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Adverse effects of psychosocial work factors on blood pressure: systematic review of studies on demand-control-support and effort-reward imbalance models

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This is the first systematic review and critical synthesis on the adverse effects of psychosocial work factors of the demand–control–support and effort–reward imbalance models on blood pressure. A more consistent effect has been observed among men than women and among studies of higher methodological quality. Evidence-based guidelines for cardiovascular diseases prevention may benefit from these findings.

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Key terms: blood pressure; cardiovascular risk factor; cardiovascular risk factor; demand–control model; demand–control–support model; effort–reward imbalance; effort–reward imbalance model; hypertension; job control; Karasek; overcommitment; psychological demand; psychosocial risk factor; Siegrist; systematic review; workplace stressor

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Adverse effects of psychosocial work factors on blood pressure: systematic review of studies on demand–control–support and effort–reward imbalance models

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Objectives A growing body of research has investigated the adverse effects of psychosocial work factors on blood pressure (BP) elevation. There is now a clear need for an up-to-date, critical synthesis of reliable findings on this topic. This systematic review aimed to evaluate the adverse effects of psychosocial work factors of both the demand–control–support (DCS) and effort–reward imbalance (ERI) models on BP among men and women, according to the methodological quality of the studies.

Methods To be eligible, studies had to: (i) evaluate at least one psychosocial work factor, (ii) evaluate BP or hypertension, (iii) comprise ≥100 workers, (iv) be written in English or French, and (v) be published in a peer-reviewed journal.

Result A total of 74 studies were included. Of these, 64 examined the DCS model, and 12 looked at the ERI model, with 2 studies considering both models. Approximately half the studies observed a significant adverse effect of psychosocial work factors on BP. A more consistent effect was observed, however, among men than women. For job strain, a more consistent effect was also observed in studies of higher methodological quality, ie, studies using a prospective design and ambulatory BP measures.

Conclusions A more consistent adverse effect of psychosocial work factors was observed among men than women and in studies of higher methodological quality. These findings contribute to the current effort of primary prevention of cardiovascular disease by documenting the psychosocial etiology of elevated BP, a major cardiovascular risk factor.

Key terms cardiovascular risk factor; hypertension; job control; Karasek; overcommitment; psychological demand; Siegrist; workplace stressor.

Cardiovascular diseases (CVD) are the leading cause of death worldwide (1). In Canada, these diseases account for one third of male and female deaths (2) and are the most costly group of health problems in terms of hospitalization (3). High blood pressure (BP) is a major risk factor for CVD (4). Indeed, it accounts for an estimated 54% of all strokes and 47% of all ischemic heart disease events globally (5). Among adults, almost one American in five (6) and one Canadian in five (7) has high BP. The risk of cardiovascular mortality grows linearly with BP from 115/75 mm Hg among adults aged 40–69 years-old with no CVD. At the population level, even a mean systolic BP that was 2 mm Hg lower would lead to a reduction in middle-age mortality from coronary heart disease and stroke of approximately 7% and 10%, respectively (8, 9). Over recent decades, a growing number of studies have investigated the adverse effects of psychosocial factors, including those of the workplace (ie, work stress), on BP elevation.

Two well-defined and internationally recognized theo-

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retical models have been used to assess the adverse effects of psychosocial work factors on BP: the demand–control–support (DCS) (10) and the effort–reward-imbalance (ERI) (11) models. The DCS model suggests that workers simultaneously experiencing high psychological demands and low job control are more likely to develop stress-related health problems (10). Psychological demands mainly refer to an excessive workload, very hard or overly fast work, and conflicting demands. Job control is a combination of skill discretion (eg, learning new things, opportunities to develop skills, creativity, a variety of activities, non-repetitive work) and decision authority (eg, taking part in decisions affecting oneself, making one's own decisions, having a say on the job, and freedom as to how the work is accomplished) (10). Johnson et al (12) introduced poor social support as a third component of the demand–control model. This component refers to a lack of help and cooperation from supervisors and coworkers. The ERI model proposes that extrinsic efforts (eg, pressure to work overtime, increasingly demanding work, constant time pressure, repeated interruptions) should be rewarded in various ways, namely: financially (income), socially (respect, esteem), and organizationally (job security, promotion prospects) (11). Workers are in a state of detrimental imbalance when high extrinsic efforts are accompanied by low reward and thus more susceptible to health problems. A third component, overcommitment, is a personal coping style that presents as being unable to withdraw from work obligations, being impatient and irritable, and having a high need for approval (13). Overcommitment may act either directly or as a modifier (ie, amplifier) of the ERI effect (13).

Two main biological pathways have been suggested to explain how psychosocial work factors contribute to BP elevation. Firstly, CVD results from a chain of events linking risk factors to cardiovascular events, which is summarized in Dzau's CVD "continuum" (14, 15). First, asymptomatic damage occurs from interactions between genetic and environmental risk factors (14, 15). This damage then amplifies over time to trigger cardiovascular events. For example, BP elevation could successively lead to hypertension, arterial stiffness, and stroke or ischemic heart disease. Several epidemiological studies have demonstrated that psychosocial stressors might contribute to the incidence of CVD. Even though possible mechanisms are not clearly defined (16), based on experimental studies, one can reasonably assume that the deleterious effects of psychosocial stressors arise from the cumulative impact of multiple and prolonged exposures. These studies have provided evidence that the sympathetic nervous system, a primary mediator of the stress response, is one of the major pathways activating the renin-angiotensin system (17, 18). Stress can therefore stimulate the secretion of renin and increase plasma levels of angiotensin II, which has a significant effect on blood vessel walls. Indeed, angiotensin II plays a crucial role in the development of CVD by causing vasoconstriction, endothelial dysfunction, cellular proliferation, and inflammation that promotes atherosclerosis (19–22). In conjunction with sympathetic activation and hypothalamo-pituitary-adrenal axis stimulation, activation of the renin–angiotensin system can lead to hypertension and cardiovascular events (17, 18). Secondly, psychosocial work factors could act more indirectly on BP through known risk factors or risk behaviors (eg, obesity, smoking, lack of physical exercise, or excessive alcohol consumption) (22–25).

Six systematic reviews have been conducted to investigate the adverse effect of psychosocial work factors on CVD (26–30, 31). These reviews concluded that these psychosocial factors play an important role in the etiology of CVD. Five reviews also reported that adverse effects were more consistently observed among men than women (26–30). A possible explanation for these gender differences is the fact that on average, CVD occur ten years later among women (32). Therefore, work-related CVD might occur at the end of or after the work period among women, leading to low statistical power to detect an effect in some studies. However, such a limitation might be of lesser importance in studies on BP, since BP elevations tend to occur earlier in life than CVD. It is also worth adding that large studies conducted in the US and Europe observed a consistently higher proportion of women exposed to adverse psychosocial work factors than men (33). High job strain and ERI are therefore a frequent psychosocial exposure among women.

Two recent literature reviews (34, 35) have presented evidence that adverse psychosocial work factors may also be a risk factor for BP elevation. Based on 22 cross-sectional studies, Landsbergis et al (35) presented higher pooled BP means of +3.43 mmHg (systolic) and +2.07 mm Hg (diastolic) among workers exposed to high job strain as compared to non-exposed workers. However, 13 of 22 studies observed no significant effects (35), thereby indicating inconsistencies. These two literature reviews were limited by the fact that they: (i) took a narrative approach (non-systematic) (34), (ii) did not systematically evaluate ERI (34, 35), and (iii) did not systematically investigate the effects on hypertension (34, 35). Therefore, no previous systematic review has investigated the adverse effects of both the DCS and ERI factors on BP level and hypertension. There is thus a need to investigate the consistency of effects according to gender and the methodological quality of studies.

The general objective of this systematic review was to evaluate the effects that the psychosocial work factors of both the DCS and ERI models had on BP among men and women. The period under study was 1979 (year of the first publication presenting the demand–control model) (10) to 4 November 2011. The following specific objectives were assessed: (i) Do workers exposed to psychosocial work factors of the DCS and ERI models have higher BP than unexposed workers? (ii) Are there gender differences in the effects of these psychosocial work factors on BP? (iii) Do studies of higher methodological quality, particularly studies with a prospective design and ambulatory BP measures, present more consistent adverse effects than studies of lesser methodological quality?

Methods

Search strategy

We conducted a systematic review to evaluate the association between adverse psychosocial work factors and BP among men and women. All relevant citations were collected and analyzed with a predefined strategy. Relevant citations were extracted from PubMed, Embase, Web of Science, and PsycInfo databases from 1979 to January 2011. Keywords and MeSH terms were combined to generate lists of publications. The databases were searched with a combination of three types of search strings (the complete search strategy is available on request) with terms related to: (i) the work setting: job, work, occupation, occupations, workplace, worker, employee; (ii) psychosocial factors: psychosocial factors, psychosocial work factors, psychosocial work-related factors, job stress, job-related stress, work stress, work-related stress, psychosocial, psychosocial stress, psychological demand, job demand, demand, job control, job control, job strain, iso-strain, social support, reward, effort–reward imbalance, effort reward, Karasek, Siegrist, psychosocial environment; (iii) BP: BP, hypertension, ambulatory BP, BP monitoring, cardiovascular responses, cardiovascular risk factors, systolic BP, and diastolic BP.

For practical reasons, publications had to be available in English or French. For scientific reasons, such as improved credibility and relevance, publications had to be available in peer-review journals. In the first step, a first reviewer selected studies on the basis of the title. In the second step, the abstracts of all the selected titles were sorted for a more detailed evaluation. Two independent reviewers read the abstracts and categorized them as relevant, not relevant, and possibly relevant. The same two reviewers fully reviewed, synthesized, and approved the relevant and possibly relevant publications. The quality and integrity of this review were optimized by following the validated PRISMA (preferred reporting items for systematic reviews and meta-analyses) recommendations (36).

Selection criteria

The selected populations had to include populations of >100 workers at baseline. Workers had to be exposed to psychosocial work factors of the DCS and/or ERI models. The comparison groups had to be composed of workers unexposed to the corresponding psychosocial work factors. The cut-offs between exposed and unexposed workers were generally determined by the median score of the study population or by the median score observed in a reference population (eg, the working population of a given country). To be included, a study must also have assessed these psychosocial work factors at the individual level. Articles based on imputed job title exposure score were therefore excluded since they are more vulnerable to misclassification (28, 29, 37).

The outcome had to be defined by (i) BP level (ie, mean or coefficient) or (ii) hypertension incidence or prevalence. Studies using office BP or ambulatory measurements were included. Office hypertension was generally defined as systolic or diastolic BP mean ≥140 mm Hg and ≥90 mm Hg, respectively. Ambulatory hypertension was generally defined as systolic or diastolic BP mean ≥135 mm Hg and ≥85 mm Hg, respectively (38). However, some studies used higher cut-offs to define hypertension (see tables A–C, available at www.sjweh.fi/data_repository.php). Studies on gestational hypertension were excluded.

Cross-sectional, prospective, and case–control studies were included. Narrative reviews and duplicates were excluded. Multiple publications based on the same study population were retained if the analyses were conducted for different exposures or outcomes.

Analysis

An effect was defined as being a statistically significant difference in BP between workers exposed to psychosocial work factors and those unexposed. Effect measures [differences in mean, beta coefficients, correlation coefficients, risk ratios (RR), and odds ratios OR)] and their P-value or 95% confidence interval (95% CI) were presented for each study, when available. Effects were presented for combinations of psychosocial work factors and for each factor taken separately. Results were also synthesized according to study design (cross-sectional, prospective, case–control), type of BP measures (office, ambulatory), and outcome (BP level, hypertension).

Results

Overview of included studies

The literature search provided 2913 citations, 161 of which were selected as potentially relevant (figure 1). After a complete review of the full articles, 87 studies were excluded because they: (i) did not measure the psychosocial work factors of the DCS and/or ERI models (N=39) (39–77), (ii) did not individually assess exposure to psychosocial factors (N=3) (78–80), (iii) comprised a population of high school students (not a working population) (N=1) (81), (iv) included <100 participants (N=13) (82–93); (v) were not written in English or French (N=4) (94–97), (vi) were not published in a peer-reviewed publication (N=16) (11, 98–112), (vii) did not measure BP (N=8) (70, 113–119), or (viii) did not distinctly evaluate exposure to psychosocial work factors (N=3) (120–122). Because these last studies evaluated interaction with multiple exposures, it was not possible to isolate the impact of the psychosocial factors that were of interest in our review (120–122). A total of 74 studies were ultimately included (23, 34, 44, 123–192).

The 74 studies were published between 1982–2011; 57 were cross-sectional, 15 prospective cohorts, and 2 case–control studies. Among the prospective studies, the follow-up durations ranged from 6 weeks to 12 years. Office and ambulatory BP measures were used in 45 and 28 studies, respectively. There were 64 studies on the DCS model (tables A and B) and 12 studies on the ERI model (table C), two studies considered both models (151, 155). Studies were conducted in 18 countries and included various working populations aged ≥15 years (representative samples of the general working population, white-collar workers, bus drivers, nurses, teachers, patrol officers, etc.; tables A–C).

Except for five studies on the DCS model (152, 160, 172, 173, 179), the studies included in this review controlled for at least one potential confounder. Potential confounders were sociodemographic (age, gender, ethnicity), socioeconomic (education, income, occupation), lifestyle risk factors (smoking, alcohol or caffeine consumption, physical activity, stressful situations, personality traits), biological risk factors (body mass index, waist circumference, known history of CVD, diabetes, medication for hypertension, menopausal status, estrogen medication, pregnancy history, sodium intake, cholesterol), and other factors (marital status, number of children, posture, stress outside work, having eaten a meal, length of time in the current job, and social support at work and outside work).

Studies on DCS model

Tables 1 and 2 summarize the results of the studies on the DCS model according to methodological characteristics and gender, while tables A and B (www.sjweh.fi/data_repository.php) detail the characteristics and results of these studies.

Overall, 21/40 studies observed a significant deleterious effect of job strain on BP level and 7/19 studies observed such an effect on hypertension (table 1). Significant deleterious effects were also observed for high psychological demands in 7/25 studies on BP level and 2/7 studies on hypertension, and for low social support in 1/9 studies on BP level. As well, a significant effect was observed for high job control (protective effect) in 9/25 studies on BP level and 3/6 studies on hypertension. However, no significant effects were observed in the three studies on iso-strain.

Of the 40 cross-sectional studies on job strain (table A, figures 2–4), 16 observed a significant deleterious effect, namely: (i) differences in systolic and diastolic BP means ranging respectively from +2–+10.2 mm Hg (129, 133, 147, 150–152, 169, 170, 172, 181) and from +2–+17.97 mm Hg (1333, 147, 151, 152, 169, 170, 172, 181); (ii) OR ranging from 1.18–2.9 (132, 177, 181); (iii) beta coefficients of systolic and diastolic BP of 4.53 (175) and 0.23 (178) respectively; and (iv) P-values <0.05 for the association between job strain and mean systolic BP (44, 139) (table 1). Two studies reported a significant protective effect of job strain on hypertension (131, 138, 157): OR for hypertension of 0.61 (157) and 0.63 (systolic hypertension) (131).

Of the 12 prospective studies on job strain (table B, figures 2–4), 9 observed a significant deleterious effect, namely: (i) differences in systolic and diastolic BP means ranging respectively from +1.2–+7.7 mm Hg (142, 155, 161, 180, 182) and from +0.8–+7 mm Hg (161, 182); (ii) a hypertension OR of 1.27 after being exposed to job strain at baseline and an OR of 2.06 for a change from low to high job strain during an 8-year follow-up (154); (iii) an RR for a systolic BP increase in the highest quintile of 1.33 among men (142); (iv) a beta coefficient of systolic BP of 0.19 (166); and (v) a P-value of <0.01 for the association between job strain and mean systolic BP (176) (tables 2 and A). Contrary to what was expected, one study observed a significant protective effect of job strain on BP (138).

The two case–control studies observed that job strain had a significant deleterious effect on hypertension (table 1) (163, 167). The OR for hypertension were 2.6 and 2.7, respectively. One of these studies also presented differences in mean BP (systolic: +6.8 mm Hg, diastolic: +2.6 mm Hg) (167). It is also worth noting that one case–control study evaluated the effect of low social support at work and observed no effect among either men or women (163).

