Job Strain and Ambulatory Blood Pressure: A Meta-Analysis and Systematic Review

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We reviewed evidence of the relationship between job strain and ambulatory blood pressure (ABP) in 29 studies (1985–2012). We conducted a quantitative meta-analysis on 22 cross-sectional studies of a single exposure to job strain. We systematically reviewed 1 case–control study, 3 studies of cumulative exposure to job strain, and 3 longitudinal studies.

Single exposure to job strain in cross-sectional studies was associated with higher work systolic and diastolic ABP. Associations were stronger in men than women and in studies of broad-based populations than those with limited occupational variance. Biases toward the null were common, suggesting that our summary results underestimated the true association.

Job strain is a risk factor for blood pressure elevation. Workplace surveillance programs are needed to assess the prevalence of job strain and high ABP and to facilitate workplace cardiovascular risk reduction interventions. (*Am J Public Health*. 2013;103:e61–e71. doi:10.2105/AJPH.2012.301153)

HYPERTENSION IS PRIMARILY a disease of industrial societies, with a very low prevalence among hunter-gatherers, herders, and traditional agricultural communities.[1,2] In industrial societies, evidence has accumulated on the key role of working conditions in blood pressure (BP) elevation. For example, mean systolic ambulatory BP (ABP) is higher by approximately 4 millimeters of mercury (mm Hg) during work than outside of work,[3,4] and mean 24-hour ABP is lower on nonwork days than on workdays.[5,6] Associations with BP elevation have also been observed for work stressors such as long work hours,[7,8] effort–reward imbalance at work,[9] and threat-avoidant vigilant work, primarily professional driving.[10,11]

The most widely studied work stressor is job strain (the combination of high psychological job demands and low job control).[12] Since 1985, numerous studies have reported a positive association between job strain and ABP or hypertension defined by level of ABP. These studies have used a range of populations and study designs of varying quality, making comparisons difficult. Several reviews of the association of job stressors and BP have been published,[13–16] but no one has conducted a quantitative meta-analysis, which allows for the estimation of an overall effect size. Although the conclusions of these reviews varied from support for[14–16] to skepticism toward[13] the hypothesis, all included measures of job stressors other than job strain, and 2 included measures of chronic stress outside the workplace.[14,15] Thus, none adequately evaluated the job strain–ABP association. Therefore, we conducted a quantitative meta-analysis and qualitative systematic review of studies of 1 primary work exposure, job strain, and ABP, as well as a systematic assessment of study validity.

The previous reviews[13–16] included studies of casual clinic BP (CCBP), as well as ABP, as the outcome. Studies of work stressors and CCBP have not shown significant associations, except in a few larger studies.[17,18] CCBP measurements "often provide a poor estimate of risk in an individual patient for reasons such as observer error, the 'white-coat' effect . . . the inherent variability of blood pressure,"[19(p2368)] and unreliability.[20] On the other hand, ABP monitoring captures dynamic BP fluctuations in relation to daily life, including physical and psychosocial stimuli at work. ABP is also a much better predictor than CCBP of target organ damage[21,22] and incident cardiovascular disease.[23,24] We therefore focused on studies that used ABP as the outcome of interest.

METHODS

We followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines in reporting the methods and results of the studies in our systematic review and meta-analysis.[25]

A study met eligibility criteria if it

1. assessed or imputed exposure to job strain via its 2 major dimensions, job psychological demands and job decision latitude, an operationalization of the concept of job control;
2. used ABP as the dependent variable (during work hours, leisure time or evening, sleep, or 24 hours) or hypertension status (if measured by ABP);
3. had a case–control, cross-sectional, or cohort design;
4. was empirical; and
5. was published in English as a full-length article in a peer-reviewed journal.

We included more than 1 publication by a single author or group if the study population, endpoint, or design differed. If 2 or more studies by the same author(s) offered complementary information but had the same design, endpoint, and study population, we combined them and analyzed them together.

Information Sources, Search Strategy, and Study Selection

We conducted an online search of the PubMed and Cambridge Scientific Abstracts PsycINFO databases with dates ranging from "earliest" to April 2012. We entered the search terms as text words in the title, abstract, keywords, or other searchable fields (e.g., MeSH terms). For the independent variable, the search terms were "job strain," "iso-strain," "psychological demands," "job demands," "workload demands," "job control," "work control," "decision latitude," "skill discretion," "decision authority,"

and "intellectual discretion." We combined these terms with 2 search terms for the dependent variable: "hypertension" and "blood pressure." We made English language a limit. A senior medical information specialist replicated this strategy. We also reviewed bibliographies of relevant articles and personal files.

We examined articles meeting eligibility criteria to determine whether more than 1 article referred to the same study group, study design, and endpoint. We combined multiple articles from the same study for assessment of validity and bias and abstraction of quantitative study results. We incorporated all eligible articles with sufficient detail for studying effect sizes in the systematic review. We abstracted all eligible articles for a quantitative meta-analysis if we found sufficient studies with a particular study design and, in the case of prospective studies, if exposure change measures and follow-up periods were similar. We separated cross-sectional studies into those with a single measure of job strain exposure and those that assessed cumulative exposure to job strain at 2 or more time points. Where studies reported cross-sectional and longitudinal results, we reviewed both types of results.

