INTRODUCTION

Recent epidemiological and clinical studies have shown that not only mean blood pressure (BP) levels but also BP variability (BPV) is associated with cardiovascular target organ damage and incident cardiovascular disease outcomes in general populations. Among several BPV parameters, short-term BPV is affected by various types of intrinsic factors, such as increased central sympathetic drive and reduced arterial and cardiopulmonary reflexes, increased arterial stiffness, and humoral and genetic factors. In general, BP levels tend to increase in response to physical

Abstract

There are no studies assessing short-term blood pressure (BP) changes induced by daily exercise load in young trained individuals. The authors enrolled 25 healthy, trained (mean age 19.7 ± 0.1 years, 36% female) and 26 healthy, untrained (mean age 20.4 ± 0.3 years, 50% female) individuals and measured BP after the Master two-step test. Among them, 42 individuals underwent echocardiography after BP measurements to assess left ventricular mass index (LVMI). The baseline systolic BP (SBP) levels of trained and untrained individuals were 122.7 ± 2.9 versus 117.4 ± 1.5 mmHg, respectively (p = .016). Trained individuals showed a significant suppression of the SBP increase soon after exercise loads and lower SBP levels at 1, 2, and 3 min after exercise loads compared with untrained individuals. The peak SBP level over the study period was also significantly lower in trained individuals than in untrained individuals: 156.4 ± 3.3 versus 183.7 ± 5.2 mmHg (p < .001). Trained individuals showed significantly higher LVMI compared with untrained individuals: 129.4 versus 101.6 g/m² (p < .001). These findings demonstrated that trained individuals showed significant suppression of short-term BP variability in response to daily exercise loads and prompt SBP recovery from acute exercise loads compared with untrained individuals. Our results would be useful to understand short-term BPV and LV hypertrophy induced by adaptive responses of the heart to regular exercise loads.
exercise, and the degree of increase in BP by exercise loads may depend on differences in age, sex, type of exercise, and exercise tolerance. Exercise is recommended not only for patients with hypertension but also for all individuals other than those with normal BP, and an increase of physical performance by continued exercise lowers the prevalence of various chronic diseases and mortality. In regard to the association between exercise and exercise-induced BPV, previous studies assessed the BP changes by maximal exercise loads in trained individuals. Those studies demonstrated that trained individuals showed higher BP responses by maximal exercise, which in turn indicated that the trained individuals might have an adaptive response that was associated with increased cardiac output. In addition, trained individuals have been reported to exhibit physiological cardiac hypertrophy by adaptive responses. On the other hand, there have been no studies assessing the BP changes induced by daily exercise loads, rather than maximal exercise, in trained individuals. If the clinical implications of short-term BPV induced by daily exercise levels were clarified, the results would be useful to elucidate the adaptive response to exercise loads in trained individuals.

In the present study, we investigated the BP changes by daily exercise loads between trained and untrained individuals. We hypothesized that trained individuals would show lesser short-term BPV in response to daily exercise loads compared with untrained individuals via an adaptive cardiac response.

2 METHODS

2.1 Study participants

Fifty-five healthy participants were recruited at the Takasaki University of Health and Welfare, Gunma Prefecture, Japan. We defined the participants who had belonged to athletic clubs and trained for more than 2 h a day more than 4 days a week as trained individuals, and those who did not belong to any athletic club and had no exercise habits as untrained individuals. The ethics committee of the Takasaki University of Health and Welfare (Takasaki, Japan) approved the study.

2.2 Blood pressure measurements

We measured ambulatory BP with a validated automatic device (TM-2441; A&D Co.) that recorded the participant’s BP using an oscillometric method. One BP reading was taken with participants in a seated position. After that, all participants underwent a Master two-step test. We measured the BP levels immediately after the exercise load, and at 1, 2, and 3 min after the exercise load, for a total of four measurements in each participant.

