# Ambulatory blood pressure monitoring and morning surge in blood pressure in adult black and white South Africans

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**Short title: Circadian variation in blood pressure**

**Key words:** hypertension, cardiovascular risk, circadian variation

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## Abstract

We examined whether there were differences in the circadian variation in blood pressure and the morning surge in blood pressure between black and white Africans. Clinic and ambulatory blood pressure data obtained from the Sympathetic Activity and Ambulatory Blood Pressure in Africans (SABPA) study was examined (n=406; 49% black African). Ambulatory blood pressure readings were fitted to a six-parameter double logistic equation to determine the power and rate of the morning surge in blood pressure. Multiple linear regression analysis was used to examine differences in blood pressure between black and white participants. Clinic and ambulatory blood pressure were higher in black participants throughout the day and night. In those taking medications blood pressure was less well controlled in black subjects. Despite the higher systolic blood pressure, the day night difference estimated by the logistic function was similar in black and white partcipants. However, the rate of rise and power in the morning surge in blood pressure was lower in black participants. We conclude that black participants of the SABPA study present with higher blood pressure throughout the day and night but have a lower power of the morning surge in blood pressure due to a slower morning rate of increase. Moreover, they had an increased prevalence of undiagnosed hypertension and, in those taking medication, were less likely to have their blood pressure controlled than their white counterparts.

**Key words:** hypertension, cardiovascular risk, circadian variation

## Introduction

Despite repeated comprehensive blood pressure screening and education programs [1], high blood pressure remains a significant contributor to the global burden of disease [2]. Importantly, while once associated with affluent countries, hypertension prevalence is now prominent in low-income countries, including those in south Asia and sub-Saharan Africa [3]. Through its impact in contributing to heart and kidney disease, stroke and dementia, the unfettered effect of high blood pressure in these communities poses serious concerns.

With the development and wider adoption of ambulatory blood pressure monitoring (ABPM), evidence has accumulated that ambulatory blood pressure measurements are a stronger predictor of all-cause and cardiovascular mortality than blood pressure recorded in the clinic [4]. Moreover, ABPM permits the evaluation of blood pressure throughout the day and night and the dynamics of changes during the transition from day to night and from night to day. This circadian variation in blood pressure has been well-described [5]. Under resting conditions blood pressure levels are lowest typically during night-time sleep and then rise prior to waking, peaking within the first few hours of daytime activity [6]. An exaggerated morning surge in blood pressure has been linked with stroke risk in older individuals with hypertension in Japan [7] and predicted cardiovascular outcome in a large study across 8 populations [8]. These data contrast with the observations of Verdecchia and colleagues who found that a blunted morning surge in blood pressure was an independent predictor of cardiovascular events in an Italian, predominantly white population [9]. Most studies have used a measure of morning blood pressure surge (MBPS) calculated as the difference between the average blood pressure during the 2 hours after awakening and the lowest night-time blood pressure [7]. However, MBPS can only be determined if waking time is reliably known. A method has been developed for determining the power of the morning surge and the rate of morning rise based on the pattern of the changes in blood pressure independent of waking [10]. Using this method Luo and colleagues found an

association between the rate of the morning surge and cardiovascular events and stroke in a Chinese population [11]. A recent analysis of the Ohasama study population revealed a U-shaped risk of cardiovascular events, mainly stroke, and the power of the morning surge where the power function was the rate multiplied by the amplitude of the morning rise [12].

The Sympathetic Activity and Ambulatory Blood Pressure in Africans (SABPA) study was designed to examine blood pressure in a cohort of black and white school teachers in South Africa. Previous reports have documented higher 24-hour and clinic blood pressure in black subjects [13]. Given the paucity of multi-ethnic data examining ABPM and the MBPS calculated using this novel approach we postulated that the power of the morning surge and rate of morning rise in blood pressure will be different in black and white participants.

## Methods

Clinic and ambulatory blood pressure measurements were obtained from the SABPA study. Details of the recruitment strategy and protocol have been published elsewhere [14]. Briefly, participants comprised black and white African, male and female urban-dwelling teachers working in the Dr Kenneth Kaunda Education District in North-West Province South Africa. The SABPA study conformed to institutional guidelines and terms of the Declaration of Helsinki and was approved by the ethics review board of the North-West University, Potchefstroom Campus (NWU0003607S6). Written informed consent was obtained prior to participation. Phase 1 assessment of SABPA was undertaken over a 1-year period in 2008–09.

