We compared the CFi and CFre estimated by ISO7933 and ISO9920. Because both air velocity and walking velocity were included in the formula for CFi, we calculated the independent effects of air velocity and walking velocity to the CFi and CFre. Because the formula for CFi changes for clothing less than 0.6 clo, CFi and CFre were compared at 0.3 clo and 1.0 clo.

There were times when the core temperature did not return to the equilibrium level associated with the metabolic rate. While the amount of heat loss needed to reduce the core temperature was computed and factored into the model, this was not sufficient to bring the core temperature all the way down (and sometimes it was too much and the core temperature was reduced below the equilibrium value for the metabolic rate). To account for this discrepancy, two steps were taken. The first was to assess the elevation of the current core temperature above the equilibrium value. If E_{req} was less than zero and there was still a core temperature elevation, the required sweat rate (SW_{req}) was adjusted proportionately to the difference. To fine tune the outcome, E_p and SW_p were set to zero when the equilibrium core temperature was reached.

Finally, as a minor modification, posture adjustments to the available surface area for convection and maximum evaporative cooling were instituted for sitting (0.7) and squatting (0.9). It was assumed that there was no conduction across the surfaces in contact with the body during sitting.

After changing the PHS code to compute CFi and CFre by either method, the code was verified using the standard test conditions in ISO7933. Values for final T_{re}, water loss, $D_{lim\ tre}$, $D_{limloss50}$, $D_{limloss95}$ by PHS model (ISO7933) and PHS_m (PHS with the changes). We also calculated $D_{lim\ tre}$ by PHS model and PHS_m using trial conditions used for high heat stress exposure conditions (safe exposure times less than 120 min) for 142 trials (Bernard and Ashley 2009).

An Excel workbook with a VBA macro was used to compute PHS and PHSm.

RESULTS

The first step in the PHS modification process was to examine the differences between CFi and CFre as specified by ISO7933 and ISO9920 as a function of air speed, walking speed, and clothing level. Fig. 1 illustrates the comparison.

CFi was not substantially different for either method, and decreased with air and walking speed. CFre by ISO9920 was larger than ISO7933, and the difference increased with either air or walking speed. As a note, the difference in methods for estimating CFre in ISO9920 was less than 5% over the same conditions.

The outcomes by PHS and PHS_m were compared using the standard test conditions in ISO7933 (Table 1). The calculation time limit was 480min. PHS_m showed higher Final T_{re} than PHS. The temperature difference between PHS and PHS_m increased when the final T_{re} increased. Maximum allowable exposure time for heat storage ($D_{lim\ tre}$) was shorter in PHS_m than PHS. In example 3 and 7, PHS did not have limitation but PHS_m had limitation time of 117 min and 196 min respectively.

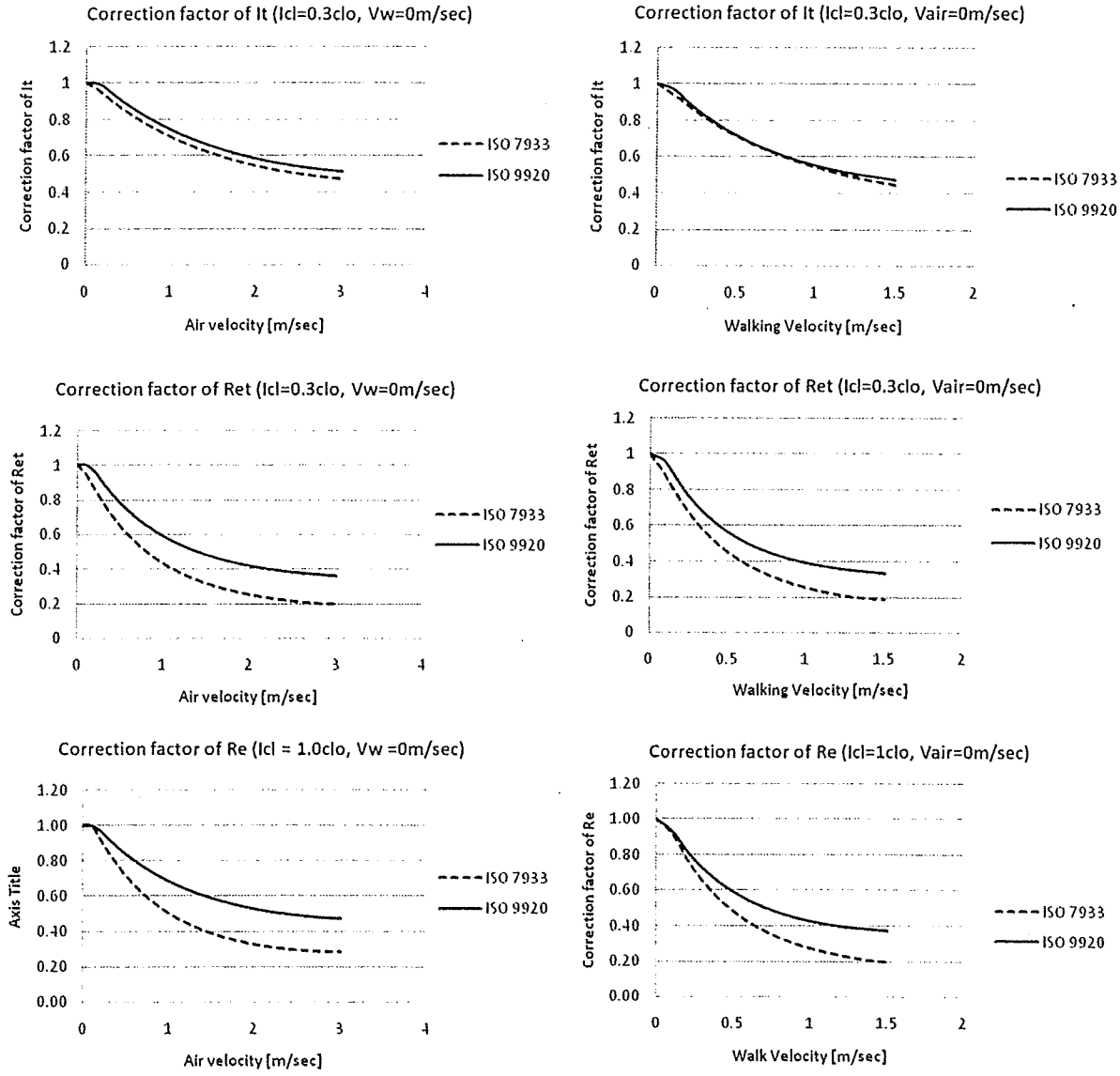

Fig. 1. Correction factors for I_{cl} and R_{et} calculated by ISO7933 and ISO9920.

Table 1. Differences in outcomes for standard test conditions

Parameters	1		2		3		4		5		6		7		8	
Acclimatized	Yes		Yes		Yes		No		No		No		Yes		Yes	
Posture	Standing		Standing		Standing		Standing		Standing		Standing		Standing		Standing	
t_a(° C)	40		35		30		28		35		34		40		40	
Pa(kPa)	2.5		4.0		3.0		3.0		3.0		3.0		3.0		3.0	
t_r(° C)	40		35		50		58		35		34		40		40	
v_a(m/s)	0.3		0.3		0.3		0.3		0.3		0.3		0.3		0.3	
M(W/m2)	150		150		150		150		206		150		150		150	
I_{cl}(clo)	0.5		0.5		0.5		0.5		0.5		1.0		0.4		0.4	
θ(degree)	0		0		0		0		0		0		0		90	
Walk speed (m/s)	0		0		0		0		0		0		0		1	
Output	PHS	PHSm	PHS	PHSm	PHS	PHSm	PHS	PHSm	PHS	PHSm	PHS	PHSm	PHS	PHSm	PHS	PHSm
Final t_{re}(° C)	37.5	37.7	39.8	41	37.7	39	41.2	42.5	39.2	41.3	41	42.5	37.5	38.4	37.6	37.6
Water loss(g)	6174	7209	6938	6973	7167	7206	5807	5807	7252	7371	5551	5642	6691	7215	5386	5541
$D_{lim\ tre}$ (min)	480	480	74	56	480	117	57	44	69	45	67	54	480	196	480	480
$D_{limloss\ 50}$ (min)	439	378	385	382	380	377	466	466	371	360	480	479	406	377	480	480
$D_{limloss\ 95}$ (min)	297	257	256	256	258	255	314	314	246	237	317	314	275	255	339	329

To compare the differences of $D_{lim\ tre}$ calculated by PHS and PHS_m, 142 experimental conditions from a previous study were used. $D_{lim\ tre}$ was longer in PHS than in PHSm (Fig. 2). When the $D_{lim\ tre}$ was below 20 min, PHS and PHS_m were about the same value. As $D_{lim\ tre}$ increased, the difference became larger.

