist. First, the Job Demand Control Model has been introduced and used as a concept that is restricted to the structural aspects of the psychosocial work environment, whereas the Effort–Reward Imbalance Model includes both structural and personal characteristics. Second, components of the Effort–Reward Imbalance Model (salaries, career opportunities and job security) are linked to more distant macroeconomic labour market conditions, while the major focus of the Job Demand Control Model is on workplace characteristics. In both models, job stress has been associated with an increased risk of CVD in both men and women.[51–53,64] However, the effect of job stress on CVD is still subject to debate.[63,65] Most, but not all, cross-sectional or short-term studies report that subjects exposed to high job stress have higher blood pressure or heart rates.

Studies of industrial workers have also suggested that psychosocial work factors independently contribute to CVD.[51,52,61] Niedhammer et al.[61] found significant associations between three psychosocial factors (psychological demands, decision latitude and social support) and CVD risks such as hypertension, hyperlipidemia and diabetes in a cohort study of 13,226 French industrial workers. The cross-sectional results underline the potential effect of psychosocial work characteristics on CVD risk factors and differences between the effects of job stress in men and women.[61] High psychological demands were significantly associated with hyperlipidemia (OR 1.32, 95% CI 1.0 to 1.72). However, psychosocial work factors and CVD risk factors were based on self-report, which reflects a partially objective work environment.

Social class, occupational status and CVD. Lower socioeconomic status is consistently associated with a wide variety of disease outcomes, including hypertension and CVD.[66] Studies found that blue collar workers had an increased risk of myocardial infarction[67] and ischemic heart disease[7,68,69] compared with white collar workers. These studies provided evidence that workers from more disadvantaged groups and low socioeconomic classes are at higher risk of ischemic heart disease. Thus, social inequality with respect to CVD has been documented in studies from working populations.[15]

In summary, the effects of job stress and social support on CVD risk are relatively well supported by the literature.[36,51–53,56] This highlights the importance of primary prevention of CVD and the significant contribution of psychosocial components to CVD risk in workers. Furthermore, socioeconomic status should not be overlooked as an independent risk factor for CVD, along with the causal criteria used in evaluating socioeconomic status as a risk factor.

Discussion

All of the research studies reviewed for this paper demonstrated several limitations, including a lack of homogeneity in research design, differences in sample size, and lack of theoretical models to guide the research. Of the studies reviewed, only one used a clinical research design.[19] Four studies enrolled less than 100 participants,[19,24,56,67] while other studies used populations based on more than 10,000 participants.[25,37,40,70–72] In addition, the lack of quality assessment of the studies in the initial stage and a criterion standard for the measurement of CVD outcome prevented the possibility of conducting a meta-analysis. These limitations made it difficult to compare outcomes across studies.

Moreover, most of the studies included in this review were cross-sectional in design. Thus, causality is usually questionable. For example, work-related risk factors may change for workers with CVD risk who move to less stressful or less physically demanding jobs because of their symptoms. This can lead to an underestimation of the association between exposure to risk factors and outcome.[18] A study by Rose et al.[59] found that the association between job stress and CVD risk was not significant within a cross-sectional study, but was significant with prospective data, leading to weakened evidence. Prospective studies can provide important information with respect to increasing or decreasing incidence and can establish estimates of CVD risk.

A considerable research effort, however, has been made to identify risk factors for CVD in workers. Single factors

might include exposure to a chemical like dioxin.[31,42,70,73] More complex psychosocial exposures might include work involving high psychological demand and low decision latitude.[51–53,56] Job stress, social support, shift work and overtime may contribute to the development of CVD in the workplace. Passive smoking may also contribute to CVD, particularly in occupations with high exposure. Combined exposures or some measure of total occupational burden may best explain why certain occupational factors consistently show high CVD risk. CVD in workers is the result of a complex set of factors involving work environment such as shift work, overtime, and noise and chemical exposures; and psychosocial factors such as job stress, social support, and social class; none of which are explained or intervened by a single risk factor. Furthermore, certain CVD risk factors aggravate or attenuate other risk factors. For example, the relationship between job stress and shift work, or job stress and social support, are affected as combined exposures.