Gender. A majority of the 40 cross-sectional studies on job strain presented results separately for men and women; 19 presented results solely for men, and 15 presented results solely for women (table 2, figures 2–4). A higher proportion of studies observed a deleterious effect among men (BP level: 6/18 studies, hypertension: 2/5 studies) than women (BP level: 1/10 studies, hyper-

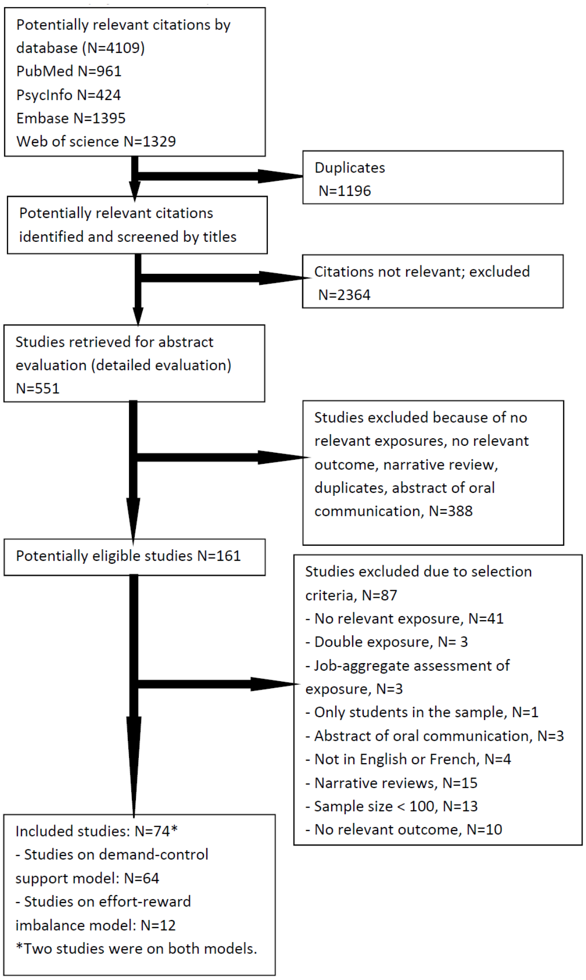

Figure 1. Summary of the selection process.

tension: 0/7 studies) (table 2, figures 2–4). Two studies on hypertension reported a significant deleterious effect among men [OR 1.18 (177) and 2.9 (181)], while none (0/7 studies) observed such an effect among women.

In addition, a slightly higher proportion of cross-sectional studies observed a deleterious effect of high psychological demands (4/11 studies) and high job control (6/10 studies) among men compared to women (demands: 2/11 studies, control: 5/11 studies) (table A).

Of the 12 prospective studies on job strain, 5 presented separate results for men and 4 presented separate results for women (table 2, figures 2–4). In studies on BP level, a higher proportion observed a deleterious effect among men (5/5 studies) compared to women (2/4 studies) (table 2). Moreover, the only study on hypertension reported a deleterious effect among men versus no effect among women (145) (table 2).

The effects of high psychological demands and low job control that were observed in prospective studies were not consistent among either men (demands: 2/7 studies, control: 2/7 studies) or women (demands: 2/5 studies, control: 3/5 studies) (table 2). In addition, the only study that evaluated the effect of low social support observed a deleterious effect among women but not men (133) (table 2).

Study design. For job strain, a higher proportion of prospective studies yielded a deleterious effect on BP mean level as compared to cross-sectional studies (significant effect in 7/9 studies as compared to 13/30 studies) (table B). However, a prospective design did not lead to a more consistent effect in studies on hypertension (significant effect in 2/5 prospective studies as compared to 3/12 in cross-sectional studies).

Type of BP measures. Office and ambulatory BP measures were used in respectively 39 and 27 studies on job strain (table 1). Overall, a higher proportion of studies using ambulatory BP measures (13/20 studies) observed an adverse effect of job strain than did studies using office measures (12/35 studies) (table 1). This observation mostly applies for cross-sectional studies. Indeed, among cross-sectional studies, 9/15 studies using ambulatory BP measures observed a significantly deleterious effect as compared to 7/27 studies using office BP measures (table 1). However, in prospective studies on BP level, ambulatory BP measures (4/5 studies) did not lead to a more consistent deleterious effect than office measures (3/4 studies), which could be due to the small number of studies (table B). Only one prospective study on hypertension used ambulatory BP measures.

Among studies evaluating the separate effects of the demand–control–support factors, the use of ambulatory or office BP measures led to inconsistent findings for high psychological demands (ambulatory BP: 3/9 studies, office BP: 2/9 studies) and high job control (ambulatory BP: 4/9 studies, office BP: 2/9 studies) (table 1). Moreover, only 1/12 studies on the separate effect of low social support observed a significant deleterious effect (table 1).

Studies on ERI model

Tables 3 and 4 summarize the results of the studies on the model according to methodological characteristics and gender, while table C details the characteristics and results of these studies.

In studies on ERI, 4/7 studies observed a significantly deleterious effect of ERI on BP level and 5/6 studies observed such an effect on hypertension (table 3). A significant deleterious effect of overcommitment on BP level was also observed in 2/4 studies (table 3).

Of the 11 cross-sectional studies of the ERI model, 7 studies observed a significant deleterious effect (table C, figures 5–6), namely: (i) differences in systolic and diastolic BP means ranged respectively from +1.86–

Table 1. Number of studies reporting a statistically significant deleterious effect / total number of studies having these methodological characteristics (reference number) reporting a deleterious effect of the demand–control–support factors on blood pressure according to study designs (cross-sectional, prospective or case–control), blood pressure (BP) measurements (office or ambulatory), and outcome (hypertension or BP level).

	Cross-sectional studies (N=50) [a]		Prospective studies (N=14)		Case-control studies (N= 2)		Total
	Office BP (N=29/50)	Ambulatory BP (N= 21/50)	Office BP (N=9/14)	Ambulatory BP (N=5/14)	Office BP (N=1/1)	Ambulatory BP (N=1/1)	
Job strain (N=52)							
Hypertension	1 [b] (177) / 10 (33, 123, 124, 131, 134, 135, 147, 149, 157, 177)	2 (132, 181) / 2 (132, 181)	2 [c] (142, 154) / 4 (23, 142, 148, 154)	0 / 1 (137)	1 (163) /1 (163)	1 (167) / 1 (167)	7 / 19
BP level	6 [d] (129, 139, 147, 152, 172, 178) / 17 (129, 136, 138, 139,145–147, 152, 158, 160, 171–173, 178, 193, 214)	7 (44, 133, 150, 151, 169, 175, 181) / 13 (44, 125, 126, 128, 135, 150,151, 153, 156, 169, 170,175, 181)	3 (142, 161, 180) / 4 (142, 148, 161, 180)	4 (155, 166, 176, 182) / 5 (137, 155, 166, 176, 182)	0 /0	1 (167) / 1 (167)	21 / 40

[a] Studies on both BP level and hypertension (137, 147, 158, 162, 167).
[b] Two studies also reported a significant protective effect of job strain (131, 157).
[c] The estimated risk ratios (RRs) for blood pressure increases in the highest quintile for each job strain group (142).
[d] One study also reported a significant protective effect of job strain (138).

Table 2. Number of studies reporting a statistically significant deleterious effect / total number of studies having these methodological characteristics (reference number) reporting a deleterious effect of job strain on blood pressure (BP) according to gender, study designs (cross-sectional, prospective or case–control), BP measurements (office or ambulatory) and outcome (hypertension or BP level).

	Cross-sectional studies (N=26)		Prospective studies (N=6)		Total
	Office BP (N=18/26)	Ambulatory BP (N= 8/26)	Office BP (N=3/6)	Ambulatory BP (N=3/6)	
Women					
Hypertension	0 / 6 (131, 134, 135, 149, 157, 177)	0 / 0	0 / 1 (142)	0 /0	0 / 7
BP level	1 (138)/ 7 (129, 138, 152, 171, 178, 193, 214)	1 (150) / 3 (126, 150, 156)	1 (180) / 3 (142, 161, 180)	1 (166) / 1 (166)	4 / 14
Men					
Hypertension	1 (177) / 4 (134, 147, 157, 177)	1 (181) / 1 (181)	1 (142) / 1 (142)	0 / 0	3 / 6
BP level	5 (129, 147, 152, 172, 178) / 11 (129, 138, 145–147, 152, 160, 171, 172, 178, 193)	2 (151, 181) / 6 (125, 126, 128, 151, 156, 181)	3 (142, 161, 180) / 3 (142, 161, 180)	2 (155, 182) / 2 (155, 182)	12 / 22

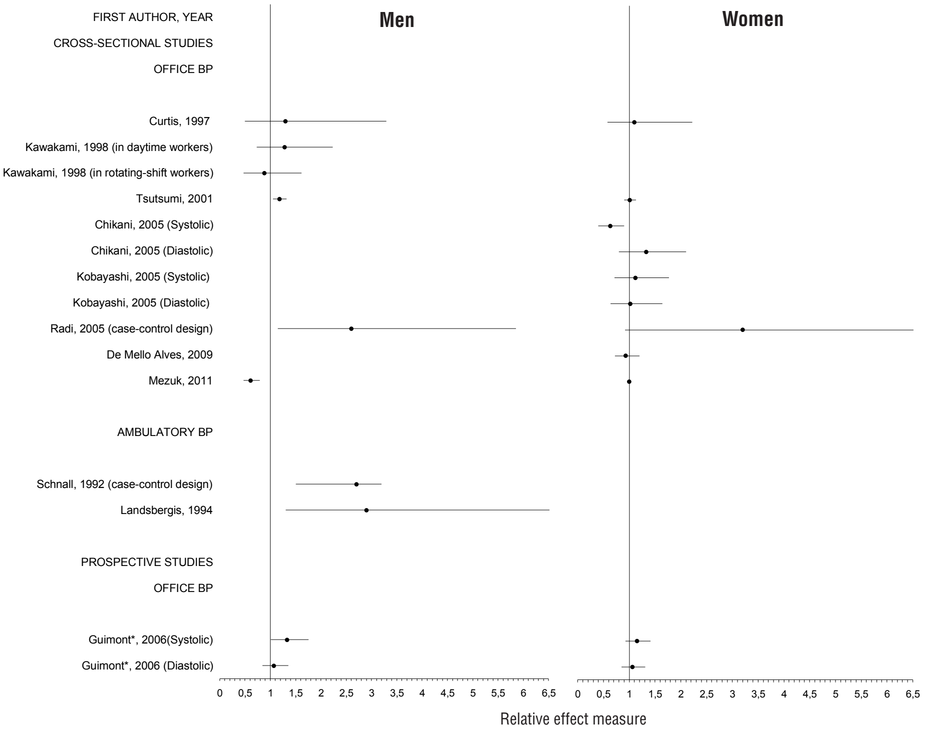

Figure 2. Relative risk of hypertention observed among studies on job strain by gender. [BP=blood pressure.]

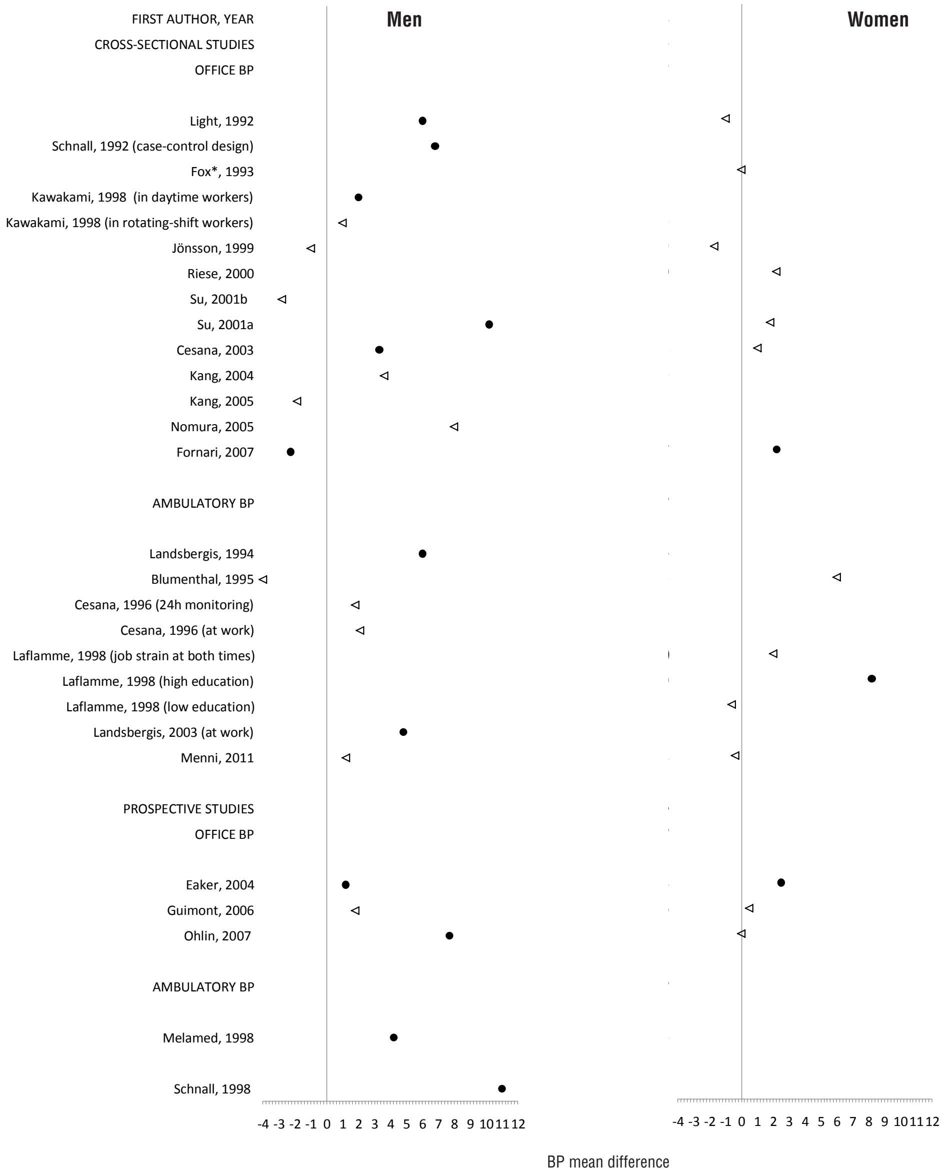

Figure 3. Systolic blood pressure (BP) mean differences observed among studies on job strain by gender. A dot designates a statistically significant result (P<0.05) while a triangle designates a non-significant result. * This difference was stated as statistically significant but no effect measure was presented.

+4.52 mm Hg and +1.31–+4.17 mm Hg (153, 188, 189) and (ii) hypertension OR ranged from 1.62–5.77 (184, 185, 186, 190). In addition, two cross-sectional studies evaluated the separate effect of effort and reward (153, 190). None of these studies observed significant results (table C).

A significant deleterious effect of overcommitment was observed in one out of three cross-sectional studies (194). This study observed a higher ambulatory systolic BP mean among men (+6.4 mm Hg) but no effects among women (194). It is also worth mentioning that no cross-sectional studies presented results for the potential modifying effect of overcommitment on the association between ERI and BP.

Only one prospective study evaluated the effect of ERI on BP (191). This study used ambulatory BP measures. Among men, no association was observed. Among women, age had a modifying effect. Women <45 years old exposed to ERI at both times (over a 3-year follow-up) had significantly higher BP means at follow-up than those unexposed (systolic: +1.86 mm Hg, diastolic: +1.48 mm Hg) (table C, figure 6). Among women ≥45 years old, the cumulative incidence of hypertension was 2.78 times higher among those exposed to ERI at both times (table C and figure 6). In this study, no modifying effects were observed for overcommitment. However, men and women in the higher tertile of overcommitment also had higher BP means than those in the lower tertile (men: systolic +1.66 mm Hg, diastolic non-significant; and women: systolic: +1.28 mm Hg, diastolic +1.02 mm Hg) (table C).

