Full title: “My blood pressure is low today, do you have the heating on?” The association between indoor temperature and blood pressure

Brief title: Indoor temperature and blood pressure

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ABSTRACT

Objectives: The independent association of indoor temperature with blood pressure is poorly understood and is not routinely considered in hypertension diagnosis or research. Questions remain as to whether the effect of indoor temperature on BP is confounded or modified by other factors.

Methods: This study used data from the Health Survey for England 2014, consisting of 4,659 community-dwelling adults aged 16 and over, interviewed from January to December. Multivariable regression models were used to determine whether indoor temperature was related to levels of blood pressure, and if these relationships were confounded by other factors, including mean monthly outdoor temperature.

Results: After controlling for confounding variables, a 1°C decrease in indoor temperature was associated with rises of 0.48 mmHg (95% CI: -0.72, -0.25) in systolic blood pressure and 0.45 mmHg (95% CI: -0.63, -0.27) in diastolic blood pressure. The magnitude of association of indoor temperature with diastolic and systolic blood pressure was modified by physical activity. The indoor temperature-blood pressure relationship was stronger in people who do not take physical activity regularly compared with people who exercise regularly.

Conclusions: The size of the independent association between indoor temperature and blood pressure suggests it should be considered in the clinical management of hypertension and in hypertension research. Room temperature should also be considered as a modifiable risk factor in hypertension-related mortality and morbidity.

INTRODUCTION

Cardiovascular disease (CVD) remains a major cause of death worldwide, associated with 30% of all global deaths in 2008 (17.3 million).[1,2] There is growing evidence that the prevalence of CVD-associated mortality and morbidity is significantly higher in winter or in cold environments.[3–5] One British study has indicated that a winter-summer difference in systolic blood pressure (SBP) of 12mmHg could contribute to a 20% increase in stroke and ischaemic heart disease mortality in winter.[6] Such relationship was also observed in a European study, reporting that for a 1°C decrease in outdoor temperature, the CVD-associated mortality increased by 1.72%.[7] Raised blood pressure (BP) is known as a leading cause of CVD, and also as an important intermediate marker of cardiovascular health.[8] Increased CVD-associated mortality in cold environments can partly be explained by the underlying association between temperature and BP.[9] Both SBP (systolic blood pressure) and DBP (diastolic blood pressure) are higher in winter than in summer in different contexts.[10–12] Additionally, the effect of indoor temperature deserves attention as people, particularly those living in cold climates, spend a lot of time indoors, which could influence their BP in the short and long term.[13] Therefore, accurately measuring BP, with ambient temperature taken into account, is of great importance to studies of hypertension and related mortality and morbidity risks.[14] As for the clinical implications, a clear identification of prognostic relevance regarding temperature-BP association can help improve the hypertension diagnosis, and inform decisions regarding the use of antihypertensive medication. For example, higher doses of drugs may be required for hypertensives in cold environments to achieve normal BP control. Moreover, social prescribing could recommend subsidies for higher indoor temperature for

those who cannot heat their home. Generally, effective management of hypertension could save medical costs and reduce CVD-associated mortality during the winter months.[15,16]

Most studies have observed a significant inverse association between ambient temperature and BP.[17–20] The association, however, is not similar in magnitude across studies due to differences in the studies' samples. One study looking at normotensive women aged 18 to 40 years in Delhi, India, indicated that the mean SBP and DBP in winter was 11.07mmHg and 6.79mmHg higher than in summer, respectively.[12] Another study, with a sample of more than 500,000 adults in China, reported that for each 10°C decrease in outdoor temperature, the mean SBP and DBP rose by 5.7mmHg and 2.0 mmHg respectively.[18] A stronger association between indoor temperature and BP compared with outdoor temperature has been identified in inter-climate analysis, and within-climate analysis.[9,21] One cohort study in Scotland indicated that a threshold of indoor temperature below 18°C could have a greater impact on blood pressure changes.[13] This threshold of indoor temperature is also suggested by Public Health England to minimise health risks in winter, particularly for people 65 years and over.[22]

The confounding or modifying effect of social determinants has been supported in the current evidence. The magnitude of the temperature-BP association is higher in the presence of the risk factors of hypertension, such as increasing age, poor mental health and reduced physical activity.[19,23,24] The effects of demographic factors, such as ethnicity and income, have also been explored.[18,25]

Despite the evidence of the ambient temperature-BP phenomenon, substantial uncertainty remains regarding the strength of this association, particularly with

regards the effect of indoor temperature. Previous studies have mostly considered indoor temperature as an interactive factor when studying the effect of outdoor temperature on seasonal BP variation.[9,10,21] The strength of the seasonal-variation-BP association could be underestimated in winter due to the use of home-heating equipment, which may prevent BP from rising.[26,27] Independent association of BP with indoor temperature is still unclear, however. Moreover, most studies have small sample sizes, and most were conducted in highly selective groups. Previous studies have also failed to clearly identify the effects of interfering factors, such as gender and anti-hypertensive medication on results. For instance, two studies from Asia have indicated that women were more sensitive to the cold than men,[28] whereas the opposite conclusion was found by two studies from Europe.[8] It is possible that differences across study samples may have caused these apparently contradictory results in relation to social determinants. Therefore, a large population-based study is needed to assess the independent relationship between indoor temperature and BP, and to clearly identify potential factors that could affect this association.