While this review included studies based on the predetermined selection criteria, the majority of included papers were international, coming from countries in Europe and Asia (Table 1). This is expected because European countries in particular are ahead of the field in this area of research. This has implications for adapting the results of European or Asian studies regarding work-related CVD factors to other working populations. Additionally, with the exception of the job stress models, there is a significant lack of theoretical models to draw on. Thus, there are no consistent results and interpretations. Future research should incorporate a theoretical model to address this methodological issue.

Implications for practice and research

To develop a comprehensive CVD risk reduction intervention for workers, it is necessary to assess work-related CVD risk factors along with a well-known cardiovascular risk profile that consists of non-modifiable risk factors (age >65; male; positive family history of CVD in a first-degree relative; and ethnicity), and modifiable risk factors.[74]

The findings of this review can apply to cardiovascular health nurses in a number of ways. The key implication for cardiovascular health nursing is the assessment of occupational factors such as work-related environmental and psychosocial factors that should be incorporated into assessment of the patient's CVD risks and intervention plan. When significant occupational risk factors are identified, targeted intervention strategies to mitigate those factors can be developed and implemented. It is also important for cardiovascular health nurses caring for working adults with CVD to coordinate treatment and health promotion plans with the occupational health providers, if they have any.[75] Moreover, cardiovascular health nurses, in collaboration with occupational health providers, should consider the implementation of stress management programmes for workers to reduce their job stress.

Cardiovascular health nurses should consider both the traditional individual determinants of CVD and work-related risk factors when conducting CVD research with working people and conduct longitudinal studies to establish solid causal relationships between reported occupational risk factors in this review and CVD. Future research should also incorporate a well-defined conceptual framework to address the effects of work-related environmental and psychosocial factors on CVD among CVD patients.

Acknowledgements

The authors acknowledge the efforts of Erika S Froelicher, University of California San Francisco, for her consulting advices throughout preparing the draft.

Funding

This study was supported by the Pacific Rim Research Program Advanced Graduate Fellowship Program, University of California.

References

1. Lloyd-Jones D, Adams RJ, Brown TM, et al. Executive summary: heart disease and stroke statistics – 2010 update: a report from the American Heart Association. *Circulation* 2010; 121: 948–954.
2. Berra K, Fletcher BJ, Hayman LL, et al. Global cardiovascular disease prevention: A call to action for nursing The global burden of cardiovascular disease. *Eur J Cardiovasc Nurs* 2011; 10(Suppl 2): S1–2.
3. Andersson P, Sjoberg RL, Krysa M, et al. Lags in behavioral change: A population based comparison of cardiovascular risk behavior in Poland and Sweden. *Cent Eur J Public Health* 2006; 14: 82–85.
4. Gomel M, Oldenburg B, Simpson JM, et al. Work-site cardiovascular risk reduction: a randomized trial of health risk assessment, education, counseling, and incentives. *Am J Public Health* 1993; 83: 1231–1238.
5. Orth-Gomer K. Job strain and risk of recurrent coronary events. *JAMA* 2007; 298: 1693–1694.
6. Williams A, Mason A and Wold J. Cultural sensitivity and day care workers. Examination of a worksite based cardiovascular disease prevention project. *AAOHN J* 2001; 49: 35–43.
7. Tuchsen F and Endahl LA. Increasing inequality in ischaemic heart disease morbidity among employed men in Denmark 1981–1993: the need for a new preventive policy. *Int J Epidemiol* 1999; 28: 640–644.
8. Hall AG, Collins KS and Glied S. *Employer-sponsor health insurance: Implications for minority workers*. New York: Commonwealth Fund, 1999.
9. Groeneveld IF, Proper KI, van der Beek AJ, et al. Design of a RCT evaluating the (cost-) effectiveness of a lifestyle intervention for male construction workers at risk for cardiovascular disease: the health under construction study. *BMC Public Health* 2008; 8: 1.