Data were collected over two days. Twenty-four hour ABPM was performed in 199 black and 207 white participants during the working day prior to the participants' clinic visit. At around 08:00 hours, participants were fitted with an ambulatory BP monitor (Meditech CE120 CardioTens; Meditech, Budapest, Hungary) on the non-dominant arm at their workplace. The ABPM device was programmed to measure blood pressure at 30 min intervals during the day

(08:00–22:00 hours) and every hour during night-time. Participants were transported at 16:30 hours to the Metabolic Unit Research Facility of the North-West University and were familiarized with the testing protocol. After receiving a standardized dinner, participants went to bed at around 22:00 hours. The following morning participants attended the laboratory for clinical assessments including anthropometric measures. Fasting blood sampling and clinic blood pressure was determined whilst in a semi recumbent position for at least 30 minutes. Blood pressure was measured two times using a mercury sphygmomanometer with a 5-minute rest between readings [13].

ABPM recording data were fitted to a 6 parameter double logistic equation as previously described [15]. The novel power function is the first derivative of the logistic curve multiplied by the amplitude which is the day night difference between plateaus [10]. Nocturnal dipping pattern was defined as: riser ≤ 0 % night-time systolic blood pressure elevation, non-dipper > 0 ≤ 10% night-time systolic blood pressure decrease, dipper > 10 ≤ 20% night-time systolic blood pressure reduction, and extreme dipper > 20% night-time systolic blood pressure fall [16]. Hypertension and hypertension status (controlled, uncontrolled) were classified after assessment of both clinic and ambulatory recordings according to the current European Society of Hypertension and European Society of Cardiology guidelines for the management of arterial hypertension and the Ambulatory blood pressure monitoring in Australia 2011 consensus position statement [17,18]. Hypertension was defined as either a clinic blood pressure ≥140/90 mmHg, daytime ABMP ≥135/85 mmHg or night-time ABPM ≥120/70.

Analyses were conducted using Stata 15.0 (StataCorp, College Station, Texas). Differences between attributes by race were determined using t-tests, Mann-Whitney U tests or chi-squared tests as appropriate. Multiple linear regression analysis was used to examine the morning surge in blood pressure between black and white participants after adjusting for confounders, namely, gender, age, BMI, plasma cholesterol, smoking status, habitual insomnia, statin and

hypertensive drugs. Standard homoscedasticity and normality checks were conducted on the residuals to assess model fit. All results are reported with 95% confidence intervals unless otherwise specified. A P-value of less than 0.05 (two-tailed) was deemed to be statistically significant.

## Results

There was an approximately equal gender mix amongst both black and white subjects (Table 1). White participants were slightly older than their black counterparts. While body weight was similar between groups, the white participants were taller resulting in black subjects having a larger BMI. There was no difference in waist circumference between groups. Although tobacco smoking was prevalent across both groups there were proportionally more white participants who smoked. Clinic blood pressure and the proportion of subjects with blood pressure over 140/90 mmHg was significantly higher in black subjects whilst white individuals had higher plasma cholesterol levels (Table 1). A larger proportion of black participants compared to white were on antihypertensive medications which comprised predominantly angiotensin converting enzyme inhibitors (11 v 2%; P=0.001), thiazide diuretics (12 v 4%; P=0.004) and calcium channel blockers (8 v 1%; P<0.001). In those subjects taking antihypertensive medications blood pressure remained uncontrolled in more black participants (46 v 22%, P<0.01). There was a trend (P=0.06) for a greater proportion of the black group to be on oral diabetes medications. Participants kept a diary during their ABPM period. There was no difference between groups with regards to self-reported habitual insomnia [awake 0.0 (0.0, 1.5) hours/night in black and 0.0 (0.0, 1.0) hours/night in white participants, P=0.10) or the proportion of participants who reported insomnia (39% black, 34% white, P=0.34).