In this study, we defined (1) the peak systolic BP (SBP) as the maximum SBP value measured over the whole study period; (2) SBP reactivity as the difference between the peak SBP and baseline SBP; and (3) SBP recovery as the difference between SBP measured 3 min after exercise and baseline SBP.

2.3 Echocardiography

After exercise loads, echocardiography was performed by two cardiologists (YI and MT) in 42 participants (17 trained individuals and 25 untrained individuals). The two-dimensional B-mode images were recorded using an ultrasound machine according to the guidelines of the American Society of Echocardiography and the European Association of Echocardiography. The LVM was obtained using the formula validated by the American Society of Echocardiography:

\[
LVM = 0.8 \times (1.04 \left( [LVd + PWT + SWT]^3 - LVd^3 \right) + 0.6 \text{g, where the } LVd \text{ is LV diameter in diastole, PWT is the posterior wall thickness in diastole, and SWT is the septal wall thickness in diastole.}
\]

The LVM was calculated using the formula LVM/BSA (g/m²), where BSA is body surface area.

2.4 Statistical analysis

All statistical analyses were performed using SPSS software ver. 24.0 (SPSS). Descriptive statistics are presented as means, standard error of the means (SEM), and proportions, where appropriate. The Wilcoxon’s rank-sum test was used to compare clinical and echocardiographic parameters between trained and untrained individuals. All statistical tests were performed using a two-tailed design with a significance level (p-value) of <.05.

3 RESULTS

The characteristics of the study participants are shown in Table 1. The mean age of study participants was 19.9 ± 0.1 years, 44% of participants were female, and 49% and 51% were trained and untrained individuals, respectively. Baseline SBP levels in trained individuals were significantly higher than those in untrained individuals (Table 1).

Figure 1 shows the differences in SBP changes after exercise loads between trained and untrained individuals. In the majority of individuals, regardless of whether trained or untrained, SBP was highest soon after exercise loads, and the SBP value gradually decreased after 1, 2, and 3 min. The increase in SBP due to the exercise loads of trained individuals was suppressed compared with that of untrained individuals (Figure 1).

The details of the SBP changes after exercise loads are shown in Table 1. After the exercise loads, SBP was significantly lower in trained individuals than untrained individuals at each time point, that is, soon after exercise, and 1, 2, and 3 min after exercise. The SBP reactivity was significantly lower in trained individuals than untrained individuals (33.6 ± 3.3 vs. 67.2 ± 4.8 mmHg, p < .001). The SBP recovery of trained
individuals was also significantly lower in trained individuals than untrained individuals (3.4 ± 2.2 vs. 30.6 ± 5.1 mmHg, *p* < .001).

A total of 42 participants were eligible for the analysis of LVMI: 17 trained individuals and 25 untrained individuals. Table 2 shows the differences of echocardiographic parameters between trained and untrained individuals. The trained individuals showed significantly higher SWT, LVDd, and LV diameter in systole (LVDs). The trained individuals showed significantly higher LVMI compared with untrained individuals (Figure 2). When this result was analyzed by sex, the LVMI elevation in trained versus untrained individuals was similar in men and women.