10. Muntaner C, Sorlie P, O'Campo P, et al. Occupational hierarchy, economic sector, and mortality from cardiovascular disease among men and women. Findings from the National Longitudinal Mortality Study. *Ann Epidemiol* 2001; 11: 194–201.
11. Oetzel JG, Ting-Toomey S and Rinderle S. Conflict communication in contexts: A social ecological perspective. In: Ting-Toomey JGOS (ed.). *The SAGE handbook of conflict communication*. Thousand Oaks, CA: Sage, 2006.
12. National Institute for Occupational Safety and Health. NIOSH Program Portfolio: Cancer, Reproductive, and Cardiovascular Diseases. Atlanta, GA: NIOSH.
13. Cesario S, Morin K and Santa-Donato A. Evaluating the level of evidence of qualitative research. *J Obstet Gynecol Neonatal Nurs* 2002; 31: 708–714.
14. Brown SA, Upchurch SL and Acton GJ. A framework for developing a coding scheme for meta-analysis. *West J Nurs Res* 2003; 25: 205–222.
15. Baigi A, Fridlund B, Marklund B, et al. Cardiovascular mortality focusing on socio-economic influence: the low-risk population of Halland compared to the population of Sweden as a whole. *Public Health* 2002; 116: 285–288.
16. Hoogendoorn WE, van Poppel MN, Bongers PM, et al. Systematic review of psychosocial factors at work and private life as risk factors for back pain. *Spine* 2000; 25: 2114–2125.
17. Karasek R, Brisson C, Kawakami N, et al. The Job Content Questionnaire (JCQ): an instrument for internationally comparative assessments of psychosocial job characteristics. *J Occup Health Psychol* 1998; 3: 322–355.
18. Copertaro A, Bracci M, Barbaresi M, et al. Assessment of cardiovascular risk in shift healthcare workers. *Eur J Cardiovasc Prev Rehabil* 2008; 15: 224–229.
19. Munakata M, Ichi S, Nunokawa T, et al. Influence of night shift work on psychologic state and cardiovascular and neuroendocrine responses in healthy nurses. *Hypertens Res* 2001; 24: 25–31.
20. Kawachi I, Colditz GA, Stampfer MJ, et al. Prospective study of shift work and risk of coronary heart disease in women. *Circulation* 1995; 92: 3178–3182.
21. Nakanishi N, Yoshida H, Nagano K, et al. Long working hours and risk for hypertension in Japanese male white collar workers. *J Epidemiol Community Health* 2001; 55: 316–322.
22. Liu Y and Tanaka H. Overtime work, insufficient sleep, and risk of non-fatal acute myocardial infarction in Japanese men. *Occup Environ Med* 2002; 59: 447–451.
23. Yang H, Schnall PL, Jauregui M, et al. Work hours and self-reported hypertension among working people in California. *Hypertension* 2006; 48: 744–750.
24. Su TC, Lin LY, Baker D, et al. Elevated blood pressure, decreased heart rate variability and incomplete blood pressure recovery after a 12-hour night shift work. *J Occup Health* 2008; 50: 380–386.
25. Fujino Y, Iso H and Tamakoshi A. A prospective cohort study of perceived noise exposure at work and cerebrovascular diseases among male workers in Japan. J Occup Health 2007; 49: 382–388.
26. Fialho G, Cavichio L, Povoa R, et al. Effects of 24-h shift work in the emergency room on ambulatory blood pressure monitoring values of medical residents. Am J Hypertens 2006; 19: 1005–1009.