Successful ABPM recordings were obtained more frequently in white participants (Table 2). The number of readings was influenced by BMI, where higher BMI was associated with less

successful readings recorded during both day (r=-0.31, P<0.001) and night (r=-0.20, P<0.001). Complete recordings during the night were obtained in 77% of white and 54% of the black participants (P<0.001). These proportions were increased to 94 and 84% respectively when considering subjects who had all but one pressure recording during the night. In all subjects combined ambulatory blood pressure was higher in the black participants at all times throughout the day (Figure 1). Although the magnitude of the difference between day and night systolic blood pressure between groups was not different, a greater proportion of white participants displayed a night-time blood pressure dipping pattern, with systolic blood pressure falling between 10-20 mmHg in more white subjects whereas more black participants displayed a non-dipping pattern (Table 2). Ambulatory heart rate was elevated in the black participants (Table 2).

In those subjects not taking antihypertensive medications blood pressure was higher in the black participants [systolic blood pressure: clinic 130 (120, 143) mmHg v 124 (116, 133) mmHg P<0.001; 24 hour ABPM 131 (120, 142) mmHg v 124 (117, 130) mmHg, P<0.001 for both; Figure 2].

While the blood pressure range (difference between estimated day and night plateaus) was no different between groups, the rate of rise and power in the morning surge in blood pressure was lower in black participants (Table 2, Supplementary Tables 1 and 2). The rate of rise of mean arterial blood pressure in the morning was independently associated with gender, age and the use of statins but was not associated with serum cholesterol concentration, smoking status or the use of antihypertensive medications (Table 3). Among those not taking statins, the rate of mean arterial blood pressure changes during the morning awakening was lower in black participants [difference in change=2.90 mmHg/hour, 95% CI (0.87, 4.93), P=0.002]. There was no difference in the rate or power of blood pressure change during the evening transition to sleep between the black and white participants (data not shown). The power of the morning

surge in mean arterial blood pressure was independently associated with race, being lower in black participants, and sex (Table 3). The calculated rate of rise in blood pressure during awakening and the power of the morning surge in blood pressure was not associated with the number of blood pressure recordings obtained.

## Discussion

In this study we report that in school teachers residing in the same region in South Africa, black participants present with higher blood pressure throughout the day and night and have a lower rate of rise and power of the morning surge in blood pressure. Moreover, they are less likely to have their blood pressure controlled when on medication and have an increased prevalence of undiagnosed hypertension. Differences in blood pressure and prevalence of hypertension, diabetes and dyslipidaemia between black and white individuals in Africa have been described previously [13,19,20]. These factors, plus smoking, account for around 90% of myocardial infarction in African populations, with a history of hypertension seeming to exert a more prominent effect in black Africans [21].

In those participants prescribed antihypertensive medications, blood pressure remained uncontrolled in significantly more black participants. The medications typically prescribed included angiotensin converting enzyme inhibitors, thiazide diuretics and/or calcium channel blockers. Whether differences in medication efficacy or compliance between groups influenced blood pressure control remains uncertain. Additionally, we observed that an increased proportion of black participants presented with previously undiagnosed, or at least currently untreated, high blood pressure. While it would be convenient to ascribe these differences in blood pressure, at least in part, to a lack of attendance to primary care it should be noted that all participants were from the same region, were in similar employment and had access to medical aid benefits. A greater proportion of black participants were prescribed

antihypertensive medications, indicating that accessing health services was unlikely the reason for the differences between the black and white participants.

Given that cardiovascular events are more prevalent in the morning, significant attention has focused on whether the morning surge in blood pressure is associated with cardiovascular morbidity and mortality. Both exaggerated [7,8] and blunted [9] surges in blood pressure have been shown to be associated with stroke and cardiovascular events. In the present report we noted a lower rate of morning rise in blood pressure in black participants but no difference in the systolic or mean blood pressure range (difference between calculated day and night plateaus). A previous report indicated that those with higher blood pressure generally have a greater rate of rise in the morning [22]. Thus, it is not clear why the rate of rise is lower in the black participants in the present report given that they had a higher blood pressure. However, our data are consistent with a recent report from the African-PREDICT Study which noted a blunted morning rise in blood pressure in young black South Africans [23] and a *post hoc* analysis of data from the PRISMA I and PRISMA II studies which examined the effects of telmisartan or ramipril on blood pressure dipping and blood pressure variability in patients with mild-to-moderate hypertension [24]. In these studies the authors estimated the MBPS from measures of the difference between mean blood pressure within 2-hours after arising and the night-time low, and as the moving peak morning systolic blood pressure (highest 1 hour moving average of consecutive systolic blood pressures between 6am and 10am) minus the moving lowest night-time systolic blood pressure (lowest 1 hour moving average of consecutive systolic blood pressures between 1am and 6am). The latter technique is limited to a peak change in consecutive values between fixed time points so is subject to influences such as variability in blood pressure. By contrast the logistic fitting procedure used in the present report makes no assumptions about when the blood pressure is surging and relies on a line of best fit based on the entire recording. Interestingly, and indicative that the sleep-trough approach and our

logistic analysis provide differing information, in a previous analysis using both methods we found that only 14% of the variance of the rate of rise and 22% of the variance in the morning power was explained by the sleep-trough MBPS [25].

