Received: 19 October 2020 | Revised: 10 December 2020 | Accepted: 28 December 2020

DOI: 10.1111/jch.14177

ORIGINAL PAPER

WILEY

Differences in exercise-induced blood pressure changes between young trained and untrained individuals

Kenji Nakamura PhD[1] | Takeshi Fujiwara MD, PhD[2] | Satoshi Hoshide MD, PhD[2] | Yusuke Ishiyama MD, PhD[2] | Mizuri Taki MD, PhD[2] | Seiji Ozawa MD, PhD[1] | Kazuomi Kario MD, PhD[2]

[1]Department of Healthcare Informatics, Faculty of Health and Welfare, Takasaki University of Health and Welfare, Takasaki, Japan

[2]Division of Cardiovascular Medicine, Department of Medicine, Jichi Medical University School of Medicine, Shimotsuke, Japan

Correspondence
Kazuomi Kario, Division of Cardiovascular Medicine, Department of Medicine, Jichi Medical University School of Medicine, 3311-1, Yakushiji, Shimotsuke, Tochigi 329-0498, Japan.
Email: kkario@jichi.ac.jp

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/m^2 (p < .001). These findings demonstrated that trained individuals showed significant suppression of short-term BP variability in response to by 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.

1 | INTRODUCTION

Recent epidemical 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.[1-4] 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,[5] increased arterial stiffness,[6,7] and humoral[8] and genetic factors.[9] In general, BP levels tend to increase in response to physical

Kenji Nakamura and Takeshi Fujiwara contributed equally to the present study.

exercise,[10] 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,[11] and an increase of physical performance by continued exercise lowers the prevalence of various chronic diseases[12] and mortality.[13] In regard to the association between exercise and exercise-induced BPV, previous studies assessed the BP changes by maximal exercise loads in trained individuals.[14,15] 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.[16,17] 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 defied (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.[18] The LVM was obtained using the formula validated by the American Society of Echocardiography: $LVM = 0.8 \times (1.04\ [(LVDd + PWT + SWT)^3 - LVDd^3]) + 0.6$ g, where the LVDd is LV diameter in diastole, PWT is the posterior wall thickness in diastole, and SWT is the septal wall thickness in diastole. The LVMI was calculated using the formula LVM/BSA (g/m^2), 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

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 exercis exercise 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.

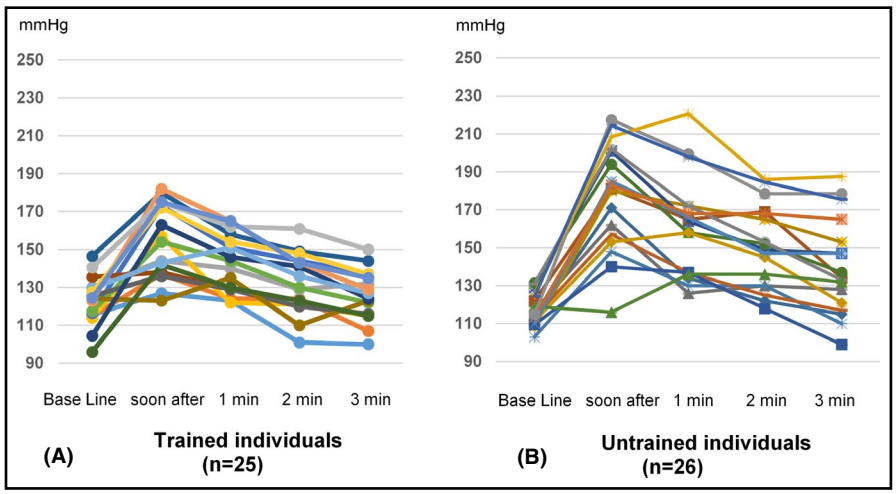

FIGURE 1 Differences in exercise-induced changes of systolic blood pressure (SBP) between trained and untrained individuals. SBP values before exercise, immediately after exercise, and 1, 2, and 3 min after exercise were plotted, and the plotted points were connected in each participant

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.

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

TABLE 2 Differences in echocardiographic parameters between trained and untrained individuals

	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.

