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## Relationship between Outdoor Temperature and Blood Pressure

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### Abstract

**Objectives**—Cardiovascular mortality has been linked to changes in outdoor temperature.

However, the mechanisms behind these effects are not well established. We aimed to study the effect of outdoor temperature on blood pressure (BP), as increased BP is a risk factor of cardiovascular deaths.

**Methods**—The study population consisted of men aged 53–100 years living in the Boston area. We used a mixed effects model to estimate the effect of three temperature variables: ambient, apparent, and dew point temperature (DPT), on repeated measures (every 3–5 years) of diastolic and systolic blood pressure. Random intercepts for subjects and several possible confounders were used in the models, including black carbon (BC) and barometric pressure.

**Results**—We found modest associations between diastolic BP and ambient temperature, and apparent temperature. In the basic models, increases in diastolic BP in association with a 5°C decrease in 7-day moving averages of temperatures were 1.01% (95% CI: –0.06 – 2.09), and 1.55% (95%, CI: 0.61 – 2.49) for ambient and apparent temperature, respectively. Excluding extreme temperatures made these associations stronger (2.13%, 95% CI: 0.66 – 3.63, and 1.65%, 95% CI: 0.41 – 2.90, for ambient and apparent temperature, respectively). Effect estimates for dew point temperature were close to null. The effect of apparent temperature on systolic BP was similar (1.30% increase (95% CI: 0.32 – 2.29) for a 5°C decrease in 7-day moving average).

**Conclusions**—Cumulative exposure to decreasing ambient and apparent temperature may increase BP. These findings suggest that increase in BP could be a mechanism behind cold-, but not heat-related cardiovascular mortality.

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## Keywords

Cardiovascular; Blood pressure; Climate; Epidemiology; Temperature

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## INTRODUCTION

Numerous study results have shown a link between high ambient temperatures and increased mortality, especially for cardiovascular diseases,[1–4] and exposure to cold temperature has also been shown to increase mortality.[5,6] Recently, associations between ambient temperature and morbidity have also been reported,[7,8] even though these associations have not always been similar in magnitude to mortality,[8] and null findings have also been reported.[9] Also, while the association between temperature and cardiovascular mortality has been reported to be U-shaped in many studies,[10] the association with cardiovascular hospitalization was reported to be linear in the US [7]. As climate change has been predicted to increase not only ambient mean temperature by 1.4 to 5.8 degrees Celsius by the end of this century, but also the variability of temperature, the occurrence of extreme weather conditions such as heat waves and sudden weather pattern changes may also increase.[11] Therefore the effects of ambient temperature on human health have recently become a target of vigorous research.[12,13]

Increased blood pressure (BP) is a risk factor for cardiovascular mortality [14,15] and for coronary heart disease and stroke morbidity [16,17]. Therefore changes in BP might also play a part in the development of cardiovascular events that are associated with changes in temperature. The effects of mild exposure to cold have been tested under controlled conditions, and the results have shown that short-term exposure to cold causes subcutaneous vasoconstriction that increases central blood volume that further increases BP.[18,19] However, the effects of temperature on BP under ambient conditions with changing air pressure and humidity, which may also include intermittent exposures as people go in and out of buildings, may differ substantially from those in chamber studies, and are not thoroughly studied. Two epidemiological studies from Europe have reported that systolic BP [20], or both systolic and diastolic blood pressure [21] decrease in association with increasing outdoor temperature. Another study has found that increases in outdoor and indoor temperatures may have independent, though similar, negative effect on systolic BP. [22] However, more studies also from North America are needed to assess the validity of these conclusions.

The mechanisms behind temperature-related cardiovascular mortality and morbidity are not fully established. As elevated BP is a known risk factor for cardiovascular disease and stroke events, BP can also be part of the mechanism leading to temperature-related deaths. We studied the effect of outdoor temperature on diastolic and systolic BP among elderly men using three different temperature variables (ambient, apparent, and dew point temperature (DPT)). In this study, we controlled for confounding by black carbon (BC), a marker of combustion particles that has been shown to have an effect on blood pressure in our cohort [23] and elsewhere.[24] Possible confounding by ozone was also studied as a suggestion of an association between ozone and blood pressure has been reported [25].