In light of existing literature we test the following research hypotheses:

A decrease in indoor temperature is associated with an increase in BP, independent of other interfering factors.

The indoor temperature-BP relationship is moderated by factors, including physical activity.

To our knowledge, this study is the first population-based study assessing the independent relationship between indoor temperature and BP in England. Findings

from this study could help improve accurate diagnosis of hypertension, promote clinical practice to reduce cold-related mortality, and suggest basic improvements in indoor environments.

METHODS

Study Design and Participants

This study is a secondary analysis using data from the Health Survey for England (HSE) 2014. The HSE is a nationally representative cross-sectional survey examining the health and wellbeing of people living in England.[29] Participants were selected through a multistage stratified random sampling procedure. Data collection included two stages. In the first stage, trained interviewers would complete a main questionnaire covering topics such as general health, smoking habits and other lifestyle factors. At the end of the interview, participants were asked to consent for a follow-up visit by a professional nurse. The nurse would undertake examinations, including BP measurements and blood samples. A total of 8,077 respondents aged 16+ were interviewed, 5,491 consented to a nurse visit. The response rate was 85% for the interviewer stage and 58% for the nurse visit stage.

Participant inclusion criteria stated that adults must be aged 16 years or over, with a valid response to BP and indoor temperature measurements (n = 4,659). To reduce non-response bias at both stages, survey weights were used in all analyses.

BP Measurement

Participants were asked, during the initial interview, to abstain from eating, smoking, drinking alcohol or engaging in vigorous exercise 30 minutes prior to the nurse's visit. Pregnant women were excluded from the measurement. Participants were

instructed to remain seated for five minutes to rest, and their BP was then measured at three one-minute intervals with the use of a digital oscillometric BP monitor (Omron HEM 907; Omron Corp, Kyoto, Japan). The device has been shown to satisfy the British Hypertension Society and American Association for the Advancement of Medical Instrumentation accuracy criteria in adult samples. [30,31] Depending on each participant's upper arm circumference, an appropriately sized cuff was used, placed on the upper right of the arm. The mean of the last two readings was used in this study.

Indoor Temperature Measurement

Indoor temperature was defined as the ambient temperature in the participants' living rooms. To measure indoor temperature, the nurses used a standard digital thermometer (Digitron 20461) during the five minutes rest time before the BP measurement. The thermometer probe was kept away from heat sources, such as radiators or sunlight, in order to ensure an accurate measurement of indoor temperature. It was recommended that the probe hung over the edge of table. When prompted by Computer Assisted Personal Interviewing (CAPI) to take a reading, the nurse turned on the thermometer and recorded the stable reading in CAPI to one decimal place.

Covariates Inclusion

Equivalised household income (taking into account family size) was grouped into five equal-sized quintiles. Area deprivation was derived from the English Index of Multiple Deprivation (IMD) 2010, which was compiled by the Department of Communities and Local Government. Respondents' neighbourhoods were split into

five quintiles (from least deprived to most deprived) based on an overall deprivation score.[32]

Individual mental health was measured using a self-completion GHQ-12 (General Health Questionnaire). Participants were asked about their quantity of alcohol consumption during the past 12 months. Consumption in units per week was classified as four risk groups: (1) none (no past drinking or <1 unit); (2) light (1-10 units for men and 1-7 units for women); (3) medium (11-21 units for men and 8-14 units for women); and (4) heavy (>21 units for men and >14 units for women).[29]

Physical activity was derived from the amount of moderate or vigorous intensive activity (MVPA) engaged in during the week prior to the interview. It was categorised as: high (30 minutes of MVPA at least five days a week); middle (30 minutes of MVPA one to four days a week); and low (less than 30 minutes of MVPA a week).[33]

Height (cm) and body weight (kg) were measured in terms of standard procedures, and used to calculate body mass index (BMI, kg/m^2). Total cholesterol (mmol/L) and urine sodium excretion (mmol/L) were measured from blood analytes. Details of the collection procedures have been described elsewhere.[29]

Monthly mean outdoor temperature was measured using UK Met Office historic station data at a least one site per government office region (GOR) in England.[34] These data were matched to HSE using the month of nurse visit and GOR of a respondent.

Other covariates included: age (in years); gender (male/female); highest educational qualification (degree or equivalent/below degree/no qualification); marital status (married/unmarried); ethnicity (white/non-white); smoking status (current/past/never) and type 2 diabetes diagnosis (yes/no).