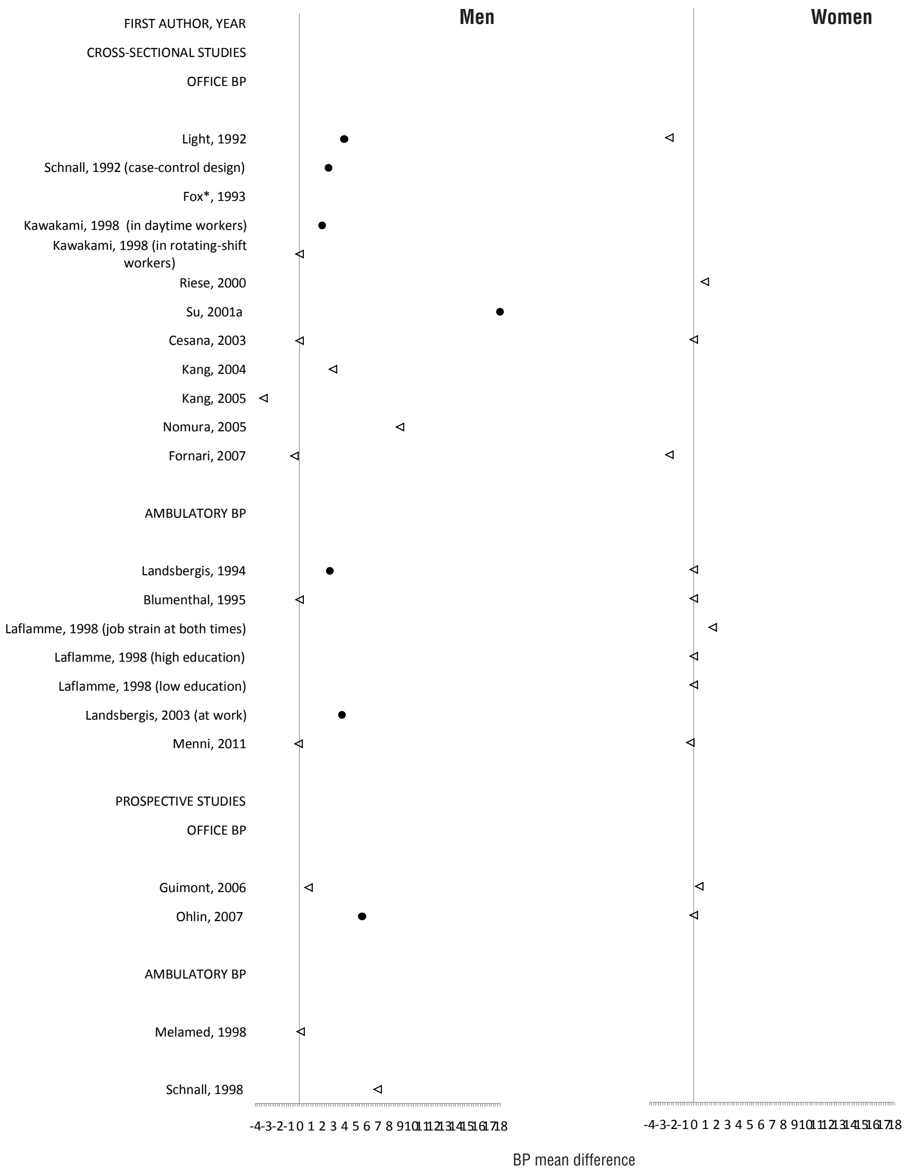

Figure 4. Diastolic blood pressure (BP) mean differences observed among studies on job strain by gender. A dot designates a statistically significant result (P<0.05) while a triangle designates a non-significant result. * This difference was stated as statistically significant but no effect measure was presented.

Gender. Of the 11 cross-sectional studies on ERI, 6 presented results separately for men and women (table C, figures 5–6). The deleterious effect of ERI was more consistent among men (5/6 studies) than women (1/6 studies) (table C, figures 5–6).

Methodological characteristics. For the ERI model, there were 11 cross-sectional studies and only 1 prospective study (table 3). More prospective studies are needed to compare results according to study designs.

A higher proportion of studies using ambulatory BP measures (3/4 studies) observed an adverse effect of ERI as compared to studies using office measures (5/8 studies, table 3). However, this comparison should be interpreted cautiously due to the small number of cross-sectional (N=3) and prospective (N=1) studies using ambulatory BP measures.

Discussion

Of the 74 studies on the adverse effects of psychosocial work factors on BP, 64 looked at the DCS model and 12 at the ERI model, with two studies considering both models (152, 156). For both models, a more consistent adverse effect has been observed for men compared to women. In studies on job strain, those of higher methodological quality (ie, studies using a prospective design and/or ambulatory BP measures) observed a more consistent effect than those of lesser quality.

Gender

In line with the results of the current review, previous reviews on BP (35) and CVD (26–30, 35) also observed

Table 3. Number of studies reporting a statistically significant deleterious effect / total number of studies having these methodological characteristics (reference number) reporting a deleterious effect of the effort-reward imbalance (ERI) factors on blood pressure (BP) according to study designs (cross-sectional, prospective, or case-control), BP measurements (office or ambulatory), and outcome (hypertension or BP level).

	Cross-sectional studies (N=11)		Prospective study (N=1)		Total
	Office BP (N=8/11)	Ambulatory BP (N=3/11)	Office BP (N=0/1)	Ambulatory BP (N=1/1)	
ERI (N=12)					
Hypertension	4 (184–186, 190) / 5 (149, 184–186, 190)	0 / 0	0 / 0	1 (191) / 1 (191)	5 / 6
BP level	1 (189) / 3 (183, 187, 189)	2 (153, 188) / 3 (153, 188, 194)	0 / 0	1 (191) / 1 (191)	4 / 7

a more consistent adverse effect of psychosocial work factors among men than women (26–30, 35). However, a recent meta-analysis of coronary heart disease European cohort studies, including 197 473 workers, observed a similar effect in both genders (31). Gender differences may be due to the fact that BP elevations tend to arise later in women's lives than men's. Indeed, until age 45, a lower percentage of women have high BP (195). Therefore, among women, age might modify the effect of psychosocial work factors on BP, leading to a stronger effect in older than younger women. Supporting this hypothesis, Gilbert-Ouimet et al (191) observed an adverse effect of ERI on hypertension among women aged ≥45 years old, while no such effect was observed among younger women. It is thus possible that studies observing no significant adverse effect (in particular studies on hypertension) would have observed such effect after stratifying on age. However, since only two studies stratified their results on women's age (both having observed an adverse effect) (130, 191), it would be important to further evaluate this potential modifying effect.

Gender differences could also be explained by women having different occupational trajectories than men (more often characterized by absences or reduced hours of paid work due to family responsibilities), resulting in less continuous exposure to psychosocial work factors. In addition, it is also possible that being exposed to adverse psychosocial work factors might only add a little adverse impact to that already encountered by experiencing the burden of large family responsibilities. As pointed out by Messing et al (196), multiple roles and complex exposures make it difficult to pin down risks for working women. In the current review, only three studies have taken family responsibilities into account (135, 142, 159). Future studies would benefit from evaluating the potential modifying or confounding effect of family responsibilities. Marital cohesion would also be of interest since previous studies have observed that a lack of it amplified (ie, modified) the adverse effect of psychosocial work factors on BP (176, 197).

Gender differences in the experience of stress (198) may also lead to differential self-reported exposures to psychosocial work factors (26). In line with this, two studies that used both self-reports and external observations to assess psychosocial work factors noted that women tended to overestimate their self-reported job control, while no such phenomenon was observed among men (78, 199). For women, this may lead to an underestimation of the prevalence of high job strain. Such non-differential misclassification could dilute the adverse effect of high job strain on BP among women.

Another potential explanation for gender differences might lie in the effect of social support at work among women. As shown for the association between job strain and depression (200), high social support at work may moderate the adverse effects of job strain among women. None of the studies included in the current review evaluated the potential modifying effect of social support on the association between psychosocial work factors and BP according to gender. It is thus possible that studies showing no significant adverse effect among women would have observed such an effect after stratifying on social support. It is also worth noting that only one study (130) evaluated the separate effect of social support. This study observed an adverse effect among women but no effect among men. More studies evaluating the separate and modifying effects of social support at work according to gender are needed.

Finally, the gender differences observed in the current review rely partly on a comparison of studies comprising solely men or women. A potential limitation of comparing such studies is that gender differences might result from inter-study differences (eg, differences in design, BP measurements, and definition of psychosocial exposures) instead of true gender differences. We therefore conducted a complementary analysis to verify this hypothesis by comparing only studies including *both* men and women. For job strain, a higher proportion of studies observed a deleterious effect among men (7/15 studies) than women (1/15 studies) (tables A and B, figures 2–4), which is in line with the findings of the overall analysis. For ERI, only three studies including both men and women presented results according to gender (188, 193, 194). A deleterious effect was observed among men and women in 2/3 and 1/3 of the studies, respectively (table C, figures 5–6). More studies on ERI that include both men and women are needed. These additional studies would allow a comparison of the consistency of the effect of ERI according to gender.

Table 4. Number of studies reporting a statistically significant deleterious effect / total number of studies having these methodological characteristics (reference number) reporting a deleterious effect of effort-reward imbalance (ERI) on blood pressure (BP) according to gender, study designs (cross-sectional, prospective or case-control), BP measurements (office or ambulatory) and outcome (hypertension or BP level).

	Cross-sectional studies (N=11)		Prospective study (N=1)		Total
	Office BP (N=8/11)	Ambulatory BP (N=3/11)	Office BP (N=0/1)	Ambulatory BP (N=1/1)	
Women					
Hypertension	0 / 3 (149, 185, 190)	0 / 0	0/0	1 (191) / 1 (191)	1 / 4
BP level	1 (189) / 2 (183, 189)	0 / 0	0/0	1 (191) / 1 (191)	2 / 3
Men					
Hypertension	4 (184–186, 190) / 4 (184–186, 190)	0 / 0	0/0	0 / 1 (191)	4 / 5
BP level	0 / 0	1 (188) / 1 (188)	0/0	0 / 1 (191)	1 / 2

Methodological characteristics

Study design. A prospective design is more appropriate than a cross-sectional one, especially for studies on hypertension. Indeed, cardiovascular alterations such as hypertension could take years to develop. A prospective design has a considerable advantage in that it allows for a time lag between exposure and outcome measurements, circumventing an eventual reverse causation bias.

In this review, studies used different study designs to evaluate the effect of psychosocial work factors on BP. For job strain, a higher proportion of prospective studies (7/9 studies) yielded a deleterious effect on BP mean level compared to cross-sectional studies (13/30 studies) (table B). However, the prospective design did not lead to a more consistent effect in studies on hypertension (significant effect in 2/5 prospective studies compared to 3/12 in cross-sectional studies). This may be due to the low number of prospective studies on hypertension (N=5) and their predominant use of office BP measures (4/5 studies) (table 1). More studies combining a prospective design and ambulatory BP measures are needed to evaluate the role of job strain in the etiology of hypertension.

For the ERI model, there was only one prospective study (191), which emphasizes the need for more studies using this design.

Types of BP measures. A higher proportion of studies using ambulatory BP measures showed an adverse effect of job strain (13/20 studies) and ERI (3/4 studies) as compared to studies using office measures (12/35 and 5/8 studies, respectively) (tables 1 and 3). Ambulatory BP measures are known to sidestep the observer error (the so-called "white-coat effect"). They also provide better precision by capturing the BP fluctuations related to daily life and make it possible to capture "masked" hypertension, defined as elevated daytime ambulatory BP (≥135/85 mmHg) in the face of normal office BP (<140/90 mm Hg). The prevalence of masked hypertension has been estimated to be between 8–30% in the general population (201–204). Several population-based studies and prospective clinical trials have provided clear evidence of the superiority of ambulatory over office BP measures in predicting cardiovascular risks (152, 205–208).

Besides comparing clinical to ambulatory BP measures, a distinction can be made according to the moment of BP collection (ie, during work, at home, over 24 hours, and during sleep). We performed a complementary analysis of studies that measured BP during and outside work (N=10) (44, 127, 128, 133, 137, 139, 151, 169, 175, 181) (tables A and B). Most of these studies were cross-sectional (N=9) and used ambulatory BP measures (N=9) (tables A and B). All the studies (except reference 44) presenting significant results found a deleterious effect in both BP at work and: (i) at home (133, 139, 151, 181), (ii) over 24 hours (133, 175), and (iii) during sleep (133). The effect magnitudes were comparable for all periods. This suggests that the deleterious effect of psychosocial work factors not only contributed to inflate daytime BP but also persist after work. It is likewise noteworthy that most studies included in this review (N=64/74) only measured daytime BP. Boggia et al (205) showed that daytime BP predicts the 10-year incidence of fatal and non-fatal strokes, cardiac, and coronary events just as well as nighttime BP. Indeed, hazard ratios for the combination of these cardiovascular events were 1.33 and 1.25 for systolic and diastolic daytime BP, respectively, compared to 1.31 and 1.28 for nighttime BP (in continuous analyses).

Limitations

Methodological choices. As Belkic et al (29) emphasize, publication bias and heterogeneity are major reasons for skepticism towards meta-analyses of non-experimental studies (29, 210, 211). There is also an increasing need for qualitative approaches and the identification of the best way of evaluating effects (29). The current review does not provide meta-analytical estimates since the available data did not meet the criteria for homogeneity in methods used to assess job strain and ERI, confounders, outcome measures, and biases potentially affecting internal validity. Our review however provides an in-depth analysis of several potential explanations for data inconsistencies (ie, gender, study design, types of BP measures, instruments for measuring psychosocial factors, categorization of exposure to psychosocial fac-

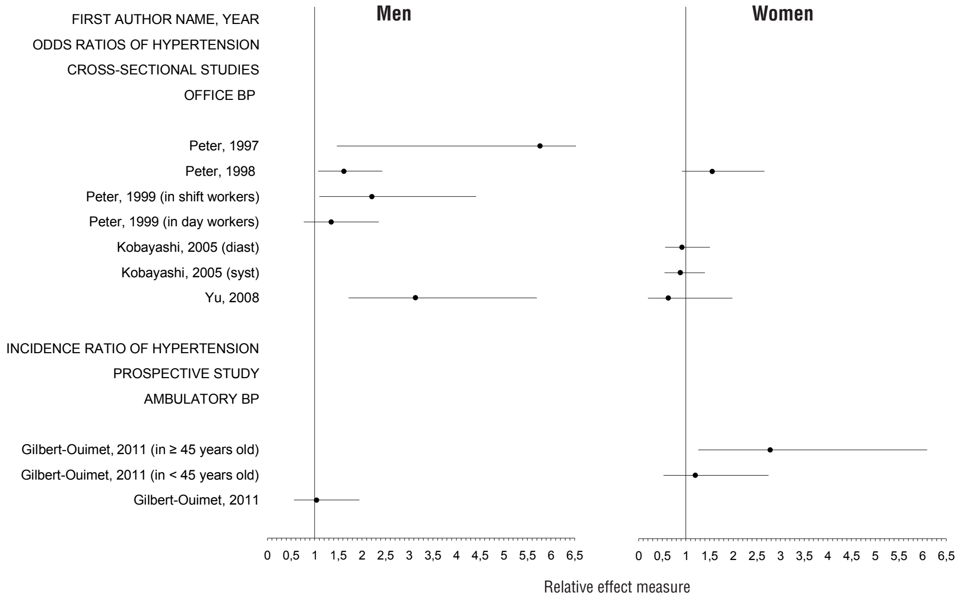

Figure 5. Relative risk of hypertension observed among studies on effort-reward imbalance by gender. [BP=blood pressure; syst=systolic; diast=diastolic]

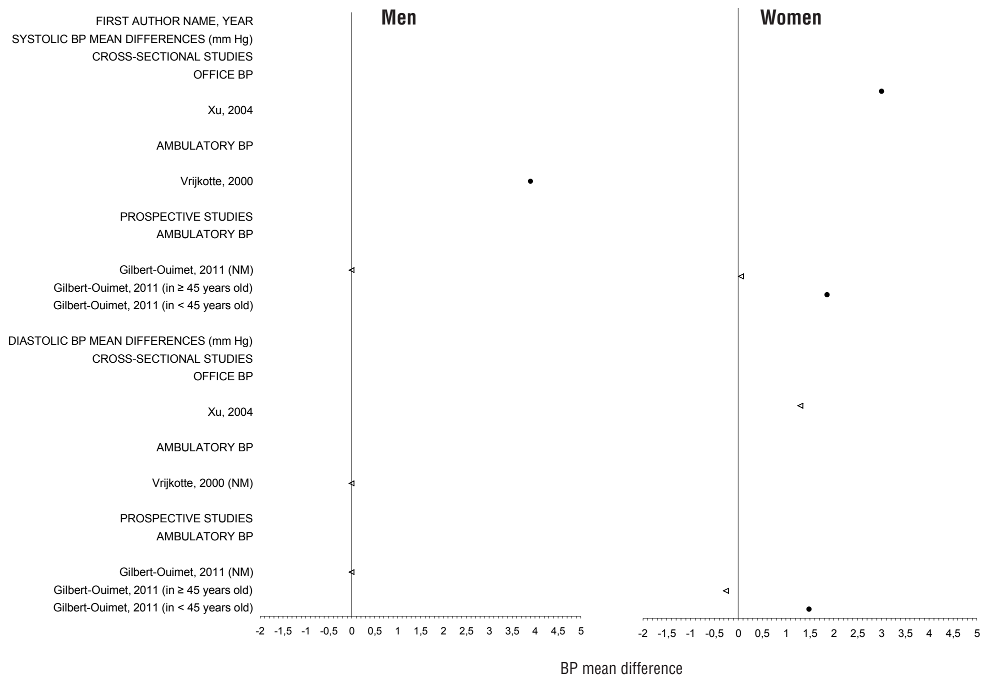

Figure 6. Systolic and diastolic blood pressure (BP) mean differences and beta coefficients observed among studies on effort-reward imbalance by gender. A dot designates a statistically significant result (P<0.05) while a triangle designates a non-significant result. [NM=effect not mentioned].

tors, control for potentially confounding factors, and participation rate). Such analysis allowed the identification of "optimal" methods to consistently observe the deleterious effect of psychosocial work factors on BP, namely the use of a prospective design and ambulatory BP measures.