Although antihypertensive medications may be associated with lower power of the MBPS [10] we found no effect of blood pressure medications in the present analysis. Similarly, plasma cholesterol has been shown to be positively associated with the MBPS [25] yet in the present analysis there was no link between plasma cholesterol and the rate or power of the MBPS. We noted that black participants presented with increased heart rate during the day and night. In the African-PREDICT Study the lower morning blood pressure rise in black participants was associated with heart rate variability-derived measures of autonomic activity [26]. Although measures of heart rate and of heart rate variability do not provide a reliable index of resting muscle [27] or cardiac sympathetic activity [28,29], we previously found a significant association between muscle sympathetic nervous activation in response to a cold pressor test and the rate and power of blood pressure rise during the morning [30]. Whether increased stress reactivity in the black participants could underline the lower MSBP observed in the present report remains unknown. Interestingly, recent findings in black participants of the SABPA study demonstrated sympathetic hyperactivity with desensitized neuroendocrine responses [31]. Whether neuroendocrine desensitization in response to chronic sympathetic activation in black participants limits blood pressure homeostatic reflexes and dampens the morning rise in blood pressure remains uncertain but merits further attention.

We found that black participants were more likely to display a non-dipping blood pressure pattern and that their blood pressure remained higher than the white subjects throughout the night. However, with the definition of dipping being a fixed percentage it is more likely that those with higher pressures are ascribed to have a non-dipping pattern as was found here. Some studies using the sleep-trough or pre-awakening blood pressure methods have found that the

magnitude of the morning surge in blood pressure is less in those with higher night-time pressure [9,23]. This was not the case in the present study, perhaps because the range in blood pressure was not different between groups. Nevertheless, the higher night blood pressure is important. Previous reports have shown that elevated nocturnal blood pressure is linked with cardiovascular events [32], and a decline of <10 mmHg in blood pressure during sleep has been associated with end organ damage [33] and increased risk of mortality [34]. Interestingly, Verdecchia and colleagues noted that interruptions to sleep due to cuff inflation interfered with the prognostic value of ABPM monitoring [35]. In 2934 untreated hypertensive subjects night-time blood pressure was higher in subjects who experienced interrupted sleep due to cuff inflation [35]. Blood pressure during the night was associated with cardiovascular events and death over a 7-year follow up but only in those subjects in whom the ambulatory recording device did not markedly interrupt sleep. The prognostic significance of higher nocturnal blood pressure on all-cause mortality disappeared in those participants with perceived sleep deprivation of more than 2 hours [35]. We noted that obtaining a complete set of ABPM recordings throughout the night was more likely in the white participants. Interestingly, in a previous report, African Americans were less likely to display a dipping blood pressure pattern and were more often awakened by inflation of the blood pressure cuff during the night [36]. Although there was no difference in self-reported insomnia between the black and white participants, we cannot be certain that there was no difference between groups in sleep quality during the actual experimental period. We noted that blood pressure was higher in the black participants throughout the day and the night. In the study by Verdecchia and colleagues [35] blood pressure during the day in those subjects who had interrupted sleep due to cuff inflations was not different to that observed in those who slept throughout the night.