Statistical Analysis

Multiple imputation using chained equations were used to impute missing covariate data. One hundred imputed datasets were estimated.[35,36]

Chi-square tests and variance analyses were used to compare the distribution of baseline characteristics across indoor temperature categories. A *P* value of all statistical analyses <0.05 was considered statistically significant.

We investigated the independent association of indoor temperature with SBP and DBP variability using a multivariable linear regression model after adjustment for confounding variables in a series of stepwise models. We presented standardised coefficients to compare the scale of the relationship between BP and the covariates in the final model.

Interaction analyses were used to investigate if the magnitude of the effect of indoor temperature on BP differed in subgroups based on the following effect modifiers: age; gender (male/female); BMI; GHQ-12 score; alcohol consumption (none/light/medium/heavy); smoking status (current/past/never); physical activity levels (high-middle/low), and total cholesterol. These effect modifiers were selected according to previous studies or our own research hypotheses.[8,18,19] Lanzinger et al. [8] find the ambient temperature blood pressure relationship to be stronger in men, aged 60 and over, obese and those who spend more time indoors. Lewington et al. [18] support the age effect modification and Chen et al.[19] support the gender effect modification, but both find a stronger association in individuals with lower BMI. Chen et al. [19] also find the relationship to be stronger in those who drink alcohol.

To analyse sensitivity, we limited the model based on untreated (including normotensive and hypertensive), normotensive (including treated and untreated) and hypertensive (including treated and untreated) to observe whether the associations remained in people without antihypertensive medication, without high blood pressure and with high blood pressure. Use of antihypertensive medication is likely to regulate the indoor temperature–BP association. Therefore, focusing on the subjects without antihypertensive medication can test the robustness of this association. To further test sensitivity we predict classification of hypertension stages (normal, pre, stage 1 and stage 2) by indoor temperature using a multinomial regression model containing all covariates in the final model.

RESULTS

Table 1 summarises the baseline characteristics of participants across indoor temperature categories. Both SBP and DBP showed a negative association with indoor temperature, with a decreased trend from lower temperature levels to higher temperature levels (both *P* values <0.001).

Mean SBP and DBP were 126.64 mmHg and 74.52 mmHg, respectively, in the lowest temperature levels, compared with 121.12 mmHg and 70.51 mmHg, respectively, in the highest temperature levels.

Among the other participant characteristics, ethnicity, alcohol consumption and diabetes diagnosis were significantly associated with indoor temperature levels.

Table 2 shows the adjusted association of indoor temperature on SBP and DBP after adding confounders. Although the association remained significant when the confounding variables were included in both models (*P* values<0.001), there was a

substantial attenuation of the effect of indoor temperature on SBP after adding outdoor temperature to the model. In the unadjusted model, 1°C increase in indoor temperature corresponded to a 0.73 (95% CI: -1.00, -0.46) mmHg and 0.46 (95% CI: -0.64, -0.28) mmHg decrease of SBP and DBP, respectively. The association was robust to the inclusion of demographics, health measurements and lifestyle factors. After accounting for all variables a 1°C decrease in indoor temperature was associated with a 0.5mmHg increase in both SBP and DBP.

Table 3 provides the results of the relative effect sizes of all covariates for BP. A one SD increase in indoor temperature was associated with a 1.10 mmHg decrease in SBP (95% CI: -1.64, -0.56) and a 1.02 mmHg decrease in DBP (95% CI: -1.43, -0.62). The size of the indoor temperature SD unit association with DBP was equivalent to the difference between female relative to male, holding all other variables constant. Interactions between indoor temperature and eight potential effect modifiers were investigated. The only significant interaction with indoor temperature was in physical activity. Figure 1a and Figure 1b show how the indoor temperature-SBP and temperature-DBP relationship, respectively is stronger in those who take less physical activity. Holding all other effects constant, when indoor temperature increased from 18°C to 22°C, predicted DBP decreased by 2.46 mmHg in the low physical activity group compared with 1.08 mmHg in the middle to high physical activity group.

Table 4 shows the associations between indoor temperature and SBP/DBP limited to people not taking antihypertensive medication, normotensives (treated and untreated) and hypertensives (treated and untreated). The associations of both SBP and DBP remained statistically significant among untreated participants (*P*

values<0.001), suggesting robustness of the results for the adjusted model. The same is true for normotensives. There was no indoor temperature-SBP association in hypertensives. The multinomial model (Table 5) predicting hypertension classification shows the probability of hypertension (stage 1 or 2) is greater if the indoor temperature is lower relative to those who have normal hypertension.

