Relationship Between Home Blood Pressure and the Onset Season of Cardiovascular Events: The J-HOP Study (Japan Morning Surge-Home Blood Pressure)

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BACKGROUND
The incidence of cardiovascular disease (CVD) increases during winter. The risk that elevated home blood pressure (BP) poses for CVD events that occur in each of 4 seasons is unclear. We conducted a *post hoc* analysis using the dataset from a nationwide cohort, the Japan Morning Surge-Home Blood Pressure (J-HOP) study, to assess the association between home BP and winter-onset CVD events.

METHODS
J-HOP participants who had cardiovascular risks conducted morning and evening home BP measurements for a 14-day period and were followed-up for the occurrence of CVD events.

RESULTS
We analyzed 4,258 participants (mean age 64.9 years; 47% male; 92% hypertensives) who were followed-up for an average of 6.2 ± 3.8 years (26,295 person-years). We divided the total of 269 CVD events (10.2/1,000 person-years) by the season of onset as follows: 82 in the winter and 187 in the other seasons (spring, summer, and autumn). In the Cox models adjusted for covariates and the season when home BPs were measured at baseline, morning home systolic BP (SBP) was associated with both winter-onset and other season-onset CVD events: hazard ratio (HR) for winter 1.22, 95% confidence interval (CI) 1.06–1.42 per 10 mm Hg; HR for other seasons 1.11, 95% CI 1.00–1.23. Evening home SBP was associated with the other season-onset CVD events (HR 1.20, 95% CI 1.08–1.33 per 10 mm Hg), but not with the winter-onset CVD events.

CONCLUSIONS
Our findings indicate that compared with evening home BP, morning home BP might be a superior predictor of winter-onset CVD events.

Keywords: blood pressure; home blood pressure; hypertension; morning home blood pressure; winter-onset cardiovascular event

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The incidence of cardiovascular disease (CVD) events has been reported to be higher in the winter compared with the summer.[1–3] Although exposure to cold temperature has been reported to be a risk factor for the increase in CVD events during the winter,[2,3] other specific risk factors for the incidence of CVD events that occur in particular seasons (such as the winter-onset CVD events) are still unclear.

Home blood pressure (BP) measurement is well accepted as a useful tool in the management of hypertension and is recommended in international guidelines and the consensus on the management of hypertension,[4–11] since home BP has been reported as a stronger predictor of future CVD events compared with office BP.[12–16] However, those findings did not take into account the seasons of the CVD events.

Cardiovascular events occur more often in the morning than in the evening.[17] In addition, exposure to cold temperature in winter activates sympathetic activity and elevates BP levels in the morning.[18–20] We thus hypothesized that compared with the evening home BP level, the morning home BP level would be more strongly associated with CVD events that occur in the winter. To test this hypothesis, we used the dataset of the Japan Morning Surge-Home Blood Pressure (J-HOP) study, a nationwide multicenter prospective study of outpatients in clinical practice. J-HOP study participants conducted home BP measurements in the morning and evening and were followed-up for CVD events. We analyzed the database of the J-HOP study to clarify the relationship between home BP and future cardiovascular events that occurred in the winter and other seasons.

METHODS

Study design

The present study was a *post hoc* analysis of the J-HOP study. Details of the J-HOP study rationale, design, and procedures have been published.[16] Briefly, 4,310 patients with a history of or risk factors for CVD were recruited at 71 institutions throughout Japan between 2005 and 2012. Participants measured their own home BP on 14 consecutive

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days at study enrollment and were then followed-up for CVD events through May 2018. The present study aimed to assess the relationship between home BP measured at study enrollment and CVD events that occurred in each of the 4 seasons. The Institutional Review Board of Jichi Medical University School of Medicine approved the methods, and all of the patients provided written informed consent to participate and to have their data published.