In comparing between black and white participants at baseline this study has the limitations of a cross-sectional analysis. It is apparent in Table 1 that the groups differed in other attributes

besides race. Although we controlled for these in the primary analysis there may be other unmeasured confounders that have influenced our results. Ambulatory and clinic blood pressures were determined on only one occasion and our ABPM protocol, with recordings obtained every 30 minutes during the day and 60 minutes at night, was on the lower end of recommendations for frequency of recordings [37]. Current clinical practice would ensure that follow up recordings be conducted in order to confirm diagnosis. Additionally, more successful blood pressure readings were obtained in white participants. While we are uncertain as to why this occurred, in line with the European Society of Hypertension position paper on ambulatory blood pressure monitoring [37], within our cohort we obtained more than 20 readings during the day and more than 7 readings during the night in both black and white groups. While a lower number of recordings may impact on the assessment of the morning surge in blood pressure a previous study compared ambulatory blood pressure at 5, 10, 15, 30 and 60-minute intervals with continuous beat to beat recordings and demonstrated that accurate blood pressure assessment can be achieved at recording intervals as much as 30 or 60 minutes apart [38]. We found no association between the number of blood pressure readings and our determination of the rate and power of the morning surge in blood pressure. The morning surge in blood pressure takes on average around 3 hours and our fitting procedure is influenced predominantly by the 5-hour period around the surge [24]. The strengths of the study are that testing was performed in a controlled setting according to a standardised protocol and participants were drawn from the same region and were matched for education and profession.

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Figure 1

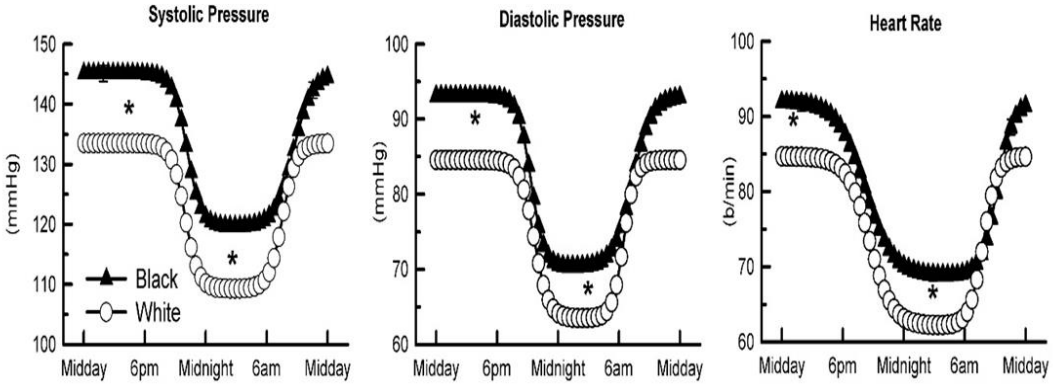
Systolic Pressure
Diastolic Pressure
Heart Rate
(mmHg)
(mmHg)
(b/min)
150
140
130
120
110
100
90
80
70
60
Black
White
Midday
6pm
Midnight
6am
Midday
*

Figure 2

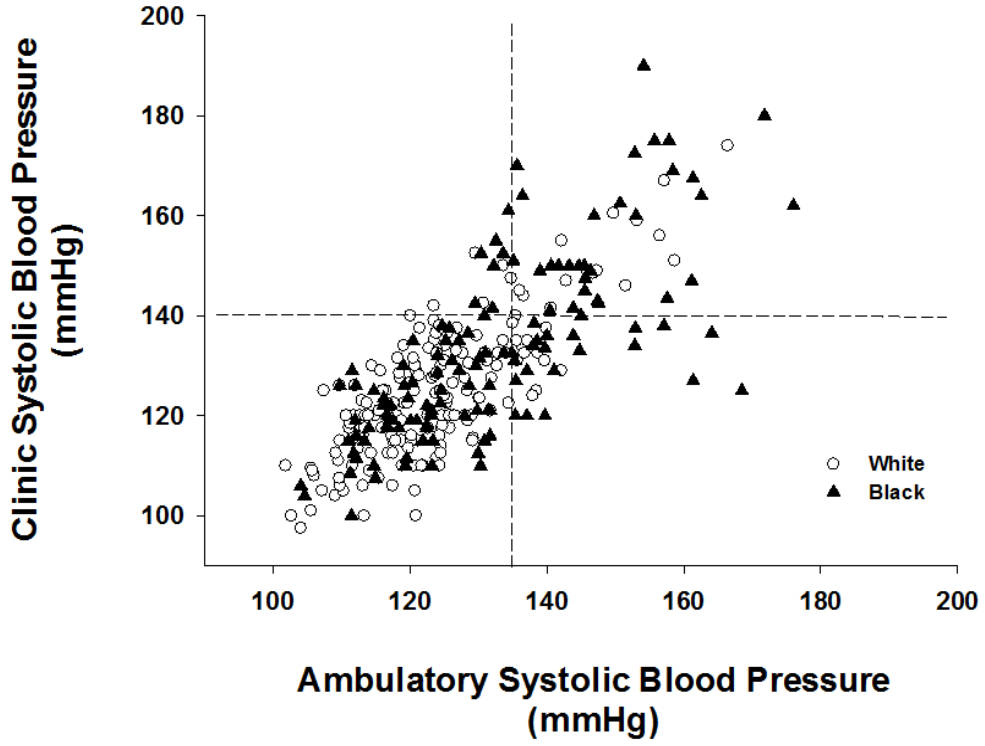
Clinic Systolic Blood Pressure (mmHg)
200
180
160
140
120
100
White
Black
100
120
140
160
180
200
Ambulatory Systolic Blood Pressure (mmHg)

