Original Article

Influence of Mental Stress on Cardiovascular Function as Evaluated by Changes in Energy Expenditure

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We assessed the influence of mental stress on cardiovascular parameters, including systolic blood pressure (SBP), heart rate (HR) and sympathetic activity (LF/HF), with the use of a quantitative indicator of energy expenditure (EE). Forty-four male university students underwent a mental arithmetic test. Their EE was 1.3-fold that at rest in response to the test. Change in EE (*Δ*EE) in response to the test was compared between subjects with high blood pressure (BP) and those without and between subjects with high salt intake and those without. Changes in SBP (*Δ*SBP), changes in HR (*Δ*HR) and changes in LF/HF (*Δ*LF/HF) in relation to *Δ*EE were represented by linear regression. Regression analysis showed that the coefficients of *Δ*EE were positively related to the dependent variables *Δ*SBP, *Δ*HR and *Δ*LF/HF. The slopes of the regression curves for the high-BP group (24-h SBP ≥127 mmHg) and the high-salt–intake group (≥11 g/day) were steeper than those for the normal BP group and the low-salt–intake group (*p*=0.11 and *p*=0.01, respectively). Thus, we were able to determine the influence of mental stress on cardiovascular function. The influence of mental stress on cardiovascular function likely differs according to a subject's environmental conditions. Our study implied that high salt intake increases the sensitivity of cardiovascular functions to mental stress. (*Hypertens Res* 2007; 30: 1019–1027)

***Key Words*: energy expenditure, mental stress, oxygen uptake, salt intake, blood pressure**

Introduction

Mental stress is a risk factor for cardiovascular and cerebrovascular disease (*1*, *2*). Several studies have estimated the influence of mental stress on cardiovascular function (*3*–*5*). Rosengren *et al*. investigated the influence of mental stress on myocardial infarction (*4*). Munakata *et al*. reported that mental stress induced by a doctor's visit increased blood pressure (BP) in patients with essential hypertension (*5*).

When the influence of mental stress is investigated, the stress should be quantified. The number of work hours is often used to quantify job-related stress (*6*). However, the physical and mental effects of a given quantity of work time differ between individuals (*7*). The influence of mental stress may also vary according to a subject's environmental conditions (*7*). Thus, to estimate the influence of mental stress, it is important to determine the quantity of a given stress load.

Energy expenditure (EE) is defined as energy burned to maintain body structures and body temperature and to perform movements and tasks (*8*), and is often used as a quantitative indicator of physical activity (*8*, 9). Based on recent reports that EE can be increased in response to mental stress (*10*–*13*), this parameter can also be used as an index of the

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Table 1. Characteristics of Study Subjects

	All subjects	High-BP group	Normal-BP group
Number	44	14	30
Age (years)	24.1±4.6	23.0±2.2	24.4±4.5
Height (cm)	172.2±4.6	173.2±5.7	171.6±4.1
Weight (kg)	66.9±8.5	72.9±8.8*	63.9±6.9
24-h SBP (mmHg)	122.9±8.5	133.9±6.1*	118.3±5.3
24-h DBP (mmHg)	70.1±8.5	74.3±7.0*	66.5±5.3
24-h salt (g/day)	10.1±3.2	11.7±2.6	9.35± 3.4
	All subjects	High-$Salt_{24}$ group	Normal-$Salt_{24}$ group
Number	44	18	26
High-BP:Normal-BP (number)	14:30	10:8†	4:22
Age (years)	24.1±4.6	23.9±4.6	24.1±3.7
Height (cm)	172.2±4.6	172.8±5.0	171.3±3.8
Weight (kg)	66.9±8.5	69.4±8.3	65.3±8.2
24-h SBP (mmHg)	122.9±8.5	123.4±9.3	121.8±9.8
24-h DBP (mmHg)	70.1±8.5	70.4±7.7	70.0±5.4
24-h salt (g/day)	10.1±3.2	13.2±2.2**	8.3±2.0

Values are expressed as mean±SD. SBP, systolic blood pressure; DBP, diastolic blood pressure. *Differences were analyzed by *t* test. $p<0.05$ High-BP group *vs*. Normal-BP group. †Differences were analyzed by χ^2 test. $p<0.05$ High-$Salt_{24}$ group *vs*. Normal-$Salt_{24}$ group. **Differences were analyzed by paired *t* test. $p<0.05$ High-$Salt_{24}$ group *vs*. Normal-$Salt_{24}$ group.