BP and other measurements

We assessed office BP and self-measured home BP. Three office BP readings were taken at 15-second intervals on 2 different occasions within 2 months, and the mean of these 6 readings was used as the participant's office BP value. Office BP was recorded on 2 different occasions: before and after the self-measured home BP. Self-measured home BP was performed according to the Japanese BP guideline.[21] Three BP readings were taken at 15-second intervals with the patient in a seated position in both the morning (within 1 hour of waking and before taking antihypertensive medications) and the evening (before going to bed) for 14 consecutive days. The first day's home BP measurements were excluded, and the averages of the remaining morning BP (37.5 ± 7.3 readings) and evening BP (36.1 ± 8.3 readings) values were calculated separately. Here, "mean home BP" is the average of the patient's morning and evening home BP. The office and home BP values were measured using the same validated, automatic, and oscillometric device (HEM-5001; Omron Healthcare, Kyoto, Japan). Additionally, to avoid reporting bias, BP data were automatically stored in the memory of the device and were downloaded to a computer by a physician or nurse during office visits. The J-HOP study had more BP readings than the previous home BP studies.[12–14,16]

The risk factors were hypertension (office systolic BP [SBP] ≥140 mm Hg and/or diastolic BP ≥90 mm Hg, or current use of antihypertensive medication), hyperlipidemia (total cholesterol ≥240 mg/dl or treated hyperlipidemia), diabetes mellitus (fasting blood sugar ≥126 mg/dl or receiving an antidiabetic drug), glucose intolerance, metabolic syndrome, chronic kidney disease (estimated glomerular filtration rate <60 ml/min/1.73 m^2), history of CVD (coronary artery disease, stroke, congestive heart failure, or aortic dissection), atrial fibrillation, current smoking, chronic obstructive pulmonary disease, and sleep apnea syndrome. Blood and spot urine samples were collected in the morning from the patient in a fasting state at study enrollment. Laboratory methods are described in Supplementary Materials online.

Outcome ascertainment and CVD events in each season

Each patient's vital status was ascertained through May 2018. Total incident CVD events during the follow-up, including stroke (fatal and nonfatal), fatal and nonfatal coronary heart disease (fatal and nonfatal), congestive heart failure, and aortic dissection events, was assessed as an outcome. The incident CVD events were categorized as follows: (i) fatal and nonfatal stroke, defined as the sudden onset of a neurological deficit persisting for ≥24 hours in the absence of any other disease that could account for the symptoms, with the findings of brain computed tomography or magnetic resonance imaging. Transient ischemic attack was not included. (ii) Fatal and nonfatal coronary heart disease, defined as acute myocardial infarction, angina pectoris requiring percutaneous coronary intervention, and sudden death within 24 hours of the abrupt onset of symptoms. (iii) Congestive heart failure, defined as hospitalization due to acute decompensated heart failure. (iv) Aortic dissection, defined as aortic dissection requiring hospitalization or invasive treatment. If events occurred on ≥2 occasions, the first occurrence was included in the analysis. Evidence of the above CVD outcomes was ascertained by ongoing reports from a general physician at each institute. The follow-up time was censored from the data of event ascertainment. Participants who did not experience CVD events were censored at the last study visit. When participants failed to come to the hospital, we interviewed them or their families by telephone. The detailed definitions of outcomes and methods of follow-up are described in Supplementary Materials online.

Subsequently, we divided the total of 269 CVD events into seasons when each event occurred: spring (n = 65), summer (n = 72), autumn (n = 50), and winter (n = 82). The seasons were defined as follows: spring = 1 March to 31 May; summer = 1 June to 31 August; autumn = 1 September to 30 November; and winter = 1 December to the last day of the next year's February.[22] Additionally, environmental factors such as the mean annual outdoor temperature and the duration of sunlight (the number of hours between sunrise and sunset) at each institution were obtained from a database maintained by the Japanese Meteorological Agency.[23]