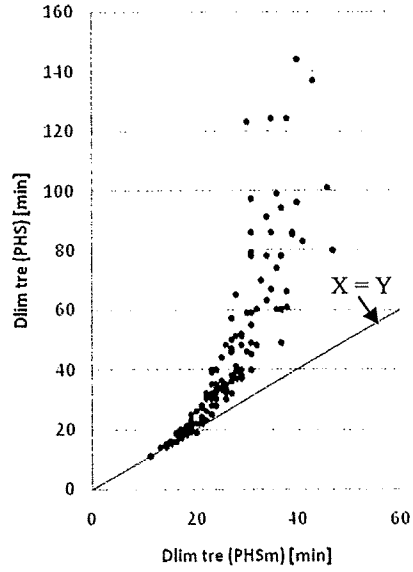

Fig. 2. Differences in predicted time limit by PHS and PHS_m based on experimental trials for short term exposures.

To illustrate the code changes for recovery to the equilibrium core temperature, T_{re} time course for an exposure and recovery by PHS, PHS_m, PHS_{ce} (core equilibrium included in PHS), PHS_{mce} (core equilibrium included in PHS_m) (see Fig. 3). The faster rate of rise was expected for PHSm. The recovery to different levels from the different levels of rectal temperature without the code adjustment was also expected.

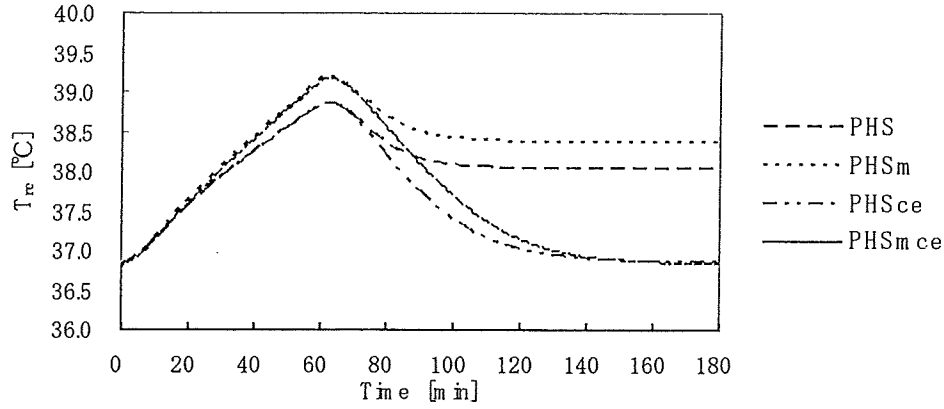

Fig. 3. Recovery of T_{re} calculated by four variations of the PHS model.

CONCLUSIONS

By introducing the dynamic correction factor of clothing insulation and evaporative resistance of ISO9920 (PHSm) into ISO7933 (PHS), there was little change in the total dynamic (resultant) insulation, but the total dynamic evaporative resistance was higher for PHSm. Thus the predicted heat strain was greater in PHS_m than PHS. This trend was seen both in the test scenerios published in ISO7933 and from a set of experimental data.

After the elevation of current core temperature above equilibrium temperature was used to adjust predicted sweat rate, T_{re} returned to the equilibrium temperature as intended.

ACKNOWLEDGMENTS

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UTILITY OF PREDICTED HEAT STRAIN TO LIMIT SHORT-TERM HEAT STRESS EXPOSURES

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INTRODUCTION

The Predicted Heat Strain (PHS) was proposed by Malchaire et al (2002) and validated against laboratory and field data (Machaire et al 2001) as part of the EU BIOMED II project. The result of the effort was an update to ISO7933 (2004). One useful purpose of PHS is to examine the pattern of heat strain that results from steady and time varying heat stress exposures. Alternative controls can be simulated on a desktop evaluation. A second use of the PHS is to limit acute heat stress exposures to the time it takes to reach a predicted physiological limit (either rectal temperature or dehydration).

Japan National Institute of Occupational Safety and Health and University of South Florida undertook several changes to PHS, mainly to account for clothing effects following the guidance of ISO9920 (2007). The purpose of this paper was to compare the predicted exposure limit from PHS and the modified PHS (called here PHSm) to observed data from trials designed to last from about 30 to 110 minutes (Bernard and Ashley 2009). From these comparisons, the ability of PHS and PHSm to limit acute exposures can be assessed.

METHODS

Twelve adults participated in the time-limited heat stress exposures. Prior to beginning the experimental trials to determine critical WBGT, they underwent a 5-day acclimatization to dry heat. Participants wore a base ensemble of shorts, tee-shirt (or sports bra for women), socks and shoes.

Three different clothing ensembles over the base ensemble were evaluated: work clothes (135 g m^{-2} cotton shirt and 270 g m^{-2} cotton pants), water-barrier, vapor-permeable ensemble (NexGen® LS 417), and vapor-barrier ensemble (Tychem QC®, polyethylene-coated Tyvek®). The limited-use coveralls had a zippered closure in the front and elastic cuffs at the arms and legs; and they did not include a hood.

Each participant walked on a treadmill at a moderate rate of work (190 W m^{-2}) at five levels of heat stress. The 15 combinations of clothing and heat stress level (see Table 1) were completed in random order. During trials, participants were allowed to drink at will. Core (rectal) temperature, heart rate and ambient conditions were monitored continuously and recorded every

5 minutes. The time at which T_{re} reached 38 °C (t@38) was noted. The safe exposure time (SET) was taken as the time at which the first of the following conditions was satisfied: (1) T_{re} reached 38.5 °C, (2) a sustained heart rate greater than 85% of the age-predicted maximum heart rate, or (3) participant wished to stop. Only trials in which t@38 was reached and trials that ended before 120 min were used to evaluate validity for t@38 and SET.

Table 1. Combinations of Clothing Ensemble and Target Levels of WBGT [°C] at 50% Relative Humidity

	Heat Stress Level				
Ensemble	HL1	HL2	HL3	HL4	HL5
Work Clothes	36	37	38	40	44
NexGen	33	34	36	38	41
Tychem QC	29	30	32	34	38

The experimental conditions for each trial were used as the heat stress conditions for the PHS and PHSm. These included dry bulb and radiant temperatures, ambient water vapor pressure, air speed, metabolic rate, walking speed, and intrinsic clothing insulation and estimated i_m from previous data as well as participant height and weight. The PHS and PHSm times were taken as the time at which predicted rectal temperature reached 38 °C. When walking speed was used, the wind direction was considered omni-directional. A second evaluation was performed in which the effective walking speed based on metabolic rate was used.

For comparison, t@38 and SET were compared to PHS and PHSm times. The outcomes were classified in three categories. On-Time (OT) was a condition in which the actual time (either t@38 or SET) occurred within a window that was plus or minus 10% of the predicted time (i.e., PHS or PHSm time). An early predicted time (E) was the outcome in which the predicted time plus 10% was earlier than the observed time. A late predicted time (L) occurred when the predicted time minus 10% was greater than the observed time.

RESULTS

There were 8 comparison groups based on observed time at two levels (t@38 and SET), predicted time by two methods (PHS and PHSm), and the use of walking speed at two levels (Yes and No). The experimental trials were not designed to have True Negatives, and thus sensitivity and specificity were not evaluated. The distribution of trials among Early, On-Time and Late was determined and provided in Table 2. If a broad view of protection including over-protection is taken, then protective accuracy can be computed as the ratio of the On-Time plus the Early over the total observations. These values were generally high but driven by the large number of Early observations.