DISCUSSION

Based on a large population-based study of 4,659 people aged 16+ in England, we observed a statistically significantly and clinically relevant inverse association between indoor temperature and BP. This finding confirmed our hypothesis that with a decrease in indoor temperature, there is an increase in BP. Additionally, the magnitude of the effect of indoor temperature on BP, especially on DBP, is robust to confounding and including and excluding those taking BP medication. Outdoor temperature had a strong confounding effect on the relationship between indoor temperature and SBP. Sensitivity analysis confirmed the probability of hypertension classification (i.e stage 1 or stage 2 vrs normal) was greater if indoor temperature is lower. Moreover, the effect of indoor temperature on blood pressure varies by physical activity, suggesting those who do not take exercise regularly are more susceptible to blood pressure variation in cooler or warmer indoor environments. As well as adding to the body of literature indicating an inverse relationship between indoor temperature and BP, our study further demonstrates that this relationship is independent of the presence of confounding factors. Indoor temperature in most previous studies was considered to be a modifier of seasonal BP variation.[21] It is often found that the effect of outdoor temperature on BP is attenuated after

controlling for indoor temperature. Nevertheless, when an independent association between indoor temperature and BP is confirmed, the magnitude of the association is similar to that reported in this study and any deviation is perhaps a result of small selective samples used in other studies. [10,21]

We investigated the interaction of this relationship to determine if the association of indoor temperature on BP would be different if potential modifiers were taken into account. Consistent with the findings of Kristal-Boneh et al. [23] we found indoor temperature had a stronger association with BP among people who take less regular physical activity. One possible reason is that people who exercise less regularly do not rise their body temperature as frequently, which itself will have a regulatory affect on BP. Therefore, our results suggest that people should be encouraged to exercise more regularly and those who do not should keep warm in cold environments to minimise BP fluctuation. Among other covariates, our results showed that the association of indoor temperature with SBP or DBP differed negligibly by age, gender, BMI, mental health, alcohol consumption, smoking status and total cholesterol.

Our study has a number of strengths. First, this is a large population-based research study looking at the association of indoor temperature with BP. Our HSE study sample was much larger, covering a number of subjects in a wider age range, than previous similar studies looking at indoor temperature.[9,21]

The second strength of our study is the accurate measurement of indoor temperature in participants' living rooms. Previous studies reported a relationship between indoor temperature and BP in workplaces, or using personal-level environmental temperatures measured by thermosensors attached to ambulatory

BP monitoring.[24] These methods have been shown to be less accurate due to the effect of clothing or skin temperature.[27]

Another strength is that we estimated the magnitude of the association between indoor temperature and BP, independent of other covariates. The information regarding most of the covariates affecting this relationship, as discussed in previous studies, was available in HSE.

There are several limitations that should be acknowledged. The first limitation of this study lies in the unavailability of information on environmental exposure, such as humidity and outdoor temperature at the point of blood pressure examination. Although we have presumed that people spend most of their time indoors and are influenced more by indoor temperature, the effect of outdoor temperature on BP variation should not be ignored, especially under extreme weather conditions and for those who spend large amounts of time in environments that are a different temperature to their living room.[14] Therefore, exposure misclassification could exist as a possible source of bias in the study as we only investigate the effect of indoor temperature and mean monthly outside temperature.

A related limitation is that we did not take into account the timing of measurement. Timing of measurement refers to two aspects: duration of staying indoors before the measurement and the time of the measurement. First, any mechanism involved in this relationship must have a long-lasting effect on BP.[11] Saeki et al.,[37] indicated that the interpretation of the magnitude of temperature-BP association should depend on the amount of time spent indoors or outdoors. The large difference between outdoors and indoors might cause BP changes in a short time, and therefore measurement bias. We could calculate the indoor exposure time by using the

average length of interview minus the time after the BP measurement, providing nurses followed the procedures to examine participants. This was still unsupported, however, as we did not know how long the participants had stayed indoors before the nurses visited. Moreover, an ideal study would have information on the time of the BP measurement, or even draw a picture of an individual's BP variation within 24 hours by taking ambulatory BP.[25] Use of ambulatory BP could provide a better description of the association between indoor temperature and BP, thereby strengthening the result.[12,18]

A final limitation regards the cross-sectional nature of the study. Cross-sectional studies preclude investigation of BP variation in the same subjects, and lack temporal evaluation of the relationship. There is more confidence in the relationship obtained from a follow-up cohort study with multiple BP measurements on the same individuals. Additionally, a follow-up cohort study may also help to explore the relationship of hypertension care status, as we can look at individuals who change from one care group to another, but keep their indoor temperature and other factors the same.