Statistical analyses

All statistical analyses were performed with SPSS software, ver. 26.0 (SPSS, Chicago, IL), and R software, ver. 3.5.1 (The R Foundation for Statistical Computing, Vienna, Austria). Data are presented as the mean ± SD (continuous variables) or as percentages (categorical variables). We used Cox proportional hazards models to examine the association between home BP values and the risks for incident CVD in each season. Model 2 included covariates such as traditional risk factors (age, sex, current smoking, diabetes mellitus, and total cholesterol), body mass index, statin use, antihypertensive medication use, preexisting CVD (i.e., angina pectoris, acute myocardial infarction, stroke, or hospitalization due to congestive heart failure), and office SBP. In Model 3, we included the season when the participants underwent home BP measurements at baseline as a covariate to adjust the effect of seasonal variation in BP values for CVD outcomes. The seasons when the home BP measurements were conducted were classified based on the first day of home BP measurements and the definition of seasons provided above.[22] We also assessed whether similar results would be obtained with only the home BP values measured during the first week. The levels of statistical significance were established at a 2-sided P value <0.05 for all tests.

RESULTS

Participants' characteristics

The 4,258 participants enrolled in the present analysis were from the J-HOP study. The mean age was 64.9 ± 10.9 years, 46.8% were men, and 91.5% were hypertensive patients. Their clinical characteristics are summarized in Table 1.

Association between home BP and total CVD events

During the average of 6.2 ± 3.8 years of follow-up (26,295 person-years), 269 CVD events (10.2/1,000 person-years) occurred. The 269 events were divided into other seasons (spring, n = 65; summer, n = 72; and autumn, n = 50) (n = 187) and winter (n = 82). The detailed breakdown of CVD events in each season is provided in Supplementary Table S1 online.

Table 2 shows the Cox proportional hazard models for a 10-mm Hg increase in office, morning and evening home SBP for the incidence of total CVD events. Both morning and evening home SBP were associated with the CVD risks after adjustment for host factors: morning home SBP, hazard ratio (HR) 1.14, 95% confidence interval (CI) 1.05–1.24; evening home SBP, HR 1.15, 95% CI 1.06–1.26 per 10 mm Hg (Model 2 in Table 2). These relationships were also significant after adjustment for the season when home BPs were measured at baseline: morning home SBP, HR 1.14, 95% CI 1.05–1.24; evening home SBP, HR 1.16, 95% CI 1.06–1.27 (Model 3 in Table 2).

Association between home BP and the CVD events that occurred in winter and the other seasons

Figures 1 and 2 show the rates of CVD events that occurred in other seasons (spring, summer, and autumn) and winter according to the respective quartiles of morning and evening home SBP. Table 3 shows the Cox proportional hazard models for the onset season of CVD events divided into winter and the other seasons. In the adjusted model for the CVD events that occurred in other seasons, both morning and evening home SBP were significantly associated with CVD risks: morning home SBP, HR 1.11, 95% CI 1.00–1.23; evening home SBP, HR 1.20, 95% CI 1.08–1.33 per 10 mm Hg (Model 3 for the CVD events that occurred in other seasons in Table 3). In the adjusted model for the CVD events that occurred in winter, morning home SBP was significantly associated with CVD risks (HR 1.22, 95% CI 1.06–1.42 per 10 mm Hg). However, evening home SBP did not show a significant relationship with the CVD events that occurred in winter (Model 3 for the CVD events that occurred in winter in Table 3). These relationships were also observed even when the analysis was adjusted for environmental factors (mean annual temperature and sunlight time) in Japan (Supplementary Table S2 online). When only the home BP values that were measured during the first week

Table 1. Clinical characteristics and BP parameters of the study participants (n = 4,258)

