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Job Strain and Family History of Hypertension as Significant Risks for Masked Hypertension at Workplace Among Japanese White-color Male Employees

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Abstract

There are still many unknown reasons for masked hypertension (HT) at workplace. 260 white-color male employees with normotension at annual physical examination were studied. 52 subjects had masked HT at their workplaces. After adjustment for cardiovascular risk factors and job stressors, the odds ratios of family history of HT and high job strain for masked HT at workplace were 3.05 (95% confidence interval (CI), 1.45-6.40, p=0.003), and 3.67 (95% CI, 1.05-12.83, p=0.042), respectively. Moreover, the combination of family history of HT and high job strain was closely associated with masked HT at workplace.

要旨

職場での仮面高血圧の要因は不明な点が多い。正常血圧で年次健康診断を受けている 260 名のホワイトカラー男性労働者を対象に検討を行った。52 名が職場での仮面高血圧者に該当した。心血管危険因子および仕事上のストレス因子を検討した結果，職場での仮面高血圧の要因として、高血圧家族歴と仕事上の高度ストレスが有意な因子であり，それぞれのオッズ比は 3. 05, 3. 67 であった。さらにこの 2 要因が複合した場合はさらなる関連を認めた。

Keywords

Masked hypertension (仮面高血圧)　Male employees (男性労働者)　Stress (ストレス)

Family history of hypertension (高血圧家族歴)

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1. Introduction

Hypertension (HT) is an important risk factor for cardiovascular disease, a significant cause of morbidity and mortality worldwide (Lewington, S, et al. 2002; Ezzati, M, et al. 2002; van den Hoogen, P.C, et al. 2000; Lim, S.S, et al. 2012). HT has been classified as sustained HT, white coat HT, or masked HT according to clinic and ambulatory blood pressure (Pickering, T.G, et al. 2007). Masked HT was the phenomenon as the variability of blood pressure and defined as a normal blood pressure (BP) in the clinic or office, but an elevated BP out of the clinic (Pickering, T.G, et al. 2007). Masked HT and sustained HT were at equivalent risk for the occurrence of cardiovascular diseases (Verberk, W.J, et al. 2007; Cuspidi, C. and Parati, G. 2007; Mancia, G, et al. 2006). Masked HT at workplace may be clarified as one type of masked HT and was associated with the increase of left ventricular mass index and the damage of target organs such as heart and brain by changes of the magnitude of cardiac and cerebral perfusions (Schnall, P.L, et al. 1990; Mancia, G. and Parati, G. 2000; Eguchi, K, et al. 2003). The worksite mental reactions were associated with masked HT at workplace have been closely examined in previous studies (Fauvel, J.P, et al. 2003; Riese, H, et al. 2004; Gilbert-Ouimet, M, et al. 2012; Guimont, C, et al. 2006; Markovitz, J.H, et al. 2004; Chandola, T, et al. 2006), however, the cause of masked HT at workplace has not been fully examined in Japanese populations from both physiological and mental risks factors.

The aim of the present study was to elucidate the association between masked HT at workplace and the risks such as anthropometric conditions and job strain in Japanese white-color male employees.

2. Methods

2-1 Participants

This is a cross-sectional occupational-based study that took place in Sapporo, Hokkaido prefecture, Japan. We contacted a total of 293 computer service male employees, aged between 34 and 60 years, who were to undergo their annual health examination in 2018. The BP values of all study participants were obtained at annual physical examination. 33 hypertensive participants (systolic BP ≥140 mmHg and/or diastolic BP ≥90 mmHg at annual physical examination) were excluded and the remaining 260 male participants were enrolled in the present study. Written informed consent was obtained from all participants before participation in the study.