PERSPECTIVES

Clinical implications of the indoor temperature-BP relationship have been confirmed in this study. We suggest that for people with borderline high BP, the association can help make the decision about starting antihypertensive treatment. Higher doses may be needed at lower temperatures for those individuals diagnosed as hypertensive, or people who are undergoing treatment. Such adjusted and accurate hypertension management will reduce mortality and medical costs in the population, especially for

vulnerable individuals. Findings from this study also suggest that basic improvements to indoor environments and better home heating would help mitigate the increase in BP due to indoor temperatures. This study also highlights the need for future studies of BP to take indoor temperature into account, for example, using a regression coefficient to standardise BP to a fixed indoor temperature. Further studies with a cohort design are needed to support the independent relationship between indoor temperature and BP, as well as roles of other factors in this relationship.

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Novelty and Significance

What is new?

- This is first study in England to use a population-based sample to estimate the association between indoor temperature and blood pressure by adjusting for potential confounders and effect modifiers.

What is relevant?

- Clinically relevant relationship demonstrated between indoor temperature and blood pressure in England. A 1°C change in indoor temperature is the equivalent of the difference in predicted DBP between men and women.
- Physical activity moderates the relationship between indoor temperature and diastolic blood pressure: those who do not exercise regularly are more sensitive to indoor temperature.

Summary

- We find a relationship between indoor temperature when taking into account outdoor temperature and other potential confounders of the relationship.

Tables and figures

Table 1. Baseline Distribution of Participants Characteristics by Indoor Temperature

Participants Characteristics	All	Indoor temperature (℃)				*P* value
		T<18	18≤T<21	21≤T<24	T≥24	
		Mean (SD)				
Age (years)	47.60 (19.08)	46.25 (17.76)	47.69 (19.32)	48.34 (19.40)	45.82 (17.83)	0.207
BMI (kg/m2)	27.17 (5.40)	26.72 (5.04)	27.07 (5.49)	27.39 (5.46)	27.46 (5.11)	0.247
GHQ-12 (scores)	1.40 (2.60)	1.39 (2.56)	1.34 (2.55)	1.48 (2.70)	1.43 (2.45)	0.581
Total cholesterol (mmol/L)	5.12 (1.11)	5.12 (1.17)	5.13 (1.11)	5.08 (1.11)	5.24 (1.01)	0.315
Urine sodium (mmol/L)	88.02 (53.43)	91.29 (53.74)	86.94 (52.66)	88.35 (54.24)	88.01 (52.54)	0.544
SBP (mmHg)	125.03 (16.86)	126.64 (16.13)	126.2 (17.22)	123.81 (16.65)	121.12 (15.67)	<0.001
DBP (mmHg)	72.47 (11.20)	74.52 (11.24)	72.81 (11.35)	71.77 (11.05)	70.51 (10.23)	<0.001
		N (%)				
Gender						0.218
Male	2056 (100.00)	225 (11.29)	999 (47.43)	706 (34.27)	126 (7.02)	
Female	2603 (100.00)	292 (11.38)	1223 (45.47)	898 (34.47)	190 (8.68)	
Highest education qualification						0.209
Degree or equivalent	1204 (100.00)	156 (13.49)	567 (44.57)	392 (33.00)	89 (8.93)	
Below degree	2423 (100.00)	260 (10.92)	1169 (47.17)	840 (34.49)	154 (7.43)	
No qualification	1026 (100.00)	101 (9.56)	482 (46.76)	371 (36.11)	72 (7.57)	
Marital status						0.050
Married	2111 (100.00)	269 (12.49)	1022 (46.82)	673 (32.14)	147 (8.55)	
Unmarried	2547 (100.00)	248 (10.21)	1200 (46.08)	931 (36.61)	168 (7.10)	
Equivalised household income						0.087
lowest quintile	628 (100.00)	67 (10.01)	291 (42.40)	218 (34.48)	52 (13.11)	

2nd lowest quintile	583 (100.00)	62 (10.87)	273 (45.87)	209 (35.53)	39 (7.73)	
middle quintile	915 (100.00)	83 (9.78)	446 (47.45)	322 (35.2)	64 (7.57)	
2nd highest quintile	903 (100.00)	108 (11.72)	461 (51.17)	277 (31.54)	57 (5.57)	
highest quintile	870 (100.00)	104 (11.68)	416 (47.04)	294 (34.27)	56 (7.01)	
Area deprivation						0.054
least deprivation	1120 (100.00)	116 (11.20)	524 (47.03)	415 (36.01)	65 (5.75)	
2nd least deprivation	962 (100.00)	124 (12.53)	477 (48.19)	315 (33.94)	46 (5.35)	
middle deprivation	949 (100.00)	106 (11.67)	453 (47.78)	335 (34.34)	55 (6.20)	
2nd most deprivation	889 (100.00)	90 (9.59)	435 (47.06)	286 (33.03)	78 (10.32)	
most deprivation	739 (100.00)	81 (11.83)	333 (41.05)	253 (34.29)	72 (12.83)	
Ethnicity						0.002
White	4222 (100.00)	475 (11.66)	2045 (47.39)	1440 (33.77)	262 (7.18)	
Non-white	435 (100.00)	41 (8.83)	176 (39.26)	164 (38.92)	54 (12.99)	
Alcohol consumption						0.010
None	1416 (100.00)	133 (9.08)	669 (44.76)	494 (36.08)	120 (10.08)	
Light	1584 (100.00)	177 (10.97)	755 (47.11)	552 (34.71)	100 (7.21)	
Medium	724 (100.00)	98 (14.28)	354 (48.24)	226 (29.63)	46 (7.84)	
Heavy	851 (100.00)	102 (13.82)	404 (46.18)	298 (34.03)	47 (5.97)	
Smoking status						0.845
Current smoking	640 (100.00)	73 (10.70)	299 (46.91)	219 (34.65)	49 (7.74)	
Past smoking	1609 (100.00)	188 (12.18)	764 (46.07)	563 (35.66)	94 (7.09)	
Never smoking	2259 (100.00)	240 (11.75)	1083 (45.25)	708 (34.10)	156 (8.90)	
Physical activity level						0.132
Low	2146 (100.00)	209 (10.11)	1018 (46.02)	756 (34.92)	163 (8.96)	
Middle	1546 (100.00)	204 (13.23)	746 (46.43)	502 (33.20)	94 (7.14)	
High	939 (100.00)	103 (11.03)	444 (47.26)	336 (34.90)	56 (6.81)	