However, the calculation of a meta-analytical estimate based on a sub-sample of studies with comparable methodological characteristics would partly circumvent the pitfalls of heterogeneity. The only subsample comprising a sufficient number of studies (N>5) of higher methodological quality (ie, studies having either a prospective design or ambulatory BP measures) would be the cross-sectional studies evaluating the association between job strain and mean level of ambulatory BP. However, Landsbergis et al (198) already calculated such an estimate in a very recent meta-analysis. Indeed, based on 22 cross-sectional studies, they presented higher pooled ambulatory BP means of +3.43 mmHg (systolic) and +2.07 mm Hg (diastolic) among workers exposed to high job strain compared to non-exposed workers (35).

Also, it is worth mentioning we could have missed potentially relevant papers in the first step of data selection when we selected citations on the basis of their titles rather than reviewing abstracts, which would have minimized the chance of introducing such bias. However, the references of all included studies and prior literature reviews have been thoroughly consulted and no additional studies were added. Even though it cannot be ruled out, a potential bias resulting from this data selection step seems unlikely.

In line with previous literature reviews on psychosocial work factors and cardiovascular health (26, 27, 29, 30), we evaluated the consistency of the effects on the basis of statistical significance. However, gainful but non-significant effects have also been observed in studies on job strain (N=13) (tables A and B) (no such effects have however been observed in studies on ERI, table C). Only three of these studies (N=3) had a sample size <200 (N=100–175, table A), which suggests that statistical power is unlikely to explain why results were not significant. It is also noteworthy that most of these studies were cross-sectional (12/13 studies) and used clinical BP measures (9/13 studies), which supports the hypothesis that poorer methodological quality leads to lower effect consistency.

Publication bias. A potential publication bias might have been introduced due to the inclusion of articles written only in English or French. To document this potential bias, a sensitivity analysis including articles written in other languages was conducted. This complementary search led to the identification of four potentially relevant articles written in Chinese, Italian, Persian, and Spanish (94–97). The potential relevance of these articles was based on the titles and abstracts, which were written in English. Three articles were on the DCS model (94, 96, 97) and two were on the ERI model (95, 97). One article included both models. Based on the abstracts, only one study (96) observed significant results. This cross-sectional study looked at the DCS model and used ambulatory BP measures. However, since it included only 30 men, it would not have been eligible for the current review (studies had to include ≥100 workers). Thus, this sensitivity analysis revealed that three possible eligible studies with negative results were omitted. Of these, two were cross-sectional (98, 100) (the other one did not mention the study design in the abstract). The abstracts did not mention the type of BP measurement used in these studies.

Another publication bias could have arisen from the fact that statistically significant results are more likely to get published than non-significant results. Such publication bias is assumed to be present if larger studies (in which it is easier for smaller effects to be significant) report smaller effects than small studies (larger effects are needed for significant findings) (209). To investigate the presence of this bias, the test for funnel plot asymmetry is generally conducted (209). However, due the diversity of effect measures, psychosocial exposures, study designs, and outcomes used in the reviewed studies, such a test could not be performed. The current review was restricted to studies including ≥100 workers. This makes it easier to achieve satisfactory statistical power, which reduces the likelihood of a publication bias due to non-significant findings. In addition, approximately one in two reviewed studies reported non-significant results, which shows that such results are frequently published in this field. It is, however, important to point out that other non-significant results might not have been presented. As mentioned above, only one study on ERI presented an investigation of the potential modifying effect of overcommitment. This however does not definitively suggest that such analyses were not performed. Presenting non-significant modifying effects is needed to further document the psychosocial etiology of BP elevation. The magnitude of such publication bias cannot be estimated.

The potential biases of the reviewed studies are detailed below.

Selection bias. A selection bias could have been introduced in studies where participants and non-participants differed with regard to both psychosocial work factor exposure and BP (210). Studies having a low participation rate at baseline and/or follow-up are particularly vulnerable to such bias (212). In a recent literature review, Galea et al (213) reported that a participation rate of ≥75% is generally considered satisfactory in epidemiological studies. In the current review, 20 studies had participation rate(s) <75% (23, 128–130, 132, 133, 141, 148, 149, 153, 159, 162, 166, 176–178, 188, 193, 194, 214,) (tables A–C). Almost half of these (9/20 studies) documented the potential differences between participants and non-participants (23, 128, 141, 148, 149, 159, 166, 176, 214). Those suspecting a differential participation (4/9 studies): (i) observed higher cardiovascular risk factor prevalence (159) combined with lower socioeconomic status (177) or with a higher prevalence of psychosocial work factors in non-participants (23, 148), and (ii) noted that, since recruitment was by advertisements, it is possible that the study attracted predominantly "stressed" subjects as volunteers (176). These observations suggest that a selection bias due to differential participation could lead to an under- or overestimation of the true effects. It is important to mention that participation rate(s) were not reported in a third of the studies (26/76 studies, 34%) (121, 125–127, 131, 134, 136, 138, 140, 143, 146, 152, 154, 156, 157, 161, 163, 165, 168, 173, 181, 183, 185, 186, 190, 194) (tables A–C). Such a high proportion of non-reporting of participation is in line with what Morton et al (212) observed in a recent review of articles published in major epidemiology journals. They noted comparable or poorer reporting of participation rates in cross-sectional (participation rate not mentioned in 41% of studies), case–control (66%), and prospective (68%) studies. Since selection bias may threaten the internal validity of epidemiologic studies, authors should report participation rate(s) consistently.

The well-documented selection bias of the "healthy worker effect" (210) might also have been introduced in some of the included studies. This bias, which is more likely to occur in cross-sectional than prospective studies (210, 215), generally leads to an underestimation of the true effect (218). In occupational studies, a healthy worker effect can arise from: (i) a differential participation at baseline or follow-up (discussed above) and (ii) the application of selection criteria. In prospective studies on hypertension, this second mechanism could for example be introduced by excluding hypertensive workers at baseline, who are "sicker" than normotensive workers. However, creating prospective cohorts free of the outcome under study at baseline is an important methodological quality since it allows causal inferences to be made by ensuring that the exposure precedes the outcome. In keeping this rationale, most prospective studies on hypertension (4/6 studies) opted to exclude hypertensive workers at baseline (137, 142, 154, 191) (tables A–C).

It is also noteworthy that, in occupational studies, the healthy worker effect has mostly been observed in studies on cardiovascular disease, diabetes, and respiratory disorders (210, 215) due to the fact that such diseases are symptomatic. Studies on BP mean level or hypertension are less prone to such bias because: (i) BP elevations and hypertension are generally asymptomatic, and (ii) over 50% of individuals with high BP are unaware of their condition (216). Nevertheless, the healthy worker effect can occur in cross-sectional studies on hypertension and generally lead to an underestimation of the true effect (210).

Information bias. An information bias might have resulted from the fact that psychosocial work factors are notoriously difficult to measure. Indeed, psychosocial work factors are known to be more difficult to measure than standard cardiovascular risk factors such as smoking, alcohol consumption, or abdominal adiposity. More specifically, the concept of psychological demands measured by the DCS model has been criticized for not measuring emotional demands, which include becoming emotionally involved during work or having to face emotionally disturbing situations (217). Thus, the concept of psychological demands might underestimate the actual "demands" to which workers are exposed. This could lead to a non-differential information bias underestimating the true adverse effect of psychological demands (210).

Another potential information bias might have resulted from the use of different instruments to measure the psychosocial work factors of both the DCS and ERI models. Of the 64 studies on the DCS model, 53 used Karasek's Job Content Questionnaire (JCQ) (10) (tables 1 and 2). A majority of these studies observed significant effects of the DCS factors (32/53) (tables A and B). Among the studies using instruments other than the JCQ, a majority (9/11 studies) also observed significant effects (tables A and B). For the ERI model, 10/12 studies used the recommended Siegrist questionnaire (13) (table C), 7 of which observed a significant effect (table C). The two studies that used other instruments partly (191) or entirely (185) also observed significant effects. However, comparing the effect consistency observed in studies on the basis of the instrument used to measure psychosocial factors is complex since studies also differ with regard to other methodological characteristics. Uniformity in measuring psychosocial work factors is nevertheless recommended to improve interstudy comparability.

A misclassification bias might also have resulted from the fact that studies on job strain used different categorizations of exposure. Some studies (19/52 studies) categorized job strain in quadrants as recommended (10) (tables A and B). These quadrants classify workers as unexposed (low demands, low control), passive (low demands, high control), active (high demands, high control), or high strain (high demands, low control). Even though quadrants are recommended, a majority of studies (28/52 studies) used a dichotomous exposure, comparing the high strain category (as "exposed") to the combination of unexposed, passive, and active categories (as "unexposed") (tables A and B). A dichotomous job strain categorization might lead to an important misclassification bias. Such a bias would lead to a dilution of the adverse effect of high job strain. A complementary analysis showed that studies using the job strain quadrants did not yield a more consistent effect than studies using a dichotomous exposure (8/19 compared to 12/25 studies, tables A and B). As mentioned previously, such a comparison is limited by the fact that studies differ in other methodological characteristics. It is also noteworthy that three studies using the job strain quadrants observed deleterious effects in the active group (166) or in both the active and passive groups (33, 163).

In the same vein, a misclassification bias might also have resulted from the use of different scales to measure the ERI factors. A majority of studies (8/12) used an agreement scale with answers varying from "strongly agree" to "strongly disagree" (153, 183, 186–188, 190, 191, 194). The four other studies (149, 184, 185, 189) used a scale measuring both the agreement and the intensity of distress experienced. In these studies, participants who agreed to a given item had to indicate the level of distress experienced, ranging from "very distressed" to "not at all distressed". Measuring both the employees' agreement and distress intensity may have led to a more acute exposure to ERI than measuring only the employees' agreement. In line with this hypothesis, a slightly higher proportion of studies combining both agreement and distress intensity observed a significant deleterious effect of ERI (3/4 versus 4/8 studies). In future studies, measuring psychosocial work factors with standardized instruments would favor interstudy comparability.

Another potential information bias might have arisen from the use of a single time-point exposure. Only 7 (137, 150, 151, 154, 166, 182, 191) of 64 studies evaluated the effect of job strain or ERI using more than a single time-point. Of these, 5 observed a significant adverse effect, which is a higher proportion than that observed in studies using a single time-point exposure (28/57 studies, table A). In line with this, data from the British Whitehall II study and the Quebec post-myocardial infarction cohort showed that a single time-point measurement underestimated the effect of job strain on first and recurrent coronary heart disease (28, 218). Measuring psychosocial work factors repeatedly makes it possible to take changes in exposure into account. It also makes it possible to identify chronically exposed subjects, who may have a higher cardiovascular risk than subjects exposed for a shorter period. There is too little empirical evidence to suggest an optimal number of measures or an ideal interval of time between psychosocial work factor measurements. According to experimental studies, it is however reasonable to assume that the deleterious effect of psychosocial stressors on BP elevations, particularly on hypertension, would arise from prolonged exposures (17–19, 219–221).

An additional information bias might have occurred in studies on BP level that did not take hypertension medication into account. Since hypertension medication leads to artificially lowered BP measures, not considering it might contribute to underestimating the true adverse effect of psychosocial work factors on BP level. A total of 18/59 studies on BP means (141, 145, 146, 158, 160, 162, 165, 167, 178–180, 182, 187, 189, 192, 193) did not take hypertension medication into consideration (ie, workers on medication were not excluded or not controlled for in analyses) (tables A–C). Of these 18, 10 studies observed an adverse effect of psychosocial work factors, which is, however, comparable to the overall proportion of studies observing such an effect.

It is also worth mentioning that: (i) 2 of 3 prospective studies on hypertension did not consider workers taking hypertension medication at follow-up as "hypertensive cases" (ie, as having the outcome under study) (table B); and (ii) of 14 cross-sectional studies on hypertension, 1 excluded workers taking hypertension medication and 5 controlled for the consumption of such medication (table A). Since workers taking hypertensive medication have the outcome under study, not considering them as "cases" leads to a misclassification that might bias the estimates toward the null.

One can also argue that another misclassification bias could have been introduced by assessing psychosocial work factors using self-reported questionnaires. In theory, self-reported data tend to introduce more misclassification bias than objective data (210). However, it has been suggested that the individual's judgment may bring about most of the deleterious effects of psychosocial work factors on health (13). In addition, job title exposure score has been shown to involve more misclassification than self-reported measures due to an incomplete capture of psychosocial work exposure (generally leading to an underestimation of estimates) (28, 29, 222).

Finally, it is worth noting that some studies included populations of workers from only one or two occupations [ie, bus drivers (123), nurses (127, 139, 166), police officers (125), and teachers (127, 169, 192)] (tables A–C). In these studies, the range of variation of exposure to psychosocial work factors might have been limited due to considerable similarity in job characteristics. Little variation due to restricted working areas may lead to lower effect estimates compared to those that would have been observed in representative samples of the active working population. Also, as Landsbergis et al (153) states, a limited range of variation in exposure due to study design might reduce the statistical power available to detect main effects of psychosocial work factors.

Confounding. Confounding biases also need to be addressed. The five studies on the DCS model (152, 160, 171, 173, 179) that did not control for any cofactors are the most prone to confounding bias. Confounding might also be present in other studies due to a lack of control for cardiovascular risk factors. For example, some studies did not control for age (125, 132, 144, 146, 174) or family history of CVD (61 studies) (23, 44, 124–129, 131–141, 153, 155–159, 161–168, 171, 174, 178, 180, 181, 183, 185–189, 192–194, 214, 223, 224) (tables A–C), which constitute major risk factors for high BP. Residual confounding might also have resulted from the fact that none of the studies on job strain controlled for ERI or vice-versa. Finally, residual confounding might have been present due to psychosocial work factors of emerging models, such as organizational injustice (225) and managerial leadership (226), which have been suggested to be causally related to cardiovascular risk (28, 226). It is worth mentioning that recent studies have presented evidence of a complementary adverse effect of job strain and ERI on BP and coronary heart disease (227 and Trudel X, Brisson C, Milot A, Vézina M, Masse B. Psychosocial work environment and ambulatory blood pressure: independent effect of demand–control and ERI models. In preparation). Similarly, a recent systematic review observed that procedural and relational injustice (ie, two components of the organizational injustice model) can be considered a different and complementary model to the DCS and ERI models. It is also possible that simultaneous exposures to psychosocial work factors would lead to an increased adverse effect on BP compared to single exposures. Such a phenomenon has been observed in job strain and ERI with regard to the risk of acute myocardial infarction among men and women of a large case–control study (N=951 cases and 1147 referents) (228).