Table 1

**Table 1: Participant demographics and medications by race**

| | Black | White | P |
|---|---|---|---|
| n | 199 | 207 | |
| Sex, n (% Female) | 101 (49.5) | 101 (51.7) | 0.66 |
| Age (years) | 44 (38, 51) | 47 (39, 53) | 0.002 |
| Height (m) | 1.64 (1.58, 1.70) | 1.73 (1.66, 1.81) | <0.001 |
| Weight (kg) | 80.0 (67.1, 94.1) | 81.3 (66.8, 97.5) | 0.52 |
| BMI ($kg/m^2$) | 30.1 (25.7, 34.7) | 26.9 (23.6, 30.3) | <0.001 |
| Waist circumference (cm) | 93.0 (81.9, 103.1) | 92.2 (80.3, 102.8) | 0.71 |
| Smoker n (%) | 144 (72) | 181 (86.6) | <0.001 |
| Total cholesterol (mmol/l) | 4.45 (3.74, 5.46) | 5.40 (4.65, 6.35) | <0.001 |
| HDL (mmol/l) | 1.08 (0.89, 1.29) | 1.15 (0.92, 1.44) | 0.30 |
| Triglycerides (mmol/l) | 1.09 (0.76, 1.56) | 0.95 (0.70, 1.45) | 0.08 |
| Antihypertensive medications n (%) | 69 (35) | 27 (13) | <0.001 |
| Uncontrolled blood pressure | 32 (46) | 6 (22) | <0.01 |
| Diabetes oral meds n (%) | 19 (10) | 10 (5) | 0.06 |
| Statins n (%) | 2 (1) | 9 (4) | 0.049 |
| Contraceptive pill n (%) | 17 (9) | 7 (3) | 0.027 |
| Anti-depressant/anxiolytic n (%) | 2 (1) | 4 (2) | 0.442 |
| Anti-inflammatory n (%) | 16 (8) | 13 (6) | 0.483 |
| Anti-histamine n (%) | 3 (2) | 12 (6) | 0.023 |

Data are displayed as n (%) for proportions or median ($25^{th}$, $75^{th}$ percentiles) for continuous variables.