Data Collection Process and Items

At least 2 of 3 authors (P. A. L., M. D., P. S.) independently reviewed eligible studies for internal validity and bias. If we did not agree on any criterion, we rereviewed the study and the criterion until we reached consensus. We abstracted relevant study design, sample, exposure measurement, and quantitative study results into Excel spreadsheets (Microsoft Corp, Redmond, WA) and Comprehensive Meta-Analysis version 2 database software (Biostat, Englewood, NJ). If sufficient detail for studying effect sizes (e.g., adjusted group means, group sample size, and SDs) was not available in the published article, we contacted the authors by e-mail and requested additional details.

We abstracted from the published articles or requested from study authors the following data: study design, study country, occupations and industries of sample, range of variation of sample (population, white collar, blue collar, or single occupation), number of men and women in the sample, type (self-report, job exposure matrix) and number of exposure measurements, type of BP outcome (work, evening, sleep, 24 hours), exposure contrasts (job strain group vs all other participants or job strain group vs low-strain group), and quantitative study results (e.g., adjusted group means, group sample size, and SDs). In addition, we abstracted data on 15 internal validity criteria and 17 potential biases.

Risk of Bias in Individual Studies

We derived the 15 internal validity criteria from Stock[26]; they addressed issues of bias, confounding, effect modification, range of variation, and exposure and outcome assessment. Complete criteria are listed in Table A (available as a supplement to this article at http://www.ajph.org). The first category, assembly of the sample, contains 3 criteria related to avoidance of selection bias, avoidance of nonresponse bias, and application of appropriate exclusion criteria. We evaluated validity of exposure variable assessment by 5 criteria related to assessment of point exposure to high psychological demands and to low control, avoidance of recall bias, analysis of job strain, adequate range of variation, and assessment of temporal aspects of exposure. The 4 criteria in the category for confounding and effect modification were adjustment for relevant demographic confounders, adjustment for relevant biomedical and behavioral confounders, appropriate consideration of gender as an effect modifier, and assessment of other dimensions of the work environment. The 3 validity criteria for the outcome variable were related to the assessment of the endpoint itself, whether assessment of outcome was blinded with respect to exposure status, and adequate range of variation of the outcome variable. We scored each criterion on a range of zero to 3 or 4 points. In subgroup analyses, we compared studies with validity scores above and below the median value.

We also assessed the overall direction in which methodological issues (e.g., bias, confounding, study design) were likely to affect associations between job strain and ABP, as follows: (1) a bias toward the null value or (2) bias possible in both directions. Complete criteria for this judgment are in Table B (available as a supplement to this article at http://www.ajph.org). Complete scores on all internal validity and bias criteria for all eligible studies are available upon request from the authors.

Synthesis, Risk of Bias, and Additional Analyses

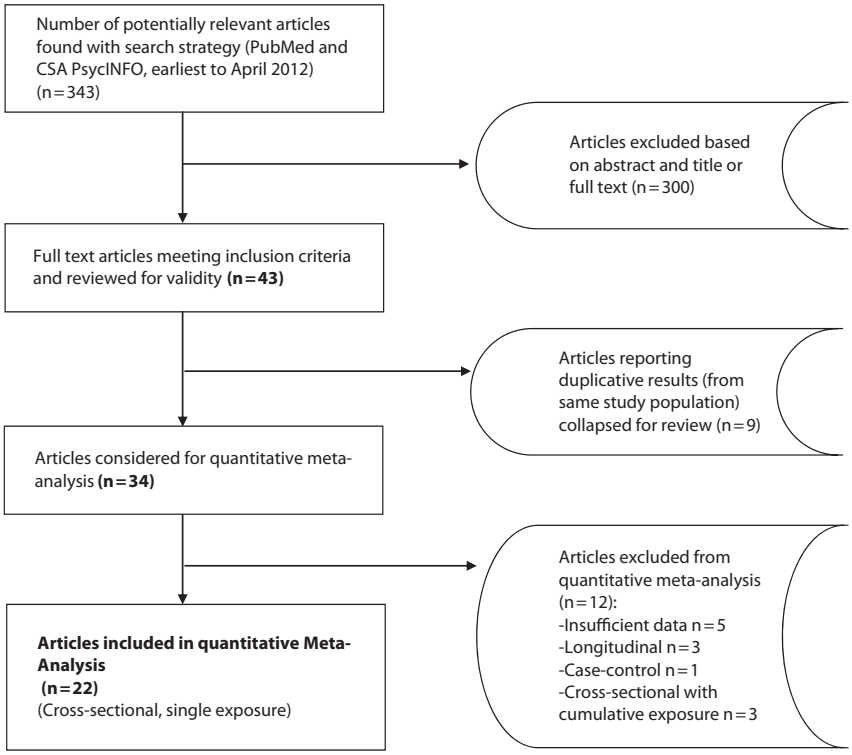
We used Comprehensive Meta-Analysis 2.0 to conduct our quantitative meta-analysis. Because of the variety of populations studied and methods used, we did not assume that the true effect size was identical in all studies. Thus, a random-effects model was appropriate. The principal summary measure was the adjusted job strain group mean difference in ABP. We computed adjusted group mean differences for each of 8 ABP outcomes when available (systolic and diastolic work, home, sleep, and 24-hour ABP). We assessed heterogeneity[27] by the P value of the fixed-effect analysis overall Q value and by the corresponding I^2.

We assessed potential publication bias by Kendall's τ (Begg and Mazumdar's rank correlation test), Egger's test of the intercept, and Duval and Tweedie's trim-and-fill procedure.[28]

We conducted prespecified, subgroup analyses comparing male and female samples, exposure contrasts (job strain group vs all other participants compared with job strain group vs low-strain group), population-based compared with white-collar or single-occupation studies, self-report of exposure compared with imputation (use of a job exposure matrix), validity score above the median compared with validity score below the median, and study biases all toward the null value compared with study biases possible in both directions. We assessed potential effect modification by the P value of the between-studies mixed-effects analysis Q value.