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 to 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.[19] 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.[20] The mechanism of this response was reported to be due to the decreases of both myocardial β-adrenergic[21] and α-adrenergic[22] 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.[23] 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.[24] Adaptive physiological hypertrophy in athletes was not associated with cardiac dysfunction, cardiac fibrosis, or the development

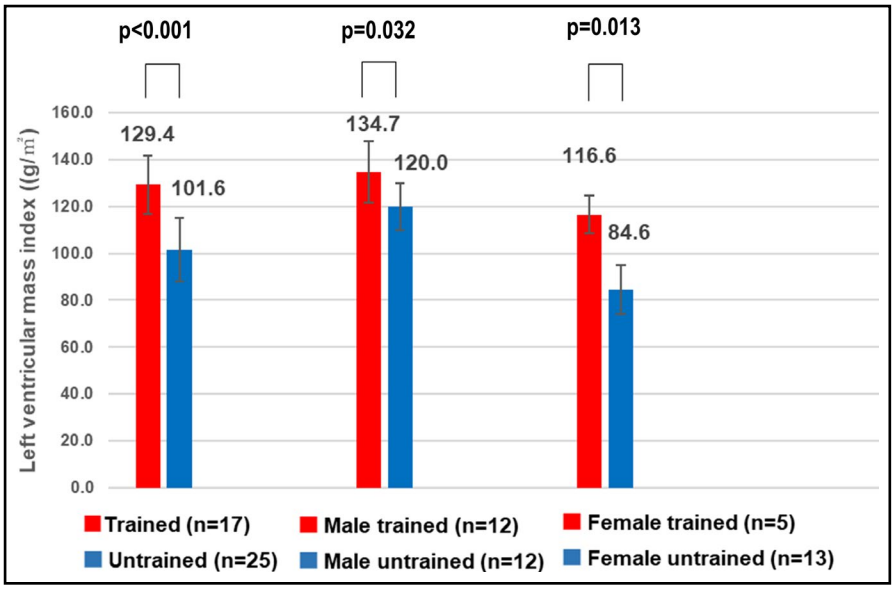

FIGURE 2 Differences in left ventricular mass index between trained and untrained individuals. Wilcoxon's rank-sum test was used to evaluate statistical differences between them

of heart failure.[25,26] Fundamental aspects of physiological cardiac hypertrophy are that it increases muscle mass by increasing cardiac myocyte size[17] 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 LVMI 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. LVMI 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.

ACKNOWLEDGEMENTS

We gratefully acknowledge all participants that took part in this study, as well as investigators involved in the study, Ms. Naoko Tomitani for the coordination and data management of this study, and Ms. Yukie Okawara and Ms. Ayako Okura for editorial assistance.

CONFLICT OF INTEREST

None.

AUTHORS CONTRIBUTION

Kazuomi 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.

ORCID

Takeshi Fujiwara https://orcid.org/0000-0003-4151-2806
Satoshi Hoshide https://orcid.org/0000-0001-7541-5751
Kazuomi Kario https://orcid.org/0000-0002-8251-4480

REFERENCES

1. Rothwell PM, Howard SC, Dolan E, et al. Prognostic significance of visit-to-visit variability, maximum systolic blood pressure, and episodic hypertension. *Lancet*. 2010;375:895-905.
2. Johansson JK, Niiranen TJ, Puukka PJ, Jula AM. Prognostic value of the variability in home-measured blood pressure and heart rate: the Finn-Home Study. *Hypertension*. 2012;59:212-218.
3. Wei FF, Li Y, Zhang L, et al. Beat-to-beat, reading-to-reading, and day-to-day blood pressure variability in relation to organ damage in untreated Chinese. *Hypertension*. 2014;63:790-796.
4. Stevens SL, Wood S, Koshiaris C, et al. Blood pressure variability and cardiovascular disease: systematic review and meta-analysis. *BMJ*. 2016;354:i4098.
5. Parati G, Saul JP, Di Rienzo M, Mancia G. Spectral analysis of blood pressure and heart rate variability in evaluating cardiovascular regulation. A critical appraisal. *Hypertension*. 1995;25:1276-1286.
6. Kotsis V, Stabouli S, Karafillis I, et al. Arterial stiffness and 24 h ambulatory blood pressure monitoring in young healthy volunteers: the early vascular ageing Aristotle University Thessaloniki Study (EVA-ARIS Study). *Atherosclerosis*. 2011;219:194-199.
7. Gómez-Marcos MA, Recio-Rodríguez JI, Patino-Alonso MC, et al. Ambulatory arterial stiffness indices and target organ damage in hypertension. *BMC Cardiovasc Disord*. 2012;12:1.
8. Parati G, Ochoa JE, Lombardi C, Bilo G. Assessment and management of blood-ressure variability. *Nat Rev Cardiol*. 2013;10:143-155.
9. Gosmanova EO, Mikkelsen MK, Molnar MZ, et al. Association of systolic blood pressure variability with mortality, coronary heart disease, stroke, and renal disease. *J Am Coll Cardiol*. 2016;68:1375-1386.
10. Domingos E, Polito MD. Blood pressure response between resistance exercise with and without blood flow restriction: a systematic review and meta-analysis. *Life Sci*. 2018;209:122-131.
11. Umemura S, Arima H, Arima S, et al. The Japanese Society of Hypertension guidelines for the management of hypertension (JSH 2019). *Hypertens Res*. 2019;42:1235-1481.
12. Willis BL, Gao A, Leonard D, et al. Midlife fitness and the development of chronic conditions in later life. *Arch Intern Med*. 2012;172:1333-1340.
13. Blair SN, Kohl HW 3rd, Paffenbarger RS Jr, et al. Physical fitness and all-cause mortality. *JAMA*. 1989;262:2395-2401.
14. Tanaka H, Bassetta DJ Jr, Turner MJ. Exaggerated blood pressure response to maximal exercise in endurance-trained individuals. *Am J Hypertens*. 1996;9:1099-1103.
15. Pressler A, Jähnig A, Halle M, Haller B. Blood pressure response to maximal dynamic exercise testing in an athletic population. *J Hypertens*. 2018;36:1803-1809.
16. Venckunas T, Mazutaitiene B. The role of echocardiography in the differential diagnosis between training induced myocardial hypertrophy versus cardiomyopathy. *J Sports Sci Med*. 2007;6:166-171.
17. Weeks KL, McMullen JR. The athlete's heart vs. the failing heart: can signaling explain the two distinct outcomes? *Physiology*. 2011;26:97-105.
18. Lang RM, Bierig M, Devereux RB, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J Ame Soc Echocardiogr*. 2005;18:1440-1463.