A large majority of studies presented effect measures adjusted for lifestyle risk factors that might have acted as mediating variables (62/77 studies) in the causal pathway linking work stress and BP (28, 29). Indeed, psychosocial work factors have been associated with lifestyle, cardiovascular risk factors such as increased smoking intensity (229), reduced leisure-time physical activity (229), unhealthy diet (230), weight gain, and obesity (28, 231). Adjusting for mediating factors may result in controlling for a part of the effect under study, which contributes to an underestimation of the overall effect of psychosocial work factors on BP (210). To avoid such a limitation, five studies (23, 124, 157, 186, 187) evaluated the additional effect of adjusting for lifestyle, cardiovascular risk factors in a supplementary statistical model (ie, sequential adjustment). In all of these studies, this additional adjustment only resulted in a slight change in the effect measures presented. Studies using structural equation modeling are, however, needed to quantify the potential causal pathway linking psychosocial work factors, lifestyle risk factors, and BP.

Generalization

The results of the current study can be generalized to working populations from various countries. Indeed, participants in a large proportion of studies (N=35/78) were recruited from representative samples of the active working population. The remaining studies included workers from various but restricted working areas (ie, public employees, bus drivers, nurses, and teachers; tables A–C), which may limit the external validity of their results (153, 176).

Concluding remarks

The present review has some strengths. It gathered and summarized empirical evidence through an explicit, systematic, and objective research strategy known for minimizing bias (36). This is also the first systematic review on the effects of both the DCS and the ERI models on BP level and hypertension. This review also provides an in-depth analysis of gender differences. In addition, the systematic approach made it possible to explore five methodological characteristics as potential explanations for the data inconsistencies observed in the literature: (i) study design, (ii) types of BP measures (office versus ambulatory), (iii) instruments for measuring psychosocial factors, (iv) categorization of exposure to psychosocial factors, (v) control for potentially confounding factors, and (vi) participation rate.

In conclusion, the present review contributes to current efforts of primary prevention of CVD by providing an up-to-date, systematic synthesis of reliable findings on the psychosocial etiology of BP, a major CVD risk factor. Overall, approximately half the studies observed a significant adverse effect of psychosocial work factors on BP. However, the extensive body of research on this topic showed a more consistent effect for men than for women. In studies on job strain, a more consistent effect was also observed in studies of higher methodological quality that is studies using: (i) a prospective design and (ii) ambulatory BP measures. The numerous evidences presented in this review supports the need for workplace intervention studies to evaluate the effect that reducing psychosocial work factors has on BP among various working populations.

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References

1. World Health Organization (WHO). Cardiovascular diseases. Fact sheet. 2011; (317): Available from: http://www.who.int/mediacentre/factsheets/fs317/en/index.html.
2. Statistiques Canada. Mortality, summary. List of causes. 2010: Available from: http://www5.statcan.gc.ca.
3. Canadian Institute for Health Information (CIHI), editor. The Cost of Acute Care Hospital Stays by Medical Condition in Canada: 2004-2005. Ottawa: CIHI; 2008.
4. Claes N, Jacobs N. The PreCardio-Study protocol - a randomisez clinical trial of a multidisciplinary electronic cardiovascular

prevention programme. BMC Cardiovasc Disord. 2007;4(7):27. http://dx.doi.org/10.1186/1471-2261-7-27.

5. Lawes C, Vander Hoorn S, Rodgers A. International Society of Hypertension. Global burden of blood pressure disease. Lancet. 2008; 371:1513. http://dx.doi.org/10.1016/S0140-6736(08)60655-8.

6. Cutler J, Sorlie P, Wolz M, Thom T, Fields L, Rocella E. Trends in hypertension prevalence, awareness, treatments, and control rates in Unites States adults between 1988-1994 and 1999-2004. Hypertension. 2008;52(5):801–2. http://dx.doi.org/10.1161/HYPERTENSIONAHA.108.113357.

7. Statistiques Canada. Enquête sur la santé dans les collectivités canadiennes - Composante annuelle (ESCC) [Canadian Community Health Survey (CCCHS) cycle 3.1]. 2005: Available from: http://www.statcan.gc.ca/concepts/health-sante/cycle3_1/.

8. MacMahon S, Peto R, Cutler J, Collins R, Sorlie P, Neaton J, et al. Blood pressure, stroke and coronary heart disease. Lancet. 1990;335:765–74. http://dx.doi.org/10.1016/0140-6736(90)90878-9.

9. Lewington S, Clarke R, Qizilbash N, Peto R, Collins R. Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. Lancet. 2002 Dec 14;360(9349):1903–13.

10. Karasek R. Job demands, job decision latitude, and mental strain: implications for job redesign. Adm Sci Quaterly. 1979;24:285–308. http://dx.doi.org/10.2307/2392498.

11. Siegrist J. Adverse health effects of high-effort/low-reward conditions. J Occup Health Psychol. 1996;1(1):27–41. http://dx.doi.org/10.1037/1076-8998.1.1.27.

12. Johnson JV, Hall EM, Theorell T. Combined Effects of Job Strain and Social Isolation on Cardiovascular Disease Morbidity and Mortality in a Random Sample of the Swedish Male Working Population. Scand J Work Environ Health. 1989;15:271–79. http://dx.doi.org/10.5271/sjweh.1852.

13. Siegrist J, Starke D, Chandola T, Godin I, Marmot M, Niedhammer I, et al. The measurement of effort–reward imbalance at work: European comparisons. Social Sience & Medicine. 2004;58:1483–99. http://dx.doi.org/10.1016/S0277-9536(03)00351-4.

14. Dzau V, Antman E, Black H, Hayes D, Manson J, Plutzky J, et al. The cardiovascular disease continuum validated: clinical evidence of improved patient outcomes: part I: Pathophysiology and clinical trial evidence. Circulation. 2006 Dec;114(25):2850–70. http://dx.doi.org/10.1161/CIRCULATIONAHA.106.655688.

15. Dzau V, Braunwald E. Resolved and unresolved issues in the prevention and treatment of coronary artery disease: a workshop consensus statement. Am Heart J. 1991 Apr;121(4 Pt 1):1244–63. http://dx.doi.org/10.1016/0002-8703(91)90694-D.

16. Chida Y, Steptoe A. Greater Cardiovascular Responses to Laboratory Mental Stress Are Associated With Poor Subsequent Cardiovascular Risk Status: A Meta-Analysis of Prospective Evidence. Hypertension. 2010 April;55(4):1026–32. http://dx.doi.org/10.1161/HYPERTENSIONAHA.109.146621.

17. Vale S. Psychosocial stress and cardiovascular diseases. Postgrad Med J. 2005;81(957):429–35. http://dx.doi.org/10.1136/pgmj.2004.028977.

18. Lambert E, Lambert G. Stress and its role in sympathetic nervous system activation in hypertension and the metabolic syndrome. Curr Hypertens Rep. 2011;13(3):244–8. http://dx.doi.org/10.1007/s11906-011-0186-y.

19. Groeschel M, Braam B. Connecting chronic and recurrent stress to vascular dysfunction: no relaxed role for the renin-angiotensin system. Am J Physiol - Renal Physiol. 2011 January;300(1):F1–F10. http://dx.doi.org/10.1152/ajprenal.00208.2010.

20. Sata M, Fukuda D. Crucial role of renin-angiotensin system in the pathogenesis of atherosclerosis. Curr Opin Nephrol Hypertens. 2010;57(1-2):12–25.

21. Stegbauer J, Coffman T. New insights into angiotensin receptor actions: from blood pressure to aging. Curr Opin Nephrol Hypertens. 2011 Jan;20(1):84–8. http://dx.doi.org/10.1097/MNH.0b013e3283414d40.

22. Brunner EJ, Chandola T, Marmot MG. Prospective effect of job strain on general and central obesity in the Whitehall II Study. Am J Epidemiol. 2007 Apr;165(7):828–37. http://dx.doi.org/10.1093/aje/kwk058.

23. Chandola T, Britton A, Brunner E, Hemingway H, Malik M, Kumari M, et al. Work stress and coronary heart disease: what are the mechanisms? Europ Heart J. 2008 Mar;29(5):640–8. http://dx.doi.org/10.1093/eurheartj/ehm584.

24. Piazza PV, Le Moal M. Glucocorticoids as a biological substrate of reward: physiological and pathophysiological implications. Brain Res Brain Res Rev. 1997 Dec;25(3):359–72. http://dx.doi.org/10.1016/S0165-0173(97)00025-8.

25. Brisson C, Larocque B, Moisan J, Vezina M, Dagenais GR. Psychosocial factors at work, smoking, sedentary behaviour and body mass index: a prevalence study among 6995 white collar workers. J Occup Environ Med. 2000;42(1):40–6. http://dx.doi.org/10.1097/00043764-200001000-00011.

26. Backe E, Seidler A, Latza U, Rossnagel K, Schumann B. The role of psychosocial stress at work for the development of cardiovascular diseases: a systematic review. Int Arch Occup Environ Health. 2012;85(1):67–79. http://dx.doi.org/10.1007/s00420-011-0643-6.

27. Eller N, Netterstrom B, Gyntelberg F, Kristensen T, Nielsen F, Steptoe A et al. Work-related psychosocial factors and the development of ischemic heart disease: a systematic review. Cardiol Rev. 2009;17(2):83–97. http://dx.doi.org/10.1097/CRD.0b013e318198c8e9.

28. Kivimaki M, Virtanen M, Elovainio M, Kouvonen A, Vaananen A, Vahtera J. Work stress in the etiology of coronary heart disease--a meta-analysis. Scand J Work Environ Health. 2006 Dec;32(6):431–42. http://dx.doi.org/10.5271/sjweh.1049.

29. Belkic KL, Landsbergis PA, Schnall PL, Baker D. Is job strain a major source of cardiovascular disease risk? Environ Health. 2004 April;30(2):85–128.

30. Hemingway H, Marmot M Evidence based cardiology: Psychosocial factors in the aetiology and prognosis of

coronary heart disease: systematic review of prospective cohort studies. BMJ. 1999;318:1460–7. http://dx.doi.org/10.1136/bmj.318.7196.1460.

31. Kivimäki M, Nyberg S, Batty GD, Fransson E, Heikkilä K, Alfredsson L, et al. Job strain as a risk factor for coronary heart disease: a collaborative meta-analysis of individual participant data. The Lancet. 2012;380(9852):1491–7. http://dx.doi.org/10.1016/S0140-6736(12)60994-5.

32. World Health Organization. World health report: reducing risks, promoting healthy life. Geneva; 2002; Available from: http://epsl.asu.edu/ceru/Documents/whr_overview_eng.pdf.

33. Trudel X, Brisson C, Milot A. Job strain and masked hypertension. Psychosom Med. 2010 Oct;72(8):786–93. http://dx.doi.org/10.1097/PSY.0b013e3181eaf327.

34. Rosenthal T, Alter A. Occupational stress and hypertension. J Am Soc Hypertens. 2012 Oct;6(1):2–22. http://dx.doi.org/10.1016/j.jash.2011.09.002.

35. Landsbergis PA, Dobson M, Koutsouras G, Schnall P. Job strain and ambulatory blood pressure: a meta-analysis and systematic review. Am J Public Health. 2013 Mar;103(3):e61–71. http://dx.doi.org/10.2105/AJPH.2012.301153.

36. Liberati A, Altman D, Tetzlaff J, Mulrow C, Gotzsche P, Loannidis J, et al. The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate health care interventions: explanation and elaboration. J Clin Epidemiol. 2009;62(10):e1–34. http://dx.doi.org/10.1016/j.jclinepi.2009.06.006.

37. Schwartz J, Pickering T, Landsbergis P. Work-related stress and blood pressure: Current theoretical models and considerations from a behavioral medicine perspective. J Occup Health Psychol. 1996;1(3):287–310. http://dx.doi.org/10.1037/1076-8998.1.3.287.

38. O'Brien E, Asmar R, Beilin L, Imai Y, Mancia G, Mengden T, et al, European Society of Hypertension Working Group on Blood Pressure Monitoring. Practice guidelines of the European Society of Hypertension for clinic, ambulatory and self blood pressure measurement. J Hypertens. 2005 Apr;23(4):697–701. http://dx.doi.org/10.1097/01.hjh.0000163132.84890.c4.

39. Alterman T, Grosch J, Chen X, Chrislip D, Petersen M, Krieg Jr E, et al. Examining associations between job characteristics and health: Linking data from the Occupational Information Network (O*NET) to two U.S. national health surveys. J Occup Environ Med. 2008;50(12):1401–13. http://dx.doi.org/10.1097/JOM.0b013e318188e882.

40. Bages N, Warwick-Evans L, Falger P. Differences between informants about Type A, anger, and social support and the relationship with blood pressure. Psychol Health. 1997;12(4):453–65. http://dx.doi.org/10.1080/08870449708406722.

41. de Gaudemaris R, Levant A, Ehlinger V, Herin F, Lepage B, Soulat JM, et al. Blood pressure and working conditions in hospital nurses and nursing assistants. The ORSOSA study. Arch Cardiovasc Dis. 2011 Feb;104(2):97–103. http://dx.doi.org/10.1016/j.acvd.2010.12.001.

42. Ducher M, Fauvel JP, Cerutti C. Risk profile in hypertension genesis: A five-year follow-up study. Am J Hypertens. 2006 Aug;19(8):775–80. http://dx.doi.org/10.1016/j.amjhyper.2005.07.019.

43. Fauvel JP, Mpio I, Quelin P, Rigaud JP, Laville M, Ducher ML. Le stress professionnel et la réactivité pressionnelle au travail ne prédisent pas la presion artérielle à 5 ans [Professional stress and blood pressure reactivity to stress do not predict blood pressure at 5 years]. Arch Mal Coeur Vaiss. 2004 Jul-Aug;97(7-8):767–71.

44. Fauvel JP, Quelin P, Ducher M, Rakotomalala H, Laville M. Perceived job stress but not individual cardiovascular reactivity to stress is related to higher blood pressure at work. Hypertension. 2001 Jul;38(1):71–5. http://dx.doi.org/10.1161/01.HYP.38.1.71.

45. Frommer MS, Edye BV, Mandryk JA, Grammeno GL, Berry G, Ferguson DA. Systolic blood pressure in relation to occupation and perceived work stress. Scand J Work Environ Health. 1986 Oct;12(5):476–85. http://dx.doi.org/10.5271/sjweh.2115.

46. Garde AH, Laursen B, Jorgensen AH, Jensen BR. Effects of mental and physical demands on heart rate variability during computer work. Eur J Appl Physiol. 2002 Aug;87(4-5):456–61. http://dx.doi.org/10.1007/s00421-002-0656-7.

47. Hlávková J, Blažková V, Procházka B, Kožená L. Cardiovascular reactions to job stress in teachers. Homeostasis Health Dis. 1998;39(1-2):32–7.

48. Ilies R, Dimotakis N, De Pater I. Psychological and physiological reactions to high workloads: Implications for well-being. Pers Psychol. 2010;63(2):407–36. http://dx.doi.org/10.1111/j.1744-6570.2010.01175.x.

49. Kario K, James GD, Marion RM, Ahmed M, Pickering TG. The influence of work- and home-related stress on the levels and diurnal variation of ambulatory blood pressure and neurohumoral factors in employed women. Hypertens Res. 2002; 25(4):499–506. http://dx.doi.org/10.1291/hypres.25.499.