2-2 Baseline physical examination

Smoking habit, alcohol intake, exercise habits (more or less than one per week), sleeping time (more or less than 6 hours), working hours, HT, diabetes mellitus, hyperlipidemia, conventional cardiovascular risk factors such as age, body mass index, systolic and diastolic BP, total cholesterol, triglyceride, high-density lipoprotein cholesterol, and fasting plasma glucose were assessed in annual physical examination in April 2015. Blood sample was obtained from antecubital vein and serum was separated. After precipitation by heparin- manganese, total cholesterol and high-density lipoprotein cholesterol were measured by the phosphotungstate method. Triglyceride was measured enzymatically. Body weight and height were measured in the morning in the fasting state and body mass index was calculated as body weight (kilograms) divided by squared height (meters squared). Subjects who had never smoked and ex-smokers were classified as “nonsmokers”. The mean BP was calculated with a standard

formula as follows: diastolic BP + 1/3 [systolic BP – diastolic BP] (Sainas, G, et al. 2016).

Job stress and social support

The Brief Job Stress Questionnaire (BJSQ) was used to evaluate two job stress dimensions (job demand and job control) and social support from supervisors and co-workers. The BJSQ has been widely used in japan for occupational health evaluation and research (Umehara, K, et al. 2007; Kawada, T. and Otsuka, T. 2011). Job demand was evaluated by 7 items as follows:

- You have to do an enormous amount of work
- You cannot complete all your work in the allotted time.
- You have to work very hard.
- Job control was evaluated by 3 items as follows:
- You can work at your own pace.
- You can decide the order in which you do your work.
- You can provide your opinions on the work strategy for your workplace.

The responses were scored by using a 4-point Likert-type response option (1-agree; 2-somewhat agree; 3-somewhat disagree, and 4-disagree). The total score for each question was used to evaluate job demand and job control, and higher score indicated higher job demand or control.

Social support from supervisors and co-workers was evaluated by 3 items as follows:

- You can often communicate with supervisors/ co-workers.
- You can strongly rely on supervisors/co-workers when you have problems.
- Your supervisors/co-workers are prepared to spend their time on dealing with your personal problems

The responses were scored by using a 4-point Likert-type response option (1-agree; 2-somewhat agree; 3-somewhat disagree, and 4-disagree). The total score for each question was used to evaluate social support, and higher scores indicated higher social support.

Job demand and job control scores were divided into two groups by dichotomizing at a median value, and job strain was categorized into three groups as follows:

- High job strain was defined as when job demand was high and job control was low.
- Medium job strain was defined as when job demand was high and job demand was high, or when job demand was low and job control was low.
- Low job strain was defined as when job demand was low and job control was high.

Cronbach's α coefficients for the BSJQ subscales of job demand, job control, social support from supervisors and social support from co-workers were 0.82, 0.75, 0.92 and 0.83, respectively.

2-3 BP measurement at workplace

The BP measurement at workplace was examined at scheduled break points during afternoon workday in a room located apart from the workplace. A well-trained nurse measured BP at the upper arm with mercury sphygmo-manometers in a sitting position after a few minutes of rest. Smoking was prohibited at least 1 hour before the BP measurement. HT was defined as systolic BP ≥140 mmHg and/or diastolic BP ≥90 mmHg, or both. BP measurement at workplace was performed twice (once per a month) in all study subjects and the average of two BP values was defined as the values of BP at workplace.

2-4 Statistical Analysis

Data were presented as means ± SD. Triglyceride was described as a median (and interquartile range) for variables with a skewed distribution. Smoking habit, alcohol intake,

exercise habits, sleeping time, HT, diabetes mellitus, and hyperlipidemia were described as a percentage. The differences of variables between two groups were examined by the Student unpaired t test for normal distributed variables, or by the Wilcoxon rank-sum test for triglyceride, and by chi-square test for the prevalence of study subjects. The logistic regression analysis was used to examine the relationship between risk factors and masked HT at workplace to assess the crude and adjusted odds ratio of masked HT at workplace. The principle model included candidate variables for age, body mass index, smoking habit, alcohol intake, exercise habits, sleeping time, mean BP, total cholesterol, triglyceride, high-density lipoprotein cholesterol, fasting plasma glucose, job strain, support from supervisor, support from co-workers, and working hours. A p value of less than 0.05 was considered to indicate statistical significance. All statistical analyses were performed using the SPSS statistical package for Windows version 21.0 (Chicago, IL, USA).