RESULTS

Our search identified 343 studies, and 43 articles met our inclusion criteria (Figure 1). We considered 9 articles to be duplicative; these had the same author(s), design, study group, and endpoint, and we combined them for the validity and bias review and data abstraction. Of the 34 remaining studies, we excluded 5 from the quantitative



Note. CSA = Cambridge Scientific Abstracts.

FIGURE 1—Flowchart of included and excluded studies of job strain and ambulatory blood pressure.

meta-analysis after contacting authors because data were not reported in sufficient detail for studying effect sizes and no further analyses were possible because authors no longer had access to the data.

Because we had only 1 case–control study, a quantitative meta-analysis of studies of this design was not possible. We excluded 3 cross-sectional studies from the quantitative meta-analysis because they assessed cumulative exposure to job strain over 2 time points (rather than a single exposure to job strain), and these 3 studies also varied in time between exposure assessments. We excluded 3 longitudinal studies from the quantitative meta-analysis because they differed in how they assessed exposure change and in follow-up periods. Instead, we assessed the case–control, prospective, and cumulative exposure studies qualitatively. Therefore, 22 cross-sectional studies of a single exposure to job strain were available for the quantitative meta-analysis. Characteristics of the 34 eligible studies are summarized in Table 1, including the summary internal validity score and directionality of biases.

Cross-Sectional Studies

The 22 cross-sectional studies provided 28 samples for a quantitative meta-analysis (5 studies reported results separately for men and women and 1 study reported results separately for normotensive and hypertensive participants). Twenty-two samples provided results for work ABP, 11 samples provided results for home and sleep ABP, and 9 samples provided results for 24-hour ABP. Results are presented in Table 2 and Figure 2. Single exposure to job strain was associated with work systolic (3.43 mm Hg; 95% confidence interval [CI] = 2.02, 4.84; $P < .001$; $I^2 = 62.3$) and diastolic (2.07 mm Hg; 95% CI = 1.17, 2.97; $P < .001$; $I^2 = 42.3$) ABP. Significant associations were also observed between job strain and home and sleep ABP but not 24-hour ABP. We found substantial heterogeneity between studies (with the exception of home ABP), supporting the use of a random-effects model.

Significant associations were observed between job strain and work, home, and sleep ABP among men, but the only significant association among women was job strain and work systolic ABP (Table 3). In this relatively small number of studies, none of the interaction terms for job strain by gender were statistically significant. As hypothesized, weaker (and mostly nonsignificant) associations were observed in the 3 single-occupation samples and the 4 samples of white-collar workers than in the 15 population-based samples. The job strain by occupational variance interaction was statistically significant for work systolic and diastolic ABP. In population-based studies, the association between job strain and work systolic ABP was 4.46 mm Hg (95% CI = 2.61, 6.30; $P < .001$).

Contrary to our expectations, we observed no consistent differences between studies that used a larger exposure contrast (comparing workers in high-strain vs low-strain jobs), and studies with a smaller exposure contrast (comparing workers in high-strain vs other jobs). In addition, contrary to our hypothesis, the 2 studies that used a job exposure matrix (imputation) to determine exposure did not have weaker associations between job strain and ABP than studies that used self-report measures of job strain; rather, they tended to have stronger associations. The 1 significant job strain by exposure measurement interaction (for sleep diastolic ABP) suggested a stronger association in the imputation (vs self-report) studies.

Contrary to our expectations, we found no consistent differences (and no significant interactions) between studies whose biases were all toward the null hypothesis and those that contained biases both toward and away from the null hypothesis, or between studies with high versus low validity scores.

We found little evidence of publication bias (smaller studies with larger effect sizes being more likely to be published) for studies

TABLE 1—Studies Eligible for Quantitative Meta-Analysis of Research on Job Strain and Ambulatory Blood Pressure