For each factor in Table 2, there were trends worth noting. Using the actual walking speed to one estimated from metabolic rate led to fewer Early classifications within each of the four combinations of observed and predicted time. For t@38 and PHS with actual walking speed, the distribution was somewhat balanced; and it shifted to Early for PHSm and for estimated walking speed. For SET, there was a general movement to Early regardless of the predicted time or walking speed method.

Table 2. Classification of outcomes for the eight comparison groups.

	Early	On-Time	Late	Total
		Actual V_{walk}		
t@38 v. PHS	55	28	71	154
t@38 v. PHSm	123	17	14	154
SET v. PHS	92	23	53	168
SET v. PHSm	157	8	3	168
		Estimated V_{walk}		
t@38 v. PHS	121	22	11	154
t@38 v. PHSm	134	15	5	154
SET v. PHS	158	8	2	168
SET v. PHSm	164	3	1	168

Figures 1 and 2 illustrate the relationships between individually observed safe exposure times (SETs) and individually determined predicted times when walking speed was used. As expected from Table 2, there was a trend for the predicted times to be less than the observed times (to the right and below of the identity line). This effect was greater for PHSm than PHS. The trial protocol called for stopping at 120 min and represented greater than 120 min.

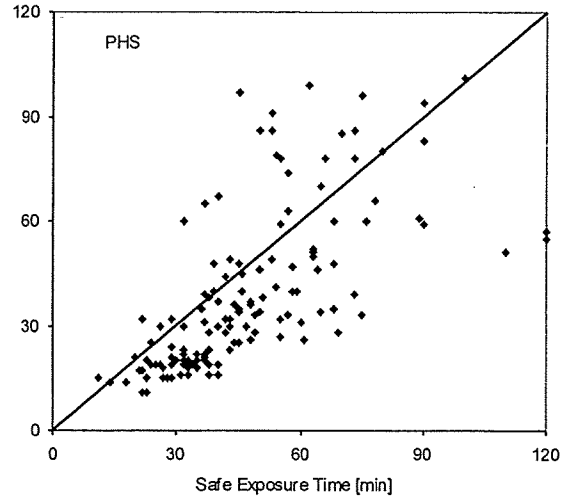

Figure 1. Predicted time from PHS compared to the observed safe exposure time.

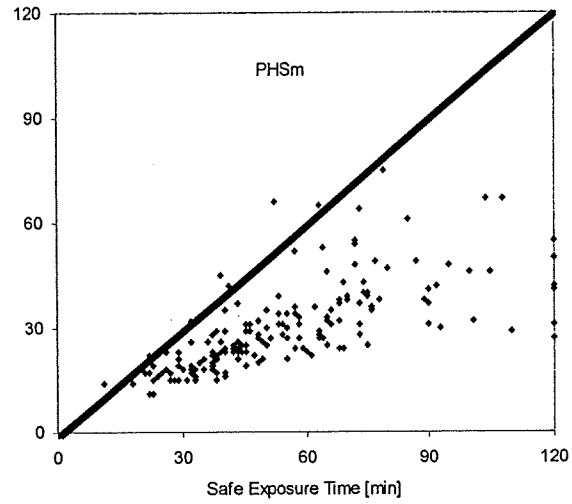

Figure 2. Predicted time from PHSm compared to the observed safe exposure time.

CONCLUSIONS

Comparing t@38 with PHS including walking speed was the approach most in line with the original intent of PHS. That is, (1) PHS used walking speed to adjust for the dynamic effects of work on clothing factors and the metabolic estimation was offered as an alternative and (2) it was validated in its ability to predict a mean rectal temperature response (Malchaire et al 2001). This comparison supported the original validation with a roughly balanced set of outcomes among Early, On-Time and Late.

The validity study also showed that the 95% confidence interval included 38.5 °C; and was up to 38.7 °C (see Fig 2 and 4 of Malchaire et al 2001). This fact means that most rectal temperatures will be less than 38.7 °C when the model predicts 38 °C. For this reason, a comparison to the observed safe exposure time at 38.5 °C (or elevated heart rate or fatigue) was appropriate. While PHS was indicative of the mean response, it was not protective when used as a method to limit heat stress exposures in a prescriptive fashion. When using the SET value, there were a large number of Late outcomes (53 of 168 or 32%).

The lower predicted times of PHSm were not intended but an outcome of updating the methods to adjust static values of insulation and evaporative resistance to dynamic (resultant) values. The result was fewer Late outcomes (3 of 168 or 2%) but more Early ones (157 of 168 or 93%). The protective accuracy was generally high for PHSm, but this was driven by a high number of Early trials. That means that PHSm was overly protective in 93% of the trials.

Overall, the use of PHS or PHSm for prescribing acute exposures appeared to be limited. Where PHS had significant risk of Late outcomes pointing out the risk for over-exposures, PHSm was characterized by a large number of Early outcomes, which may reduce the utility for exposure planning.

It was clear from the data that substituting a metabolic rate for actual walking speed led to a systematic lowering of the predicted times. This may be an area for further investigation.

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Industrial Health 2009, 47, 589–602

Review Article

Heart Rate Variability in Occupational Health —A Systematic Review

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Abstract: This systematic review evaluates and summarizes the evidence of association between work-related factors and heart rate variability (HRV) in workers. We reviewed English articles indexed in MEDLINE under the key words: work, worker, occupational, industrial, and heart rate variability. Studies were included if one or more of the dependent variables was one of the time- or frequency-domain indexes of HRV [standard deviation of all normal-to-normal (NN) intervals (SDNN), mean of the 5-min standard deviations of NN intervals calculated over several hours (SDNN index), the root mean squared differences of successive NN intervals (RMSSD), integrated spectral powers of high (HF, > 0.15 Hz) and low frequency (LF, 0.04–0.15 Hz) HRV, and the LF/HF ratio] as recommended by the European Society of Cardiology and the North American Society of Pacing Electrophysiology. Physical and chemical work environments (i.e. exposure to occupational toxicants and hazardous environments), psychosocial workload (i.e. job stressors), and working time (i.e. shift work) had been examined and identified as having associations with low HF power. These findings may indicate that research into parasympathetic nervous system activity should be focused to protect cardiovascular health at work. We also propose the use of very low and ultralow frequency HRV components in autonomic research for workers' health.

Key words: Physical and chemical work environment, Psychosocial workload, Shift work, Fatigue, Cardiovascular disease, Autonomic nervous system, HRV

Background

Cardiovascular disease (CVD) is the leading cause of mortality and morbidity in industrialized countries[1]. In a working population, occupational factors are believed to pose a threat to workers' cardiovascular health. Substantial research has shown that adverse physical and psychosocial work environments and working conditions, such as shift work and excessive workload, are related to CVD. To examine the acute and chronic effects of such work-related factors on the cardiovascular system, cardiac autonomic nervous system activity during work and rest has been assessed by heart rate variability (HRV).

Heart rhythm is recorded by electrocardiography. The measured signal shows a series of waves that present electrical events in the four chambers and the conduction pathways within the heart. The time interval between consecutive R waves that correspond to the contraction of the ventricles is called the RR interval (RRI). HRV is a time series of RRIs that fluctuate beat by beat in healthy humans because of time-to-time changes in activities of sympathetic and parasympathetic nerves innervating to the sinus node.

HRV is generated in part by periodic inputs of both respiration and blood pressure variability into the medullary cardiovascular centers[2]. These periodic modulations are clearly identified within the power spectrum of HRV as peaks at the respiratory frequency (≈ 0.25 Hz) and at the frequency of the well-known Mayer wave (0.1 Hz) in blood pressure variability[1, 3].