50. Kaufmann G, Beehr T. Interactions between job stressors and social support: some counterintuitive results. J Appl Psychol. 1986 Aug;71(3):522–6. http://dx.doi.org/10.1037/0021-9010.71.3.522.

51. Kawakami N, Haratani T, Kaneko T, Araki S. Perceived job-stress and blood pressure increase among Japanese blue collar workers: one-year follow-up study. Ind Health. 1989;27(2):71–81. http://dx.doi.org/10.2486/indhealth.27.71.

52. Kayaba K, Yazawa Y, Natsume T, Yaginuma T, Hosaka T, Hosoda S, et al. The relevance of psychosocial factors in acute ischemic heart disease. A case-control study of a Japanese population. Jpn Circ J. 1990 Apr;54(4):464–71. http://dx.doi.org/10.1253/jcj.54.464.

53. King A, Oka R, Young D. Ambulatory blood pressure and heart rate responses to the stress of work and caregiving in older women. J Gerontol. 1994;49(6):M239–M45. http://dx.doi.org/10.1093/geronj/49.6.M239.

54. Kjeldsen SE, Knudsen K, Ekrem G, Fure TO, Movinckel P, Erikssen JE. Is there an association between severe

job strain, transient rise in blood pressure and increased mortality? Blood Press. 2006;15(2):93–100. http://dx.doi.org/10.1080/08037050600750157.

55. Kornitzer M, Kettel F, Dramaix M, de Backer GU. Job stress and coronary heart disease. Adv Cardiol. 1982;29:56–61.
56. Kozena L, Frantik E, Horvath M. Cardiovascular reaction to job stress in middle-aged train drivers. Int J Behav Med. 1998;5(4):281–94.
57. Light KC, Brownley KA, Turner JR, Hinderliter AL, Girdler SS, Sherwood A, et al. Job status and high-effort coping influence work blood pressure in women and blacks. Hypertension. 1995 Apr;25(4 Pt 1):554–9. http://dx.doi.org/10.1161/01.HYP.25.4.554.
58. Light KC, Girdler SS, Sherwood A, Bragdon EE, Brownley KA, West SG, et al. High stress responsivity predicts later blood pressure only in combination with positive family history and high life stress. Hypertension. 1999 Jun;33(6):1458–64. http://dx.doi.org/10.1161/01.HYP.33.6.1458.
59. Lindquist TL, Beilin LJ, Knuiman M. Effects of lifestyle, coping and work-related stress on blood pressure in office workers. Clin Exp Pharmacol Physiol. 1995 Aug;22(8):580–2. http://dx.doi.org/10.1111/j.1440-1681.1995.tb02069.x.
60. Lindquist TL, Beilin LJ, Knuiman MW. Influence of lifestyle, coping, and job stress on blood pressure in men and women. Hypertension. 1997 Jan;29(1 Pt 1):1–7. http://dx.doi.org/10.1161/01.HYP.29.1.1.
61. Lovallo WR, al'Absi M, Pincomb GA, Everson SA, Sung BH, Passey RB, et al. Caffeine and behavioral stress effects on blood pressure in borderline hypertensive Caucasian men. Health Psychol. 1996 Jan;15(1):11–7. http://dx.doi.org/10.1037/0278-6133.15.1.11.
62. Ming EE, Adler GK, Kessler RC, Fogg LF, Matthews KA, Herd JA, et al. Cardiovascular reactivity to work stress predicts subsequent onset of hypertension: the Air Traffic Controller Health Change Study. Psychosom Med. 2004 Jul-Aug;66(4):459–65. http://dx.doi.org/10.1097/01.psy.0000132872.71870.6d.
63. Nedic O, Belkic K, Filipovic D, Jocic N. Job stressors among female physicians: relation to having a clinical diagnosis of hypertension. Int J Occup Environ Health. 2010 Jul-Sep;16(3):330–40. http://dx.doi.org/10.1179/107735210799160165.
64. Nyklicek I, Vingerhoets A, VanHeck GL, Kamphuis PL, VanPoppel J, VanLimpt M. Blood pressure, self-reported symptoms and job-related problems in schoolteachers. J Psychosom Res. 1997 Mar;42(3):287–96. http://dx.doi.org/10.1016/S0022-3999(96)00299-1.
65. Parkka J, Merilahti J, Mattila E, Malm E, Antila K, Tuomisto M, et al. Relationship of Psychological and Physiological Variables in Longterm self-monotored data during Work Ability Rehabilitation Program. IEEE Trans Inf Technol Biomed. 2009 Aug 15;13(2):141–51. http://dx.doi.org/10.1109/TITB.2008.2007078.
66. Peltzer K, Shisana O, Zuma K, Van Wyk B, Zungu-Dirwayi N. Job stress, job satisfaction and stress-related illnesses among South African educators. Stress Health. 2009;25(3):247–57. http://dx.doi.org/10.1002/smi.1244.
67. Piros S, Karlehagen S, Lappas G, Wilhelmsen L. Psychosocial risk factors for myocardial infarction among Swedish railway engine drivers during 10 years follow-up. J Cardiovasc Risk. 2000;7(5):389–94.
68. Rau R. The association between blood pressure and work stress: The importance of measuring isolated systolic hypertension. Work Stress. 2006 Mar;20(1):84–97. http://dx.doi.org/10.1080/02678370600679447.
69. Rod NH, Gronbaek M, Schnohr P, Prescott E, Kristensen TS. Perceived stress as a risk factor for changes in health behaviour and cardiac risk profile: a longitudinal study. J Intern Med. 2009 Nov;266(5):467–75. http://dx.doi.org/10.1111/j.1365-2796.2009.02124.x.
70. Seibt R, Boucsein W, Scheuch K. Effects of different stress settings on cardiovascular parameters and their relationship to daily life blood pressure in normotensives, borderline hypertensives and hypertensives. Ergonomics. 1998 May;41(5):634–48. http://dx.doi.org/10.1080/001401398186801.
71. Sims J. Individual differences in the perception of occupational stress and their association with blood pressure status. Work Stress. 1995;9(4):502–12. http://dx.doi.org/10.1080/02678379508256896.
72. Steptoe A. Stress, social support and cardiovascular activity over the working day. Intern J Psychophysiol. 2000 Sep;37(3):299–308. http://dx.doi.org/10.1016/S0167-8760(00)00109-4.
73. Steptoe A, Cropley M. Persistant high job demands and reactivity to mental stress predict future ambulatory blood pressure. J Hypertens. 2000;18(5):581–6. http://dx.doi.org/10.1097/00004872-200018050-00011.
74. Steptoe A, Cropley M, Joekes K. Task demands and the pressures of everyday life: associations between cardiovascular reactivity and work blood pressure and heart rate. Health Psychol. 2000 Jan;19(1):46–54. http://dx.doi.org/10.1037/0278-6133.19.1.46.
75. Steptoe A, Fieldman G, Evans O. An experimental study of the effects of control over work pace on cardiovascular responsivity. J Psychophysiol. 1993;7(4):290–300.
76. Steptoe A, Roy MP, Evans O, Snashall D. Cardiovascular stress reactivity and job strain as determinants of ambulatory blood pressure at work. J Hypertens. 1995 Feb;13(2):201–10. http://dx.doi.org/10.1097/00004872-199502000-00007.
77. Wellens B, Smith A. Combined workplace stressors and their relationship with mood, physiology, and performance. Work Stress. 2006 Jul-Sep;20(3):245–58. http://dx.doi.org/10.1080/02678370601022712.
78. Rau R. Job strain or healthy work: a question of task design. J Occup Health Psychol. 2004 Oct;9(4):322–38. http://dx.doi.org/10.1037/1076-8998.9.4.322.
79. Weidner G, Boughal T, Connor SL, Pieper C, Mendell NR. Relationship of job strain to standard coronary risk factors and psychological characteristics in women and men of the Family Heart Study. Health Psychol. 1997 May;16(3):239–47. http://dx.doi.org/10.1037/0278-6133.16.3.239.

80. Theorell T, de Faire U, Johnson J, Hall E, Perski A, Stewart W. Job strain and ambulatory blood pressure profiles. Scand J Work Environ Health. 1991 Dec;17(6):380–5. http://dx.doi.org/10.5271/sjweh.1690.

81. Hutt J, Weidner G. The effects of task demand and decision latitude on cardiovascular reactivity to stress. Behav Med. 1993;18(4):181–8. http://dx.doi.org/10.1080/08964289.1993.9939113.

82. Brown DE, James GD. Physiological stress responses in Filipino-American immigrant nurses: the effects of residence time, life-style, and job strain. Psychosom Med. 2000 May-Jun;62(3):394–400.

83. Evans O, Steptoe A. Social support at work, heart rate, and cortisol: a self-monitoring study. J Occup Health Psychol. 2001 Oct;6(4):361–70. http://dx.doi.org/10.1037/1076-8998.6.4.361.

84. Karlin WA, Brondolo E, Schwartz J. Workplace social support and ambulatory cardiovascular activity in New York City traffic agents. Psychosom Med. 2003 Mar-Apr;65(2):167–76. http://dx.doi.org/10.1097/01.PSY.0000033122.09203.A3.

85. Knox SS, Theorell T, Svensson JC, Waller D. The relation of social support and working environment to medical variables associated with elevated blood pressure in young males: a structural model. Soc Sci Med. 1985;21(5):525–31. http://dx.doi.org/10.1016/0277-9536(85)90036-X.

86. Melin B, Lundberg U, Soderlund J, Granqvist M. Psychological and physiological stress reactions of male and female assembly workers: a comparison between two different forms of work organization. J Organ Behav. 1999 Jan;20(1):47–61. http://dx.doi.org/10.1002/(SICI)1099-1379(199901)20:1<47::AID-JOB871>3.0.CO;2-F.

87. O'Connor DB, O'Connor RC, White BL, Bundred PE. Job strain and ambulatory blood pressure in British general practitioners: A preliminary study. Psychol Health Med. 2000;5(3):241–50. http://dx.doi.org/10.1080/713690191.

88. O'Connor DB, O'Connor RC, White BL, Bundred PE. Are occupational stress levels predictive of ambulatory blood pressure in British GPs? An exploratory study. Fam Pract. 2001 Feb;18(1):92–4. http://dx.doi.org/10.1093/fampra/18.1.92.

89. Schaubroeck J, Merritt DE. Divergent effects of job control on coping with work stressors: The key role of self-efficacy. Acad Manag J. 1997 Jun;40(3):738–54. http://dx.doi.org/10.2307/257061.

90. Theorell T, Hjemdahl P, Ericsson F. Psychosocial and physiological factors in relation to blood pressure at rest - A study of Swedish men in their upper twenties. J Hypertens. 1985;3(6):591–600. http://dx.doi.org/10.1097/00004872-198512000-00004.

91. Theorell T, Perski A, Akerstedt T, Sigala F, Ahlberg-Hulten G, Svensson J, et al. Changes in job strain in relation to changes in physiological state. A longitudinal study. Scand J Work Environ Health. 1988 Jun;14(3):189–96. http://dx.doi.org/10.5271/sjweh.1932.

92. Theorell T, Ahlberg-Hulten G, Jodko M, Sigala F, de la Torre B. Influence of job strain and emotion on blood pressure in female hospital personnel during workhours. Scand J Work Environ Health. 1993 Oct;19(5):313–8. http://dx.doi.org/10.5271/sjweh.1469.

93. Van Egeren LF. The relationship between job strain and blood pressure at work, at home, and during sleep. Psychosom Med. 1992 May-Jun;54(3):337–43.

94. Lorusso A, Bruno S, Caputo F, de Nichilo G, Minunni V, Sciannamblo G, et al. Job strain e pressione arteriosa in operatori sanitari [Job strain and blood pressure levels in health care workers]. G Ital Med Lav Ergon. 2007 Jul-Sep;29(3 Suppl):810–1.

95. Yadegarfar G, Alinia T, Asl RG, Allahyari T, Sheikhbagloo R. Study of association between job stress and cardiovascular disease risk factors among urmia petrochemical company personell. J Isfahan Med School. 2010;28(112).

96. Yu SF, Zhou WH, Jiang KY, Qiu Y, Gu GZ, Meng CM, Wang S. Effect of occupational stress on ambulatory blood pressure. Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi. 2009 Dec;27(12):711–5.

97. Gomez V, Moreno L. Psychosocial Job Factors (Demand-Control and Effort–reward Imbalance), Mental Health and Blood Pressure: A Study with High-School Teachers in Bogota, Colombia. Univ Psychol. 2010 May-Aug;9(2):393–407.

98. Byrne DG, Espnes GA. Occupational stress and cardiovascular disease. Stress Health. 2008 Aug;24(3):231–8. http://dx.doi.org/10.1002/smi.1203.

99. Carels RA, Sherwood A, Blumenthal JA. Psychosocial influences on blood pressure during daily life. Int J Psychophysiol. 1998 Mar;28(2):117–29. http://dx.doi.org/10.1016/S0167-8760(97)00090-1.

100. Gafarov VV, Gromova EA, Gagulin IV, Pilipenko PI. Изучение факторов риска возникновения инсульта по программе ВОЗ «MONICA-psychosocial [A study of the risk factors of stroke development in the framework of WHO program «MONICA-psychosocial»]. Zh Nevrol Psikhiatr Im S S Korsakova. 2005(Suppl 13):36–41.

101. Ganster DC, Schaubroeck J. Work Stress and Employee Health. J Manage. 1991 Jun;17(2):235–71. http://dx.doi.org/10.1177/014920639101700202.

102. Houtman I, Kornitzer M, De Smet P, Koyuncu R, De Backer G, Pelfrene E, et al. Job stress, absenteeism and coronary heart disease European cooperative study (the JACE study) - Design of a multicentre prospective study. Eur J Public Health. 1999 Mar;9(1):52–7. http://dx.doi.org/10.1093/eurpub/9.1.52.

103. Nowack KM, Schnall P, Melamed S, Froom P, Fisher J, Belkic K. Screening and management of the workplace for CVD risk. Occup Med-State Art. 2000 Jan-Mar;15(1):231–56.

104. Pickering T. The effects of occupational stress on blood pressure in men and women. Acta Physiol Scand Suppl. 1997;640:125–8.

105. Pickering T. Cardiovascular pathways: socioeconomic status and stress effects on hypertension and cardiovascular function. Ann N Y Acad Sci. 1999;896:262–77. http://dx.doi.org/10.1111/j.1749-6632.1999.tb08121.x.

106. Saruhara H, Ishida J, Ohno H, Kijima G, Karube Y, Takeda K, et al. Job-strain induced workplace hypertension is associated with obesity and high normal blood pressures at the physical checkup. J Am Coll Cardiol. 2006 Feb;47(4):350A–1A.

107. Siegrist J. Emotions and health in occupational life: new scientific findings and policy implications. Patient Educ Couns. 1995 Jul;25(3):227–36. http://dx.doi.org/10.1016/0738-3991(95)00805-A.

108. Siegrist J, Peter R, Motz W, Strauer BE. The role of hypertension, left ventricular hypertrophy and psychosocial risks in cardiovascular disease: prospective evidence from blue-collar men. Eur Heart J. 1992 Sep;13 Suppl D:89–95.

109. Slaby A. Psychophysiological risk factors of cardiovascular diseases: Psychosocial stress, personality, and occupational specificity. Act Nerv Super. 1982 Mar;24(1):34–8.

110. Steptoe A, Marmot M. The role of psychobiological pathways in socio-economic inequalities in cardiovascular disease risk. Eur Heart J. 2002 Jan;23(1):13–25. http://dx.doi.org/10.1053/euhj.2001.2611.

111. Strike PC, Steptoe A. Psychosocial factors in the development of coronary artery disease. Prog Cardiovasc Dis. 2004 Jan-Feb;46(4):337–47. http://dx.doi.org/10.1016/j.pcad.2003.09.001.

112. Wilkins K, Beaudet MP. Work stress and health. Health Rep. 1998;10(3):47–62 (ENG); 49–66 (FRE).

113. Edin-Liljegren A, Hassler S, Sjolander P, Daerga L. Risk factors for cardiovascular diseases among Swedish Sami--a controlled cohort study. Int J Circumpolar Health. 2004;63 Suppl 2:292–7. http://dx.doi.org/10.3402/ijch.v63i0.17922.