### TABLE 1 Differences in exercise-induced changes of SBP between trained and untrained individuals

|                      | Entire group (n = 51) | Trained individuals (n = 25) | Untrained individuals (n = 26) | *p*-value |
|----------------------|-----------------------|-----------------------------|-------------------------------|-----------|
| Age, years           | 19.9 ± 0.1            | 19.7 ± 0.1                  | 20.4 ± 0.3                    |           |
| Male, n (%)          | 29 (56)               | 16 (64)                     | 13 (50)                       |           |
| BMI                  | 21.9 ± 0.3            | 22.4 ± 0.3                  | 21.7 ± 0.5                    |           |
| BP parameters, mmHg  |                       |                             |                               |           |
| Baseline SBP         | 119.6 ± 1.6           | 122.7 ± 2.9                 | 117.4 ± 1.5                   | .016      |
| SBP just after exercise | 169.3 ± 4.4           | 154.2 ± 4.6                 | 180.1 ± 5.9                   | .003      |
| SBP 1 min after exercise | 155.9 ± 3.7           | 142.8 ± 3.6                 | 165.1 ± 5.1                   | .002      |
|                      |                       |                             |                               |           |
| SBP 2 min after exercise | 145.4 ± 3.4           | 131.9 ± 3.6                 | 154.9 ± 4.3                   | <.001     |
| SBP 3 min after exercise | 138.3 ± 3.9           | 125.2 ± 3.1                 | 147.5 ± 5.6                   | <.001     |
| Peak SBP             | 169.6 ± 4.0           | 156.4 ± 3.3                 | 183.7 ± 5.2                   | <.001     |
| SBP reactivity       | 49.8 ± 4.2            | 33.6 ± 3.3                  | 67.2 ± 4.8                    | <.001     |
| SBP recovery         | 16.5 ± 3.7            | 3.4 ± 2.2                   | 30.6 ± 5.1                    | <.001     |

Note: All values are shown as the mean ± SEM. Wilcoxon’s rank-sum test was used to evaluate statistical differences of SBP mean under various conditions between trained and untrained individuals.

Abbreviations: BMI, body mass index; BP, blood pressure; SBP, systolic blood pressure; SEM, standard error of the mean.

### FIGURE 1 Differences in exercise-induced changes of systolic blood pressure (SBP) between trained and untrained individuals. SBP values before exercise, immediately after exercise, and 1, 2, and 3 min after exercise were plotted, and the plotted points were connected in each participant.

4 | DISCUSSION

In this observational study on short-term BPV induced by daily exercise loads in trained and untrained individuals, we demonstrated that BP increases in response to exercise loads were significantly suppressed in trained individuals compared with untrained individuals. In addition, trained individuals showed significantly faster BP recovery from acute exercise loads compared with untrained individuals. In echocardiography examination, the prevalence of cardiac
Hypertrophy was higher in trained individuals than untrained individuals. This is the first study to show that short-term BPV induced by daily exercise loads was suppressed in trained individuals. The results of this study should help elucidate the physiological mechanisms of short-term BPV.

We have shown that trained individuals exhibited a smaller BP increase immediately after acute exercise loads compared with untrained individuals. SBP reactivity, an index of the magnitude of BPV provided by acute exercise loads, was significantly lower in trained individuals than untrained individuals. In addition, SBP recovery, an index of the rate of BP recovery from acute exercise loads, was much faster in trained individuals compared with untrained individuals, which means that the ability of trained individuals to recover from acute exercise loads was extremely high. These results indicated that BP increase due to sympathetic nervous activation would be suppressed in trained individuals compared with untrained individuals. Generally, the autonomic nervous system elicits a cardiovascular response by an increase in sympathetic nerve activity, which causes the increase in BP during exercise. Regular dynamic exercise increases the aerobic capacity and the oxygen consumption of skeletal muscles, which is followed by a mitigated response of the cardiovascular system to catecholamines at rest and on exercise. The mechanism of this response was reported to be due to the decreases of both myocardial β-adrenergic and α-adrenergic receptor density and sensitivity. Therefore, the expected increase in BP immediately after acute exercise loads, including SBP reactivity, would be reduced in trained individuals, and they would show faster BP recovery when compared with untrained individuals. We also found that the increase of circulating blood flow to skeletal muscle during exercise was due to the vasodilation of muscles, the increase of cardiac output and the suppression of blood flow to inactive sites such as the skin and internal organs. Trained individuals would have ability to coordinate vasodilation of the skeletal muscles and changes in cardiovascular function by regular exercise, which would lead to suppression of the increase in BP during and after exercise. These adaptive responses allow trained individuals to efficiently increase blood flow to active muscles.

In this study, we assessed BP response by exercise loads using the Master two-step test. Such exercise loads level are close to the exercise loads in everyday life, and they would be low-grade for trained individuals, causing the BP increase to be suppressed more markedly in trained individuals compared with untrained individuals.