Descriptive variables	
Age, years	64.9 ± 10.9
Male, %	46.8
BMI, kg/m^2	24.3 ± 3.5
Current smoking, %	12.2
Hypertension, %	91.5
Use of antihypertensive medication, %	79.2
Calcium-channel blocker, %	50.9
ACE inhibitor, %	6.6
Angiotensin receptor blocker, %	51.7
Diuretic, %	26.0
α-Blocker, %	4.9
β-Blocker, %	13.7
Diabetes, %	24.4
Preexisting CVD, %	13.8
Statin treatment, %	23.6
Total cholesterol, mg/dl	202.4 ± 32.9
HDL cholesterol, mg/dl	57.5 ± 15.2
Season of baseline home BP measurement	
Spring, % (n)	24.8 (1,058)
Summer, % (n)	23.0 (980)
Autumn, % (n)	28.7 (1,221)
Winter, % (n)	23.5 (999)
BP parameters, mm Hg	
Office SBP	141.3 ± 16.4
Office DBP	81.2 ± 10.6
Morning home SBP	138.4 ± 15.8
Morning home DBP	79.1 ± 10.0
Evening home SBP	130.1 ± 14.9
Evening home DBP	72.6 ± 9.7
Mean morning and evening home SBP	134.3 ± 14.3
Mean morning and evening home DBP	75.9 ± 9.3
Total number of home BP readings for analysis[a]	
Morning home BP readings	37.5 ± 7.3
Evening home BP readings	36.1 ± 8.3

Data are mean ± SD or percentage. Preexisting CVD includes angina pectoris, acute myocardial infarction, stroke, and prehistory of hospitalization due to heart failure. Abbreviations: ACE, angiotensin-converting enzyme; BMI, body mass index; BP, blood pressure; CVD, cardiovascular disease; DBP, diastolic blood pressure; HDL, high-density lipoprotein; PR, pulse rate; SBP, systolic blood pressure.

[a]Total number of home BP readings: 3 BP readings were taken at 15-second intervals in both the morning and evening for 14 consecutive days. The first day's home BP measurements were excluded from the analysis.

Table 2. Cox proportional hazard models of BP parameters for the total CVD events (n = 4,258)

Objective variables	Total CVD events	
No. of cardiovascular events	269	
	HR (95% CI)	*P* value
Model 1, unadjusted		
Office SBP, 10 mm Hg	1.08 (1.01–1.17)	0.033
Morning home SBP, 10 mm Hg	1.23 (1.15–1.32)	<0.001
Evening home SBP, 10 mm Hg	1.19 (1.10–1.28)	<0.001
Mean home SBP, 10 mm Hg	1.25 (1.15–1.35)	<0.001
Model 2		
Office SBP, 10 mm Hg	1.05 (0.97–1.13)	0.217
Morning home SBP, 10 mm Hg	1.14 (1.05–1.24)	0.002
Evening home SBP, 10 mm Hg	1.15 (1.06–1.26)	0.001
Mean home SBP, 10 mm Hg	1.18 (1.08–1.30)	<0.001
Model 3		
Office SBP, 10 mm Hg	1.05 (0.97–1.13)	0.205
Morning home SBP, 10 mm Hg	1.14 (1.05–1.24)	0.002
Evening home SBP, 10 mm Hg	1.16 (1.06–1.27)	0.001
Mean home SBP, 10 mm Hg	1.19 (1.08–1.31)	<0.001

The HRs (95% CI) associated with each BP parameter in all patients are shown. Mean home SBP represents the mean of morning and evening home SBP. Model 2 included adjusted host factors as follows: demographic variables (age and sex), clinical and behavioral characteristics (BMI, preexisting of CVD [angina pectoris, myocardial infarction, stroke, or prehistory of hospitalization due to heart failure], current smoking, total cholesterol, prevalence of diabetes, use statin, use of antihypertensive medication). In the analysis of home SBP parameters, office SBP was adjusted. Model 3 added covariates of seasons (when BP was measured at baseline) to Model 2. Abbreviations: BMI, body mass index; BP, blood pressure; CI, confidence interval; CVD, cardiovascular disease; HR, hazard ratio; SBP, systolic blood pressure.

were used for the analysis, the same relationships were observed (Supplementary Table S3 online).

When the results of the analysis of CVD events that occurred in other seasons were divided into those for each of spring, summer, and autumn, morning home SBP was associated with the CVD events that occurred in each of these 3 seasons in the unadjusted model. However, in the adjusted models, morning home SBP was not significantly associated with the CVD events that occurred in the spring, summer, or autumn. In contrast, evening home SBP was associated with the CVD events that occurred in summer even in the adjusted model (HR 1.20, 95% CI 1.02–1.42 per 10mm Hg), but it was not significantly associated with the CVD events that occurred in the spring or autumn (Supplementary Table S4 online).