Study	Exposure Measurements	Country	Sample	Sample Type	Men, No.	Women, No.	Job Strain Measure	Validity Score[a]	Direction of Bias
Included studies[b]									
Blumenthal et al.[29]	Single	US	Patients with mild hypertension	Population	61	38	Self-report	31	Null
Brown et al.[30,31]	Single	US	Nurses	Single occupation	. . .	59	Self-report	31	Null
Cesana et al.[32]	Single	Italy	Community residents	Population	527	. . .	Self-report	38	Null
Clays et al.[33,34]	Single	Belgium	Employees of 9 public- and private-sector firms	Population	109	69	Self-report	36	Null
Fan et al.[35]	Single	US	Patients with mild hypertension	Population	72	50	Self-report	34	Both directions
Fauvel et al.[36]	Single	France	Chemical company employees	Population	235	68	Self-report	32	Null
Laflamme et al.[37]; Brisson et al.[38]	Single	Canada	Employees of 8 firms	White-collar	. . .	210	Self-report	38	Null
Light et al.[39]	Single	US	Community residents	Population	65	64	Self-report	36	Null
Maina et al.[40]	Single	Italy	Call center operators	Single occupation	26	74	Self-report	32	Both directions
Menni et al.[41]	Single	Italy	Community residents	Population	. . .	351	Self-report	30	Both directions
Melamed et al.[42]	Single	Israel	Industrial workers	Population	79	. . .	Self-report	35	Null
Rau et al.[43]	Single	Sweden	Community residents	Population	170	. . .	Imputation	34	Null
Rau and Triemer[44]	Single	Germany	Employees of 4 public- and private-sector firms	White-collar	111	77	Expert	37	Null
Landsbergis et al.[45–48]	Single	US	Employees of 8 public- and private-sector firms	Population	264	. . .	Self-report	38.25	Both directions
Steptoe et al.[49,50]	Single	UK	Primary and high school teachers	Single occupation	60	102	Self-report	37	Both directions
Steptoe and Willemsen[51]	Single	UK	Government employees	White-collar	106	92	Self-report	30	Both directions
Theorell et al.[52]	Single	Sweden	Community residents	Population	161	. . .	Imputation	36	Null
Theorell et al.[53]	Single	Sweden	Nurses and nurse's aides	Population	. . .	56	Self-report	33	Both directions
Theorell et al.[54]; Knox et al.[55]	Single	Sweden	Military draftees	Population	71	. . .	Self-report	27.5	Null
Tobe et al.[56,57]	Single	Canada	Hospital employees and community residents	Population	113	135	Self-report	28	Both directions
Trudel et al.[58]	Single	Canada	Employees of 3 public insurance institutions	White-collar	910	1447	Self-report	34	Both directions
van Egeren[59]	Single	US	University employees	Population	17	20	Self-report	31	Null
Fauvel et al.[60]	Cumulative	France	Chemical workers	Population	278	25	Self-report	35	Null
Riese et al.[61]	Cumulative	Netherlands	Nurses	Single occupation	. . .	159	Self-report	37	Null
Schnall et al.[62]	Cumulative	US	Employees of 8 public- and private-sector firms	Population	195	. . .	Self-report	40	Null
Excluded studies									
Data unavailable									
Bishop et al.[63]	Single	Singapore	Police officers	Single occupation	118	. . .	Self-report	29	Null
Gallo et al.[64]	Single	US	Community residents	Population	. . .	108	Self-report	36	Both directions
Goldstein et al.[65]	Single	US	Nurses	Single occupation	. . .	138	Self-report	32	Null
Steptoe et al.[66]	Single	UK	Firefighter recruits	Single occupation	49	. . .	Self-report	35	Null
O'Connor et al.[67]	Single	UK	Physicians	Single occupation	17	10	Self-report	27	Both directions
Longitudinal design									
Schnall et al.[62]	Repeated	US	Employees of 8 public- and private-sector firms	Population	195	. . .	Self-report	40	Null
Theorell et al.[68]	Repeated	Sweden	Employees in 6 occupations	Population	40	18	Self-report	32	Both directions
Tobe et al.[69]	Single	Canada	Community residents	Population	106	123	Self-report	32	Null
Case–control design: Schnall et al.[70]	Case Control	US	Employees of 8 public- and private-sector firms	Population	215	. . .	Self-report	36	Both directions

Note. Ellipses indicate no participants of relevant gender.
[a]Maximum score = 51.
[b]All cross-sectional studies.

TABLE 2—Random-Effects Model Associations of Job Strain and Ambulatory Blood Pressure: Meta-Analysis of 28 Samples From 22 Cross-Sectional Studies

Location and type of ABP	Samples, No.	mm Hg (95% CI)	*P*	I^2 (*P*)
Work SBP	22	3.43 (2.02, 4.84)	< .001	62.3 (< .001)
Work DBP	22	2.07 (1.17, 2.97)	< .001	42.3 (.02)
Home SBP	11	2.55 (1.21, 3.90)	< .001	0 (.52)
Home DBP	11	1.90 (0.89, 2.91)	< .001	0 (.62)
Sleep SBP	11	3.67 (1.43, 5.90)	.001	66.5 (.001)
Sleep DBP	11	2.06 (0.51, 3.60)	.009	49.1 (.03)
24-h SBP	9	1.34 (–0.15, 2.83)	.079	41.2 (.09)
24-h DBP	7	0.57 (–0.27, 1.40)	.19	44.9 (.09)

Note. ABP = ambulatory blood pressure; DBP = diastolic blood pressure; SBP = systolic blood pressure; CI = confidence interval. I^2 refers to the percentage of variation across studies that is attributable to heterogeneity rather than chance.

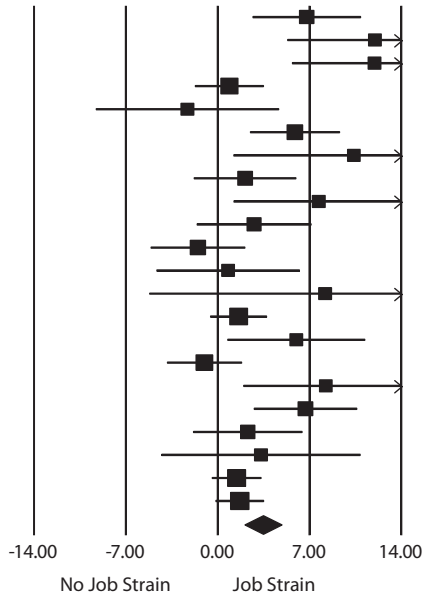
of work, home, and sleep ABP. Kendall's τ (Begg and Mazumdar's rank correlation test) and Egger's test of the intercept were statistically significant only for work systolic ABP. Duval and Tweedie's trim-and-fill procedure reduced the point estimate for work systolic ABP to 2.47 mm Hg (95% CI = 0.99, 3.96) and sleep systolic ABP to 2.65 mm Hg (95% CI = 0.39, 4.90). Among the 15 population-based studies, the point estimate for work systolic ABP was reduced to 3.1 mm Hg (95% CI = 1.3, 5.0). Thus, even after adjustment for potential publication bias, associations between job strain and work, home, and sleep ABP remained statistically significant and substantial.