## METHODS

### Study Population

The study population consisted of the Normative Aging Study cohort of aging men established by the Veterans Administration in 1963, when 2,280 men from the Greater Boston area (21–80 years of age) confirmed to be free of known chronic medical conditions were enrolled.[26] Subjects were asked to return for physical examinations and to complete questionnaires every 3–5 years. Blood pressure was measured during each visit between January 1990 and June 2009 for participants (n=1200) still presenting for examination.

Study visits were conducted in the morning after an over-night fast and abstinence from smoking. Details of the physical examination have been previously described.[23] Blood pressure measurements were taken by a physician using a standard mercury sphygmomanometer with a 14-cm cuff. Systolic blood pressure (SBP) and fifth-phase diastolic blood pressure (DBP) were measured in each arm when the subject was seated. The mean of SBP from left and right arm, and similarly for DBP, was used in the analyses. Covariate data (age, medication, body mass index, alcohol consumption, smoking, etc.) were updated at each examination. All subjects provided written informed consent prior to attending the research, and this investigation has been approved by the Institutional Review Boards of Harvard School of Public Health and Normative Aging Study, Veterans Affairs Boston Healthcare System.

### Meteorological and Air pollution Measurements

Ambient temperature, dew point temperature, relative humidity and barometric pressure measurements were derived from the Boston Logan airport weather station. We used only one temperature measurement site for our analyses. However, the correlation between ambient temperatures at Boston Logan airport and T.F. Green Airport (Warwick, RI), which is 100 km away, was 0.96. This suggests that day to day variation in temperature at Boston airport is a good surrogate for day to day variation at the locations of the participants (mean distance from airport of 18 km). Apparent temperature, defined as a person's perceived air temperature, was calculated with the following formula:  $-2.653 + (0.994 \times \text{air temperature } (^{\circ}\text{C}) + (0.0153 \times \text{dew point temperature}^2 (^{\circ}\text{C}))$ .[27] Black carbon was measured using an aethalometer (Magee Scientific, Berkeley, CA) at the Harvard School of Public Health monitoring site, 1 km from the exam site. Black carbon measurements were available for January 1995 - December 2008. Ambient ozone was measured continuously at four monitoring sites in the Greater Boston area that were located in the cities of Boston, Chelsea, Lynn and Waltham. All monitors conformed to US Environmental Protection Agency (EPA) standards. In the analyses we used the average of the four measurements.

After excluding all missing data, we had 2343 clinic visits from 928 participants for the analyses. Since measurements for indoor temperature of the examination room did not start until in November 2000, there was a considerable amount of missing data for room temperature, and therefore it was not used in the analyses.

## Statistical Analyses

Blood pressure measurements were  $\log_{10}$ -transformed to improve the normality and stabilize variance. In the mixed effects model we examined whether ambient temperature influences BP, adjusting for *a priori* chosen known or plausible predictors of blood pressure. In this method, a random intercept is fitted for each subject, so differences across subjects are controlled for and the estimates of associations are effectively from within-subject differences. All models examining BP included fixed effects for personal characteristics: body mass index (BMI), age, cigarette smoking (never/former/current), alcohol consumption (  $\geq 2$  drinks/day, yes/no), use of any antihypertensive medication and statins (yes/no), diabetes (yes/no), fasting blood glucose, race, and years of education. Temporal variables used were: indicator variables for season (warm; May-September, cold; October-April) and weekday, and sine and cosine terms for day of year to capture seasonality more effectively.

In all models we used BC as a possible confounder. We previously reported that relevant associations between blood pressure and BC are the strongest over longer averaging times. We found a 1.46 (95% CI: 0.10 – 2.82) and 0.87 (95% CI: 0.15 – 1.59) mmHg increase in systolic and diastolic BP, respectively, for a 0.43  $\mu\text{g}/\text{m}^3$  increase in BC over 7-day moving average,[23] and therefore we used 7-day moving average for BC in the current models. It was also found in the study by Mordukhovich et al.[23] that BC, but not  $\text{PM}_{2.5}$ , was associated with blood pressure in this cohort. Additionally, all models controlled for barometric pressure 24 hours prior to study visit, and in the model for ambient temperature, the 24-hour mean of relative humidity was considered as a possible confounder. We controlled for relative humidity or used exposure measures incorporating humidity, because high humidity together with high temperature adds to the discomfort and heat stress.