19. Rowell LB, O'Leary DS. Reflex control of the circulation during exercise: chemoreflexes and mechanoreflexes. *J Appl Physiol*. 1990;69:407-418.
20. Duncker DJ, Bache RJ. Regulation of coronary blood flow during exercise. *Physiol Rev*. 2008;88:1009-1086.
21. Barbier J, Reland S, Ville N, et al. The effects of exercise training on myocardial adrenergic and muscarinic receptors. *Clin Auton Res*. 2006;16:61-65.
22. Williams RS. Role of receptor mechanisms in the adaptive response to habitual exercise. *Am J Cardiol*. 1985;55:68D-73D.
23. Pelliccia A, Maron MS, Maron BJ. Assessment of left ventricular hypertrophy in a trained athlete: differential diagnosis of physiologic athlete's heart from pathologic hypertrophy. *Prog Cardiovasc Dis*. 2012;54:387-396.
24. Mihl C, Dassen WRM, Kuipers H. Cardiac remodelling: concentric versus eccentric hypertrophy in strength and endurance athletes. *Neth Heart J*. 2008;16:129-133.
25. Maron BJ, Pelliccia A. The heart of trained athletes: cardiac remodeling and the risks of sports, including sudden death. *Circulation*. 2006;114:1633-1644.
26. Janikowska G, Żebrowska A, Kochańska-Dziurowicz A, Mazurek U. Differences in echocardiography, blood pressure, stroke volume, maximal power and profile of genes related to cardiac hypertrophy in elite road cyclists. *Adv Clin Exp Med*. 2017;26:999-1004.
27. Pelliccia A, Maron BJ, De Luca R, et al. Remodeling of left ventricular hypertrophy in elite athletes after long-term deconditioning. *Circulation*. 2002;105:944-949.
28. Hein S, Arnon E, Kostin S, et al. Progression from compensated hypertrophy to failure in the pressure-overloaded human heart: structural deterioration and compensatory mechanisms. *Circulation*. 2003;107:984-991.
29. Cohn JN, Ferrari R, Sharpe N. Cardiac remodeling—concepts and clinical implications: a consensus paper from an international forum on cardiac remodeling. *J Am Coll Cardiol*. 2000;35:569-582.
30. de Luca N, Asmar RG, London GM, et al. Selective reduction of cardiac mass and central blood pressure on low-dose combination perindopril/indapamide in hypertensive subjects. *J Hypertens*. 2004;22:1623-1630.

How to cite this article: Nakamura K, Fujiwara T, Hoshide S, et al. Differences in exercise-induced blood pressure changes between young trained and untrained individuals. *J Clin Hypertens*. 2021;23:843–848. https://doi.org/10.1111/jch.14177