In a study at 8 New York City worksites, men experiencing job strain both at baseline and at a 3-year follow-up had ABP of 11 mm Hg systolic and 7 mm Hg diastolic higher than men with no job strain at both times. Results were similar for work, home, and sleep ABP. Men whose exposure to job strain changed over time had intermediate levels of ABP.[62] However, in a 5-year follow-up study of employees of a French chemical company,[60] and in a 1-year follow-up study of Dutch nurses,[61] work and nonwork ABP were not significantly different for those experiencing job strain both at baseline and at follow-up, or for those whose exposure to job strain changed over time, than for participants with no job strain at both times.

Longitudinal Studies

In the analysis of men at 8 New York City worksites, those with job strain at baseline, but not at a 3-year follow-up, had a significant decrease in work ABP (–5.3 mm Hg systolic; –3.2 mm Hg diastolic) and home ABP (–4.7 mm Hg systolic; –3.3 mm Hg diastolic). Decreases were larger (–11.3/–5.8 mm Hg) for men with hypertension at baseline. However, those with no job strain at baseline but with job strain at the 3-year follow-up, or those with exposure at

Study	Outcome	Difference in means	Lower limit	Upper limit	*P*-Value
Schnall et al.[64]	Work systolic	6.800	2.642	10.958	.001
Van Egeren[59]	Work systolic	12.000	5.282	18.718	<.001
Rau and Triemer[44]	Work systolic	11.970	5.648	18.292	<.001
Laflamme et al.[37] Brisson et al.[38]	Work systolic	0.900	-1.758	3.558	.507
Brown et al.[30]	Work systolic	-2.300	-9.308	4.708	.52
Clays et al.[33]	Work systolic	5.900	2.445	9.355	.001
Melamed et al.[42]	Work systolic	10.400	1.183	19.617	.027
Cesana et al.[32]	Work systolic	2.100	-1.842	6.042	.296
Tobe et al.[56] (male)	Work systolic	7.718	1.185	14.252	.021
Tobe et al.[56] (female)	Work systolic	2.781	-1.624	7.186	.216
Steptoe et al.[49]	Work systolic	-1.500	-5.135	2.135	.419
Theorell et al.[54] (normotensive)	Work systolic	0.800	-4.695	6.295	.775
Theorell et al.[54] (hypertensivetensive)	Work systolic	8.200	-5.247	21.647	.232
Theorell et al.[52]	Work systolic	1.600	-0.596	3.796	.153
Light et al.[39] (male)	Work systolic	6.000	0.704	11.296	.026
Light et al.[39] (female)	Work systolic	-1.000	-3.889	1.889	.497
Rau et al.[43]	Work systolic	8.260	1.934	14.586	.01
Theorell et al.[53]	Work systolic	6.700	2.740	10.660	.001
Fauvel et al.[36]	Work systolic	2.300	-1.895	6.495	.283
Maina et al.[40]	Work systolic	3.313	-4.315	10.941	.395
Trudel et al.[58] (male)	Work systolic	1.450	-0.471	3.371	.139
Trudel et al.[58] (female)	Work systolic	1.690	-0.184	3.564	.077
Combined		3.433	2.024	4.842	<.001



Note. CI = confidence interval.

FIGURE 2—Forest plot of job strain and work systolic ambulatory blood pressure (mm Hg).

TABLE 3—Random-Effects Model Associations of Job Strain and Ambulatory Blood Pressure: Subgroup Analyses in Meta-Analysis of 28 Samples From 22 Cross-Sectional Studies

Exposure Group	All Studies			Interaction P^a
	Sample, No.	mm Hg (95% CI)	*P*	
Gender				
Men				
Work SBP	10	3.85 (1.94, 5.77)	< .001	.43
Work DBP	10	1.97 (0.74, 3.20)	.002	.87
Home SBP	4	3.40 (0.51, 6.28)	.02	.2
Home DBP	4	2.75 (0.83, 4.67)	.005	.24
Sleep SBP	4	5.30 (2.32, 8.29)	< .001	.1
Sleep DBP	4	3.06 (1.39, 4.74)	< .001	.13
24-h SBP	3	2.49 (−2.93, 7.91)	.37	.51
24-h DBP	2	1.98 (−2.33, 6.28)	.37	.46
Women				
Work SBP	7	2.58 (0.05, 5.11)	.05	
Work DBP	7	1.78 (−0.18, 3.74)	.08	
Home SBP	2	0.90 (−1.65, 3.44)	.49	
Home DBP	2	−0.54 (−5.71, 4.64)	.84	
Sleep SBP	4	1.20 (−2.61, 5.01)	.54	
Sleep DBP	4	0.44 (−2.55, 3.43)	.77	
24-h SBP	4	0.59 (−1.16, 2.34)	.51	
24-h DBP	3	0.28 (−0.92, 1.49)	.64	
Exposure group contrast				
High strain vs low strain				
Work SBP	8	3.43 (1.46, 5.39)	.001	.99
Work DBP	8	2.50 (0.95, 4.04)	.002	.55
Home SBP	2	1.84 (−2.12, 5.79)	.36	.69
Home DBP	2	4.28 (1.13, 7.43)	.01	.12
Sleep SBP	3	4.07 (−0.44, 8.57)	.08	.86
Sleep DBP	3	4.96 (2.34, 7.58)	< .001	.02
24-h SBP	2	0.14 (−1.92, 2.20)	.9	.19
24-h DBP	2	−0.12 (−0.67, 0.42)	.66	.02
High strain vs other				
Work SBP	14	3.44 (1.34, 5.54)	.001	
Work DBP	14	1.91 (0.80, 3.03)	.001	
Home SBP	9	2.69 (1.13, 4.30)	.001	
Home DBP	9	1.63 (0.56, 2.69)	.003	
Sleep SBP	8	3.60 (0.76, 6.44)	.01	
Sleep DBP	8	1.35 (−0.19, 2.88)	.09	
24-h SBP	7	1.97 (0.12, 3.81)	.04	
24-h DBP	5	1.41 (0.25, 2.56)	.02	
Occupational variance				
Population-based studies				
Work SBP	15	4.46 (2.61, 6.30)	< .001	.01
Work DBP	15	2.75 (1.61, 3.89)	< .001	.03
Home SBP	7	3.47 (1.75, 5.19)	< .001	.23
Home DBP	7	2.44 (1.09, 3.79)	< .001	.49
Sleep SBP	7	4.21 (1.47, 6.94)	.003	.65