The present study revealed that trained individuals showed a significantly higher LVMI compared with untrained individuals. This result was also confirmed in both sexes. These structural changes are adaptations to regular exercise training, commonly known as athlete’s heart. The athlete’s heart is a physiological condition that can be defined as a morphological consequence of systematic training in athletes with the following features: increase in maximal cardiac output, increase in stroke volume, decrease in resting heart rate, and electrocardiographic changes in conduction and repolarization. Adaptive physiological hypertrophy in athletes was not associated with cardiac dysfunction, cardiac fibrosis, or the development of heart failure.

### TABLE 2 Differences in echocardiographic parameters between trained and untrained individuals

| Parameter          | Trained individuals (n = 17) | Untrained individuals (n = 25) | p-value |
|--------------------|-----------------------------|--------------------------------|---------|
| SWT, mm            | 8.2 ± 0.2                   | 7.9 ± 0.2                      | .013    |
| PWT, mm            | 8.1 ± 0.3                   | 7.6 ± 0.2                      | .211    |
| LVDD, mm           | 48.0 ± 0.9                  | 43.9 ± 1.2                     | .037    |
| LVDS, mm           | 30.8 ± 1.0                  | 27.9 ± 0.8                     | .030    |
| LVEF, %            | 65.0 ± 2.0                  | 65.8 ± 1.3                     | .602    |

Note: All values are shown as the mean ± SEM. Wilcoxon's rank-sum test was used to evaluate statistical differences of echocardiographic parameters between trained and untrained individuals.

Abbreviations: LVDD, left ventricular diameter in diastole; LVDS, left ventricular diameter in systole; LVEF, left ventricular ejection fraction; PWT, posterior left ventricular wall thickness; SEM, standard error of the mean; SWT, septal wall thickness in diastole.

### FIGURE 2 Differences in left ventricular mass index between trained and untrained individuals.

Wilcoxon's rank-sum test was used to evaluate statistical differences between them.
of heart failure.25,26 Fundamental aspects of physiological cardiac hypertrophy are that it increases muscle mass by increasing cardiac myocyte size17 and that it is reversible following detraining.27

In terms of the pathological response, although increased LVM is initially an adaptive response that allows the heart to maintain cardiac output, chronic high BP level or volume overload can lead to further remodeling, which involves dilatation of the left ventricle, cardiac fibrosis, and loss of cardiac myocytes.28 These changes reduce cardiac output and contribute to the progression to heart failure.29

Generally, pathological LV hypertrophy (LVH) is significantly associated with increased BPV.30 However, the present study demonstrated that trained individuals showed higher physiological LVM than untrained individuals, and also showed decreased BPV compared with untrained individuals. These differences would be due to the mechanism of LVH pathogenesis—in particular, they might reflect the degree of myocardial fibrosis. Further studies are needed to elucidate the association between physiological or pathological LVH and BPV increase.

The present study has some limitations. First, we included a small number of study participants. Second, we performed the Master exercise test and subsequent BP measurements only once. The reproducibility of these results cannot be evaluated. Lastly, our findings may not be generalizable to other age groups.

5 | CONCLUSIONS

We have demonstrated that trained individuals showed significantly greater suppression of short-term BPV in response to daily exercise loads and faster SBP recovery from acute exercise loads compared with untrained individuals. LVM was significantly higher in trained individuals than in untrained individuals. Our results would be useful to assess the difference between physiological and pathological BPV and LVH. Further large-scale studies are warranted to elucidate the hemodynamic and morphological changes to exercise in trained individuals.

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CONFLICT OF INTEREST

None.

AUTHORS CONTRIBUTION

Kazumi Kario had the primary responsibility of writing this paper. Takeshi Fujiwara wrote the essential part of the manuscript. Kenji Nakamura, Satoshi Hoshide, and Seiji Ozawa reviewed/edited the manuscript. Yusuke Ishiyama and Mizuri Taki collected echocardiographic parameters.

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