27. Haus E and Smolensky M. Biological clocks and shift work: circadian dysregulation and potential long-term effects. *Cancer Causes Control* 2006; 17: 489–500.
28. Caruso CC, Hitchcock EM, Dick RB, et al. *Overtime and extended work shifts: recent findings on illnesses, injuries, and health behaviors*. Cincinnati, OH: NIOSH, 2004.
29. Park J, Kim Y, Cho Y, et al. Regular overtime and cardiovascular functions. *Ind Health* 2001; 39: 244–249.
30. Lin YC, Hsiao TJ and Chen PC. Shift work aggravates metabolic syndrome development among early-middle-aged males with elevated ALT. *World J Gastroenterol* 2009; 15: 5654–5661.
31. Humblet O, Birnbaum L, Rimm E, et al. Dioxins and cardiovascular disease mortality. *Environ Health Perspect* 2008; 116: 1443–1448.
32. Felber Dietrich D, Schwartz J, Schindler C, et al. Effects of passive smoking on heart rate variability, heart rate and blood pressure: an observational study. *Int J Epidemiol* 2007; 36: 834–840.
33. Gignon M, Manaouil C, Jarde O, et al. Passive smoking in France: science, law and judge's point of view. *Revue des Maladies Respiratoires* 2007; 24: 133–143.
34. Krause N, Brand RJ, Kaplan GA, et al. Occupational physical activity, energy expenditure and 11-year progression of carotid atherosclerosis. *Scand J Work Environ Health* 2007; 33: 405–424.
35. Fransson E, De Faire U, Ahlbom A, et al. The risk of acute myocardial infarction: interactions of types of physical activity. *Epidemiology* 2004; 15: 573–582.
36. Tobe SW, Kiss A, Sainsbury S, et al. The impact of job strain and marital cohesion on ambulatory blood pressure during 1 year: the double exposure study. *Am J Hypertens* 2007; 20: 148–153.
37. Tomei G, Fioravanti M, Cerratti D, et al. Occupational exposure to noise and the cardiovascular system: a meta-analysis. *Sci Total Environ* 2010; 408: 681–689.
38. Maschke C, Rupp T and Hecht K. The influence of stressors on biochemical reactions – a review of present scientific findings with noise. *Int J Hyg Environ Health* 2000; 203: 45–53.
39. Lee JH, Kang W, Yaang SR, et al. Cohort study for the effect of chronic noise exposure on blood pressure among male workers in Busan, Korea. *Am J Ind Med* 2009; 52: 509–517.
40. Davies HW, Teschke K, Kennedy SM, et al. Occupational exposure to noise and mortality from acute myocardial infarction. *Epidemiology* 2005; 16: 25–32.
41. Kristensen TS. Cardiovascular diseases and the work environment. A critical review of the epidemiologic literature on chemical factors. *Scand J Work Environ Health* 1989; 15: 245–264.
42. Persson B, Magnusson A, Westberg H, et al. Cardiovascular mortality among Swedish pulp and paper mill workers. *Am J Ind Med* 2007; 50: 221–226.
43. He Y, Lam TH, Jiang B, et al. Passive smoking and risk of peripheral arterial disease and ischemic stroke in Chinese women who never smoked. *Circulation* 2008; 118: 1535–1540.
44. Lopez MJ, Perez-Rios M, Schiaffino A, et al. Mortality attributable to passive smoking in Spain, 2002. *Tob Control* 2007; 16: 373–377.