Hypertension care outcome						<0.001
Normotensive	3088 (100.00)	343 (11.17)	1440 (45.50)	1076 (34.70)	225 (8.63)	
Hypertensive untreated	685 (100.00)	102 (15.61)	338 (49.10)	218 (31.13)	27 (4.15)	
Hypertensive controlled	557 (100.00)	43 (8.08)	267 (45.69)	202 (38.16)	45 (8.07)	
Hypertensive uncontrolled	329 (100.00)	29 (8.83)	173 (52.36)	108 (31.68)	19 (7.14)	
Type2 diabetes diagnosis						0.008
Yes	332 (100.00)	16 (5.15)	167 (49.14)	126 (38.65)	23 (7.06)	
No	4326 (100.00)	501 (11.75)	2054 (46.23)	1478 (34.09)	293 (7.93)	
Total	4659 (100.00)	517 (11.34)	2222 (46.42)	1604 (34.37)	316 (7.87)	

Note. T=indoor temperature; SD=standard deviation.

Table 2. Multivariable Associations of Indoor Temperature with Blood Pressure among England Adults

	SBP		DBP	
	β (95% CI)	**Adjusted R^2**	**β (95% CI)**	**Adjusted R^2**
Crude	-0.73 (-1.00, -0.46)	0.01	-0.46 (-0.64, -0.28)	0.01
Model 1	-0.70 (-0.91, -0.49)	0.22	-0.48 (-0.65, -0.30)	0.02
Model 2	-0.70 (-0.91, -0.49)	0.23	-0.48 (-0.65, -0.30)	0.04
Model 3	-0.71 (-0.91, -0.51)	0.26	-0.51 (-0.67, -0.35)	0.17
Model 4	-0.48 (-0.72, -0.25)	0.27	-0.45 (-0.63 -0.27)	0.18

Model 1: adjusted for age, gender, marital status and ethnicity
Model 2: model 1 +, education, income and area deprivation.
Model 3: model 2 + BMI, GHQ-12, type2 diabetes diagnosis, total cholesterol, sodium excretion, alcohol consumption, smoking status and physical activity level.
Model 4: model 3 + outdoor temperature
All regression coefficients are statistically significant (*P* value<0.001)

Table 3. Standardised coefficients of predictors for blood pressure in the final model [a]

	SBP		DBP	
Predictors	**Beta (95% CI)**	***P* value**	**Beta (95% CI)**	***P* value**
Age (years)	5.94 (5.36, 6.52)	<0.001	0.25 (-0.17, 0.68)	0.246
BMI (kg/m2)	2.27 (1.74, 2.81)	<0.001	3.14 (2.76, 3.53)	<0.001
GHQ-12 (scores)	-0.39 (-0.86, 0.08)	0.125	0.01 (-0.32, 0.34)	0.965
Total cholesterol (mmol/L)	1.84 (1.25, 2.43)	<0.001	2.30 (1.86, 2.73)	<0.001
Urine Sodium (mmol/L)	0.38 (-0.09, 0.86)	0.268	0.03 (-0.35, 0.4)	0.880
Outdoor temperature (℃)	-1.06 (-1.62, -0.5)	<0.001	-0.28 (-0.68, 0.12)	0.170
Indoor temperature (℃)	-1.10 (-1.64, -0.56)	<0.001	-1.02 (-1.43, -0.62)	<0.001
Gender				
Male [b]				
Female	-7.57 (-8.44, -6.70)	<0.001	-1.11 (-1.77, -0.45)	0.001
Highest education qualification				
Degree or equivalent [b]				
Below degree	1.66 (0.56, 2.76)	0.003	-0.20 (-1.06, 0.66)	0.652
No qualification	1.69 (0.06, 3.32)	0.042	-2.30 (-3.48, -1.13)	<0.001
Marital status				
Married [b]				
Unmarried	-2.59 (-3.53, -1.65)	<0.001	0.86 (0.14, 1.57)	0.020
Equivalised household income				
lowest quintile [b]				
2nd lowest quintile	-0.77 (-2.71, 1.17)	0.437	-0.75 (-2.23, 0.74)	0.323
Middle deprivation	-0.94 (-2.72, 0.83)	0.296	-0.76 (-2.12, 0.6)	0.273
2nd highest quintile	-0.76 (-2.61, 1.08)	0.417	-0.35 (-1.74, 1.04)	0.618
highest quintile	-0.82 (-2.69, 1.06)	0.392	0.40 (-1.06, 1.85)	0.593