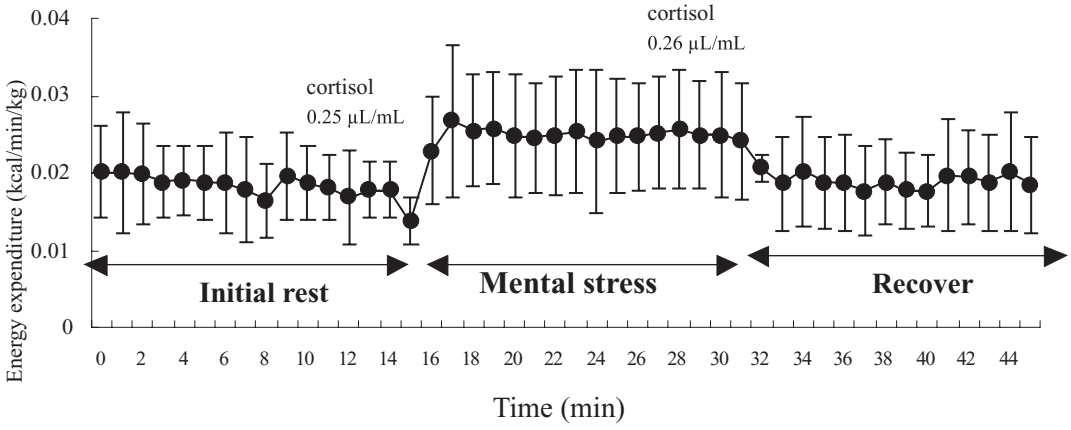

Fig. 1. *Changes in energy expenditure (EE) during the mental stress test.*

effects of mental stress.

We assessed the influence of mental stress on cardiovascular parameters including BP, heart rate (HR) and sympathetic activity with the use of a quantitative indicator of EE. We also investigated whether the influence of mental stress on cardiovascular function differs according to physical condition; the influence of mental stress was compared between subjects with high BP and those without and between subjects with a high salt intake and those without.

Methods

Subjects

Subjects of this study were 44 healthy male volunteers who were medical school students (mean age, 24.1 years; standard deviation, 4.6 years). According to the results of 24-h BP monitoring (24-h BP) conducted prior to a mental stress test, the subjects were categorized into one of two groups, a High-BP group or a Normal-BP group. High BP was defined as a mean 24-h systolic BP (SBP) ≥127 mmHg (*14*).

According to the results of a 24-h urine test with samples collected on the experimental day by means of a portable

Urinemate P (Sumitomo Bakelite Co., Ltd., Tokyo, Japan) (*15*), subjects were also assigned to one of two salt intake groups, a high-sodium–intake group (High-$Salt_{24}$ group) or a normal-sodium–intake group (Normal-$Salt_{24}$ group). The average salt intake per person in Japan is 11–12 g/day, and 90% of ingested salt is excreted in the urine (*16*). We considered 12 g/day of salt intake high and assigned subjects with 24-h urinary sodium excretion ≥11 g to the High-$Salt_{24}$ group. The Ethics Committee of Yokohama City University School of Medicine approved this study, and all subjects provided written informed consent.

Mental Stress Test

Subjects were fitted with an ambulatory BP monitoring (ABPM) device (TM2425-ECG multibiomedical recorder; A&D Co., Ltd., Tokyo, Japan) (*7*) and an indirect portable calorimeter (Metavine-N; Vine Co., Ltd., Tokyo, Japan) (*17*, *18*) and were allowed to rest for 30 min before undergoing a mental stress test (arithmetic test) for 15 min, followed by a 15-min recovery period. The mental stress test involved serial subtraction of 17 from 8,500; thus, subjects calculated 8,500 – 17, 8,483 – 17 and so on (*19*). The saliva cortisol level was determined for each subject at the end of the initial rest period and after the test with a cotton roll (*20*).