DISCUSSION

The present *post hoc* analysis of a nationwide practice-based cohort observational study demonstrated that morning home BP was associated with the future CVD events that occurred in both the winter and other seasons even when adjusted for the season when the home BP measurements were conducted at baseline. However, evening home BP was associated with the future CVD events that occurred in the other seasons, but not with the winter-onset CVD events. Although it has been reported that home BP is a predictor of incident CVD events,[12–16] there have been no data to show whether home BP poses a risk of CVD events occurring in particular seasons, such as the winter. The present study is the first to observe a relationship of morning and evening home BP with the CVD outcomes classified by the onset in each season. Our findings indicate that compared with evening home BP, morning home BP might be a superior predictor of winter-onset CVD events.

Similar to the results of previous studies, we observed that morning, evening, and mean morning and evening home SBP were also related to the total CVD risks unrelated to the season of onset even after adjustment for the season during which the home BP measurements were conducted at baseline. Home BP parameters have been reported to show seasonal variation, being higher in winter and lower in summer.[24–26] However, most of the previous studies reporting the relationships between home BP and CVD risks were not adjusted for the seasons when home BP measurements were conducted.[12–16] Our present findings suggest that home BP might be associated with CVD risks regardless of which season the home BP was measured.

Our results demonstrated that morning home SBP was associated with winter-onset CVD events, whereas evening home SBP was not a risk of the CVD events that occurred in the winter. Elevation of BP in the morning is an important therapeutic target in the management of hypertension.[27–30]

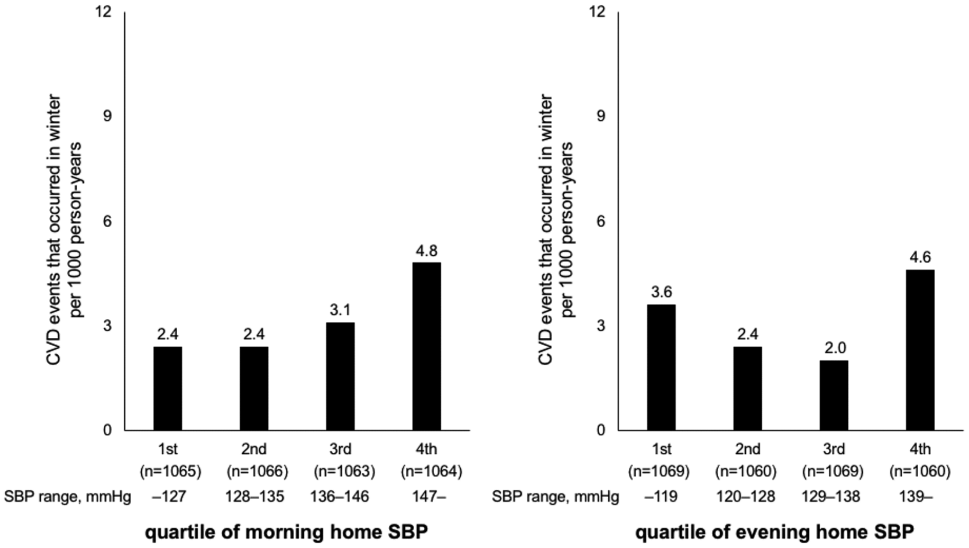

Figure 1. The number of CVD events that occurred in winter according to each quartile of morning and evening home SBP. The number of CVD events that occurred during the winter is presented per 1,000 person-years. Abbreviations: CVD, cardiovascular disease; SBP, systolic blood pressure.

Morning home BP was reported to be a superior predictor for incident CVD events compared with evening home BP.[16,31] In studies using home or ambulatory BP monitoring, morning BP was strongly affected by temperature and also showed strong seasonal variation (such as an increase from summer to winter) compared with evening BP.[19,32,33] In one of our earlier investigations, morning home BP had stronger relationships with factors of target organ damage such as B-type natriuretic peptide and the urine albumin–creatinine ratio in the winter than other seasons, whereas these relationships were not observed for evening home BP.[34] Our present finding that home BP in the morning but not in the evening had a relationship with winter-onset CVD events is thus reasonable. Our findings might reinforce the concept that compared with evening home BP, morning home BP is a high-priority therapeutic target to prevent winter-onset CVD events.