The effects of temperature on mortality and morbidity have been seen over a lag period up to 7–10 days,[7] but more strongly at shorter lags. We therefore chose *a priori* to analyze lag days 0 to 7, and the moving averages of 2, 5, and 7 days. The analyses were performed using statistical software R 2.10.1. and its linear and non-linear mixed effects models library (nlme).[28]

As a sensitivity analysis, we ran a model that included separate dummy variables for each drug likely to influence blood pressure, i.e. the use of  $\beta$ - and  $\alpha$ -blockers, calcium channel blockers, angiotensin-converting enzyme inhibitors, angiotensin receptor antagonists, and diuretics. Including these variables into the model instead of a single variable for the use of any antihypertensive medication (yes/no) did not affect the results. Therefore, only the variable for the use of any antihypertensive medication (yes/no) was used in the final models. We also studied the possible confounding effect of ozone, because even though the association between ozone and blood pressure is not evident,[29,30] some studies have suggested ozone exposure has an effect on blood pressure [25]. The influence of extreme temperatures on our associations was studied by excluding 2.5% of the hottest and coldest temperatures from the analyses, and we did visual inspection of the linearity of the association between temperature and blood pressure using plots created by penalized spline models, using the generalized additive mixed model (gamm) function in R.

As secondary analyses, we studied possible effect modification of three variables. First, interactions between temperature variables and season were studied because blood pressure has been found to vary seasonally,[20] and because the association between cardiovascular mortality and temperature has often been described to be J-, U-, or V-shaped.[10] Second, we studied interactions between temperature and obesity (BMI>30) because people with more body fat may have more insulation against cold and worse capability of cooling their body than leaner people,[31] and therefore the thermoregulation between obese and lean persons may differ. Third, because the use of antihypertensive medication was common among the study subjects, the interaction between temperature and antihypertensive medication use was also studied.

## RESULTS

The total number of clinic visits was 2343 of which 1319 were during the warm season. A detailed description of the health variables at the first and the last visit is summarized in Table 1.

The variation in BP between seasons was minimal: mean DBP in mmHg (sd) was 75.4 (10.5) and 75.7 (11.0) during warm and cold season, respectively, and mean SBP was 130.0 (17.4) and 130.3 (18.4) during warm and cold season, respectively. On average, DBP was slightly higher among obese (76.6 mmHg, sd 10.7) than non-obese (75.1, sd 10.7). Difference in SBP was even smaller: SBP among obese was 129.8 mmHg (sd 16.9), and among non-obese 130.2 (sd 18.2).

The mean ambient, apparent, and dew point temperatures (sd) were 12.7 °C (8.7), 12.0 °C (9.8), and 6.4 °C (9.4), respectively, for the whole year (Table 2). Variation in the mean room temperature (23.9 °C) in the period from November 2000 to December 2008 was small with standard deviation of 1.6.

We found mainly negative associations between temperature variables and BP and therefore the results are presented as a percent change of the arithmetic mean of blood pressure (untransformed) for a 5 °C decrease in the temperature with 95% confidence interval (CI).

The association between a 5°C decrease in ambient temperature and DBP was the strongest at lag day 5 with 0.68% (95% CI: 0.04 – 1.33) increase in DBP. Significant associations were observed also between apparent temperature and DBP on lag days 0 and 5, and over the 2-, 5- and 7-day moving averages, the 7-day moving average having the strongest association with 1.55% increase in DBP (95% CI: 0.61 – 2.49) (Table 3). The effect estimates for DPT were somewhat different from the other 2 exposure variables, being closer to null. The only significant association was observed between the previous day DPT and SBP (Table 3). We found significant associations also between apparent temperature and SBP with similar lag structure to DBP (Table 3).