Monitoring of Energy Expenditure, Blood Pressure, Heart Rate and Sympathetic Activity

EE for each subject was measured by the minute and calculated from oxygen uptake per min ($\dot{V}O_2$). The $\dot{V}O_2$ volume was determined from expired gas measured with a Metavine-N calorimeter (*17*, *18*). EE was expressed as adjusted $\dot{V}O_2$ according to body weight (*18*, *21*). ECG and HR were recorded beat-to-beat during the mental stress test. BP was measured every 3 min during the test. Low-frequency (LF; 0.04–0.15 Hz) and high-frequency (HF; 0.15–0.40 Hz) components of the ECG-RR interval power spectrum derived from the TM2425-ECG recorder were analyzed for each 5-min period during the test. The LF/HF ratio was regarded as an index of sympathetic activity (*22*). Mean HR, mean SBP and mean LF/HF were calculated for the initial resting period, the stress test and the recovery period. Changes in EE, HR, BP and LF/HF induced by the mental stress test were calculated by subtracting values obtained during the stress test from values obtained during the initial rest period.

Statistical Analysis

Differences between groups were evaluated by Student's *t*-test and χ^2 test. Influences of mental stress, as indicated by changes in EE (ΔEE), on cardiovascular function were investigated by a linear regression model:

$$\Delta X = \alpha + \beta \Delta \mathrm{EE} \qquad (1)$$

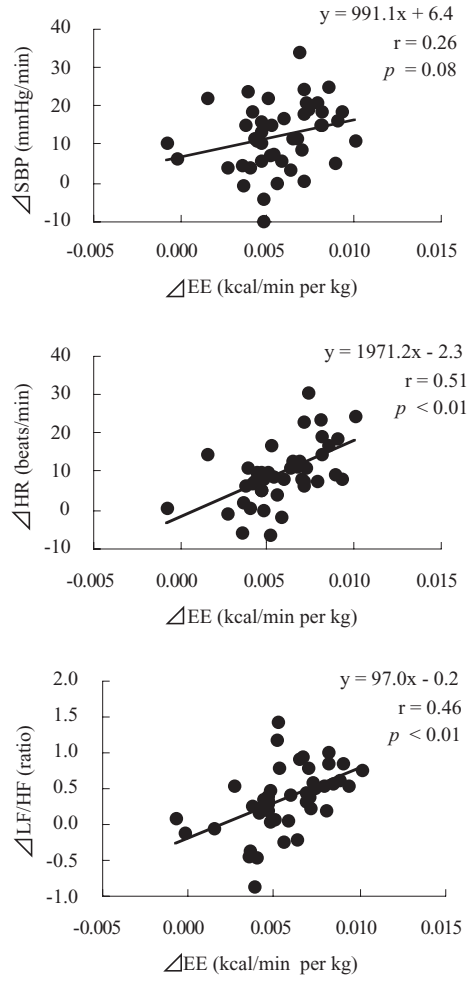

Fig. 2. *Relations between changes in energy expenditure (EE) and changes in systolic blood pressure (SBP), heart rate (HR) and sympathetic activity (LF/HF) for all subjects. ΔEE = mean EE during mental stress – mean EE during initial rest; ΔSBP = mean SBP during mental stress – mean SBP during initial rest; ΔHR = mean HR during mental stress – mean HR during initial rest; ΔLF/HF = mean LF/HF during mental stress – mean LF/HF during initial rest.*

where ΔX is the change in SBP (ΔSBP), change in heart rate (ΔHR), or change in sympathetic activity (ΔLF/HF) induced by the mental stress test; α and β are estimated parameter vectors.

To investigate differences in the sensitivity of cardiovascular parameters to mental stress between the High-BP and Normal-BP groups, a statistical representation of the linear form was determined. The regression equation was

$$\Delta X = \alpha + (\beta_0 + \beta_{\mathrm{HBP}} D_{\mathrm{HBP}}) \Delta \mathrm{EE} + \gamma D_{\mathrm{HBP}}$$

or

$$\Delta X = \alpha + \beta_0 \Delta \mathrm{EE} + \beta_{\mathrm{HBP}} D_{\mathrm{HBP}} \Delta \mathrm{EE} + \gamma D_{\mathrm{HBP}} \qquad (2)$$