3. Results

Table 1

Characteristics of the study subjects at annual physical examination

	Total	Masked hypertension at workplace		P value
		Yes	No	
	(n=260)	(n=52)	(n=208)	
Age (years)	54 ± 5	55 ± 4	54 ± 5	0.213
Body mass index (kg/m^2)	23.1 ± 3.1	23.9 ± 2.9	22.9 ± 3.1	0.038
Smoking (n, %)	138 (53.1%)	27 (51.9%)	111 (53.4%)	0.878
Alcohol (n, %)	120 (46.2%)	28 (53.8%)	92 (44.2%)	0.219
Exercise (≥1/week) (n, %)	56 (21.5%)	13 (25.0%)	43 (20.7%)	0.572
Hypertension (n, %)	50 (10.8%)	16 (30.8%)	34 (16.3%)	0.617
Diabetes mellitus (n, %)	28 (19.2%)	4 (7.7%)	24 (11.5%)	0.029
Hyperlipidemia (n, %)	24 (9.2%)	8 (15.4%)	16 (7.7%)	0.106
Family history of hypertension (n, %)	106 (40.8%)	32 (61.5%)	74 (35.6%)	0.001
Sleep time (<6 hours/day) (n, %)	73 (28.1%)	16 (30.8%)	57 (27.4%)	0.610
Systolic blood pressure (mmHg)	119 ± 12	128 ± 9	116 ± 12	<0.001
Diastolic blood pressure (mmHg)	77 ± 8	82 ± 6	75 ± 8	<0.001
Mean blood pressure (mmHg)	91 ± 9	98 ± 7	89 ± 9	<0.001
Total cholesterol (mg/dL)	203 ± 34	209 ± 36	202 ± 32	0.180
Triglyceride (mg/dL)	106 (75 - 152)	119 (85 - 151)	100 (74 - 157)	0.296
HDL-cholesterol (mg/dL)	60 ± 16	61 ± 17	59 ± 16	0.482
Fasting plasma glucose (mg/dL)	102 ± 21	103 ± 16	102 ± 22	0.663

Values are presented as mean±SD, median (interquatile range) for skewed variables, or percentage.

HDL; high density lipoprotein, SD; standard deviation.

Table 1 shows the characteristics of the study subjects at annual physical examination. 52 (20.0%) subjects had masked HT at workplace among the total of 260 subjects. The subjects with masked HT at workplace had a higher prevalence of family history of HT. The body mass index and systolic, diastolic and mean BP values were significantly higher in subjects with masked HT at workplace than those without it.

Table 2

Job stress characteristics of the study subjects according to BJSQ

	Total	Hypertension incidence at workplace		P value
		Yes	No	
	(n=260)	(n=52)	(n=208)	
Demand	6.9 ± 2.1	7.0 ± 2.2	6.8 ± 2.1	0.300
Control	7.2 ± 1.9	7.6 ± 1.9	7.1 ± 1.9	0.120
Support from supervisor	7.8 ± 1.7	7.7 ± 1.7	7.8 ± 1.6	0.710
Support from co-workers	7.0 ± 1.7	7.0 ± 1.6	7.0 ± 1.6	0.969
Working hours	7.0 ± 1.6	7.1 ± 1.7	7.0 ± 1.6	0.789
Job strain				
low	94 (36.2%)	13 (25.0%)	81 (38.9%)	
medium	142 (54.6%)	30 (57.7%)	112 (53.8%)	
high	24 (9.2%)	9 (17.3%)	15 (7.2%)	0.031

BJSQ; Brief Job Stress Questionanaire.

Values are presented as mean±SD, median (interquatile range) for skewed variables, or percentage.

Table 2 shows the job stress characteristics of study subjects according to BJSQ. Subjects with masked HT at workplace were more likely to have higher demand and control scores. The support from supervisor, support from co-workers, and working hours did not differ in subjects between with and without masked HT at workplace.