We also observed that both morning and evening home SBP were associated with the CVD events that occurred in spring, summer, and autumn. In the analysis of the CVD events that occurred during the summer, although morning home SBP was not a risk for CVD events, evening home SBP was significantly associated with the summer-onset CVD events. An epidemiological report indicated that some types of stroke (such as nonischemic cerebral infarct, and evening and nighttime-onset stroke) were observed more frequently in the summer than the winter.[35,36] Acute myocardial infarction that occurred at nighttime was also reported to increase in summer compared with other seasons.[37,38] Evening and nighttime-onset CVD events might thus occur more frequently in summer compared with winter. In consideration of these previous reports, our present observation that evening home SBP was associated with summer-onset CVD events is not contradictory.

The strengths of the present study include its recruitment from a nationwide study and the inclusion of a large number of clinical practice patients with CVD risk factors. We used the same validated device to measure home BP, applied standardized home BP measurement schedules, used automatic monitors equipped with a memory, and had a high patient retention rate. However, our findings need cautious interpretation. We observed the opposite predictive value of morning and evening home SBP for the CVD events that occurred in winter and those that occurred in the other seasons. Although morning home SBP was significantly associated with both CVD events in the winter and the other seasons, the mean of the morning and evening home SBP was also associated with both the CVD events in winter and those in the other seasons. These findings suggest that home BP measurements taken on 2 occasions, i.e., morning and evening, may be still important for risk stratification in the management of hypertension. Moreover, some potential limitations should be addressed.

First, the most notable is that our observations were cross-sectional BP profiles of participants in each season; that is, we did not conduct home BP monitoring for the same subjects throughout the 4 seasons. The present study thus did not examine the association between changes in home BP in each season in the same individuals with the risks of CVD events that occurred in each season. Second, various environmental factors differ regionally in Japan, and these differences might affect the seasonal variation of BP. Our findings may thus not be completely generalized to patients living in other regions. Third, changes in the patients' use of antihypertensive medications during the follow-up period were not considered, and thus the effect of changes in BP phenotypes and the use of antihypertensive medication during the follow-up on outcomes could not be assessed in this study. Fourth, the covariates used in the present analysis were limited and residual confounders such as socioeconomic status may exist. Fifth, our findings may not be generalized to other racial or ethnic groups.

Our findings indicate that compared with evening home SBP, elevated morning home SBP might be strongly