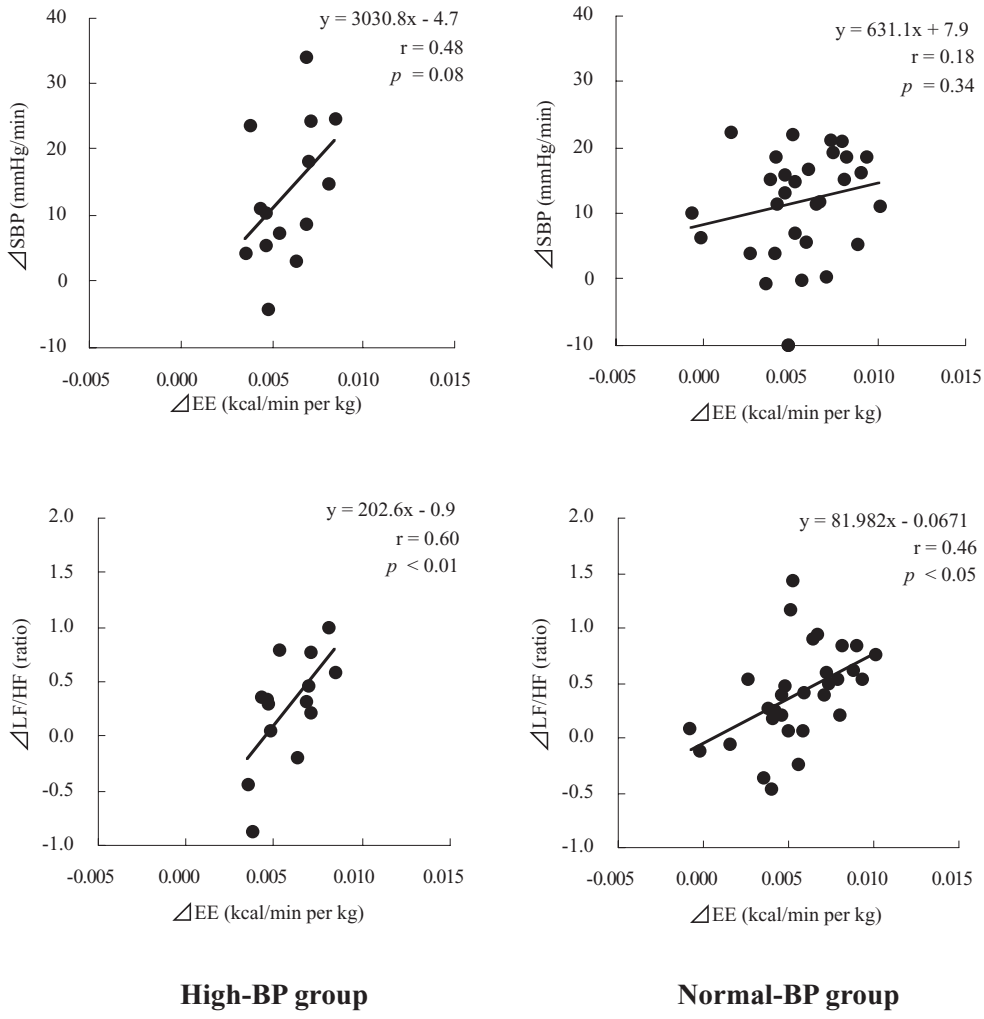

Fig. 3. *Relations between changes in energy expenditure (EE) and changes in systolic blood pressure (SBP) and sympathetic activity (LF/HF). ΔEE = mean EE during mental stress – mean EE during initial rest; ΔSBP = mean SBP during mental stress – mean SBP during initial rest; ΔHR = mean HR during mental stress – mean HR during initial rest; ΔLF/HF = mean LF/HF during mental stress – mean LF/HF during initial rest.*

where $\beta_{HBP}D_{HBP}$ represents a slope coefficient dummy (*23–25*) for subjects in the High-BP group ($D_{HBP}=1$, otherwise $D_{HBP}=0$). The slope of the regression curve for the High-BP group was the sum of the incremental slope coefficient of ΔEE (*i.e.*, β_{HBP}) and the basal slope coefficient of ΔEE (*i.e.*, β_0).

Differences in the sensitivity of cardiovascular parameters to mental stress between the High-Salt$_{24}$ group and the Normal-Salt$_{24}$ group were analyzed similarly with the following regression equation:

$$\Delta X = \alpha + \beta_0 \Delta EE + \beta_{HSL} D_{HSL} \Delta EE + \gamma D_{HSL} \quad (3)$$

where $\beta_{HSL}D_{HSL}$ represents a slope coefficient dummy for subjects in the High-Salt$_{24}$ group. The slope of the regression curve for the High-Salt$_{24}$ group was the sum of the incremental slope coefficient of ΔEE (*i.e.*, β_{HSL}) and the basal slope coefficient of ΔEE (*i.e.*, β_0).