45. Venn A and Britton J. Exposure to secondhand smoke and biomarkers of cardiovascular disease risk in never-smoking adults. *Circulation* 2007; 115: 990–995.
46. Zhang J, Liu Y, Shi J, et al. Side-stream cigarette smoke induces dose-response in systemic inflammatory cytokine production and oxidative stress. *Exp Biol Med (Maywood)* 2002; 227: 823–829.
47. Belkic K, Schnall P and Ugljesic M. Cardiovascular evaluation of the worker and workplace: a practical guide for clinicians. *Occup Med* 2000; 15: 213–222, iv.
48. Hu G, Jousilahti P, Borodulin K, et al. Occupational, commuting and leisure-time physical activity in relation to coronary heart disease among middle-aged Finnish men and women. *Atherosclerosis* 2007; 194: 490–497.
49. Kristal-Boneh E, Silber H, Harari G, et al. The association of resting heart rate with cardiovascular, cancer and all-cause mortality. Eight year follow-up of 3527 male Israeli employees (the CORDIS Study). *Eur Heart J* 2000; 21: 116–124.
50. Ramey SL. Cardiovascular disease risk factors and the perception of general health among male law enforcement officers: encouraging behavioral change. *AAOHN J* 2003; 51: 219–226.
51. Kang MG, Koh SB, Cha BS, et al. Job stress and cardiovascular risk factors in male workers. *Prev Med* 2005; 40: 583–588.
52. Ducher M, Cerutti C, Chatellier G, et al. Is high job strain associated with hypertension genesis? *Am J Hypertens* 2006; 19: 694–700.
53. Bosma H, Peter R, Siegrist J, et al. Two alternative job stress models and the risk of coronary heart disease. *Am J Public Health* 1998; 88: 68–74.
54. Eller NH, Netterstrom 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.
55. Guimont C, Brisson C, Dagenais GR, et al. Effects of job strain on blood pressure: a prospective study of male and female white-collar workers. *Am J Public Health* 2006; 96: 1436–1443.
56. Steptoe A. Stress, social support and cardiovascular activity over the working day. *Int J Psychophysiol* 2000; 37: 299–308.
57. Christenfeld N and Gerin W. Social support and cardiovascular reactivity. *Biomed Pharmacother* 2000; 54: 251–257.
58. Hughes BM and Howard S. Social support reduces resting cardiovascular function in women. *Anxiety Stress Coping* 2009; 22: 537–548.
59. Rose G, Kumlin L, Dimberg L, et al. Work-related life events, psychological well-being and cardiovascular risk factors in male Swedish automotive workers. *Occup Med (Lond)* 2006; 56: 386–392.
60. Kunz-Ebrecht SR, Kirschbaum C, Marmot M, et al. Differences in cortisol awakening response on work days and weekends in women and men from the Whitehall II cohort. *Psychoneuroendocrinology* 2004; 29: 516–528.
61. Niedhammer I, Goldberg M, Leclerc A, et al. Psychosocial work environment and cardiovascular risk factors in an occupational cohort in France. *J Epidemiol Community Health* 1998; 52: 93–100.
62. Orth-Gomer K. Psychosocial and behavioral aspects of cardiovascular disease prevention in men and women. *Curr Opin Psychiatry* 2007; 20: 147–1-51.
63. Ferris PA, Sinclair C and Kline TJ. It takes two to tango: personal and organizational resilience as predictors of strain and cardiovascular disease risk in a work sample. *J Occup Health Psychol* 2005; 10: 225–238.
64. Peter R, Siegrist J, Hallqvist J, et al. 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.
65. Fauvel JP, M'Pio I, Quelin P, et al. Neither perceived job stress nor individual cardiovascular reactivity predict high blood pressure. *Hypertension* 2003; 42: 1112–1116.
66. Landsbergis PA, Schnall PL, Pickering TG, et al. Lower socioeconomic status among men in relation to the association between job strain and blood pressure. *Scand J Work Environ Health* 2003; 29: 206–215.
67. Netterstrom B, Nielsen FE, Kristensen TS, et al. Relation between job strain and myocardial infarction: a case-control study. *Occup Environ Med* 1999; 56: 339–342.
68. Netterstrom B, Kristensen TS and Sjol A. Psychological job demands increase the risk of ischaemic heart disease: a 14-year cohort study of employed Danish men. *Eur J Cardiovasc Prev Rehabil* 2006; 13: 414–420.
69. Chen JD, Cheng TJ, Lin YC, et al. Job categories and acute ischemic heart disease: a hospital-based, case-control study in Taiwan. *Am J Ind Med* 2007; 50: 409–414.
70. Laden F, Hart JE, Smith TJ, et al. Cause-specific mortality in the unionized U.S. trucking industry. *Environ Health Perspect* 2007; 115: 1192–1196.
71. Pizzi C, Evans SA, De Stavola BL, et al. Lifestyle of UK commercial aircrews relative to air traffic controllers and the general population. *Aviat Space Environ Med* 2008; 79: 964–974.
72. Sisson SB, Camhi SM, Church TS, et al. Leisure time sedentary behavior, occupational/domestic physical activity, and metabolic syndrome in U.S. men and women. *Metab Syndr Relat Disord* 2009; 7: 529–536.
73. Axelson O, Selden A, Andersson K, et al. Updated and expanded Swedish cohort study on trichloroethylene and cancer risk. *J Occup Med* 1994; 36: 556–562.
74. D'Agostino RB, Sr, Vasan RS, Pencina MJ, et al. General cardiovascular risk profile for use in primary care: the Framingham Heart Study. *Circulation* 2008; 117: 743–753.
75. Eshah NF, Bond AE and Froelicher ES. The effects of a cardiovascular disease prevention program on knowledge and adoption of a heart healthy lifestyle in Jordanian working adults. *Eur J Cardiovasc Nurs* 2010; 9: 244–253.