Continued

both time points, exhibited very little change in ABP.[62]

In a study of workers from 6 different occupations in Stockholm, which assessed job strain and ABP 4 times over the course of 1 year, work systolic ABP was 4 mm Hg higher during the assessment with the highest than with the lowest reported job strain. Similar results were observed in the subsample of men in this study (results were not reported separately for women).[68] A study of Toronto-area employees followed for 1 year, which assessed job strain exposure only at baseline, observed no change in ABP in women. However, the increase in 24-hour ABP in men approached statistical significance (+4.1 mm Hg systolic ABP; $P = .1$; +2.8 mm Hg diastolic ABP; $P = .11$).[69]

DISCUSSION

A quantitative meta-analysis confirmed our hypothesis that job strain is associated with ABP when BP is measured during work, home, and sleep hours. These associations are not likely explained by confounding because most of the studies reviewed controlled for the major potential risk factors for hypertension, such as age, body mass index, race, work physical activity, and alcohol use. Similarly, bias is a highly unlikely explanation for these findings because in 13 of the 22 studies in our quantitative meta-analysis, all study biases identified were in the direction of the null hypothesis. This finding of substantial bias toward the null suggests that our quantitative meta-analysis underestimated true effect sizes. Restriction of range in blood pressure values occurred in all but 3 studies,[49,53,54] also likely leading to underestimates of true effect sizes.[71] In only 1 cohort

TABLE 3—*Continued*

Sleep DBP	7	2.37 (0.31, 4.43)	.02	.37
24-h SBP	7	1.82 (−0.44, 4.08)	.11	.53
24-h DBP	5	0.64 (−0.50, 1.78)	.27	.86
White-collar studies				
Work SBP	4	2.52 (0.06, 4.98)	.04	
Work DBP	4	1.48 (0.01, 2.94)	.05	
Home SBP	1	1.00 (−1.72, 3.72)	.47	
Home DBP	1	1.60 (−0.47, 3.67)	.13	
Sleep SBP	2	4.33 (−5.07, 13.73)	.37	
Sleep DBP	2	2.94 (−1.81, 7.68)	.23	
24-h SBP	2	0.89 (−0.94, 2.72)	.34	
24-h DBP	2	0.79 (−0.15, 1.57)	.11	
Single-occupation studies				
Work SBP	3	−0.91 (−3.89, 2.06)	.55	
Work DBP	3	−0.96 (−3.53, 1.91)	.46	
Home SBP	3	1.09 (−2.63, 4.81)	.57	
Home DBP	3	0.23 (−3.45, 3.91)	.9	
Sleep SBP	2	1.42 (−3.85, 6.69)	.6	
Sleep DBP	2	−1.00 (−5.47, 3.47)	.66	
24-h SBP	0			
24-h DBP	0			
Exposure measurement				
Self-report				
Work SBP	19	3.05 (1.60, 4.50)	< .001	.72
Work DBP	19	1.71 (0.87, 2.56)	< .001	.24
Home SBP	10	2.63 (1.22, 4.04)	< .001	.73
Home DBP	10	1.64 (0.59, 2.70)	.002	.1
Sleep SBP	8	2.69 (0.19, 5.18)	.04	.46
Sleep DBP	8	1.14 (−0.34, 2.63)	.13	.02
24-h SBP	9	1.34 (−0.15, 2.83)	.08	
24-h DBP	7	0.57 (−0.27, 1.40)	.19	
Imputation				
Work SBP	2	4.24 (−2.14, 10.63)	.19	
Work DBP	2	3.95 (0.31, 7.59)	.03	
Home SBP	1	1.80 (−2.76, 6.36)	.44	
Home DBP	1	4.65 (1.19, 8.11)	.01	
Sleep SBP	2	5.29 (−1.20, 11.78)	.11	
Sleep DBP	2	4.88 (2.15, 7.61)	< .001	
24-h SBP	0			
24-h DBP	0			
Validity score				
High (≥ 35)				
Work SBP	10	3.39 (1.11, 5.67)	.004	.9
Work DBP	10	2.22 (0.80, 3.64)	.002	.81
Home SBP	5	2.79 (0.80, 4.79)	.006	.73
Home DBP	5	2.21 (1.08, 3.34)	< .001	.24
Sleep SBP	5	3.66 (0.86, 6.46)	.01	.98
Sleep DBP	5	3.06 (1.17, 4.95)	.002	.11