The sensitivity of cardiovascular parameters to mental stress was estimated simultaneously for the BP and salt intake groups. The statistical formula was

$$\Delta X = \alpha + (\beta_0 + \beta_{HBP} D_{HBP} + \beta_{HSL} D_{HSL}) \Delta EE + \gamma D_{HBP} + \delta D_{HSL}$$

or

$$\Delta X = \alpha + \beta_0 \Delta EE + \beta_{HBP} D_{HBP} \Delta EE + \beta_{HSL} D_{HSL} \Delta EE + \gamma D_{HBP} + \delta D_{HSL} \quad (4)$$

Ordinary least squares regression analyses were conducted with SPSS 12.0J software for Windows (SPSS Japan, Tokyo, Japan). Two-tailed tests were applied, and a p value <0.05 was considered statistically significant.

Table 2. Influence of High Blood Pressure on the Relation between Changes in EE and Changes in SBP, HR, and LF/HF

Dependent variable	Independent variable	Slope (β)		p value	95% confidence interval		r^2
					Lower	Upper	
ΔSBP	ΔEE	($=\beta_0$)	563	0.366	−680.3	1,805.9	0.063
	ΔEE High-BP dummy	($=\beta_{HBP}$)	2,615	0.110	−615.3	5,844.5	
ΔHR	ΔEE	($=\beta_0$)	1,886	0.001*	803.8	2,968.8	0.302
	ΔEE High-BP dummy	($=\beta_{HBP}$)	302	0.816	−2,300.3	2,903.7	
ΔLF/HF	ΔEE	($=\beta_0$)	9.3	0.010*	20.0	138.6	0.226
	ΔEE High-BP dummy	($=\beta_{HBP}$)	110	0.158	−44.3	263.9	

EE, energy expenditure; SBP, systolic blood pressure; HR, heart rate; LF/HF, sympathovagal balance. ΔSBP = mean SBP during mental stress test − mean SBP during initial rest; ΔHR = mean HR during mental stress test − mean HR during initial rest; ΔLF/HF = mean LF/HF during mental stress test − mean LF/HF during initial rest; r^2: adjusted multiple coefficient of determination. *$p < 0.05$.