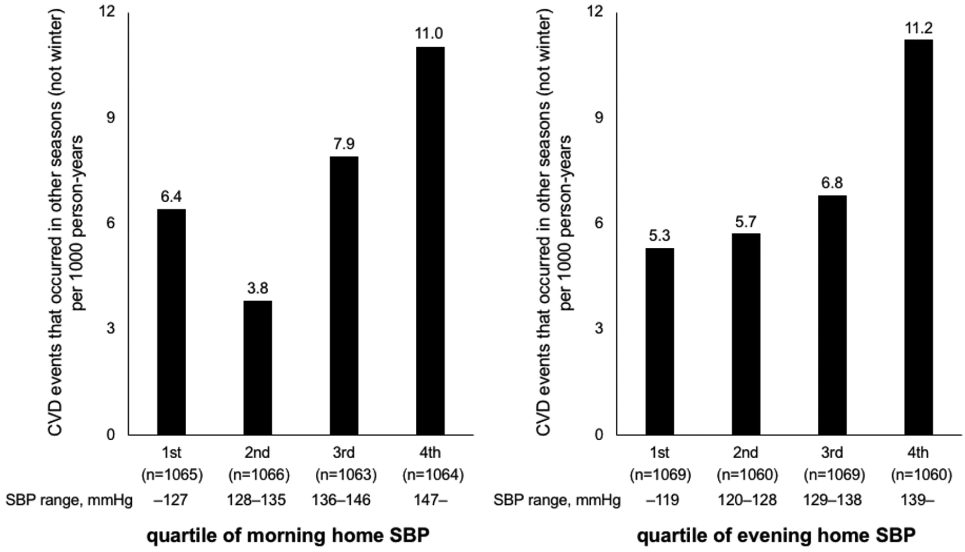

Figure 2. The numbers of CVD events that occurred in seasons other than winter (spring, summer, and autumn) according to each quartile of morning and evening home SBP. The numbers of CVD events that occurred in other seasons (spring, summer, and autumn) are presented per 1,000 person-years. Abbreviations: CVD, cardiovascular disease; SBP, systolic blood pressure.

Table 3. Cox proportional hazard models of BP parameters for the CVD events that occurred in winter and other seasons, *n* = 4,258

Objective variables	CVD events occurred in other seasons (not winter)		CVD events occurred in winter	
No. of cardiovascular events	187		82	
	HR (95% CI)	*P* value	HR (95% CI)	*P* value
Model 1, unadjusted				
Office SBP, 10 mm Hg	1.11 (1.01–1.21)	0.026	1.03 (0.90–1.18)	0.630
Morning home SBP, 10 mm Hg	1.22 (1.12–1.32)	<0.001	1.26 (1.11–1.42)	<0.001
Evening home SBP, 10 mm Hg	1.23 (1.12–1.35)	<0.001	1.09 (0.94–1.26)	0.252
Mean home SBP, 10 mm Hg	1.26 (1.15–1.38)	<0.001	1.21 (1.05–1.40)	0.008
Model 2				
Office SBP, 10 mm Hg	1.07 (0.98–1.16)	0.159	1.01 (0.88–1.15)	0.928
Morning home SBP, 10 mm Hg	1.11 (1.00–1.23)	0.041	1.21 (1.05–1.41)	0.010
Evening home SBP, 10 mm Hg	1.19 (1.08–1.32)	0.001	1.07 (0.91–1.26)	0.434
Mean home SBP, 10 mm Hg	1.18 (1.06–1.32)	0.003	1.18 (1.00–1.40)	0.057
Model 3				
Office SBP, 10 mm Hg	1.07 (0.98–1.17)	0.152	1.01 (0.88–1.16)	0.898
Morning home SBP, 10 mm Hg	1.11 (1.00–1.23)	0.042	1.22 (1.06–1.42)	0.008
Evening home SBP, 10 mm Hg	1.20 (1.08–1.33)	0.001	1.08 (0.92–1.27)	0.362
Mean home SBP, 10 mm Hg	1.19 (1.06–1.33)	0.002	1.20 (1.01–1.42)	0.041

The HRs (95% CI) of the association between each BP parameter and the CVD events that occurred in winter (during 3 months) and others (not winter, i.e., spring, summer, and autumn: during 9 months) in the whole patient group are shown. Mean home SBP represents the mean of morning and evening home SBP. Model 2 included adjustments for the following host factors: demographic variables (age and sex), clinical and behavioral characteristics (BMI, preexisting of CVD [angina pectoris, myocardial infarction, stroke, or prehistory of hospitalization due to heart failure], current smoking, total cholesterol, prevalence of diabetes, use statin, use of antihypertensive medication). In the analysis of home SBP parameters, office SBP was adjusted. Model 3 added covariates of seasons (when BP was measured at baseline) to Model 2. Abbreviations: BMI, body mass index; BP, blood pressure; CI, confidence interval; CVD, cardiovascular disease; HR, hazard ratio; SBP, systolic blood pressure.

associated with winter-onset CVD events. In light of these findings, medical practitioners should be aware that morning home BP may be a high-priority therapeutic target to prevent the winter increase in CVD events.

SUPPLEMENTARY MATERIAL

Supplementary data are available at *American Journal of Hypertension* online.

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DISCLOSURE

K. Kario received research funding from Omron Healthcare, Fukuda Denshi, and A&D.

DATA AVAILABILITY

The data underlying this article cannot be shared publicly due to ethical reasons.

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