Heart rate increases and decreases during inspiration and expiration, respectively. This is mainly caused by the physiological fact that parasympathetic output from the cardiovascular center is inhibited during inspiration[4]. Respiratory rhythm during rest is about 0.25 Hz. Therefore,

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the amplitude of variations in HRV related to respiration (i.e. respiratory sinus arrhythmia) is considered to reflect the fluctuation in tonic parasympathetic input, and the integrated spectral power of high frequency (HF, > 0.15 Hz) HRV is used as an index of the level of parasympathetic activity[5]. The Mayer wave is a 10-s oscillation of blood pressure. The wave is mediated mainly by the sympathetic nervous system. The wave elicits a 10-s oscillation in HRV by modulating both sympathetic and parasympathetic nerve activities via baroreceptors. As a result, low-frequency (LF, 0.04–0.15 Hz) power of HRV reflects both sympathetic and parasympathetic activity and the LF/HF ratio is considered to mirror sympathovagal balance[5]. Therefore, the frequency analysis of HRV is able to provide results that are more easily interpretable in terms of physiological regulation. To standardize physiological and clinical studies, short-term recordings of 5 min under physiologically stable conditions are recommended for HRV frequency-domain methods[5]. For long-term (> 5 min) recordings, the frequency analysis should be performed in 5-min segments or over the total 24-h record[5]. The HF and LF components of power spectral density obtained from the entire 24-h record are not different from the mean HF and LF components obtained from 5-min segments over the entire 24-h period.

For descriptive statistical methods, several time-domain measures are recommended by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (Task Force)[5]. The measures for overall variation include the standard deviation of all normal-to-normal (NN) intervals (SDNN) and the HRV triangular index, which is the number of NN intervals divided by the number of NN intervals within the modal bin of the NN interval histogram. The mean of the 5-min standard deviations of NN intervals calculated over several hours (SDNN index) is the measure of variation due to cycles shorter than 5 min. The measure of variation due to cycles longer than 5 min is the standard deviation of the average NN interval calculated over 5 min (SDANN). The estimates for short-term variation are the root mean squared differences of successive NN intervals (RMSSD).

Low HRV has been utilized in clinical studies of patients with CVD to predict a variety of cardiovascular outcomes[5], and in healthy populations' all cause and cardiac mortality[6–8], as well as new onset of hypertension[9]. Low HF power of the HRV spectrum is also a risk for mortality in patients, who have had myocardial infarction[10–12] and who have coronary artery disease[13] or congestive heart failure[14, 15].

The goal of this paper was to review studies of HRV concerning occupational health. Given the functional utility of HRV analysis, it may inform us of the autonomic effects of many factors at the workplace, such as work environments, workloads, and working time. The information quantified by HRV may help to determine effective strategies for health assessment and promotion at work and also future research needs. This systematic review included only studies which used one or more of the above mentioned indices of HRV, which are recommended by the Task Force[5], as a dependent variable.

Methods

We conducted a systematic review of all English articles using MEDLINE (Ovid, Pubmed) to identify all observational studies assessing the association between work and HRV. Key words used for the computer searches were: work, worker, occupational, industrial, and heart rate variability. Studies were included in our review if one or more of the dependent variables was one of the time- or frequency-domain indexes of HRV recommended by the Task Force[5], as mentioned in the Background. Studies in which participants were not workers (subjects without occupational exposure) were not included. Forty-six studies published from 1994 to July 2007 were included in the review. Sample sizes of these studies ranged from 6 to 2,197, with a median of 51.5. It is notable that only 2 of 46 studies (4%) utilized longitudinal study designs. Factors of occupational exposure were classified as a) physical and chemical work environment, b) psychosocial workload, or c) working time. Table 1 presents a summary of the studies reviewed.

Results

Physical and chemical work environment

Particulate matter

Magari *et al.*[16] found that mean RRI and SDNN on workdays decreased with increase in a 4-h moving average of fine particle (particulate matter with a mean aerodynamic diameter ≤ 2.5 μm, $PM_{2.5}$) concentration after controlling for time of day, age, and urinary nicotine levels in male boilermakers and a pipe fitter. Lead and vanadium concentrations as a component of $PM_{2.5}$ were positively associated with the SDNN index during work in the male boilermakers after controlling for mean RRI, age, and smoking status[17].

Exposure to high levels of $PM_{2.5}$, mainly from welding fume was significantly associated with reduced mean RRI, SDNN, RMSSD, and HF and LF components on workdays in the male boilermakers after controlling for time of day and time-varying activities[18]. The responses were greater in subjects with a high CVD risk profile than in subjects with a low CVD risk profile.

Particles of motor vehicle origin, as an occupational

Table 1. Studies of industrial environment and heart rate variability in workers

First author, year	Population	N (men/women or total)	Duration of recording	Condition at recording	Data length for analysis	Comparison	Outcome						
							Mean RRI	SDNN (TP)	SDNNI	RMSSD	HF	LF	LF/HF
Physical and chemical work environment													
	Particular matter												
Magari *et al.* 2001[16]	boilermakers and a pipe fitter	33/0	24 h × 2 d	workday	every 5 min	$PM_{2.5}$	↓	↓	-	-	-	-	-
Magari *et al.* 2002[17]	boilermaker construction workers	39/0	8–10 h	workday	every 5 min	$PM_{2.5}$	n.s.	-	↑	-	-	-	-
Chen *et al.* 2006[18]	boilermakers	10/0	24 h	workday	every 5 min	$PM_{2.5}$	↓	↓	-	↓	↓	↓	n.s.
Riediker *et al.* 2004[19]	highway patrol troopers	9/0	20 min	resting in the morning on workday	last 10 min	$PM_{2.5}$	↑	↑	-	-	-	-	-
Riediker *et al.* 2004[20]	highway patrol troopers	9/0	20 min	resting in the morning on workday	last 10 min	$PM_{2.5}$	↑	↑	-	-	↑	n.s.	↓
	Carbon disulfide												
Bortkiewicz *et al.* 1997[21]	a. workers at a chemical fibre plant b*. healthy workers	a. 152/0 b. 93/0	512 points	resting in the morning after the night rest	512 points	a vs. b	↓	(↓)	-	-	↓	↓	n.s.
Jhun *et al.* 2003[22]	a. workers with carbon disulfide poisoning b*. healthy workers	a. 71/0 b. 127/0	3 min	resting	3 min	a vs. b	n.s.	(↓)	-	-	n.s.	↓	-
	Lead												
Murata *et al.* 1995[23]	a. glass workers b*. textile workers	a. 0/36 b. 0/15	300 points	resting	300 points	a vs. b	n.s.	-	-	-	-	-	↓
Gajek *et al.* 2004[24]	a. copper foundry workers b*. healthy workers	a. 22/0 b .13/0	24 h	daytime after at least 20 min rest and night (0400–0415)	15 min × 2 sessions	a vs. b	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.	n.s.
Böckelmann *et al.* 2002[25]	a. lead-exposed workers b*. healthy workers	a. 109/0 b. 27/0	-	resting, psychometric strain test (number memory test), and recovery periods in the morning	-	a vs. b at baseline	n.s.	(n.s.)	-	-	n.s.	n.s.	-
						baseline vs. 4 yr later	↓	(↓)	-	-	↓	-	-
	Manganese												
Barrington *et al.* 1998[26]	a. manganese alloy workers b*. healthy workers or published normals	a. 8 b. 8 or 274	24 h	-	24 h	a vs. b	↓	n.s.	-	↓	↓	↓	↓
	Smoking												
Kobayashi *et al.* 2005[27]	taxi drivers	20/0	18 h	workday (0800–0200)	every 5 min	smoking	-	-	-	-	n.s.	-	↑
	Electromagnetic field												
Bortkiewicz *et al.* 1996[28]	a. workers on AM broadcast stations b*. workers of radio link stations	a. 71 b. 22	512 points	resting in the morning	512 points	a vs. b	↑	n.s.	-	-	n.s.	n.s.	n.s.
Wilén *et al.* 2007[29]	a. radiofrequency operators b*. healthy workers	a. 18/17 b. 14/23	24 h	workday	every 1 h	a vs. b	-	(↑)	-	-	n.s.	↑	-
Bortkiewicz *et al.* 2006[30]	a. switchyard substations b*. radio link stations	a. 63/0 b. 42/0	512 points	resting in the morning	512 points	a vs. b	n.s.	↓	-	-	n.s.	n.s.	n.s.
	Vibration												
Lasker and Harada, 1999[31]	a. chainsaw operators and tunnel construction workers with hand-arm vibration syndrome b*. healthy workers	a. 21/0 b. 10/0	2 min	resting	2 min	a vs. b	n.s.	n.s.	-	-	↓	↓	-
Lasker *et al.* 1997[32]	a. patients with hand-arm vibration syndrome with VWF b. patients with hand-arm vibration syndrome without VWF c*. healthy workers	a. 16/0 b. 13/0 c. 12/0	4 min	supine deep breathing before and after psychological test	2 min × 2 sessions	b vs. c	n.s.	↓	-	↓	-	-	-
Sakakibara *et al.* 2002[33]	a. patients with VWF b*. healthy workers	a. 21/0 b. 17/0	50 min	resting, cold water immersion, and recovery	128 s × 8 sessions	a vs. b	↑	-	-	-	n.s.	↑	↑
	Cold environment												
Bortkiewicz *et al.* 2006[34]	cold storage workers	63/39	512 points	resting in the morning	512 points	cold microclimate	n.s.	n.s.	-	-	n.s.	n.s.	n.s.

