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Beyond Heatwaves and Cold Spells: Long-Term Exposure to Short-Duration Extremes, Behavioural Pathways, and Hypertension Risk in a National Chinese Cohort

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Data availability

The China Health and Nutrition Survey (CHNS) data can be accessed at <https://chns.cpc.unc.edu/>. Temperature data are available at <https://zenodo.org/records/5502275>, and air pollution data can be accessed at <https://zenodo.org/records/6398971> and <https://zenodo.org/records/16883507>.

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CRedit authorship contribution statement

Zhe Pang: Conceptualization, Data curation, Formal analysis, Methodology, Writing-original draft, Writing-review & editing. **Zihao An:** Conceptualization, Methodology, Supervision, Writing-original draft, Writing-review & editing. **Yuchun Guan:** Data curation, Formal analysis. **Hao Xiang:** Writing-review & editing. **Bin Chen:** Conceptualization. **Bo Xie:** Conceptualization, Supervision, Writing-original draft, Writing-review & editing.

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43

44 **Ethics approval**

45 All participants provided written informed consent, and the CHNS study protocols were
46 approved by the institutional review boards at the University of North Carolina at Chapel
47 Hill and the National Institute of Nutrition and Food Safety in China.

48

49 **Declaration of competing interest**

50 The authors declare that they have no known competing financial interests or personal
51 relationships that could have appeared to influence the work reported in this paper.

Highlights

- We examined the impacts of long-term extreme temperature event (ETE) exposure on hypertension risk.
- We demonstrated that existing identification strategies for examining the health impacts of long-term ETE exposure fail to yield practically relevant results.
- A new identification strategy was developed, highlighting that four conceptually related yet temporally independent ETEs — including sustained and short-duration types — should be considered jointly.
- Short-duration heat and cold extremes were more robust risk factors for hypertension than sustained ETEs such as heatwaves and cold spells.
- In interaction with ETE exposure, short sleep and long sedentary time amplified, whereas low-level physical activity mitigated hypertension risk.

Beyond Heatwaves and Cold Spells: Long-Term Exposure to Short-Duration Extremes, Behavioural Pathways, and Hypertension Risk in a National Chinese Cohort

ABSTRACT

Background: Extreme temperature events (ETEs) are increasing in frequency, duration, and intensity under climate change. Evidence on whether long-term accumulated ETE exposure contributes to chronic cardiovascular disease remains limited, and behavioural pathways linking such exposure to cardiovascular risk are still insufficiently understood.

Methods: We demonstrated that commonly used identification strategies for estimating cumulative health impacts of long-term ETE exposure may yield biased estimates with limited interpretability when a complete set of temporally exclusive ETE categories within the exposure window is not jointly modelled. We therefore developed a new identification strategy emphasising that at least four conceptually related yet temporally exclusive ETEs – cold spells, short-duration cold extremes (SD-CEs), heatwaves, and short-duration heat extremes (SD-HEs) – should be examined simultaneously. Within this framework, we applied Cox models and four-way decomposition to examine long-term ETE exposure effects on hypertension and interactions with lifestyle behaviours, using national prospective cohort data (n = 8,147) from the China Health and Nutrition Survey.

Results: Higher SD-CE and SD-HE exposure was associated with increased hypertension risk (HR range: 1.05–1.06 and 1.04–1.05, respectively). SD-CEs additionally showed a significant non-linear association, with greater risk at higher exposure levels. Cold spells were associated with higher risk

only under higher, more frequent cold thresholds, whereas heatwaves were not significantly associated with hypertension risk. ETE exposure primarily exerted its effects through direct pathways. Interactions with lifestyle behaviours were also evident: low physical activity attenuated hypertension risk, whereas short sleep duration and prolonged sedentary time amplified it.

Conclusions: Short-duration extremes may be an overlooked but relevant dimension of temperature exposure for climate-sensitive hypertension prevention. While the associated risk is primarily driven by exposure-induced physiological load, interactions with lifestyle behaviours exert an additional influence. Our research also advances the methodologies for assessing the cumulative effects of long-term ETE exposure on chronic cardiovascular outcomes.

KEYWORDS

Extreme temperature events; Short-duration extremes; Hypertension; Behavioural pathways; Identification strategy; Cohort study

1 Introduction

Climate change is shifting populations away from moderate temperature conditions toward more frequent, prolonged, and intensive exposure to extreme temperature events (ETEs), with growing impact on population health (Cowan et al., 2014; Meehl and Tebaldi, 2004). Cardiovascular outcomes account for a substantial share of temperature-related disease burden, making ETEs a salient concern for cardiovascular prevention and health system resilience (Hundessa et al., 2024; Siddiqui et al., 2025; Yin et al., 2024). A large body of literature has documented that short-term or concurrent exposure to ETEs is associated with increased risks of cardiovascular outcomes, including mortality, morbidity, and hospitalisation (Ni et al., 2025; Singh et al., 2024). In contrast, whether repeated and long-term ETE exposure accumulates over months to years to influence the development of chronic cardiovascular conditions remains relatively unexplored.