114. Fletcher BC, Jones F. A Refutation of Karasek Demand-Discretion Model of Occupational Stress with a Range of Dependent Measures. J Organ Behav. 1993 Jul;14(4):319–30. http://dx.doi.org/10.1002/job.4030140404.

115. Gyntelberg F, Suadicani P, Jensen G, Schnohr P, Netterstrom B, Kristensen TS, et al. Job strain and cardiovascular risk factors among members of the Danish parliament. Occup Med. 1998 Jan;48(1):31–6. http://dx.doi.org/10.1093/occmed/48.1.31.

116. Kamarck TW, Schwartz JE, Shiffman S, Muldoon MF, Sutton-Tyrrell K, Janicki DL. Psychosocial stress and cardiovascular risk: what is the role of daily experience? J Pers. 2005 Dec;73(6):1749–74. http://dx.doi.org/10.1111/j.0022-3506.2005.00365.x.

117. Leitner K, Resch MG. Do the effects of job stressors on health persist over time? A longitudinal study with observational stressor measures. J Occup Health Psych. 2005 Jan;10(1):18–30. http://dx.doi.org/10.1037/1076-8998.10.1.18.

118. Patel C. Identifying Psychosocial and Other Risk-Factors in Whitehall-Ii Study. Homeostasis Health Dis. 1994;35(1-2):71–83.

119. Vrijkotte T, Van Doornen L, de Geus E. Overcommitment to work is associated with changes in cardiac sympathetic regulation. Psychosom Med. 2004 Sep-Oct;66(5):656–63. http://dx.doi.org/10.1097/01.psy.0000138283.65547.78.

120. Goldstein IB, Shapiro D, Chicz-DeMet A, Guthrie D. Ambulatory blood pressure, heart rate, and neuroendocrine responses in women nurses during work and off work days. Psychosom Med. 1999 May-Jun;61(3):387–96.

121. Landsbergis PA, Schnall PL, Pickering TG, Warren K, Schwartz JE. Lower socioeconomic status among men in relation to the association between job strain and blood pressure. Scand J Work Environ Health. 2003 Jun;29(3):206–15. http://dx.doi.org/10.5271/sjweh.723.

122. Ohlin B, Berglund G, Nilsson PM, Melander O. Job strain, job demands and adrenergic beta1-receptor-polymorphism: a possible interaction affecting blood pressure in men. J Hypertens. 2008 Aug;26(8):1583–9. http://dx.doi.org/10.1097/HJH.0b013e328303df5f.

123. Albright CL, Winkleby MA, Ragland DR, Fisher J, Syme SL. Job strain and prevalence of hypertension in a biracial population of urban bus drivers. Am J Public Health. 1992 Jul;82(7):984–9. http://dx.doi.org/10.2105/AJPH.82.7.984.

124. Alfredsson L, Hammar N, Fransson E, de Faire U, Hallqvist J, Knutsson A, et al. Job strain and major risk factors for coronary heart disease among employed males and females in a Swedish study on work, lipids and fibrinogen. Scand J Work Environ Health. 2002 August;28(4):238–48. http://dx.doi.org/10.5271/sjweh.671.

125. Bishop G, Enkelmann H, Tong E, Why Y, Diong S, Ang J, Khader M. Job Demands, Decisional Control, and Cardiovascular Responses. J Occup Health Psych. 2003 April;8(2):146–56. http://dx.doi.org/10.1037/1076-8998.8.2.146.

126. Blumenthal JA, Thyrum ET, Siegel WC. Contribution of job strain, job status and marital status to laboratory and ambulatory blood pressure in patients with mild hypertension. J Psychosom Res. 1995 Feb;39(2):133–44. http://dx.doi.org/10.1016/0022-3999(94)00087-L.

127. Brown DE, James GD, Mills PS. Occupational differences in job strain and physiological stress: female nurses and school teachers in Hawaii. Psychosom Med. 2006 Jul-Aug;68(4):524–30. http://dx.doi.org/10.1097/01.psy.0000222356.71315.8e.

128. Cesana G, Ferrario M, Sega R, Milesi C, DeVito G, Mancia G, et al. Job strain and ambulatory blood pressure levels in a population-based employed sample of men from northern Italy. Scand J Work Environ Health. 1996 Aug;22(4):294–305. http://dx.doi.org/10.5271/sjweh.144.

129. Cesana G, Sega R, Ferrario M, Chiodini P, Corrao G, Mancia G. Job strain and blood pressure in employed men and women: a pooled analysis of four northern italian population samples. Psychosom Med. 2003 Jul-Aug;65(4):558–63. http://dx.doi.org/10.1097/01.PSY.0000041473.03828.67.

130. Chapman A, Mandryk JA, Frommer MS, Edye BV, Ferguson DA. Chronic Perceived Work Stress and Blood-Pressure among Australian Government Employees. Scand J Work Environ Health. 1990 Aug;16(4):258–69. http://dx.doi.org/10.5271/sjweh.1786.

131. Chikani V, Reding D, Gunderson P, McCarty CA. Psychosocial work characteristics predict cardiovascular disease risk factors and health functioning in rural women: the Wisconsin Rural Women's Health Study. J Rural Health. 2005;21(4):295–302. http://dx.doi.org/10.1111/j.1748-0361.2005.tb00098.x.

132. Clays E, Van Herck K, De Buyzere M, Kornitzer M, Kittel F, De Backer G, et al. Behavioural and psychosocial correlates of nondipping blood pressure pattern among middle-aged men and women at work. J Hum Hypertens. 2011 Jun; 26(6):381–7. http://dx.doi.org/10.1038/jhh.2011.42.

133. Clays E, Leynen F, De Bacquer D, Kornitzer M, Kittel F, Karasek R, et al. High job strain and ambulatory blood pressure in middle-aged men and women from the Belgian job stress study. J Occup Environ Med. 2007 Apr;49(4):360–7. http://dx.doi.org/10.1097/JOM.0b013e31803b94e2.

134. Curtis AB, James SA, Raghunathan TE, Alcser KH. Job strain and blood pressure in African Americans: the Pitt County Study. Am J Public Health. 1997 Aug;87(8):1297–302. http://dx.doi.org/10.2105/AJPH.87.8.1297.

135. de Mello A, Chor D, Faerstein E, Werneck G, Lopes C. Job strain and hypertension in women: Estudo Pro-Saude [Pro-Health Study]. Rev Saude Publ. 2009 Oct;43(5):893–6.

136. Ducher M, Cerutti C, Chatellier G, Fauvel JP. Is high job strain associated with hypertension genesis? Am J Hypertens. 2006 Jul;19(7):694–700. http://dx.doi.org/10.1016/j.amjhyper.2005.12.016.

137. Fauvel JP, M'Pio I, Quelin P, Rigaud JP, Laville M, Ducher M. Neither perceived job stress nor individual cardiovascular reactivity predict high blood pressure. Hypertension. 2003 Dec;42(6):1112–6. http://dx.doi.org/10.1161/01.HYP.0000102862.93418.EE.

138. Fornari C, Ferrario M, Menni C, Sega R, Facchetti R, Cesana GC. Biological consequences of stress: conflicting findings on the association between job strain and blood pressure. Ergonomics. 2007 Nov;50(11):1717–26. http://dx.doi.org/10.1080/00140130701674208.

139. Fox ML, Dwyer DJ, Ganster DC. Effects of stressful job demands and control on physiological and attitudinal outcomes in a hospital setting. Acad Manage J. 1993 Apr;36(2):289–318. http://dx.doi.org/10.2307/256524.

140. Gallo LC, Bogart LM, Vranceanu AM, Walt LC. Job characteristics, occupational status, and ambulatory cardiovascular activity in women. Ann Behav Med. 2004 Aug;28(1):62–73. http://dx.doi.org/10.1207/s15324796abm2801_8.

141. Greenlund KJ, Liu K, Knox S, McCreath H, Dyer AR, Gardin J. Psychosocial work characteristics and cardiovascular disease risk factors in young adults: The cardia study. Soc Sci Med. 1995;41(5):717–23. http://dx.doi.org/10.1016/0277-9536(94)00385-7.

142. Guimont C, Brisson C, Dagenais GR, Milot A, Vezina M, Masse B, et al. Effects of job strain on blood pressure: a prospective study of male and female white-collar workers. Am J Public Health. 2006 Aug;96(8):1436–43. http://dx.doi.org/10.2105/AJPH.2004.057679.

143. Kamarck TW, Janicki DL, Shiffman S, Polk DE, Muldoon MF, Liebenauer LL, et al. Psychosocial demands and ambulatory blood pressure: a field assessment approach. Physiol Behav. 2002 Dec;77(4-5):699–704. http://dx.doi.org/10.1016/S0031-9384(02)00921-6.

144. Kamarck T, Shiffman S, Smithline L, Goodie J, Paty J, Gnys M, et al. Effects of task strain, social conflict, and emotional activation on ambulatory cardiovascular activity: Daily life consequences of recurring stress in a multiethnic adult sample. Health Psychol. 1998 Jan;17(1):17–29. http://dx.doi.org/10.1037/0278-6133.17.1.17.

145. Kang M, Koh S, Cha B, Park J, Baik S, Chang S. Job stress and cardiovascular risk factors in male workers. Prev Med. 2005 May;40(5):583–8. http://dx.doi.org/10.1016/j.ypmed.2004.07.018.

146. Kang M, Koh S, Cha B, Park J, Woo J, Chang S. Association between job stress on heart rate variability and metabolic syndrome in shipyard male workers. Yonsei Med J. 2004 Oct 31;45(5):838–46.

147. Kawakami N, Haratani T, Araki S. Job strain and arterial blood pressure, serum cholesterol, and smoking as risk factors for coronary heart disease in Japan. Int Arch Occup Env Hea. 1998 Sep;71(6):429–32. http://dx.doi.org/10.1007/s004200050302.

148. Kivimaki M, Head J, Ferrie LE, Shipley MJ, Steptoe A, Vahtera J, et al. Hypertension is not the link between job strain and coronary heart disease in the Whitehall II study. Am J Hypertens. 2007 Nov;20(11):1146–53.

149. Kobayashi Y, Hirose T, Tada Y, Tsutsumi A, Kawakami N. Relationship between two job stress models and coronary risk factors among Japanese part-time female employees of a retail company. J Occup Health. 2005 May;47(3):201–10. http://dx.doi.org/10.1539/joh.47.201.

150. Laflamme N, Brisson C, Moisan J, Milot A, Masse B, Vezina M. Job strain and ambulatory blood pressure among female white-collar workers. Scand J Work Environ Health. 1998 Oct;24(5):334–43. http://dx.doi.org/10.5271/sjweh.353.

151. Landsbergis PA, Schnall PL, Pickering TG, Warren K, Schwartz JE. Life-course exposure to job strain and ambulatory blood pressure in men. Am J Epidemiol. 2003 Jun 1;157(11):998–1006. http://dx.doi.org/10.1093/aje/kwg095.

152. Light KC, Turner JR, Hinderliter AL. Job strain and ambulatory work blood pressure in healthy young men and women. Hypertension. 1992 Aug;20(2):214–8. http://dx.doi.org/10.1161/01.HYP.20.2.214.

153. Maina G, Bovenzi M, Palmas A, Prodi A, Filon FL. Job strain, effort–reward imbalance and ambulatory blood pressure: results of a cross-sectional study in call handler operators. Int Arch Occup Env Hea. 2011 Apr;84(4):383–91. http://dx.doi.org/10.1007/s00420-010-0576-5.

154. Markovitz JH, Matthews KA, Whooley M, Lewis CE, Greenlund KJ. Increases in job strain are associated with incident hypertension in the CARDIA Study. Ann Behav Med. 2004 Aug;28(1):4–9. http://dx.doi.org/10.1207/s15324796abm2801_2.

155. Melamed S, Kristal-Boneh E, Harari G, Froom P, Ribak J. Variation in the ambulatory blood pressure response to daily work load - The moderating role of job control. Scand J Work Environ Health. 1998 June;24(3):190–6. http://dx.doi.org/10.5271/sjweh.298.

156. Menni C, Bagnardi V, Padmanabhan S, Facchetti R, Sega R, Ferrario MM, et al. Evaluation of how gene-job strain interaction affects blood pressure in the PAMELA study. Psychosom Med. 2011 May;73(4):304–9. http://dx.doi.org/10.1097/PSY.0b013e318212e0be.

157. Mezuk B, Kershaw K, Hudson D, Lim K, Ratliff S. Job strain, workplace discrimination, and hypertension among older workers: The Health and Retirement Study. Race Soc Prob. 2011;3(1):38–50. http://dx.doi.org/10.1007/s12552-011-9041-7.

158. Netterstrom B, Kristensen TS, Damsgaard MT, Olsen O, Sjol A. Job strain and cardiovascular risk factors: a cross sectional study of employed Danish men and women. Br J Ind Med. 1991 Oct;48(10):684–9.

159. Niedhammer I, Goldberg M, Leclerc A, David S, Bugel I, Landre MF. Psychosocial work environment and cardiovascular risk factors in an occupational cohort in France. J Epidemiol Commun H. 1998 Feb;52(2):93–100. http://dx.doi.org/10.1136/jech.52.2.93.

160. Nomura K, Nakao M, Karita K, Nishikitani M, Yano E. Association between work-related psychological stress and arterial stiffness measured by brachial-ankle pulse-wave velocity in young Japanese males from an information service company. Scand J Work Environ Health. 2005;31(5):352–9. http://dx.doi.org/10.5271/sjweh.918.

161. Ohlin B, Berglund G, Rosvall M, Nilsson PM. Job strain in men, but not in women, predicts a significant rise in blood pressure after 6.5 years of follow-up. J Hypertens. 2007 Mar;25(3):525–31. http://dx.doi.org/10.1097/HJH.0b013e32801220fa.

162. Pelfrene E, De Backer G, Mak R, de Smet P, Kornitzer M. Job stress and cardiovascular risk factors. Results from the BELSTRESS study. Arch Pub Health. 2002;60(34):245–68.

163. Radi S, Lang T, Lauwers-Cances V, Diene E, Chatellier G, Larabi L, et al. Job constraints and arterial hypertension: different effects in men and women: the IHPAF II case control study. Occup Environ Med. 2005 Oct;62(10):711–7. http://dx.doi.org/10.1136/oem.2004.012955.

164. Rau R, Georgiades A, Fredrikson M, Lemne C, de Faire U. Psychosocial work characteristics and perceived control in relation to cardiovascular rewind at night. J Occup Hea Psychol. 2000;6(3):171–81. http://dx.doi.org/10.1037/1076-8998.6.3.171.

165. Reed DM, LaCroix AZ, Karasek RA, Miller D, MacLean CA. Occupational strain and the incidence of coronary heart disease. Am J Epidemiol. 1989 Mar;129(3):495–502.

166. Riese H, Van Doornen L, Houtman I, De Geus E. Job strain in relation to ambulatory blood pressure, heart rate, and heart rate variability among female nurses. Scand J Work Environ Health. 2004 Dec;30(6):477–85. http://dx.doi.org/10.5271/sjweh.837.

167. Schnall PL, Schwartz JE, Landsbergis PA, Warren K, Pickering TG. Relation between job strain, alcohol, and ambulatory blood pressure. Hypertension. 1992 May;19(5):488–94. http://dx.doi.org/10.1161/01.HYP.19.5.488.

168. Song YK, Lee KK, Kim HR, Koo JW. Job demand and cardiovascular disease risk factor in white-collar workers. Ind Health. 2010;48(1):12–7. http://dx.doi.org/10.2486/indhealth.48.12.

169. Steptoe A, Cropley M. Job strain, blood pressure and responsivity to uncontrollable stress. Int J Psychophysiol. 1998 Sep;30(1-2):89–90. http://dx.doi.org/10.1016/S0167-8760(98)90227-6.

170. Steptoe A, Willemsen G. The influence of low job control on ambulatory blood pressure and perceived stress over the working day in men and women from the Whitehall II cohort. J Hypertens. 2004 May;22(5):915–20. http://dx.doi.org/10.1097/00004872-200405000-00012.