First author, year	Population	N (men/women or total)	Duration of recording	Condition at recording	Data length for analysis	Comparison	Outcome						
							Mean RRI	SDNN (TP)	SDNNI	RMSSD	HF	LF	LF/HF
Psychosocial workload													
	Job strain												
Collins *et al.* 2005[36]	day workers	36/0	24 h × 2 d	workday and the next rest day	every 5 min	job strain	↓	-	-	-	↓	-	↑
van Amelsvoort *et al.* 2000[37]	workers in the integrated circuit manufacturing industry, waste incinerator plants, or in hospitals	135	24 h	morning or day shift	every 5 min	job strain	↑	n.s.	-	-	n.s.	↑	-
Riese *et al.* 2004[38]	nurses	0/159	24 h × 2 d	the third morning shift (0730–1615) and rest day	every 30 s	job strain	n.s.	-	-	n.s.	-	-	-
Kang *et al.* 2004[39]	workers employed in the ship-building industry aged over 40	167/0	5 min	resting	5 min	job stress	-	n.s.	-	-	n.s.	n.s.	n.s.
Kageyama *et al.* 1998[40]	white collar workers	223/0	5 min	supine and standing rest during late morning or early afternoon	3 and 2 min	job stress	n.s.	-	-	-	n.s.	n.s.	-
Vrijkotte *et al.* 2000[42]	white collar workers	109/0	24 h × 3 d	2 workdays and a rest day	every 30 s	work stress	↓	-	-	↓	-	-	-
	Employment												
Hemingway *et al.* 2005[43]	clerical civil servants	2,197/0	5 min	resting	5 min	employment grade	↓	↓	-	-	↓	↓	-
	Organizational injustice												
Elovainio *et al.* 2006[45]	workers in long term care homes	0/57	5 min	resting	5 min	injustice	-	-	-	-	↓	-	-
	White-collar office work for hypertensive												
Kobayashi *et al.* 2001[46]	white-collar workers	22/0	24 h	workday	512 points (every 30 min)	hypertension	n.s.	-	-	-	↓	↓	↑
	Fatigue												
Pichot *et al.* 2002[47]	a. garbage collectors b*. healthy sedentary workers	a. 6/0 b. 8/0	4 h × 8 d	nocturnal sleeping (0000–0400) on workday	every 256 points	a vs. b	↓	n.s.	n.s.	↓	↓	↓	n.s.
Watanabe *et al.* 2002[48]	workers of an electric instrument corporation	52/0	5 min	resting in the morning	5 min	vital exhaustion	-	-	-	-	↓	n.s.	↓
Sasaki *et al.* 1999[49]	engineers	188/0	3 min	resting in the afternoon on workday	3 min	working hours	-	-	-	-	-	-	n.s.
Sasaki *et al.* 1999[50]	engineers	147/0	3 min	resting in the afternoon on workday	3 min	working hours	n.s.	-	-	-	-	-	n.s.
Karita *et al.* 2006[51]	information service workers	413/0	10 min	resting	last 150 points	subjective symptoms	-	-	-	-	↓	n.s.	n.s.
Aasa *et al.* 2006[52]	ambulance personnel	24/2	24 h × 3 d	24-h shift (1700–1700) and the next two rest days	(mean values of 1 h intervals)	health complaints	n.s.	-	-	-	↓	↑	-
	Train driving												
Myrtek *et al.* 1994[53]	train drivers	23/0	1,387 min	workday	every 1 min	speed	↑	-	-	↓	-	-	-
Working time													
	Night shift work												
Ito *et al.* 2001[54]	nursing staff	0/10	24 h × 2 d	a. day shift (0800–1700) b. night shift (2140–0840)	512 s (each hour)	a vs. b	n.s.	-	-	-	n.s.	n.s.	n.s.
Freitas *et al.* 1997[55]	oil refinery security workers	12/0	24 h × 2 d	a. day shift (0600–1400) b. night shift (2200–0600)	512 points (each hour)	a vs. b	n.s.	n.s.	-	-	n.s.	n.s.	n.s.
Adams *et al.* 1998[56]	emergency physician	8/4	24 h	1500–1700, the middle of night shift (2300–0700), and after the shift	2 h × 3 sessions	night work	↓	↓	-	-	↓	-	↑
Ishii *et al.* 2005[57]	a. shift nurses b*. non-shift nurses	0/91	300 points	resting in the evening (1700–1900) following day work	300 points	a vs. b	n.s.	-	-	-	↓	-	↑
Murata *et al.* 2005[58]	a. shift workers b*. day workers	a. 153/0 b. 87/0	300 points	resting in the morning (0800–1200) of day shift or rest day	100 points with minimum SD	a vs. b	n.s.	-	-	-	n.s.	↓	n.s.
Ha *et al.* 2001[59]	a firm manufacturing diapers and feminine hygienic materials	134/0	5 min × 3 sessions	resting on the second or third day of the 3-day period of morning, afternoon, and night shift	middle 3 min × 3 sessions	shift work	-	-	-	-	↓	↓	-

First author, year	Population	N (men/women or total)	Duration of recording	Condition at recording	Data length for analysis	Comparison	Outcome						
							Mean RRI	SDNN (TP)	SDNNI	RMSSD	HF	LF	LF/HF
van Amelsvoort *et al.* 2000[37]	a. shift workers b*. day workers (all workers in the integrated circuit manufacturing industry, waste incinerator plants, or in hospitals)	a. 102 b. 32	24 h	morning or day shift	every 5 min	a vs. b	↓	↓	-	-	n.s	n.s	-
van Amelsvoort *et al.* 2001[60]	a. shift workers b*. day workers (all workers in the integrated circuit manufacturing industry or waste incinerator plants)	a. 67/8 b. 29/3	24 h × 2 d	morning or day shift at baseline and 1 yr later	every 5 min	a vs. b at baseline	↓	-	↓	-	↓	↓	-
						baseline vs. 1 yr later	↑	-	n.s.	-	n.s	n.s	-
	24-h shift work												
Mitani *et al.* 2006[61]	ambulance personnel	9/0	24 h × 2 d	24-h shift (0900-0900) and rest day	every 5 min	24-h work	n.s.	-	-	-	↓	-	↑
Takeyama *et al.* 2005[62]	firefighters	12/0	24 h	24-h shift	every 5 min	24-h work (0500–0700)	↓	-	-	-	n.s	-	↑
Sato *et al.* 2001[63]	long distance truck drivers	6/0	14.8–93.5 h	workday	every 2 min	24-h work (0000–1200)	-	-	-	-	-	-	↓

↑, ↓ indicate increase or decrease (statistically significant at $p < 0.05$). n.s. indicates no change ($p \geq 0.05$). -, data is not provided. *, unexposed workers; vwf, vibration-induced white finger; $PM_{2.5}$, particulate matter with a mean aerodynamic diameter ≤ 2.5 μm; RRI, RR interval; SDNN, standard deviation of normal-to-normal (NN) intervals; TP, total power; SDNNI, SDNN index; RMSSD, root mean squared differences of successive NN intervals; HF, high frequency (> 0.15 Hz); LF, low frequency (0.04–0.15, 0.01–0.15, 0.02–0.15, or 0.05–0.15 Hz).

source of $PM_{2.5}$, were not associated with a decrease in HRV. Riediker *et al.*[19, 20] reported an association between in-vehicle $PM_{2.5}$ during work and HRV at rest on the next morning in non-smoking male highway patrol troopers. The $PM_{2.5}$ was significantly positively associated with mean RRI, SDNN, and the HF component, and negatively associated with the LF/HF ratio[20]. Among sources of in-vehicle $PM_{2.5}$, the "speed-change" factor was dominated by copper, sulfur, and aldehydes were significantly associated with mean RRI and SDNN[19].