In all models, black carbon was a highly significant confounder. In post hoc analyses excluding black carbon, the association between ambient temperature and DBP became weaker with non-significant effect estimate for lag day 5 (0.48%, 95% CI: -0.17 – 1.14). Associations between apparent temperature and DBP also became weaker, and only the

association at lag day 5 remained significant (0.60%, 95% CI: 0.02 – 1.19, lag day 5, and 0.26%, 95% CI: –0.65 – 0.18 7-day moving average). Another post hoc analysis was performed for relative humidity. However, no associations were found between relative humidity and blood pressure (Online only supplement).

The associations we found were the strongest between DBP and apparent temperature over 5- and 7-day moving averages, but we observed association also on the current day. We chose lag 0 and moving average of 7 days for the more detailed analyses. In these analyses, lag day 0 was representing more acute effects, and the moving average of 7 preceding days, accumulated effects.

In the sensitivity analyses, we found that controlling for ozone had minor effect on the observed associations. The association between ambient temperature and DBP at lag 0 remained the same as in the main analysis (0.64%, 95% CI: –0.04 – 1.33). The increase in DBP in association with decrease in apparent temperature at lag 0 and over the 7-day moving average was 1.00% (95% CI: 0.35 – 1.66), and 1.42% (95% CI: 0.44 – 2.42), respectively. Ozone did not confound the association between apparent temperature and SBP (1.25%, 95% CI: 0.21 – 2.29 for 7-day moving average).

We observed little change in the results when the hottest (N=57) and coldest (N=50) 2.5% of the temperatures were excluded from the data. The increase in DBP was 0.73% (95% CI: 0.01 – 1.46), for a 5°C decrease in ambient temperature on the current day, and 1.54% (95% CI: 0.54 – 2.55) for a 5°C decrease in 7-day moving average of apparent temperature. Dew point temperature had slightly stronger effect on DBP (0.66%, 95% CI: 0.09 – 1.23, lag 0). The association between 7-day moving average of apparent temperature and SBP became weaker (1.09%, 95% CI: 0.04 – 2.15).

As another sensitivity analysis, we tested the linearity assumption by fitting penalized splines for apparent temperature using a generalized additive mixed model. The model chose the optimized degrees of freedom for temperature using generalized cross validation, which were 2 and 1 degrees of freedom for the 7-day moving average of apparent temperature in diastolic and systolic BP models, respectively, supporting linear effect.

In the secondary analyses, we found a significant interaction between all exposure variables and obesity (p-value <0.05) in the DBP, but not in the SBP models. We observed stronger associations among obese than leaner people (Table 4). Temperature variables had no interactions with season or with the antihypertensive medication use in our models.

## DISCUSSION

In this study, we found that diastolic blood pressure among elderly men increases in association with decreasing ambient and apparent temperature. These increases were not due to particulate pollution, ozone or extreme temperatures. We found weaker associations between temperature and systolic blood pressure.

We found an increase in diastolic BP in association with cumulative exposure to decreasing outdoor temperature. Our findings are similar in direction to the recently published findings



of the effects of temperature on SPB among elderly,[20] even though their result was not an effect controlling for air pollution. They also found seasonal variation in BP with higher values occurring during winter, whereas we did not find much difference in BP measurements between warm and cold season. Seasonal variation in BP was also found in a Norwegian study, but it disappeared after adjusting for outdoor temperature.[21] Consistently with our findings, Madsen and Nafstad [21] reported an increase in DBP and SBP in association with decreasing outdoor temperature. The multi-city Monica study [22] also reported findings consistent with those of our study, as have chamber studies on modest cold exposure.[18,19] Given that increased BP is a known risk factor of cardiovascular mortality and morbidity, these findings suggest that increase in blood pressure could be related to cold-induced cardiovascular mortality.

In this cohort, the associations between temperature and DBP were also stronger among obese than leaner people. This may partly be due to on average higher DBP among obese compared to non-obese. However, the hypothesis that greater amount of body fat provides better insulation, and therefore leads to smaller changes in BP when exposed to low temperatures could not be supported. In the study of Alperovich [20] the decrease in SBP in association with increasing temperature was also greater for obese; however, they did not report findings for DBP.