Table 3 shows the crude and adjusted odds ratio of masked HT at workplace by risk factors. Smoking, family history of HT, and high job strain were significantly related to masked HT at workplace in crude analysis. Family history of HT and high job strain were significant and independent risks for masked HT at workplace in multivariate analysis. The adjusted odds ratio of family history of HT and was 3.05 (95% confidence interval (CI), 1.45-6.40, p=0.003). That of high job strain was 3.67 (95% CI, 1.05-12.83, p=0.042), compared to low job strain.

Table 3

Crude and adjusted odds ratio of masked hypertension at workplace by risk factors.

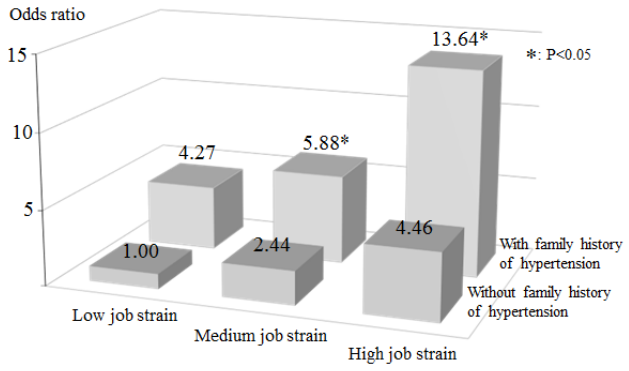
Variables	Crude	95% CI	P value	Adjusted OR	95% CI	P value
Body mass index	1.10	1.00 - 1.21	0.042	1.10	0.96 - 1.26	0.163
Smoking	0.94	0.51 - 1.73	0.852	2.13	0.95 - 4.79	0.066
Alcohol	1.47	0.80 - 2.71	0.215	1.49	0.71 - 3.11	0.294
Exercise	1.28	0.63 - 2.61	0.498	1.25	0.51 - 3.06	0.631
Sleep time (<6 hours/day)	0.85	0.44 - 1.65	0.629	0.98	0.44 - 2.19	0.960
Family history of hypertension	2.90	1.55 - 5.42	0.001	3.05	1.45 - 6.40	0.003
Total cholesterol (one SD increase)	1.23	0.91 - 1.65	0.181	1.09	0.73 - 1.64	0.666
Triglyceride (one SD increase)	0.99	0.73 - 1.35	0.960	0.89	0.53 - 1.48	0.643
HDL-cholesterol (one SD decrease)	0.79	0.59 - 1.05	0.102	0.72	0.47 - 1.12	0.143
Fasting plasma glucose (one SD increase)	1.07	0.80 - 1.43	0.662	0.91	0.62 - 1.33	0.614
Job strain						
low	1.00			1.00		
medium	1.67	0.82 - 3.40	0.158	1.70	0.73 - 3.96	0.219
high	3.74	1.36 - 10.29	0.011	3.67	1.05 - 12.83	0.042

Adjueted for age, body mass index, smoking, alcohol, exercise, family history of hypertension, sleep time, mean blood pressure, total cholesterol, triglyceride, fasting plasma glucose, high-density lipoprotein cholesterol, working hours, support from supervisor, support from co-workers, and job strain.

CI; confidence interval, SD; standard deviation.

Figure 1 shows the relationship between masked HT at workplace and clustering of risk factors including family history of HT and job strains. Subjects who had the clustering of these variables had more risk for masked HT at workplace. The combinations of medium job strain and family history of HT, and high job strain and family history of HT were significant risks for family history of HT, compared to the combination of low job strain and no family history of HT.

Figure 1.



4. Discussion

The present study demonstrated that 1) 20% of white-color male employees had masked HT at workplace, 2) Family history of HT and high job strain were significant and independent risks for masked HT at workplace by multiple regression analysis, 3) The combination of high job strain and family history of HT was an increasing the risk for masked HT at workplace.