**Table 2: Clinic and ambulatory blood pressure monitoring**[†]

| | Black | White | P |
|---|---|---|---|
| Clinic Systolic BP (mmHg) | 135 (120, 150) | 130 (120, 138) | <0.001 |
| Clinic Diastolic BP (mmHg) | 90 (80, 100) | 84 (80, 90) | <0.001 |
| Clinic BP >140/90 n (%) | 99 (51) | 58 (29) | <0.001 |
| Clinic heart rate (bpm) | 68 (61, 76) | 66 (58, 74) | 0.08 |
| **AMBP Monitoring** | | | |
| 24 hrs Number of readings (n) | 30 (27, 32) | 36 (33, 37) | <0.001 |
| 24 hrs Systolic BP (mmHg) | 131 (122, 125) | 124 (117, 129) | <0.001 |
| 24 hrs Diastolic BP (mmHg) | 83 (76, 90) | 75 (71, 82) | <0.01 |
| 24 hrs BP >130/80 n (%) | 107 (54) | 48 (23) | <0.01 |
| 24 hrs Heart rate (bpm) | 79 (72, 81) | 74 (67, 81) | <0.001 |
| Day Number of readings (n) | 22 (19, 24) | 27 (24, 29) | <0.001 |
| Day Systolic BP (mmHg) | 137 (127, 135) | 129 (123, 135) | <0.001 |
| Day Diastolic BP (mmHg) | 88 (81, 95) | 82 (76, 87) | <0.01 |
| Day BP >135/85 n (%) | 106 (54) | 51 (24) | <0.01 |
| Day Heart rate (bpm) | 83 (76, 85) | 78 (71, 86) | <0.001 |
| Night number of readings (n) | 9 (8, 9) | 9 (9, 9) | <0.001 |
| Night Systolic BP (mmHg) | 124 (112, 116) | 113 (106, 120) | <0.001 |
| Night Diastolic BP (mmHg) | 74 (66, 82) | 66 (62, 71) | 0.22 |
| Night BP >120/70 n (%) | 115 (58) | 51 (24) | <0.01 |
| Heart rate night (bpm) | 71 (65, 76) | 65 (59, 71) | <0.001 |
| **Dipping pattern** | | | |
| Day-Night difference Systolic BP (mmHg) | 14 (8, 25) | 15 (10, 20) | 0.06 |
| Day-Night difference Diastolic BP (mmHg) | 14 (9, 20) | 14 (11, 19) | 0.43 |
| Riser n (%) | 13 (7) | 6 (3) | 0.15 |
| Non dipper n (%) | 89 (46) | 69 (35) | 0.03 |
| Dipper n (%) | 83 (43) | 117 (59) | 0.002 |
| Extreme dipper n (%) | 10 (5) | 17 (9) | 0.25 |
| **Morning surge in BP** | | | |
| Systolic BP Range (mmHg) | 24.2 (18.4, 38.1) | 23.0 (17.4, 29.9) | 0.32 |
| Systolic BP Rate Morning (mmHg/hour) | 5.8 (1.9, 17.2) | 9.1 (2.8, 18.6) | 0.005 |
| Systolic BP Power Morning ($mmHg^2$/hour) | 168 (53, 760) | 231 (72, 512) | 0.06 |
| Mean BP Range | 22.1 (17.0, 29.7) | 20.6 (16.0, 26.4) | 0.72 |
| Mean BP Rate Morning (mmHg/hour) | 5.7 (2.0, 17.8) | 10.6 (4.5, 18.6) | <0.001 |
| Mean BP Power Morning ($mmHg^2$/hour) | 125 (47, 725) | 238 (86, 461) | 0.04 |

[†]Data are displayed as n (%) for proportions or median ($25^{th}$, $75^{th}$ percentiles) for continuous variables. Abbreviations used: BP (blood pressure), bpm (beats per minute), Morning [change from dark to light ie. morning (waking) transition]

**Table 3: Association with morning surge in mean arterial blood pressure**

| | Rate | | | Power | | |
|---|---|---|---|---|---|---|
| | Coefficient | 95% CI | P | Coefficient | 95% CI | P |
| Race<br>Black (ref)<br>White | | | | 1.0<br>74.2 | 10.2, 138.2 | 0.02 |
| Gender<br>Male (ref)<br>Female | 1.00<br>-2.58 | (-4.35, -0.80) | 0.005 | 1.00<br>-79.1 | -135.7, -22.5 | 0.006 |
| Age (yrs) | -0.11 | (-0.2, 0.003) | 0.04 | -3.1 | -6.4, 0.3 | 0.07 |
| BMI | -0.02 | (-0.16, 0.12) | 0.78 | 0.4 | -4.2, 4.9 | 0.87 |
| Serum cholesterol (mmol/l) | 0.29 | (-0.43, 1.00) | 0.43 | 4.1 | -18.7, 26.8 | 0.72 |
| Smoking status<br>Non (ref)<br>Smoker | 1.00<br>-0.19 | (-2.44, 2.03) | 0.86 | 1.0<br>-18.2 | -89.2, 52.8 | 0.61 |
| Anti-hypertensive drugs<br>Yes (ref)<br>No | 1.00<br>-0.19 | (-2.39, 2.01) | 0.86 | 1.0<br>-8.0 | -137.6, 266.4 | 0.44 |
| Statin across race<br>Statin Black<br>Statin White<br>No statin Black<br>No statin white | -<br>16.60<br>8.76<br>11.65 | -<br>(10.97, 22.21)<br>(7.40, 10.12)<br>(10.33, 12.99) | -<br><0.001<br><0.001<br><0.001 | Yes (ref):<br>1.0<br>No: -65.1 | -231.9, 101.8 | 0.53 |