Black carbon was a significant confounder for the associations between temperature and blood pressure and inability to control for BC would have resulted in biased effect estimates. This finding is not surprising as an association between BC and blood pressure has already been seen in our study cohort.[23] In general, traffic-related air pollution and particles from combustion sources may have even greater adverse effects on cardiovascular health than PM<sub>10</sub> or PM<sub>2.5</sub> (particles with diameter <10 and <2.5 µm, respectively).[32–34] Therefore, the possible confounding of particles specifically from these sources should be controlled for, but several studies assessing the health effects of temperature have failed to do this. [1,7,20,35,36] Even though confounding by particles has not been observed in all studies of the health effects of temperature,[2,4] the evidence that PM<sub>10</sub> may act as a confounder in temperature-related cardiovascular mortality,[37,38] and the current finding for BC underline the importance of considering ambient particles as confounders, when the effects of temperature on blood pressure and other cardiovascular outcomes are evaluated.

Of the three temperature variables, we found the strongest associations for apparent temperature, but the effect of ambient temperature was very similar and the effect lag structures of these variables were consistent. Apparent temperature is an exposure variable that is used to describe how people perceive the combination of temperature and humidity. At warm temperatures, high humidity increases the feeling of discomfort and heat stress, and therefore apparent temperature may be more sensitive exposure variable for physiologic effects than ambient temperature. However, based on our findings, both of these exposure variables are useful when estimating the health effects of outdoor temperature. Dew point temperature had slightly different effect estimates from ambient and apparent temperatures, especially with the cumulative exposures, which may be due to the close relationship between DPT and relative humidity. DPT reflects temperature well on days when humidity is high, but if humidity is low it may be a worse proxy of temperature than humidity.

There are several limitations related to our study. One is that the study population consisted of elderly men from one metropolitan area, which is why the results are not representative for the general population, or applicable for regions with different climate conditions. For example, one study has suggested that vulnerability to cold-related mortality by is higher among elderly men than women,[39] and others have found regional differences in cold-related mortality.[5,40] Elderly people may also be more vulnerable to cold and heat exposures than younger populations due to changes in thermoregulation. As an example, older people may have poorer vasoconstrictor response to cold exposure than younger people, which leads to greater heat loss,[41] or they can suffer from reduced skin blood flow when under heat stress leading to inadequate heat loss.[42] However, controlling for the effects of sedentary lifestyle and chronic conditions may diminish the effect that aging has on thermal tolerance.[31]

Another limitation is that we were not able to control for the possible confounding effect of indoor temperature or clothing. Earlier it has been noticed that air conditioning and behavior can modify the adverse impacts of temperature extremes.[35] We had no data on the subjects' home indoor temperature, and the temperature in the examination room was measured for 5 years shorter time period than other variables. However, when indoor temperature was measured, it had generally little variation. Additional limitations are that we had only one measurement of BP for each clinic visit that may have been affected by many external stimuli, and even though we had data from several clinic visits for most of the subjects, some had only one visit during this study period. Mixed effect models are used especially for studies with repeated measures, but do not require repeated measures for all subjects. Even though we note that while the estimated random intercepts for subjects with only one measurement are noisily estimated, the inferences for the fixed effects, such as temperatures, are appropriate in these models. Moreover, these models have been used for the same population in previous studies.[23,43]

We also had data from only one measurement site for temperature and BC, which may cause some exposure misclassification. However, we did find high correlation between temperatures measured at airports 100 km apart, which suggests that variation in temperature in the study area is small. For BC, it seems likely that using one measurement station leads to an underestimation rather than overestimation of the health (or confounding) effects, because most of the measurement error would be so called Berkson error, which reduces power to reveal significant effect.[44] Furthermore, the goal of this paper was not to estimate the effect of BC, but of temperature and BC was examined only as a potential confounder.

In conclusion, we found that decrease in outdoor temperature can cause increase in diastolic blood pressure among elderly men. These results suggest that BP may increase with decreasing temperature therefore possibly playing part in cold-, but not heat-related mortality. However, more research on the effects of outdoor temperature on BP is needed using diverse study cohorts and personal measurements to confirm our findings. Ambient and apparent temperature can be used as exposure variables when investigating the health effects of temperature.



## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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### What this paper adds

- The mechanisms behind temperature-related cardiovascular deaths are not well understood.
- Because increased blood pressure is a risk factor for cardiovascular events, we studied the possible associations between outdoor temperature and blood pressure.
- We found that blood pressure increases in association with cumulative exposure to decreasing ambient and apparent temperature among elderly men.
- These results suggest that increase in blood pressure may be a mechanism triggering cold-, but not heat-related deaths.
- Apparent temperature may be more sensitive exposure variable for physiological effects than ambient temperature, but both exposures provided consistent findings in this study.

**Table 1**

Descriptive statistics for the first and last clinic visit and the number of clinic visits by subject

<b>Health Variable</b>	<b>First visit Mean (sd)</b>	<b>Last visit Mean (sd)</b>
Systolic blood pressure (mmHg)	135.5 (17.1)	128.0 (19.1)
Diastolic blood pressure (mmHg)	81.0 (9.1)	69.4 (9.9)
Age (years)	70.8 (7.1)	72.5 (11.2)
Body mass index (kg/m <sup>2</sup> )	28.0 (4.1)	27.9 (4.3)
Education (years)	14.4 (2.7)	14.5 (2.8)
	N of subjects (%)	(Total N=928)
Body mass index >30	240 (25.9)	243 (26.2)
Smoking status		
Never	266 (28.7)	263 (28.3)
Former	608 (65.5)	632 (68.1)
Current	54 (5.8)	33 (3.6)
Statin use (yes)	244 (26.3)	435 (46.9)
Use of antihypertensive medication (yes)	493 (53.1)	622 (67.0)
Diabetes (yes)	107 (11.5)	152 (16.4)
Race		
Non-hispanic white	907 (97.7)	907 (97.7)
Non-hispanic black	15 (1.6)	15 (1.6)
Hispanic white	5 (0.5)	5 (0.5)
Hispanic black	1 (0.1)	1 (0.1)
Alcohol intake (≥ 2 drink/day, yes)	207 (22.3)	177 (19.1)
Weekday of the visit		
Monday	208 (22.4)	52 (6.5)
Tuesday	67 (7.2)	201 (21.7)
Wednesday	378 (40.7)	567 (61.1)
Thursday	275 (29.6)	108 (11.6)
Number of visits by subject		
1	255 (27.5)	
2	196 (21.1)	
3	245 (26.4)	
4	199 (21.4)	
5	33 (3.6)	

**Table 2**

Descriptive statistics of the environmental variables.

<b>Weather Variables</b>	<b>Mean (sd)</b>	<b>Spearman Correlation with BC</b>
Ambient temperature (°C)	12.7 (8.7)	0.32
Apparent temperature (°C)	12.0 (9.8)	0.33
Dew point temperature (°C)	6.4 (9.4)	0.42
Relative humidity (%)	68.2 (15.5)	0.37
Barometric pressure (mbar)	1016.0 (7.7)	0.10
Black carbon (µg/m <sup>3</sup> )	0.96 (0.55)	–
Ozone (ppb)	23.8 (12.2)	–0.12

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**Table 3**

The percent change in diastolic and systolic blood pressure for a 5 °C decrease in the mean of ambient, apparent and dew point temperature.