Kario indicated that masked HT, which was defined as the increment of BP values in daily life in spite of normotension in the clinic or office, had three types such as morning HT, nocturnal HT, and stress-induced HT (Kario, K, et al. 2006). HT occurrence at workplace may be considered as one of stress-induced HT. Masked HT developed an adverse prognosis including cardiovascular events and increased target organ damages (Pickering, T.G, et al. 2007). Masked HT and sustained HT were at equivalent risk for the occurrence of cardiovascular diseases (Verberk, W.J, et al. 2007;

Cuspidi, C, et al. 2007; Mancia, G, et al. 2006). Therefore, we should extend the research to investigate the medical risks for masked HT at workplace.

Several epidemiological studies reported that the prevalence of masked HT was from 9 to 21% in a general population (Sega, R, et al. 2001; Bombelli, M, et al. 2005; Liu, J.E, et al. 1999). In Japan, Harada K, Karube Y, et al. (2006) elucidated that the prevalence of workplace HT was 23%, and that of workplace hypertensive subjects with a normal BP in the clinic or office was 21% in public officials. The prevalence of masked HT at workplace subjects in the present study was 20.0%, which was similar to that of the previous studies. Several previous studies demonstrated that risk factors such as male, age, smoking, alcohol intake, and high body mass index were associated with masked HT (Schnall, P.L, et al. 1992; Bobrie, G, et al. 2004; Trudel, X, et al. 2009). Ferrier C, Cox H, et al. (1993) observed that the resting arterial plasma noradrenaline concentration was higher in normotensive subjects with a family history of essential HT than those with no family history of HT. Noll G, Wenzel RR, et al. (1996) showed that the activity of sympathetic activity systems during mental stress was more likely to increase in subjects with family history of HT than those with no family history of HT. Ohlin B, berglund G, et al. (2008) demonstrated that stress-induced HT was affected by α2β-adrenergic receptors which were located in the central nervous system and in periphery. These results could indicate that subjects with family history of HT had the dys-function of sympathetic nervous systems to regulate normal BP at workplace. Several previous studies indicated that high work stress affected unhealthy behaviors such as smoking, lack of exercise, and excessive alcohol intake (Liu, Y, and Tanaka, H, 2002; Sokejima, S, and Kagamimori, S, 1998; Chandola, T, et al. 2008). Moreover, the autonomic nervous system activated by repeated high work stress affected dysregulation of the hypothalamic-pituitary-adrenal axis (Kunz-Ebrecht, S.R, et al. 2004), which disturbed the circadian rhythm of cortisol and the development of obesity, glucose intolerance, and high BP (Brunner, E.J, et al. 2002). Subjects with family history of HT were more likely to have high body mass index and alcohol intake habit in the present study, which conformed the previous studies. Thus, the high work stress at workplace might be associated with the masked HT at workplace.

Bobrie G, Clerson P, et al. (2008) demonstrated that the factors leading to masked HT were closely associated with the cardiovascular risk factors and psychological aspects. Schnall PL, Pieper C, et al. (1990) demonstrated that work stress such as high psychological demands plus low decision latitude at work caused masked workplace HT at seven urban work sites of 215 employed men aged 30 to 60 years without evidence of coronary heart disease. Harada K, Karube Y, et al. (2006) elucidated that obesity and family history of hypertension were closely associated with workplace HT in civilian employees. However, the previous studies could not investigate the cause of HT occurrence at workplace from the combination of physical and psychological aspects. We demonstrated that the combination of high job strain and family history of hypertension had significantly higher odds ratio for HT occurrence at workplace, which differed from previous studies.

There are several limitations that should be acknowledged in this study. First, because it was cross-sectional, a cause–effect relationship cannot be inferred. If more stressed persons had been prone to take sick leaves or to quit, the influence of stress on mental health may have been attenuated.

Second, our study subjects ranged from 34 to 60 years old in one public office. Therefore, we have to be cautious in extending the present results to the community. Second, we investigated work stress conditions of study subjects once in the present study. Thus, variations in work stress might be occurred in the study period.