Carbon disulfide

In male workers at chemical fiber plants who worked under conditions of continuous exposure to carbon disulfide (CS_2), mean RRI, total power (TP) of HRV power spectral density, the normalized HF component (HF as a percentage of TP), the normalized LF component (LF as a percentage of TP), and the normalized very low frequency (VLF, 0.0033–0.04 Hz) component (VLF as a percentage of TP) during rest were significantly lower than those in age-matched, unexposed, healthy male controls[21]. In retired male workers with CS_2 poisoning, the LF component and TP during rest were significantly lower than those of age-matched control subjects who had no history of organic-solvent exposure and CVD[22].

Lead

The LF/HF ratio during rest was significantly lower in female glass workers exposed to lead (mean blood lead concentration: 556 μg/l) than in female textile workers unexposed to lead (mean blood lead concentration: 63 μg/l)[23]. In contrast, Gajak *et al.*[24] did not find a significant difference in the LF/HF ratio, assessed in 15-min RRIs during rest in the daytime and at night, between male copper foundry workers (mean blood lead concentration: 410 μg/l) and age-matched healthy male controls (mean blood lead concentration: 36 μg/l). Among the subjects exposed to lead, a negative correlation was found between the lead concentration and the ratio of nighttime HF to daytime HF[24]. In a longitudinal study that examined the effect of 4 yr of lead exposure on HRV[25], mean blood lead levels of lead-exposed workers increased from 390 μg/l to 430 μg/l over 4 yr. Mean RRI and HF component during rest, the psychometric strain test (number memory test), and recovery conditions in the morning were significantly lower than the initial values in the follow-up investigation 4 yr later.

Manganese

Mean RRI, RMSSD, and HF and LF components in manganese alloy workers were significantly smaller than in healthy controls while the LF/HF ratio was significantly larger in the workers than in the controls, although whether RRIs were collected on workdays or rest days was not specified[26].

Smoking

The LF/HF ratio significantly increased immediately after smoking at night in taxi drivers[27]. Smoking caused acute effects on HRV under their ordinary work conditions[27].

Electromagnetic field

A statistically significant negative correlation was found between the maximum exposure level of electromagnetic field (EMF) intensity and the HF component during rest in workers occupationally exposed to medium frequency (0.7–1.5 MHz) EMF, although some frequen-

cy-domain parameters of HRV (HF and LF components, TP, and LF/HF ratio) did not differ between exposed (radiofrequency exposure: 113.3 Vh/m/d) and unexposed workers (radiofrequency exposure: 0 Vh/m/d)[28]. In contrast, TP and the VLF component among operators of radiofrequency (RF) plastic sealers (mean electric field strength: 88 V/m) during the nighttime were significantly increased when compared with a control group[29]. One study found that the relative risk of decreased SDNN (SDNN < 27 ms) during rest was significantly higher in workers of switchyard substations who were occupationally exposed to 50 Hz EMF (EMF exposure: 0.2–15.2 kVh/m/d) when compared with unexposed workers (EMF exposure: 0 kVh/m/d)[30]. The VLF component was significantly higher in the exposed group and was accompanied by a significant increase in arterial blood pressure which was correlated with the exposure level[30].

Vibration

Laskar *et al.* reported that workers with hand-arm vibration syndrome showed significantly decreased HF and LF components during rest[31] and significantly decreased SDNN and RMSSD during rest after a psychological test when compared with the healthy controls[32]. Among operators with vibration-induced white finger, the LF/HF ratio increased significantly during immersion of the hand in cold water at 10°C, but did not increase in healthy controls[33].

Cold environment

Any selected HRV parameters (SDNN and HF, LF, and VLF components) during rest in the morning did not differ between workers who were chronically exposed to different microclimate conditions (–26 to 20°C)[34].

Psychosocial workload

Job strain

Work stress is usually defined as job strain according to the model of Karasek *et al.*[35]. The relationship between job strain and 5-min HRV over 48 h, a work day and a rest day, was examined in subjects covering a wide range of occupations in a range of industry sectors[36]. Subjects in high-strain jobs held positions such as letter carrier, laborer, printer, quality technician, and tele/data communications, while subjects in low-strain jobs held positions such as teacher, business agent, retirement specialist, and buyer. Job strain and low decision latitude were associated with decreases in the HF component. Job strain was also associated with increases in the LF/HF ratio during working hours[36]. Among daytime and shift workers, van Amelsvoort *et al.*[37] found a significantly elevated normalized LF component during work in the low demands and low control group, high demands and high control group, and high demands and low control group when compared with a group reporting low job demands and high work control after controlling for age, gender, smoking status, leisure time physical activity, and mean values during sleep. Three studies have been performed on a homogeneous group of healthy workers: one reported no effect of job strain by itself or in interaction with social support on 30-s HRV (RMSSD) over 24 h on a workday and a rest day in female nurses[38]; the second did not find significant differences in parameters of 5-min HRV (SDNN, HF, LF, and VLF components, TP, and LF/HF ratio) during rest between high- and low-strain groups in male workers employed in the shipbuilding industry[39]; and the third found that subjects who complained of poor sleep quality, which is associated with job stress, exhibited a reduced HF component during standing rest, although job stressors were not correlated with HRV in male workers employed by a private company[40].

When chronic work stress was defined according to Siegrist's model of effort-reward imbalance[41] as (a) high imbalance, a combination of high effort and low reward at work, or (b) high overcommitment, an exhaustive work-related coping style indexing the inability to unwind, high imbalance was associated with a lower RMSSD during work, leisure time, and sleep on workdays and a rest day in middle-aged white-collar workers working at the same computer company[42].

Employment

Among healthy clerical civil servants working in the London offices of 20 departments, low employment grade was associated with low HRV (SDNN, and HF and LF components) during rest after controlling for age[43].

Organizational injustice

Organizational justice for women working in long-term care homes was measured using the scale of Moorman[44]. The low perceived justice group showed an 80% excess risk of reduction in the HF component during rest when compared with the high perceived justice group[45].

White-collar office work for hypertensive

The LF/HF ratio during work of white-collar workers with hypertension was significantly higher than that of normotensives[46]. As a result, LF/HF during 24 h including a workday of hypertensives was significantly higher than that of normotensives[46].

Fatigue

One study examined the effects of cumulative fatigue on HRV. The HRV of six garbage collectors was measured twice a week during three consecutive weeks of work, and during the following week of rest[47]. During

the weeks of work, there was a significant progressive decrease in mean RRI, RMSSD, and the HF and LF components, while there was an increase, although not significant, in the LF/HF ratio during nocturnal sleeping. In the resting period, there were significant recoveries in mean RRI, RMSSD, and the HF and LF components of HRV during nocturnal sleeping[47].

Another study examined the effects of overtime and frequent business trips on 5-min HRV during rest in the morning in healthy male workers and found that the HF component was lower in the high vital exhaustion group[48]. Sasaki *et al.* reported increases in the LF/HF ratio during rest and the fatigue (drowsiness, dullness, and feeling of local physical abnormality) complaint rate with increase in working hours, but there were no differences among short (working hours: 53.1 h/wk), medium (working hours: 60.0 h/wk), and long (working hours: 67.1 h/wk) working hour groups[49], and they did not find significant relationships between working hours and mean RRI or the LF/HF ratio during rest in engineers[50].