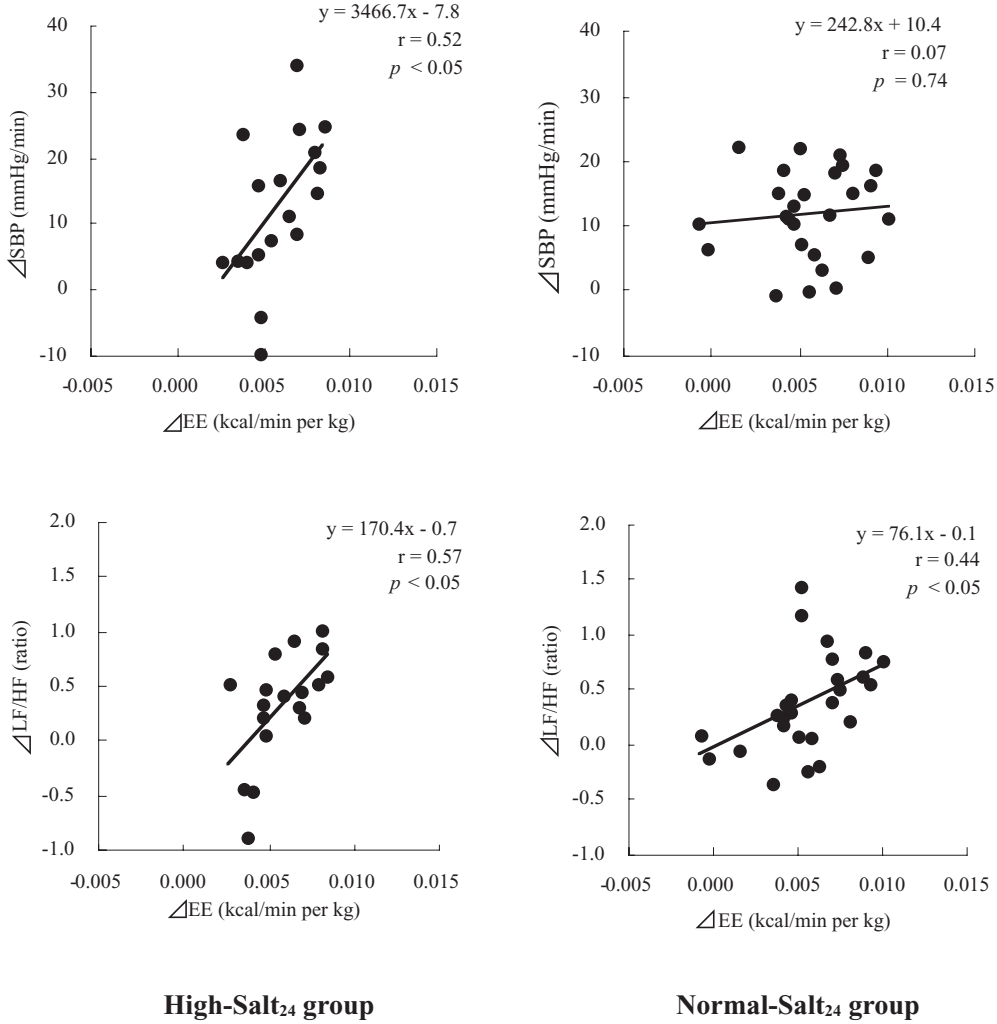

Fig. 4. *Relations between changes in energy expenditure (EE) and changes in systolic blood pressure (SBP) and sympathetic activity (LF/HF). ΔEE = mean EE during mental stress − mean EE during initial rest; ΔSBP = mean SBP during mental stress − mean SBP during initial rest; ΔHR = mean HR during mental stress − mean HR during initial rest; ΔLF/HF = mean LF/HF during mental stress − mean LF/HF during initial rest.*

Table 3. Influence of High Salt Intake on the Relation between Changes in EE and Changes in SBP, HR and LF/HF

Dependent variable	Independent variable	Slope (β)		p value	95% confidence interval		r^2
					Lower	Upper	
ΔSBP	ΔEE	($=\beta_0$)	179	0.772	−1,061.0	1,419.7	0.150
	ΔEE High-Salt$_{24}$ dummy	($=\beta_{HSL}$)	3,510	0.010*	889.4	6,131.0	
ΔHR	ΔEE	($=\beta_0$)	1,663	0.005*	533.7	2,792.0	0.269
	ΔEE High-Salt$_{24}$ dummy	($=\beta_{HSL}$)	1,044	0.345	−1,163.8	3,252.0	
ΔLF/HF	ΔEE	($=\beta_0$)	73.7	0.023*	10.5	136.8	0.200
	ΔEE High-Salt$_{24}$ dummy	($=\beta_{HSL}$)	91.8	0.172	−41.7	225.3	

EE, energy expenditure; SBP, systolic blood pressure; HR, heart rate; LF/HF, sympathovagal balance. ΔSBP = mean SBP during mental stress test − mean SBP during initial rest; ΔHR = mean HR during mental stress test − mean HR during initial rest; ΔLF/HF = mean LF/HF during mental stress test − mean LF/HF during initial rest; r^2: adjusted multiple coefficient of determination. *$p<0.05$.

Table 4. Influence of High Blood Pressure and High Salt Intake on the Relations between Changes in EE and Changes in SBP, HR and LF/HF

Dependent variable	Independent variable	Slope (β)		p value	95% confidence interval		r^2
					Lower	Upper	
ΔSBP	ΔEE	($=\beta_0$)	174.4	0.784	−1,104.6	1,453.3	0.107
	ΔEE High-BP dummy	($=\beta_{HBP}$)	193.5	0.923	−3,846.5	4,233.6	
	ΔEE High-Salt$_{24}$ dummy	($=\beta_{HSL}$)	3,375.8	0.055	−69.3	6,820.9	
ΔHR	ΔEE	($=\beta_0$)	1,671.2	0.006	514.2	2,828.3	0.244
	ΔEE High-BP dummy	($=\beta_{HBP}$)	−599.7	0.715	−3,898.1	2,698.6	
	ΔEE High-Salt$_{24}$ dummy	($=\beta_{HSL}$)	1,449.5	0.308	−1,390.3	4,289.3	
ΔLF/HF	ΔEE	($=\beta_0$)	71.7	0.028	8.1	135.2	0.200
	ΔEE High-BP dummy	($=\beta_{HBP}$)	63.4	0.527	−137.8	263.9	
	ΔEE High-Salt$_{24}$ dummy	($=\beta_{HSL}$)	66.5	0.437	−104.7	237.8	

ΔSBP = mean SBP during mental stress test − mean SBP during initial rest; ΔHR = mean HR during mental stress test − mean HR during initial rest; ΔLF/HF = mean LF/HF during mental stress test − mean LF/HF during initial rest; r^2: adjusted multiple coefficient of determination.