In conclusion, risk factors such as family history of HT and high job strain are independent and significant risks, more importantly, clustering of these risk factors increased more risks for workplace HT.

5. Figure legend

Figure 1

The relationship between masked HT at workplace and clustering of risk factors including family history of HT and job strains. Adjusted for age, body mass index, alcohol, exercise, sleep time, mean BP, total cholesterol, triglyceride, high-density lipoprotein cholesterol, fasting plasma glucose, working hours, and job strains.

*: <0.05 vs. the reference group of low job strain and no family history of HT.

References

Bobrie G, Chatellier G, et al. (2004). Cardio-vascular prognosis of "masked hypertension" detected by blood pressure self-measurement in elderly treated hypertensive patients." JAMA, Vol. 291, No. 11, pp. 1342- 9.

Bobrie G, Clerson P, et al. (2008). Masked hypertension: a systematic review. J Hypertens, Vol. 26, No. 9, pp. 1715-25.

Bombelli M, Sega R, et al. (2005). Prevalence and clinial significance of a greater ambulatory versus office blood pressure ('reversed white coat' condition) in a general population. J Hypertens, Vol. 23, No. 3, pp.513-20.

Brunner EJ, Hemingway H, et al. (2002). Adrenocortical, autonomic, and inflammatory causes of the metabolic syndrome: nested case-control study. Circulation, Vol. 106, No. 21, pp.2659-65.

Chandola T, Brunner E, et al. (2006). Chronic stress at work and the metabolic syndrome: prospective study. BMJ, Vol. 332, No. 7540, pp.521-5.

Chandola T, Britton A, et al. (2008). Work stress and coronary heart disease: what are the mechanisms? Eur Heart J, Vol. 29, No. 5, pp.640-8.

Cuspidi, C. and Parati, G. (2007). Masked hypertension: an independent predictor of organ damage. J Hypertens, Vol. 25, No. 2, pp.275-9.

Eguchi K, Kario K, et al. (2003). Greater impact of coexistence of hypertension and diabetes on silent cerebral infarcts. Stroke, Vol. 34, No 10, p.2471-4.

Ezzati M, Alan DL, et al. (2002). Selected major risk factors and global and regional burden of disease. Lancet, Vol. 360, No 9343, pp.1347-60.

Fauvel JP, M'Pio I, et al. (2003). Neither perceived job stress nor individual cardiovascular reactivity predict high blood pressure. Hypertension, Vol. 42, No 6, pp.1112-6.

Ferrier C, Cox H, et al. (1993). Elevated total body noradrenaline spillover in normotensive members of hypertensive families. Clin Sci (Lond), Vol. 84, No. 2, pp.225-30.

Gilbert-Ouimet M, Brisson C, et al. (2012). Repeated exposure to effort-reward imbalance, increased blood pressure, and hypertension incidence among white-collar workers: effort-reward imbalance and blood pressure. J Psychosom Res, Vol. 72, No. 1, pp.26-32.

Guimont C, Brisson C, et al. (2006). Effects of job

strain on blood pressure: a prospective study of male and female white-collar workers. Am J Public Health, Vol. 96, No. 8, pp.1436-43.

Harada K, Karube Y, et al. (2006). Workplace hypertension is associated with obesity and family history of hypertension. Hypertens Res, Vol. 29, No. 12, pp.969-76.

Kario K, Ishikawa J, et al. (2006). Morning hypertension: the strongest independent risk factor for stroke in elderly hypertensive patients. Hypertens Res, Vol. 29, No. 8, pp.581-7.

Kawada T. and Otsuka T. (2011). Relationship between job stress, occupational position and job satisfaction using a brief job stress questionnaire (BJSQ). Work, Vol. 40, No. 4, pp.393-9.

Kunz-Ebrecht SR, Kirschbaum C, et al. (2004). Work stress, socioeconomic status and neuroendocrine activation over the working day. Soc Sci Med, Vol. 58, No. 8, pp.1523-30.