Continued

study[68] and 1 cross-sectional study[53] was there self-report of exposure and self-measurement of ABP. In others (e.g., Brisson et al.,[38] Trudel et al.,[58] Schnall et al.[62]), ABP assessors were not aware of the job strain status of participants, and participants were not aware of their ABP when completing study questionnaires on job demands and job control, thus precluding information bias. In only 2 other studies was it unclear whether ABP values were known to participants prior to exposure assessment.[51,56] Previous research also provides evidence of the reliability and validity of the measurement of job demands and job control with the Job Content Questionnaire, particularly for job control.[47,70,72–74]

Only 2 studies used a job exposure matrix (imputation), a more objective measure of exposure than self-report, to assess job demands and job control. Although this method has inherent measurement error and a bias toward the null, associations between job strain and ABP derived with this method were not weaker than results from studies that used self-reports of exposure. Thus, use of job exposure matrices when feasible, in addition to self-report questionnaires, is recommended for future studies. Finally, study validity scores derived from 15 criteria, which ranged from 27.5 to 40, were not related to strength of association between job strain and ABP. This finding suggests that, above a certain threshold, study validity was adequate for these published studies. Some subgroup analyses were based on fewer than 5 studies and, thus, should be interpreted with caution.[75]

Results from the 3 studies of cumulative exposure to job strain were not consistent. However, the study of Dutch nurses (with null

TABLE 3—*Continued*

24-h SBP	2	0.98 (−0.87, 2.82)	.3	.6
24-h DBP	2	0.37 (−0.39, 1.12)	.34	.41
Low (≤ 34)				
Work SBP	12	3.57 (1.72, 5.42)	< .001	
Work DBP	12	1.99 (0.77, 3.21)	.001	
Home SBP	6	2.25 (−0.14, 4.65)	.07	
Home DBP	6	0.71 (−1.52, 2.93)	.53	
Sleep SBP	6	3.61 (−0.48, 7.69)	.08	
Sleep DBP	6	0.59 (−1.77, 2.95)	.62	
24-h SBP	6	2.58 (0.29, 4.87)	.03	
24-h DBP	4	1.82 (0.05, 3.58)	.04	
Direction of study biases				
All toward the null				
Work SBP	14	3.83 (1.75, 5.92)	< .001	.59
Work DBP	14	2.33 (0.96, 3.69)	.001	.51
Home SBP	8	2.18 (0.68, 3.69)	.01	.42
Home DBP	8	1.78 (0.42, 3.14)	.01	.8
Sleep SBP	7	4.16 (1.36, 6.95)	.004	.62
Sleep DBP	7	2.98 (0.98, 4.99)	< .001	.14
24-h SBP	5	1.02 (−0.55, 2.60)	.2	.47
24-h DBP	3	0.67 (−0.38, 1.71)	.21	.91
Toward or away from the null				
Work SBP	8	3.04 (1.06, 5.02)	.003	
Work DBP	8	1.73 (0.55, 2.90)	.004	
Home SBP	3	3.81 (0.14, 7.48)	.04	
Home DBP	3	2.08 (0.31, 3.84)	.02	
Sleep SBP	4	2.81 (−1.73, 7.36)	.23	
Sleep DBP	4	0.68 (−1.61, 2.97)	.56	
24-h SBP	4	2.33 (−0.80, 5.46)	.15	
24-h DBP	4	0.78 (−0.87, 2.43)	.35	

Note. CI = confidence interval; DBP = diastolic blood pressure; SBP = systolic blood pressure.
[a]Interaction between exposure groups, *P* value of *Q*.

results) was a single-occupation study and thus was expected to produce weaker associations between job exposures and health outcomes.[61] The study of French chemical company workers (also with null results) incorporated both white-collar and blue-collar workers.[60] However, as a study of 1 employer, some restriction of the range of job characteristics (relative to population-based studies of multiple employers) may have biased results to some extent toward the null. Thus, we have greater confidence in the results of the New York City study of 8 employers, which found stronger associations when cumulative job strain exposure was assessed, than in analyses with only a single measurement of job strain exposure.[62] It is believed that chronic exposure to work stressors leads to increases in BP.[76] Thus, cumulative exposure measurements, as approximations of chronic exposure, are preferred.

The 3 longitudinal studies had different methods and follow-up periods; thus, it is difficult to draw conclusions about the magnitude of the impact of job strain on change in ABP over time. One study measured exposure only at baseline,[69] and another compared the period of highest exposure with the period of lowest exposure in whatever order they occurred.[68] The third study (the New York City study) measured both job strain and ABP at 2 periods 3 years apart.[62] However, all 3 studies indicated some association between job strain or job strain change and ABP change on the order of 4 to 5 mm Hg systolic and 3 mm Hg diastolic. In addition, case-control results from the New York City study showed that job strain increased the risk of hypertension and was associated with ABP.[70]

Two major neuroendocrine systems are central to the stress response: the sympathoadrenal medullary system and the hypothalamic–pituitary–adrenal cortical system. Under demanding conditions where organisms can exert control, epinephrine levels increase and cortisol levels may decline.[77] However, in demanding but low-control situations (analogous to job strain), both epinephrine and cortisol are elevated.[78] Cortisol enhances and prolongs the effect of epinephrine,[79] and the combination of these hormones appears to promote BP elevation.[78] For example, a high-effort–low-control laboratory task elicited a rise in catecholamines, cortisol, and BP.[80]