Results

Characteristics of the subjects in each group are summarized in Table 1. Fourteen subjects were assigned to the High-BP group, and 30 subjects were assigned to the Normal-BP group. Eighteen subjects, including 10 subjects with high BP, were assigned to the High-Salt$_{24}$ group. The remaining 26 subjects, including 4 subjects with high BP, were assigned to the Normal-Salt$_{24}$ group.

The mean 24-h SBP, mean 24-h diastolic BP (DBP) and mean weight were significantly greater in the High-BP group than in the Normal-BP group. There was no significant difference between these groups with respect to age or height. There was no significant difference between the High-Salt$_{24}$ group and the Normal-Salt$_{24}$ group with respect to age, height or weight, although the proportion of High-Salt$_{24}$ subjects who belonged to the High-BP group was significantly greater than the proportion of Normal-Salt$_{24}$ subjects who belonged to the High-BP group ($p=0.008$).

Mean EE values before, during and after the mental stress test are shown in Fig. 1. Increased EE was observed during the stress period, from 0.018 kcal/min per kg to 0.023 kcal/min per kg; the difference was statistically significant ($p<0.001$). In total, EE was increased 1.3-fold compared to baseline in response to mental stress. SBP, HR and LF/HF were also increased in response to mental stress. SBP was increased an average of 11.4 mmHg ($p<0.001$), HR was increased an average of 8.4 beats/min ($p<0.001$) and LF/HF was increased an average of 0.33 ($p<0.001$). Saliva cortisol was increased 0.029 μg/mL in response to mental stress, but statistical significance was not achieved ($p=0.140$).

The quantity of stress induced by the stress test is indicated by ΔEE in the x-axis in Fig. 2. All of the slope coefficients of ΔEE estimated by Eq. (1) were positively related to the dependent variables ΔSBP, ΔHR and ΔLF/HF; statistical significance was achieved for the regression analyses of ΔHR ($p<0.001$) and ΔLF/HF ($p=0.002$).

Relations between ΔEE and cardiovascular parameters for the High-BP and Normal-BP groups are shown in Fig. 3. The slopes of the regression curves were steeper in the High-BP group than in the Normal-BP group, indicating that the car-

diovascular parameters of subjects with high BP are more sensitive to mental stress than those of subjects with normal BP. Regression analysis with Eq. (2) was conducted to investigate whether the slopes of the regression curves for the High-BP group were significantly steeper than those for the Normal-BP group (Table 2). The incremental coefficient of ΔEE by high BP (*i.e.*, β_{HBP} in Eq. (2)) was positively related to ΔSBP, but the association was not statistically significant ($p=0.110$). The incremental coefficient of ΔEE by high BP was also not significantly correlated with ΔHR ($p=0.816$) or ΔLF/HF ($p=0.158$).

Relations between ΔEE and cardiovascular parameters are shown for the High-$Salt_{24}$ and Normal-$Salt_{24}$ groups in Fig. 4. The slopes of the regression curves were steeper in the High-$Salt_{24}$ group than in the Normal-$Salt_{24}$ group. Regression analysis with Eq. (3) was conducted to investigate whether the slopes of the regression curves for the High-$Salt_{24}$ group were significantly steeper than those for the Normal-$Salt_{24}$ group (Table 3). The incremental coefficient of ΔEE by high salt intake (*i.e.*, β_{HSL} in Eq. (3)) was significantly related to ΔSBP ($p=0.010$); thus, the slopes of the regression curves for ΔSBP were significantly steeper in the High-$Salt_{24}$ group than in the Normal-$Salt_{24}$ group.

The proportion of subjects in the High-BP group who belonged to the High-$Salt_{24}$ group differed significantly from the proportion of subjects in the High-BP group who belonged to the Normal-$Salt_{24}$ group (Table 1). Because high salt intake might be confounded with high BP, we conducted multiple regression analysis with dummy values for high salt intake and high BP (Eq. (4)) (Table 4). The results showed that salt intake had a larger influence than BP with respect to the relation between mental stress and cardiovascular parameters. The incremental coefficient of ΔEE by high salt intake (*i.e.*, β_{HSL} in Eq. (4)), with the dependent variable ΔSBP was greater than the incremental coefficient of ΔEE by high BP, although statistical significance was not achieved ($p=0.055$). The incremental coefficient of ΔEE by high salt intake was also greater than that by high BP (*i.e.*, β_{HBP}) in Eq. (4) with the dependent variables ΔHR and ΔLF/HF, but these relations also were not statistically significant.