171. Su CT. Association between job strain status and cardiovascular risk in a population of Taiwanese white-collar workers. Jpn Circ J. 2001 Jun;65(6):509–13. http://dx.doi.org/10.1253/jcj.65.509.

172. Su CT, Yang HJ, Lin CF, Tsai MC, Shieh YH, Chiu WT. Arterial blood pressure and blood lipids as cardiovascular risk factors and occupational stress in Taiwan. Int J Cardiol. 2001 Dec;81(2-3):181–7. http://dx.doi.org/10.1016/S0167-5273(01)00565-4.

173. Thomas KS, Nelesen RA, Ziegler MG, Bardwell WA, Dimsdale JE. Job strain, ethnicity, and sympathetic nervous system activity. Hypertension. 2004 Dec;44(6):891–6. http://dx.doi.org/10.1161/01.HYP.0000148499.54730.0d.

174. Thomas C, Power C. Do early life exposures explain associations in mid-adulthood between workplace factors and risk factors for cardiovascular disease? Int J Epidemiol. 2010 Jun;39(3):812–24. http://dx.doi.org/10.1093/ije/dyp365.

175. Tobe SW, Kiss A, Szalai JP, Perkins N, Tsigoulis M, Baker B. Impact of job and marital strain on ambulatory blood pressure results from the double exposure study. Am J Hypertens. 2005 Aug;18(8):1046–51. http://dx.doi.org/10.1016/j.amjhyper.2005.03.734.

176. Tobe SW, Kiss A, Sainsbury S, Lesin M, Geerts R, Baker B. The impact of job strain and marital cohesion on ambulatory blood pressure during 1 year: The double exposure study. Am J Hypertens. 2007 Feb;20(2):148–53. http://dx.doi.org/10.1016/j.amjhyper.2006.07.011.

177. Tsutsumi A, Kayaba K, Tsutsumi K, Igarashi M. Association between job strain and prevalence of hypertension: a cross sectional analysis in a Japanese working population with a wide range of occupations: the Jichi Medical School cohort study. Occup Environ Med. 2001 Jun;58(6):367–73. http://dx.doi.org/10.1136/oem.58.6.367.

178. Tsutsumi A, Tsutsumi K, Kayaba K, Theorell T, Nago N, Kario K, et al. Job strain and biological coronary risk factors: a cross-sectional study of male and female workers in a Japanese rural district. Int J Behav Med. 1998;5(4):295–311. http://dx.doi.org/10.1207/s15327558ijbm0504_4.

179. Winnubst J, Marcelissen F, Kleber R. Effects of social support in the stressor-strain relationship: A Dutch sample. Soc Sci Med. 1982;16(4):475–82. http://dx.doi.org/10.1016/0277-9536(82)90056-9.

180. 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 May 15;159(10):950–8. http://dx.doi.org/10.1093/aje/kwh127.

181. Landsbergis PA, Schnall PL, Warren K, Pickering TG, Schwartz JE. Association between ambulatory blood pressure and alternative formulations of job strain. Scand J Work Environ Health. 1994 Oct;20(5):349–63. http://dx.doi.org/10.5271/sjweh.1386.

182. Schnall PL, Schwartz JE, Landsbergis PA, Warren K, Pickering TG. A longitudinal study of job strain and ambulatory blood pressure: results from a three-year follow-up. Psychosom Med. 1998 Nov-Dec;60(6):697–706.

183. Bellingrath S, Weigl T, Kudielka BM. Chronic work stress and exhaustion is associated with higher allostatic load in female school teachers. Stress. 2009;12(1):37–48. http://dx.doi.org/10.1080/10253890802042041.

184. Peter R, Siegrist J. Chronic work stress, sickness absence, and hypertension in middle managers: general or specific sociological explanations? Soc Sci Med. 1997 Oct;45(7):1111–20. http://dx.doi.org/10.1016/S0277-9536(97)00039-7.

185. Peter R, Alfredsson L, Hammar N, Siegrist J, Theorell T, Westerholm P. High effort, low reward, and cardiovascular risk factors in employed Swedish men and women: baseline results from the WOLF Study. J Epidemiol Com Health. 1998 Sep;52(9):540–7. http://dx.doi.org/10.1136/jech.52.9.540.

186. Peter R, Alfredsson L, Knutsson A, Siegrist J, Westerholm P. Does a stressful psychosocial work environment mediate the effects of shift work on cardiovascular risk factors? Scand J Work Environ Health. 1999 Aug;25(4):376–81. http://dx.doi.org/10.5271/sjweh.448.

187. Irie M, Tsutsumi A, Shioji I, Kobayashi F. Effort–reward imbalance and physical health among Japanese workers in a recently downsized corporation. Int Arch Occup Environ Health. 2004 Aug;77(6):409–17. http://dx.doi.org/10.1007/s00420-004-0533-2.

188. Vrijkotte T, Van Doornen L, De Geus E. Effects of work stress on ambulatory blood pressure, heart rate, and heart rate variability. Hypertension. 2000 April;35(4):880–6. http://dx.doi.org/10.1161/01.HYP.35.4.880.

189. Xu L, Siegrist J, Cao W, Li L, Tomlinson B, Chan J. Measuring job stress and familly stress in Chinese working women: A validation study focusing on blood pressure and psychosomatic symptoms. Women Health. 2004;39(2):31–46. http://dx.doi.org/10.1300/J013v39n02_03.

190. Yu SF, Zhou WH, Jiang KY, Gu GZ, Wang S. Job stress, gene polymorphism of beta2-AR, and prevalence of hypertension. Biomed Env Sci. 2008 Jun;21(3):239–46. http://dx.doi.org/10.1016/S0895-3988(08)60036-7.

191. Gilbert-Ouimet M, Brisson C, Vezina M, Milot A, Blanchette C. Repeated Effort-Reward Imbalance Exposure, Increased Blood Pressure, and Hypertension Incidence among White-Collar Workers. J Psychosom Res. 2012 Jan;72(1):26-32. http://dx.doi.org/10.1016/j.jpsychores.2011.07.002

192. Steptoe A. Job control, perceptions of control, and cardiovascular activity - An analysis of ambulatory measures collected over the working day. J Psychosom Res. 2001 Feb;50(2):57–63. http://dx.doi.org/10.1016/S0022-3999(00)00201-4.

193. Jonsson D, Rosengren A, Dotevall A, Lappas G, Wilhelmsen L. Job control, job demands and social support at work in relation to cardiovascular risk factors in MONICA 1995, Goteborg. J Cardiovasc Risk. 1999 Dec;6(6):379–85.

194. Steptoe A, Siegrist J, Kirschbaum C, Marmot M. Effort–reward imbalance, overcommitment, and measures of cortisol and blood pressure over the working day. Psychosom Med. 2004 May-Jun;66(3):323–9. http://dx.doi.org/10.1097/01.psy.0000126198.67070.72.

195. American heart association. High blood pressure: Heart disease and stroke statistics. Circulation. 2013;127:e6–e245.

196. Messing K. Multiple roles and complex exposures: hard to pin down risks for working women. In: Goldman MB, Hatch MC, editors. Women and health. Californie: Academic Press. 2000; p. 455–62. http://dx.doi.org/10.1016/B978-012288145-9/50042-5.

197. Tobe S, Baker B, Kiss A, Sainsbury S. Marital cohesion moderates the elevation of ambulatory blood pressure due to job strain. Am J Hypertens. 2005 May;18(5):151A–2A. http://dx.doi.org/10.1016/j.amjhyper.2005.03.421.

198. de Smet P, Sans S, Dramaix M, Boulenguez C, de Backer G, Ferrario M, et al. Gender and regional differences in perceived job stress across Europe. Eur J Public Health. 2005 Oct;15(5):536–45. http://dx.doi.org/10.1093/eurpub/cki028.

199. Waldenstrom K, Ahlberg G, Bergman P, Forsell Y, Stoetzer U, Waldenstrom M, et al. Externally assessed psychosocial work characteristics and diagnoses of anxiety and depression. Occup Environ Med. 2008 Feb;65(2):90–7. http://dx.doi.org/10.1136/oem.2006.031252.

200. Ertel KA, Koenen KC, Berkman LF. Incorporating home demands into models of job strain: findings from the work, family, and health network. J Occup Environ Med. 2008 Nov;50(11):1244–52. http://dx.doi.org/10.1097/JOM.0b013e31818c308d.

201. Pickering TG, Eguchi K, Kario K. Masked hypertension: a review. Hypertens Res. 2007 Jun;30(6):479–88. http://dx.doi.org/10.1291/hypres.30.479.

202. Verberk WJ, Kessels AG, Leeuw PW. Prevalence, causes, and consequences of masked hypertension: a meta-analysis. Am J Hypertens. 2008;21(9):969–75. http://dx.doi.org/10.1038/ajh.2008.221.

203. Bobrie G, Clerson P, Menard J, Postel-Vinay N, Chatellier G, Plouin PF. Masked hypertension: a systematic review. J Hypertens. 2008 Sep;26(9):1715–25. http://dx.doi.org/10.1097/HJH.0b013e3282fbcedf.

204. Cuspidi C, Parati G. Masked hypertension: an independent predictor of organ damage. J Hypertens. 2007 Feb;25(2):275–9. http://dx.doi.org/10.1097/HJH.0b013e32801da2d2.

205. Boggia J, Li Y, Thijs L, Hansen TW, Kikuya M, Bjorklund-Bodegard K, et al. Prognostic accuracy of day versus night ambulatory blood pressure: a cohort study. Lancet. 2007 Oct;370(9594):1219–29. http://dx.doi.org/10.1016/S0140-6736(07)61538-4.

206. Liu JE, Roman MJ, Pini R, Schwartz JE, Pickering TG, Devereux RB. Cardiac and Arterial Target Organ Damage in Adults with Elevated Ambulatory and Normal Office Blood Pressure. Ann Inter Med. 1999;131(8):564–72. http://dx.doi.org/10.7326/0003-4819-131-8-199910190-00003.

207. Devereux R, Pickering T. Relationship between the level, pattern and variability of ambulatory blood pressure and target organ damage in hypertension. J Hypertens Suppl. 1991 Dec;9(8):S34–8.

208. Verdecchia P, Porcellati C, Schillaci G, Borgioni C, Ciucci A, Battistelli M, et al. Ambulatory blood pressure. An independent predictor of prognosis in essential hypertension. Hypertension. 1994 Dec;24(6): 793–801. http://dx.doi.org/10.1161/01.HYP.24.6.793.

209. Sterne JA, Egger M. Funnel plots for detecting bias in meta-analysis: guidelines on choice of axis. J Clin Epidemiol. 2001 Oct;54(10):1046–55. http://dx.doi.org/10.1016/S0895-4356(01)00377-8.

210. Rothman KJ, Greenland S, Last TL. Modern epidemiology. 3rd ed. Philadelphia: Wolters Kluwer l Lippincott Williams & Wilkins; 2008.

211. Spitzer W. Meta-meta-analysis: unanswered questions about aggregating data. J Clin Epidemiol. 1991;44:103–7. http://dx.doi.org/10.1016/0895-4356(91)90258-B.

212. Morton LM, Cahill J, Hartge P. Reporting participation in epidemiologic studies: a survey of practice. Am J Epidemiol. 2006 Feb 163(3):197–203. http://dx.doi.org/10.1093/aje/kwj036.

213. Galea S, Tracy M. Participation rates in epidemiologic studies. Ann Epidemiol. 2007 Sep;17(9):643–53. http://dx.doi.org/10.1016/j.annepidem.2007.03.013.

214. Riese H, Van Doornen L, Houtman I, De Geus E. Job strain and risk indicators for cardiovascular disease in young female nurses. Health Psychol. 2000;19(5):429–40. http://dx.doi.org/10.1037/0278-6133.19.5.429.

215. Checkoway H, Pearce NE, Kriebel D. Research methods in Occupational epidemiology. 2th ed. New York: Oxford University Press; 2004. http://dx.doi.org/10.1093/acprof:oso/9780195092424.001.0001.

216. Costanzo S, Di Castelnuovo A, Zito F, Krogh V, Siani A, Arnout J, et al. Prevalence, awareness, treatment and control of hypertension in healthy unrelated male-female pairs of European regions: the dietary habit profile in European communities with different risk of myocardial infarction--the impact of migration as a model of gene-environment interaction project. J Hypertens. 2008 Dec;26(12):2303–11. http://dx.doi.org/10.1097/HJH.0b013e328311ce04.

217. Kristensen TS, Borg V. Copenhagen Psychosocial Questionnaire (COPSOQ)- A questionnaire on psychosocial working conditions, health and well-being in three versions. In: Psychosocial Department, editor. Copenhagen: The National Institute of Occupational Health; 2003.

218. Aboa-Eboule C, Brisson C, Maunsell E, Masse B, Bourbonnais R, Vezina M, et al. Job strain and risk of acute recurrent coronary heart disease events. JAMA. 2007 Oct 10;298(14):1652–60. http://dx.doi.org/10.1001/jama.298.14.1652.

219. de Kloet ER, Joels M, Holsboer F. Stress and the brain: from adaptation to disease. Nat Rev Neurosci. 2005;6(6):463–75. http://dx.doi.org/10.1038/nrn1683.

220. Lucassen P, Heine V, Muller M, van der Beek E, Wiegant V, Ron D, et al. Stress, Depression and Hippocampal Apoptosis. CNS Neurol Disord Drug Targets. 2006;5(5):531–46. http://dx.doi.org/10.2174/187152706778559273.

221. Lee AL, Ogle WO, Sapolsky RM. Stress and depression: possible links to neuron death in the hippocampus. Bipolar Disorders. 2002;4(2):117–28. http://dx.doi.org/10.1034/j.1399-5618.2002.01144.x.

222. Karasek RA, Theorell T, Schwartz JE, Schnall PL, Pieper CF, Michela JL. Job characteristics in relation to the prevalence of myocardial infarction in the US Health Examination Survey (HES) and the Health and Nutrition Examination Survey (HANES). Am J Public Health. 1988 Aug;78(8):910–8. http://dx.doi.org/10.2105/AJPH.78.8.910.

223 Peter R. Job stress and cardiovascular risk factors. Results from the BELSTRESS study. Psychol Beitr. 1995;37(1-2):40–5.

224. Steptoe A. Psychological factors and cardiovascular disease. Curr Opin Psychiatr. 1998 Nov;11(6):655–60. http://dx.doi.org/10.1097/00001504-199811000-00010.

225. Moorman R. Relationship between Organizational Justice and Organizational Citizenship Behavior: Do Fairness Perceptions Influence Employee Citizenship? Journal of Applied Psychology. 1991;76:845–55. http://dx.doi.org/10.1037/0021-9010.76.6.845.

226. Nyberg A. The impact of Managerial Leadership on Stress and Health Among Employees. Stockholm: Karolinska Institutet; 2009.

227. Bosma H, Peter R, Siegrist J, Marmot M. Two alternative job stress models and the risk of coronary heart disease. Am J Public Health. 1998 Jan;88(1):68–74. http://dx.doi.org/10.2105/AJPH.88.1.68.

228. Peter R, Siegrist J, Hallqvist J, Reuterwall C, Theorell T. Psychosocial work environment and myocardial infarction: improving risk estimation by combining two complementary job stress models in the SHEEP Study. J Epidemiol Commun H. 2002 Apr;56(4):294–300. http://dx.doi.org/10.1136/jech.56.4.294.

229. Kouvonen A, Kivimaki M, Elovainio M, Virtanen M, Linna A, Vahtera J. Job strain and leisure-time physical activity in female and male public sector employees. Prev Med. 2005 Aug;41(2):532–9. http://dx.doi.org/10.1016/j.ypmed.2005.01.004.

230. Wardle J, Gibson E. Impact of stress on diet: processes and implications. In: Stansfeld S, Marmot M, editors. Stress and the heart: psychosocial pathways to coronary heart disease. London: BMJ book; 2002. p. 129–49.

231. Dallman MF, Pecoraro N, Akana SF, La Fleur SE, Gomez F, Houshyar H, et al Chronic stress and obesity: a new view of «comfort food». Proc Natl Acad Sci U S A. 2003 Sep 30;100(20):11696–701. http://dx.doi.org/10.1073/pnas.1934666100.

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