Two reviewed studies provided evidence of dose–response relationships between job strain and ABP. In the New York City study, with 21% of the sample defined as high job strain, the effect of job strain was 6.7 mm Hg systolic ABP and 2.7 mm Hg diastolic ABP. However, a more stringent definition of job strain, classifying 8% of a sample as high job strain, was associated with 11.5 mm Hg systolic ABP and 4.1 mm Hg diastolic ABP.[46] In a Belgian study, systolic and diastolic ABP increased across 4 job strain groups (from low demand–high control, to high demand–high control, to low demand–low control to high demand–low control) and across increasing levels of job demands and decreasing levels of job control.[33] However, inconsistent dose–response relationships were seen in 2 other population-based studies.[32,52] An alternative method of computing job strain, job demands divided by job decision latitude, which creates a continuous measure of exposure, should be further explored in future research. In the New York

City study, such a measure enabled researchers to examine associations between continuous exposure and continuous ABP, as well as to dichotomize the continuous exposure measure at various cutpoints to assess possible thresholds of effect.[46]

Future Research

Despite the evidence from cross-sectional studies that job strain is a risk factor for BP increase, only 3 longitudinal studies of this association have been conducted. Clearly, more longitudinal (including intervention) research is needed to assess the impact of repeated exposure and change in exposure to job strain.

Further research is also needed on job strain and other occupational predictors of related risk factors for cardiovascular disease, such as blunted (< 10%) nighttime BP dipping[34,35] and masked hypertension (i.e., elevated daytime or 24-hour blood pressure but normal CCBP).[58,76,81,82] In addition, further longitudinal research is needed to test our hypothesis that work stressors such as job strain lead directly to increases in ABP, but are only later reflected in increases in CCBP.[76]

Modifiers of the effect of job strain on ABP need to be further explored. Several studies have observed a stronger association of job strain and ABP in older workers,[33,83] workers in lower socioeconomic positions,[47,64] and workers with family responsibilities[38] or low marital cohesion.[69] Although 1 study found stronger associations among white-collar women with higher education,[37,38] white-collar women without higher education who had previously been exposed to job strain changed jobs at a high rate and were significantly more likely than others to have been excluded from the study, creating a strong bias toward the null in the less educated group. In this study, ABP during waking hours was highest in university-educated women in the group with both job strain and high child and domestic responsibilities. No such interaction was seen, however, among the women without a university degree. In light of the small number of studies of job strain and ABP among women, further research is clearly required, including assessment of nonwork stressors in women.

Further research is needed on the association of ABP and other work stressors, such as long work hours,[7,8] effort–reward imbalance at work,[9] threat-avoidant vigilant work (primarily professional driving),[10,11] organizational injustice,[84] and work–family imbalance.[38] Recent research has implicated work stressors in development of obesity,[85–88] a risk factor for hypertension. Thus, further research is needed on the relationship between work stressors, obesity, hypertension, and cardiovascular disease. Finally, in light of our exclusion of 5 studies because data were not reported in published articles in sufficient detail for studying effect sizes, as well as our need to contact some authors of included studies to obtain data necessary for a meta-analysis, we strongly encourage future researchers on the association between work stressors and ABP or other health outcomes to clearly provide data (e.g., effect sizes, sample sizes, SDs in exposed and unexposed groups, stratification by gender) that will permit meta-analyses to be conducted.

Conclusions and Implications

The development of hypertension and cardiovascular disease as global epidemics has occurred in conjunction with urbanization and industrialization and, more recently, economic globalization.[1,2,89,90] Our meta-analysis and systematic review found that job strain, a common feature of industrial workplaces, plays an important role in BP elevation. The association between job strain and ABP, as well as the possible role of other psychosocial workplace stressors (e.g., effort–reward imbalance and threat-avoidant vigilance) in elevating ABP, may help to explain the previously observed association between job strain and cardiovascular disease,[91,92] providing a partial explanation for the causation of these global epidemics. Because the prevalence of job strain appears to be increasing,[93] job strain may also be a potential contributor to the increasing prevalence of hypertension worldwide.[90]

In the face of the high prevalence of hypertension and emerging insights concerning the relationship of work to hypertension, individual clinicians could become overwhelmed if they attempt to prevent work-related hypertension by themselves. In addition to an active role for clinicians, a public health approach is needed, in which BP and workplace risk factors for BP elevation are systematically evaluated on a large scale by appropriately trained health workers. Occupations with a high prevalence of hypertension (e.g., professional drivers,[10] police,[94] firefighters[95]) could be initially targeted for further evaluation. Clinicians can play an active public health role in this process by identifying clusters of work-related hypertension as potential occupational sentinel health events.[96]

Interventions to reduce the prevalence of job strain can be carried out at many levels: the individual, the job, the occupation, the organization, and from outside the organization through laws and regulations.[97,98] Workplace interventions to increase job control (and therefore to reduce job strain), either through organizational-level[99] or task-restructuring[100] interventions, have shown health benefits. Therefore, an important step in the primary prevention of hypertension is the large-scale assessment, control, and prevention of job strain and possibly other psychosocial job stressors. ■

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P. A. Landsbergis, M. Dobson, and P. Schnall originated and designed the research, acquired the data, drafted the article, and made critical revisions. P. A. Landsbergis, M. Dobson, and G. Koutsouras analyzed and interpreted the data. P. Schnall was responsible for funding and supervision.

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Institutional review board approval was not needed for this systematic review and meta-analysis because no participants were enrolled.

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