Area deprivation				
least deprivation [b]				
2nd least deprivation	0.44 (-0.88, 1.75)	0.514	0.23 (-0.82, 1.27)	0.671
middle	0.85 (-0.58, 2.28)	0.246	0.80 (-0.28, 1.88)	0.146
2nd most deprivation	-0.53 (-1.96, 0.91)	0.471	0.58 (-0.57, 1.74)	0.322
most deprivation	0 (-1.7, 1.7)	0.996	0.74 (-0.5, 1.99)	0.243
Ethnicity				
White [b]				
Non-white	-0.71 (-2.32, 0.89)	0.382	1.71 (0.34, 3.08)	0.014
Type2 diabetes diagnosis				
Yes [b]				
No	-2.40 (-4.46, -0.35)	0.022	1.99 (0.57, 3.41)	0.006
Alcohol consumption				
None [b]				
Light	-0.05 (-1.22, 1.13)	0.935	0.06 (-0.83, 0.95)	0.888
Medium	0.77 (-0.61, 2.15)	0.276	0.75 (-0.32, 1.82)	0.169
Heavy	2.94 (1.47, 4.41)	<0.001	2.13 (1.01, 3.25)	<0.001
Smoking status				
Never smoking [b]				
Past smoking	-1.07 (-2.08, -0.06)	0.038	-0.24 (-0.97, 0.49)	0.525
Current smoking	-0.7 (-2.1, 0.7)	0.325	1.06 (-0.06, 2.18)	0.064
Physical activity level				
Low [b]				
Middle	0.23 (-0.82, 1.28)	0.665	0.05 (-0.76, 0.86)	0.899
High	1.02 (-0.23, 2.27)	0.11	-0.18 (-1.1, 0.74)	0.697

[b] Reference groups.

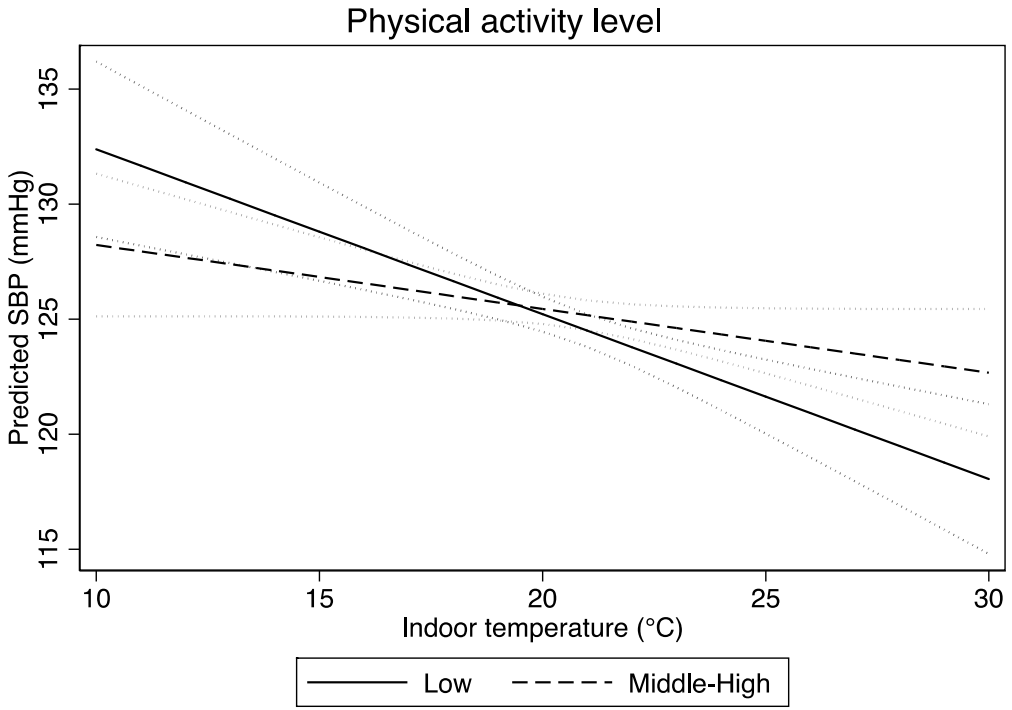

Figure 1a. Association of indoor temperature and SBP by physical activity. Interaction was statistically significant (*P* value<0.05).