Discussion

To assess the influence of mental stress on cardiovascular function, it is important to determine the quantity of stress received from a mental load rather than the quantity of load given. BP, HR, sympathetic activity, and catecholamine and cortisol levels are often used as indicators of the quantity of stress received (*5*, *7*, *10*, *16*, *19*, *20*). However, BP, HR and sympathetic activity are direct indicators of cardiovascular function. In addition, they have feedback systems for stabilization and tolerance to stress, which can alter measurements of mental stress (*12*). To minimize the impact of these factors, we retested 9 of the subjects on another day. The change of SBP in the second stress test was significantly smaller than the change of EE in the first stress test, and the change of EE was not significantly different between the first and second stress tests. Use of plasma catecholamine and plasma cortisol as indicators of mental stress can also alter results because continuous measurement by blood sampling can increase stress. Measurement of saliva cortisol may be a useful indicator of mental stress (*20*, *21*). However, in the present study, saliva cortisol was found to be unsuitable because of large variations between study subjects.

In the present study, we used change in EE (ΔEE) as an indicator of mental stress and estimated the influence of mental stress on cardiovascular function. EE is not a direct indicator of cardiovascular function and is free of feedback systems, in contrast to HR and BP. Recent studies have shown that EE is increased in response to mental stress (*10–13*) and in response to administration of epinephrine (*26*). In the present study, EE was affected by mental stress (Fig. 1). EE was increased 1.3-fold compared to baseline in response to mental stress, similar to the results of Seematter *et al*. (*10*, *11*). In the present study, we described the relations between ΔEE and cardiovascular parameters including BP, HR and LF/HF. The value of ΔEE induced by the mental stress test was positively related to all three parameters.

Our results indicate that the influence of mental stress on cardiovascular function differs according to a subject's physical condition. Several studies have reported that the mental stress–induced increase in BP is greater in subjects with essential hypertension than in subjects with normal BP (*27*, *28*). However, whether the sensitivity of BP to mental stress differs between subjects with borderline hypertension and those with normal BP has not been determined conclusively (*29–31*). Our results did not show a statistically significant difference in the sensitivity of SBP to mental stress between subjects with high BP and those with normal BP.

There have been a limited number of studies investigating the response of BP to mental stress under different conditions of salt intake (*31*, *32*), and the influence of salt intake on the sensitivity of BP to mental stress has not been determined. Falkner *et al*. (*31*) reported that salt intake significantly increased stress-induced SBP and DBP in female adolescents with a family history of hypertension and resulted in no change in cardiovascular response to stress in female adolescents without a family history of hypertension. West *et al*. (*32*) reported that mental stress increased BP, but the influence of mental stress on BP did not differ according to the level of salt intake. In the present study, we investigated the influence of high salt intake on the sensitivity of cardiovascular parameters to mental stress by categorizing our subjects into two groups. A statistically significant influence of high salt intake on the sensitivity of SBP to mental stress was observed in the high-salt–intake group ($p=0.010$, Table 3). However, when the value was adjusted by the factor of high BP, the influence was not statistically significant ($p=0.055$, Table 4). This implies that restriction of salt intake is important for the control of BP at rest as well as under conditions of

mental stress. However, to clarify the influence of high salt intake on the sensitivity of BP to mental stress, further research is required.

There are limitations to this study. First, we investigated the influence of high salt intake on the sensitivity of cardiovascular parameters to mental stress between two groups. To clearly show the influence of high salt intake, it should be evaluated in the same subjects under conditions of low and high salt intake. Second, the sample size was not sufficiently large to clarify the influence of high salt intake and high BP on the sensitivity of cardiovascular parameters to mental stress. Further research is needed in this regard as well.

In summary, we used the change of EE as an indicator of a given stress load and assessed the influence of mental stress on cardiovascular function in healthy young subjects. EE was increased by mental stress and correlated with cardiovascular function. The influence of mental stress on cardiovascular function likely differs according to the physical condition of subjects. Our study implies that high salt intake increases the sensitivity of cardiovascular functions to mental stress; however, further research is needed.

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