The HF component during rest was lower in workers with symptoms such as dullness, fatigue, backache, diarrhea, sleep disorders, and irritation than in those without these symptoms and was negatively associated with the presence of any these symptoms once a week or more frequently[51]. Mean HF and LF components during a 24-h shift and on the next two work-free days significantly differed between ambulance personnel with many health complaints and few health complaints[52]. The subjects with many health complaints showed lower HF and higher LF components with less circadian variation during both the work and the work-free days[52].

Train driving

In train drivers, RMSSD decreased during driving when compared with that during sleep, while it increased a little during standing[53].

Working time

Night-shift work

The within-individual effects of night-shift work on HRV have been examined. Two studies[54, 55] compared 24-h HRV between day (working from 0800 to 1700 or from 0600 to 1400) and night (working from 2140 to 0840 or from 2200 to 0600) shifts. All selected HRV parameters in frequency-domain (TP, HF and LF components, and LF/HF ratio) during the awake, work, and sleep periods were not statistically different between shifts. The LF/HF ratio was lower in the sleeping period and higher in the waking and working periods of workers on both shifts[54, 55], and the HF component was higher during the sleeping period and lower in the waking and working periods of workers on both shifts[54, 55]. Among emergency physicians, post-night shift (0700–1500) values for mean RRI, SDNN, and the HF component were significantly higher than both pre- (1500–2300) and mid-night shift (2300–0700) values[56]. The LF/HF ratio during post-night shift was significantly lower than the pre- and mid-night shift values[56].

The LF/HF ratio and the HF component during rest in the evening following day work were significantly larger and smaller, respectively, in nurses working shifts compared to nurses who were not[57]. Shift workers displayed significantly decreased RRI during work and SDNN during sleep at night on the morning shift day, compared with those of the daytime workers[37]. One study reported that among male shift workers, who were employed at a copper-smelting plant, mean RRI, HF component, and LF/HF ratio during rest in the morning on the day shift or work-free days were not different from those of day workers[58].

The HF and LF components during rest showed decreasing trends in accordance with increase in shift work duration[59]. In a longitudinal study, one year changes in HRV were measured in shift workers who started a new job[60]. The HF and LF components on the morning or day shift decreased after 1 yr, but those changes were not different from changes in day workers[60].

24-h shift work

One study found differences in HRV between 24-h shift workdays and work-free days. The circadian rhythms of the LF/HF ratio and normalized HF component of 5-min RRIs were disturbed on a 24-h shift workday, while the rhythm was well preserved on the next work-free day[61]. Although the mean value of the LF/HF ratio and normalized HF component differed significantly between the waking and sleeping times on the work-free day, they did not differ on the workday[61]. In firefighters, the LF/HF ratio of 5-min RRIs during night duty was significantly higher in workers who had to wake up from 0500 to 0700 than in workers who had to wake up from 0315 to 0515[62]. In long-distance truck drivers, during a workday including long distance driving, the LF/HF ratio during 0000–1200 was significantly lower than during 1600–2400[63].

Discussion

Physical and chemical work environment

Particulate matter

Particulate air pollution with $PM_{2.5}$ is associated with several adverse health outcomes, particularly death and hospital admissions from cardiopulmonary disease[64–66]. Although the mechanisms responsible for the cardiac mor-

bidity and mortality associated with particulate air pollution have not been fully elucidated, adverse effects on HRV in response to occupational exposure to $PM_{2.5}$ have been demonstrated[16, 18]. Welding fume, which has a rich content of ultrafine particles (diameter ≤ 0.1 μm) and transition metals, as an occupational source of $PM_{2.5}$ emerged as having an unfavorable influence on HRV[18]. However, some studies found the positive influences on HRV[17, 19, 20], indicating that the mechanisms of the effects of $PM_{2.5}$ on the autonomic system might depend on the characteristics of the content of $PM_{2.5}$, such as substance and diameter.

Carbon disulfide

CS_2 is reported to have a toxic effect on the cardiovascular and autonomic nervous systems[67, 68]. Studies on the effects of occupational exposure to CS_2 on HRV[21, 22] found adverse changes in HRV. The changes last after the termination of the exposure[22]. These adverse effects on the autonomic nervous system can partly explain the fact that chronic exposure to CS_2 increases the mortality rate from myocardial infarction above that of the general population[69, 70].

Lead

Lead exerts toxic effects. Long-term exposure may cause complex neurobehavioral and neuroendocrine abnormalities. Lead inhibits neuronal conduction in the nervous systems, which use dopamine, noradrenaline, serotonin, or acetylcholine as neurotransmitters. In the peripheral nervous system, lead inhibits neurotransmission at the level of autonomic system synapses and neuro-muscle junction[71]. Studies of HRV[24, 25] show that the HF component or the LF/HF ratio was reduced by occupational exposure to lead, indicating that lead damages the vagal nerve more than other cardiac nerves, supporting the hypothesis that lead-induced neuronal alterations are mainly present in long neurons, which have more binding sites, than short ones[71].

Manganese

Manganese is an essential element for humans. However, overexposure to manganese causes irreversible neuromotor damage as in Parkinson's disease. Reduced nerve conduction velocities in manganese-exposed workers were reported in the motor fibers of the median and peroneal nerves[72]. HRV in manganese alloy workers was significantly different from healthy controls[26], indicating that the autonomic nerve function might also be altered by manganese exposure. Occupational exposure to manganese may influence autonomic nerves and have neurotoxic effects.

Smoking

Smoking acutely increases sympathetic activities and increases plasma catecholamine levels[73, 74]. This has been recognized as a major mechanism of CVD in smokers. An increase in the LF/HF ratio immediately after smoking in taxi drivers[27] indicates that sympathomimetoric and parasympatho-withdrawal responses to smoking may play additional roles in increasing cardiac risk and partly explain the high incidence and hospitalization rate of CVD among taxi drivers[75, 76].

Electromagnetic field

The possible influence of electromagnetic fields on the circulatory and nervous systems has been a subject of research for many years, as in theory the autonomic system may be affected by the electric impulses generated by an external EMF. Since the circulatory and nervous systems are composed of electrically excitable tissues, it is plausible that they can be stimulated by EMF. However, studies of HRV have not found clear effects of occupational exposure to EMF on the HF and LF components[28–30]. This might be due to the level of exposure in the occupational settings, which did not exceed admissible values. Wilen *et al.*[29] indicated that an increase in TP and the VLF component of exposed workers might be associated with increased parasympathetic activity due to an adaptation of the thermoregulatory system and cardiac modulation to long-term low-level thermal exposure in RF operators[29].

Vibration

Prolonged vibration exposure can cause vibration-induced white finger, which is one of the most common types of hand-arm vibration syndrome found among operators of hand-held vibratory tools. The blanching attacks are induced by pathological vasospasm of the finger, usually following exposure of the whole body to cold conditions in winter[77]. Such enhanced vasospastic response to cold is considered to result from an exaggerated central sympathetic vasoconstrictor reflex and local changes in the digital vessels[78]. This explanation is supported by the increase seen in the LF/HF ratio during immersion of the hand of workers with white finger in cold water[33]. Workers with hand-arm vibration syndrome showed decreased HF and LF components during rest when compared with healthy workers[31]. In addition, SDNN and RMSSD decreased in workers with hand-arm vibration syndrome without vibration-induced white finger[32]. Hand-transmitted vibration may also cause persistent damage to nerves and blood vessels in the upper limbs or the higher centers of the autonomic nervous system.

Cold environment

One of the health hazards of cold exposure is hypertension[79]. Blood pressure is higher in winter than in summer because of the increase in peripheral resistance at lower environmental temperatures[80], and this may be one of the mechanisms underlying the higher mortality from CVD in winter. Long-term occupational exposure to cold environment increases blood pressure in male workers working in cold areas more than once per day[79]. Bortkiewicz *et al.*[34] also found higher blood pressure in workers with exposure to ambient temperatures varying from 0 to 10°C for 8 h/d than in workers with exposure to ambient temperatures between 10°C and 14°C for 8 h/d. However, the chronic effect of occupational exposure to a cold microclimate on HRV was not significant, indicating that HRV might not be a sensitive marker for capturing the adverse effects of exposure to a cold environment on the cardiovascular system.