In subjects not taking statins rate of rise in mean arterial pressure: black 5.7 (2.1, 18.3) mmHg/hour, white 10.5 (4.2, 18.3) mmHg/hour, power of morning surge in mean arterial pressure: black 128 (49, 490), white 230 (83, 450) $mmHg^2$/hour

**Supplementary Table 1: Rate and power of morning surge in blood pressure by race and gender**

| | Black | | White | |
|---|---|---|---|---|
| | Male | Female | Male | Female |
| Systolic BP Range (mmHg) | 24.6 (17.8, 32.6) | 23.6 (18.5, 34.0) | 23.8 (19.0, 29.9) | 22.5 (16.5, 30.1) |
| Systolic BP Rate Morning (mmHg/hour) | 5.1 (1.8, 14.4) *† | 6.6 (2.2, 15.2)* | 9.1 (2.2, 19.0) | 9.5 (3.3, 18.5) |
| Systolic BP Power Morning ($mmHg^2$/hour) | 140.5 (49.4, 371.1) | 177.8 (66.1, 388.3) | 206.8 (66.2, 536.1) | 237.2 (81.9, 453.3) |
| Mean BP Range (mmHg) | 21.8 (16.4, 26.8) | 22.2 (17.6, 27.7) | 20.1 (14.8, 26.8) | 19.9 (14.1, 25.9) |
| Mean BP Rate Morning (mmHg/hour) | 4.7 (1.8, 13.3)*† | 6.0 (2.1, 15.2)* | 11.8 (2.6, 19.4) | 10.5 (4.8, 17.2) |
| Mean BP Power Morning ($mmHg^2$/hour) | 115.7 (34.0, 340.0)* | 138.8 (56.5, 348.1)* | 245.6 (68.5, 503.1) | 223.3 (101.8, 408.1) |

Data are displayed as median ($25^{th}$, $75^{th}$ percentiles). Abbreviations used: BP (blood pressure). * $P<0.05$ comparing same gender across race, † $P<0.05$ different to values observed in white women

**Supplementary Table 2: Rate and power of morning surge in blood pressure by race and age**

| | Black | | | White | | |
|---|---|---|---|---|---|---|
| | <41 years | 41-<49 years | ≥49 years | <41 years | 41-<49 years | ≥49 years |
| Systolic BP Range (mmHg) | 22.7 (17.7, 29.5)* | 23.4 (17.1, 29.1) | 27.3 (21.1, 35.6)[a] | 21.1 (16.0, 27.9) [a] | 22.8 (17.6, 27.7) | 24.3 (19.2, 30.9) |
| Systolic BP Rate Morning (mmHg/hour) | 7.2 (2.1, 15.7) | 5.1 (1.8, 14.2) | 5.7 (2.3, 14.9) | 9.1 (3.4, 16.0) | 9.0 (3.1, 19.5) | 10.7 (2.2, 20.0) |
| Systolic BP Power Morning ($mmHg^2$/hour) | 173 (59, 380) | 143 (47, 301) | 188 (66, 402) | 190 (65, 323) | 252 (74, 541) | 246 (8, 564) |
| Mean BP Range | 22.3 (17.6, 27.9) | 19.7 (15.6, 25.5) | 22.6 (18.2, 29.8) | 20.1 (16.2, 24.4) | 20.1 (14.2, 27.1) | 21.9 (17.5, 27.1) |
| Mean BP Rate Morning (mmHg/hour) | 7.3 (2.8, 17.6)* | 4.9 (1.6, 12.6) [a] | 3.5 (1.7, 14.4)[b] | 10.0 (3.2, 17.3) | 10.6 (4.8, 18.0) [a,b] | 11.9 (4.0, 19.1) [a] |
| Mean BP Power Morning (mmHg2/hour) | 192 (67, 398) | 103 (30, 303)[a] | 103 (46, 328) | 224 (79, 417) | 241 (125, 476) [a] | 241 (79, 485) [a] |

Data are displayed as median ($25^{th}$, $75^{th}$ percentiles). Abbreviations used: BP (blood pressure). * P<0.05 across age groups within race, [a or b] P<0.05 different to corresponding value across race

Age groups were determined using tertiles.