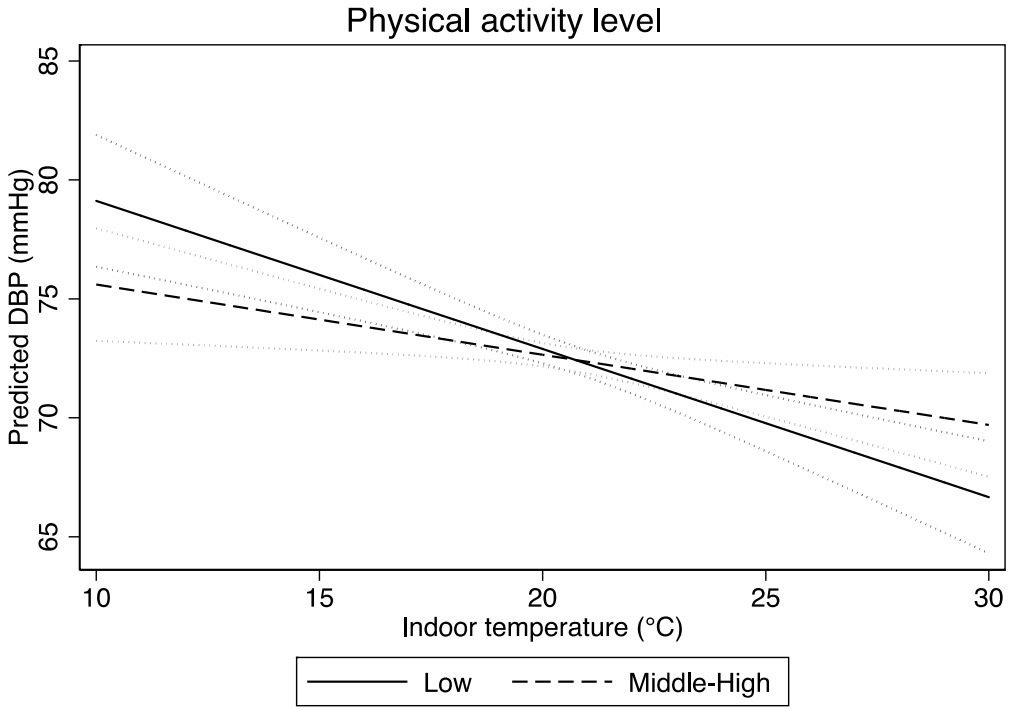

Figure 1b. Association of indoor temperature and DBP by physical activity. Interaction was statistically significant (*P* value<0.05).

Table 4. Association between indoor temperature and blood pressure among untreated (including normotensive and hypertensive), normotensive (including treated and untreated) and hypertensive (including treated and untreated)

		SBP		DBP	
		β (95% CI)	***P* value**	**β (95% CI)**	***P* value**
Untreated	**Crude model**	-0.80 (-1.07, -0.53)	<0.001	-0.46 (-0.66, -0.27)	<0.001
	Adjusted [a] model	-0.51 (-0.76, -0.26)	<0.001	-0.46 (-0.65, -0.26)	<0.001
Normotensive	**Crude model**	-0.40 (-0.61, -0.19)	<0.001	-0.24 (-0.41, -0.07)	0.006
	Adjusted [a] model	-0.24 (-0.43, -0.05)	0.015	-0.24 (-0.40, -0.08)	0.004
Hypertensive	**Crude model**	0.10 (-0.27, 0.47)	0.613	-0.43 (-0.79, -0.06)	0.022
	Adjusted [a] model	-0.32 (-0.75, 0.11)	0.140	-0.38 (-0.68, -0.08)	0.013

[a] Adjusted for age, gender, marital status, education, income, area deprivation, ethnicity, BMI, GHQ-12, cholesterol, sodium, type2 diabetes, smoking status, alcohol consumption, physical activity and outdoor temperature.

Table 5. Multinomial regression model for adjusted association between temperature and hypertension classification.

	β (95% CI)	***P* value**
Normal		
Pre	-.041 (-.081, -.002)	0.039
Stage 1	-.115 (-.167, -.064)	<0.001
Stage 2	-.133 (-.214, -.051)	<0.001

Adjusted for age, gender, marital status and ethnicity, education, income, area deprivation, BMI, GHQ-12, type2 diabetes diagnosis, total cholesterol, sodium excretion, alcohol consumption, smoking status and physical activity level and outdoor temperature