Heat and organic solvents

There is a possibility that occupational exposure to heat and some organic solvents, such as n-hexane, xylene, and toluene, effect the cardiovascular system. It has been reported that the additional stimulus of a heated environment during exercise increased sympathetic activity during exercise[81]. Murata *et al.* found a decreased coefficient of variation for the high-frequency component (0.15–0.3 Hz) of HRV in workers exposed to n-hexane, xylene, and toluene[82]. These studies were not included in this review because either the participants were not workers or the dependent variables were not the indices of HRV recommended by the Task Force[5]. Parameters of HRV recommended by the Task Force[5] might be able to detect adverse changes in the cardiovascular systems of workers with occupational exposure to heat and the organic solvents.

Psychosocial workload

Job strain

Work stress relates to the elevation of blood pressure and self-perceived psychological stress and is an independent risk factor of CVD. When subjects consisted of workers from different positions or working conditions, adverse effects on HRV associated with work strain were observed[36, 37], while in a homogeneous group of healthy workers, HRV was not associated with work strain[38–40].

Employment

Low employment grade was associated with adverse changes in HRV[43]. Part of this association is explained by behavioral factors such as smoking, and part by psychosocial factors. In the context of a working population, low job control predicts coronary incidence and may mediate the relationship between low employment grade and adverse changes in HRV.

Organizational injustice

The influence of justice at work on employee health has been gaining attention recently. It has been reported that there is an association between justice at work and employee health[83]. However, the pathways and mechanisms underlying this association are not clear. An increased risk of reduction in the HF component was reported for workers self-reporting low perceived justice[45], indicating that organizational injustice is a risk factor of adverse effects on the cardiovascular system.

White-collar office work for hypertensive

Hypertension is one of the risk factors of CVD. The LF/HF ratio during work of hypertensives was significantly higher than that of normotensive subjects[46], suggesting that hypertension enhances the response of HRV to workload and that workload contributes to the progression of CVD in hypertensives.

Fatigue

Excess fatigue has attracted a great deal of attention as one of the major occupational health problems, especially in Japan. Two studies clearly showed an association between cumulative or excess fatigue and adverse changes in HRV[47, 63]. When the fatigue complaint rate did not differ, the LF/HF ratio during rest also did not differ between workers with short, medium, and long working hours and relationships between working hours and mean RRI or the LF/HF ratio during rest were not statistically significant[50]. Symptoms were associated with adverse changes in HRV[51, 52]. It has been assumed that exhaustion is related to autonomic nervous dysfunction in patient populations. Workers with excess fatigue or intense subjective symptoms might also show objective signs of sympathovagal imbalance.

Train driving

Train driving contains physical and mental workload components[53]. Decreased RMSSD during train driving[53] indicates that physical or mental workload during train driving may cause adverse effects on HRV.

Working time

Night shift work

Several studies have shown altered circadian rhythms of HRV in night shift workers[54–56]. The circadian pattern of HRV and cardiac autonomic activity seem to be predominantly related to the level of physical activity or sleep (supine) and wakefulness (standing), and remains independent of the night-day cycle[54, 55]. The altered cir-

cadian rhythm of HRV during night shift work may last and cause adverse changes in HRV even on a day without a night shift[37, 57]. Furthermore, the longer the night shift work duration is, the smaller the HF and LF components are[59, 60]. These results may explain positive associations between night shift work and CVD[84–86], and between the duration of night shift work and the risk of CVD[87].

24-h shift work

Twenty-four-hour shift work can also disturb the circadian rhythm of HRV[61–63] as the autonomic system associates with wake-sleep and activity-rest rhythms[88]. As shown for night shift workers, a disturbed circadian rhythm of HRV could increase CVD risk. Sato *et al.*[63] found that the LF/HF ratio was lower in the morning than at midnight in 24-h shift truck drivers, suggesting that parasympathetic nervous system activities are more dominant than sympathetic nervous system activities in the morning after midnight work, and that this situation might cause drowsiness during driving in the morning.

Future directions

As CVD can be caused by a number of occupational factors, HRV has been examined in workers from a wide range of occupations. HRV is a useful tool for revealing adverse effects on the cardiovascular system in the occupational setting. Nevertheless, to get a clear and deeper understanding of the effects of work-related factors on HRV, it might be better to consider the following points in the future studies.

For short-term recordings, only four studies controlled or measured the breathing rhythm during the recording of RRI of resting workers. Since respiration potentially influences the HF component[5], breathing rhythm should be measured or controlled when HRV is measured in a controlled circumstance. This would permit identification and exclusion of participants with breathing rhythms outside the HF range.

For long-term recordings, frequency analysis has been performed in 5-min segments, and mean HF and LF components were evaluated in most studies where continuous recording over several hours was performed on workdays or rest days. In frequency analysis of the total record of continuous recordings over several hours, the lower frequency components, i.e. VLF (0.0033–0.04 Hz) and ultralow frequency (ULF, < 0.0033 Hz) components can also be evaluated. These components have not been examined in previous studies concerning occupational health and they might become promising markers according to Bigger *et al.*[10] who demonstrated decreases in the VLF and ULF components as better predictors of all-cause mortality in post-infarction patients. However, it is noted that HRV at frequencies lower than $10^{-3.5}$ ($\approx 3.16 \times 10^{-4}$) Hz (a period > 1 h) are strongly influenced by behavioral factors such as physical activity, sleep-wake cycle, and circadian and ultradian rhythms. Therefore, the ULF component should be examined controlling for behavioral factors. Another approach to adjust for the effects of behavioral factors is to change the frequency divisions of the Task Force[5] and evaluate a "new" VLF ($10^{-3.5}$–0.04 Hz) component suggested by Aoyagi *et al.*[89]. Figure 1 depicts the representative power spectral densities of a healthy young man and an elderly man during waking with normal daily activities. Power for the young man at frequencies higher than $10^{-3.5}$ Hz is higher than that for the elderly man, which might be related to the deterioration of the cardiovascular system due to aging. This implies that the "new" VLF component might be sensitive to undesirable changes in the cardiovascular system. Also, the division of VLF and ULF components at 0.0033 Hz, as suggested by the Task Force[5], is arbitrary.

Finally, the literature on the effects of work-related factors on HRV is predominantly based on cross-sectional studies. To deduce chronic effects on the autonomic system and the causal relationships between the hypothesized exposures and HRV, longitudinal research is highly recommended.

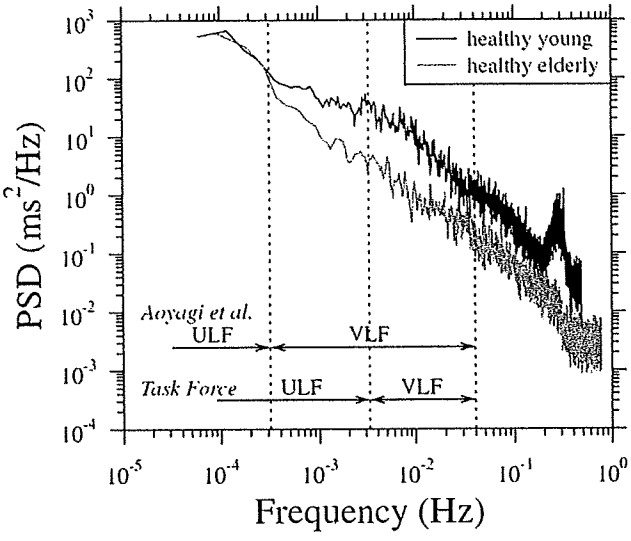

Fig. 1. Examples of power spectral density (PSD) of long-term heart rate variability calculated using a fast Fourier transform during waking with normal daily activity for a healthy young man and a healthy elderly man. Vertical dotted lines indicate boundary frequencies of the very low (VLF) and ultralow frequency (ULF) bands defined in the Task Force report[5] or suggested by Aoyagi *et al.*[89].

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