Lewington S, Clarke R, et al. (2002). 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, Vol. 360, No. 9349, pp.1903-13.

Lim SS, Vos T, et al. (2012). A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet, Vol. 380, No. 9859, pp.2224-60.

Liu JE, Roman MJ, et al. (1999). Cardiac and arterial target organ damage in adults with elevated ambulatory and normal office blood pressure. Ann Intern Med, Vol,131, No. 8, pp.564-72.

Liu Y, and Tanaka H, (2002). Overtime work, insufficient sleep, and risk of non-fatal acute myocardial infarction in Japanese men. Occup Environ Med, Vol. 59, No. 7, pp.447-51.

Mancia G, Facchetti R, et al. (2006). Long-term risk of mortality associated with selective and combined elevation in office, home, and ambulatory blood pressure. Hypertension, Vol. 47, No. 5, pp.846- 53.

Mancia G, and Parati G, (2000). Ambulatory blood pressure monitoring and organ damage. Hypertension, Vol. 36, No.5, pp.894-900.

Markovitz JH, Matthews KA, et al. (2004). Increases in job strain are associated with incident hypertension in the CARDIA Study. Ann Behav Med, Vol. 28, No. 1, pp.4-9.

Noll G, Wenzel RR, et al. (1996). Increased activation of sympathetic nervous system and endothelin by mental stress in normotensive offspring of hypertensive parents. Circulation, Vol. 93, No. 5, pp.866-9.

Ohlin B, berglund G, et al. (2008). Job strain, job demands and adrenergic beta1- receptor-polymorphism: a possible interaction affecting blood pressure in men. J Hypertens, Vol. 26, No. 8, pp.1583-9.

Pickering TG, Eguchu K, et al. (2007). Masked hypertension: a review." Hypertens Res, Vol. 30, No. 6, pp. 479- 88.

Riese H, Van Doornen LJ, et al. (2004). Job strain in relation to ambulatory blood pressure, heart rate, and heart rate variability among female nurses. Scand J Work Environ Health, Vol. 30, No. 6, pp.477-85.

Sainas G, Milia R, et al. (2016). Mean blood pressure assessment during post-exercise: result from two different methods of calculation. J Sports Sci Med, Vol. 15, No. 3, pp.424-433.

Schnall PL, Pieper C, et al. (1990). The relationship between 'job strain,' workplace diastolic blood pressure, and left ventricular mass index. Results of a case-control study. JAMA, Vol. 263, No. 14, pp.1929-35.

Schnall PL, Schwartz JE, et al. (1992). Relation between job strain, alcohol, and ambulatory blood pressure. Hypertension, Vol. 19, No. 5, pp.488-94.

Sega R, Trocino G, et al. (2001). Alterations of cardiac structure in patients with isolated office, ambulatory, or home hyper- tension: Data from the general population (Pressione Arteriose Monitorate E Loro Associazioni [PAMELA] Study). Circulation, Vol. 104, No. 12, pp.1385-92.

Sokejima S, and Kagamimori S, (1998). Working hours as a risk factor for acute myo-cardial infarction in Japan: case-control study. BMJ, Vol. 317, No.7161, pp.775-80.

Trudel X, Brisson C, et al. (2009). Masked hypertension: different blood pressure measure ment methodology and risk factors in a working population. J Hypertens, Vol. 27, No. 8, p.560-7

Umehara K, Ohya Y, et al. (2007). Association of work-related factors with psychosocial job stressors and psychosomatic symptoms among Japanese pediatricians. J Occup Health, Vol. 49, No. 6, pp.467-81.

van den Hoogen PC, Feskens EJ, et al. (2000). The relation between blood pressure and mortality due to coronary heart disease among men in different parts of the world. Seven Countries Study Research Group. N Engl J Med, Vol. 342, No. 1, pp.1-8.

Verberk WJ, Thien T, et al. (2007). Masked hypertension, a review of the literature. Blood Press Monit, Vol. 12, No. 4, pp.267-73.