	Diastolic Blood Pressure		Systolic Blood Pressure	
	% Change	95% CI	% Change	95% CI
<b>Ambient temperature †</b>				
Lag 0	0.63	-0.02 1.29	0.53	-0.16 1.22
Lag 1	0.08	-0.60 0.77	0.47	-0.25 1.19
Lag 2	0.22	-0.47 0.90	0.43	-0.29 1.15
Lag 3	0.24	-0.41 0.90	0.16	-0.52 0.85
Lag 4	0.37	-0.26 1.01	0.40	-0.26 1.06
Lag 5	0.68*	0.04 1.33	0.53	-0.14 1.21
Lag 6	0.34	-0.28 0.97	0.22	-0.43 0.87
Lag 7	0.35	-0.29 1.00	0.19	-0.48 0.87
2-d moving average	0.47	-0.28 1.23	0.63	-0.16 1.43
5-d moving average	0.69	-0.28 1.68	0.87	-0.16 1.90
7-d moving average	1.01	-0.06 2.09	1.04	-0.08 2.18
<b>Apparent temperature §</b>				
Lag 0	0.89*	0.30 1.49	0.56	-0.06 1.19
Lag 1	0.39	-0.20 0.99	0.51	-0.12 1.14
Lag 2	0.32	-0.26 0.90	0.44	-0.17 1.06
Lag 3	0.46	-0.12 1.04	0.31	-0.30 0.92
Lag 4	0.67	0.10 1.25	0.60*	0.00 1.20
Lag 5	0.94*	0.36 1.52	0.78*	0.17 1.39
Lag 6	0.51	-0.04 1.06	0.34	-0.24 0.92
Lag 7	0.50	-0.06 1.07	0.25	-0.34 0.84
2-d moving average	0.80*	0.14 1.47	0.67	-0.03 1.37
5-d moving average	1.14*	0.29 1.99	1.00*	0.12 1.90
7-d moving average	1.55*	0.61 2.49	1.30*	0.32 2.29
<b>Dew Point Temperature §</b>				
Lag 0	0.39	-0.13 0.91	0.24	-0.31 0.78

	Diastolic Blood Pressure		Systolic Blood Pressure	
Ambient temperature †	% Change	95% CI	% Change	95% CI
Lag 1	0.19	-0.36 0.73	0.71 *	0.14 1.29
Lag 2	0.14	-0.38 0.67	0.45	-0.10 1.00
Lag 3	-0.08	-0.57 0.40	0.04	-0.47 0.55
Lag 4	0.09	-0.38 0.56	0.09	-0.41 0.59
Lag 5	0.40	-0.09 0.89	0.08	-0.43 0.60
Lag 6	0.28	-0.22 0.77	0.00	-0.52 0.51
Lag 7	0.36	-0.14 0.86	0.12	-0.41 0.64
2-d moving average	0.39	-0.22 1.02	0.63	-0.02 1.28
5-d moving average	0.33	-0.46 1.13	0.69	-0.14 1.53
7-d moving average	0.61	-0.28 1.51	0.66	-0.28 1.60

\* p-value <0.05

† Model adjusted for black carbon, 24-h humidity, 24-h barometric pressure, season, time trend, weekday, age, smoking, years of education, alcohol intake ( 2 drinks per day), BMI, statin use, use of any antihypertensive medication, diabetes, fasting blood glucose, and race

‡ Model adjusted for black carbon, 24-h barometric pressure, season, time trend, weekday, age, smoking, years of education, alcohol intake ( 2 drinks per day), BMI, statin use, use of any antihypertensive medication, diabetes, fasting blood glucose, and race

**Table 4**

The percent change in diastolic blood pressure for a 5 °C decrease in temperature among obese and non-obese.

	Non-Obese			Obese		
	% Change	95% CI	% Change	95% CI	% Change	95% CI
<b>Ambient Temperature</b> †						
Lag 0	0.37	-0.31 1.05	1.24 *	0.43	2.07	
7-d moving average	0.76	-0.33 1.86	1.69 *	0.51	2.89	
<b>Apparent Temperature</b> §						
Lag 0	0.67 *	-0.11 *	0.06 1.29	1.38 *	0.65 2.12	
7-d moving average	1.31 *	0.35 2.28	2.10 *	1.07	3.15	
<b>Dew Point Temperature</b> §						
Lag 0	0.22	-0.33 0.76	0.79	0.10	1.49	
7-d moving average	0.36	-0.55 1.28	1.19 *	0.18	2.22	

\* p-value <0.05

† Model adjusted for black carbon, 24-h humidity, 24-h barometric pressure, season, time trend, weekday, age, smoking, years of education, alcohol intake ( 2 drinks per day), statin use, use of any antihypertensive medication, diabetes, fasting blood glucose, and race

§ Model adjusted for black carbon, 24-h barometric pressure, season, time trend, weekday, age, smoking, years of education, alcohol intake ( 2 drinks per day), statin use, use of any antihypertensive medication, diabetes, fasting blood glucose, and race