A clear distinction between concurrent, short-term, and long-term ETE exposure categories is important because these designs imply different interpretations of exposure timing and therefore target different risk contrasts (**Fig. 1**). Concurrent exposure studies relate ETEs and health outcomes measured within the same observational window (e.g., ETE exposure and mortality rates within the same month (Khatana et al., 2022; Kollanus et al., 2021)). While a temporal order exists in reality, this analytical framework typically does not distinguish the relative timing of exposure and outcome. Short-term exposure studies focus on ETEs occurring in a limited period immediately preceding an acute cardiovascular event, such as hospitalisation following one-week ETE exposure (Cheng et al., 2021; Hu et al., 2024; Phung et al., 2017; Ponjoan et al., 2017). Long-term exposure studies instead quantify accumulated ETE exposure over extended periods, spanning several months to years, beginning from an outcome-free baseline during which chronic cardiovascular outcomes may develop. Only a few recent prospective cohort studies have adopted this long-term perspective (Peng et al., 2024; Tang et

al., 2025; Tuerdi et al., 2025; Zhou et al., 2023). These studies typically operationalised exposure as the cumulative number of days in sustained ETEs, most commonly heatwaves and cold spells. Their findings suggest that greater long-term exposure to sustained ETEs may be associated with elevated risks of chronic cardiovascular outcomes such as stroke, hypertension, and cardiometabolic multimorbidity.

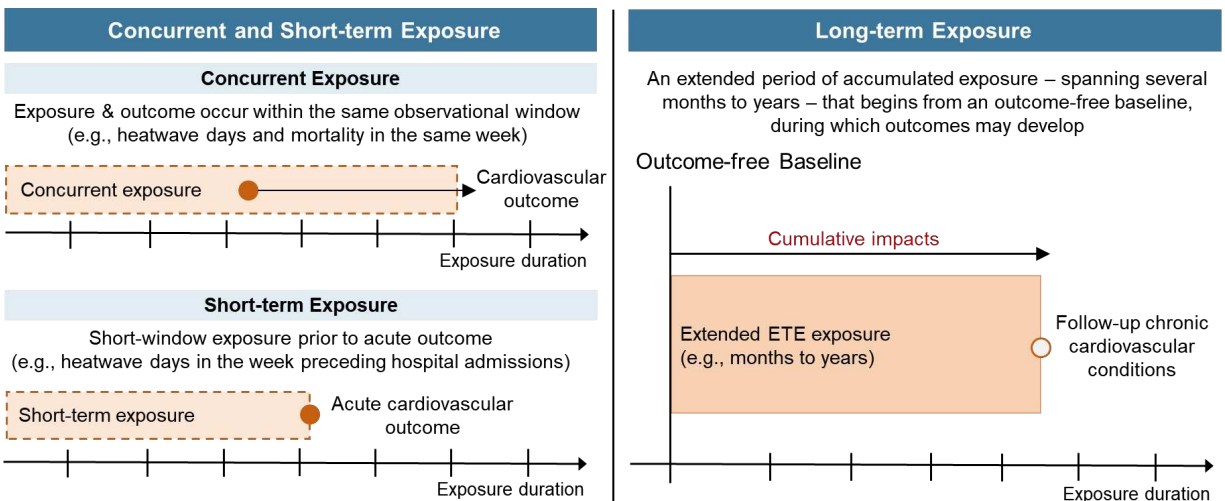


Fig. 1 Differences between studies focusing on concurrent, short-term, and long-term ETE exposure.

However, these studies share three critical limitations. First, the identification strategies used to estimate cumulative effects of ETEs are structurally limited in providing practically interpretable estimates. Long-term ETE exposure is measured over a fixed exposure window, within which ETE and non-ETE conditions jointly define the exposure structure. From a structural perspective, an estimate is interpretable only when it is linked to a clearly defined and meaningful reference condition, here, genuinely non-ETE conditions. Interpretability therefore depends not only on the numerical magnitude or statistical significance of the coefficient, but also on whether the coefficient represents the intended contrast. If health-relevant and temporally exclusive ETE categories are omitted from the model, the coefficient for the target ETE may no longer provide a clear contrast with non-ETE conditions. As shown in **Section 2.5 and Appendix A**, such omission can introduce an omitted-category

contamination component into the estimated coefficient, thereby obscuring the intended risk contrast.

Second, under this narrow focus on a limited set of ETE categories, the exposure domain in long-term studies has been defined primarily around sustained events (Peng et al., 2024; Tang et al., 2025; Tuerdi et al., 2025; Zhou et al., 2023), while short-duration ETEs have been largely overlooked. This omission may be consequential because the continuity of an extreme event plausibly shapes physiological and behavioural responses. Sustained ETEs are often more salient and allow anticipatory actions, such as adjusting clothing and activity plans, and may permit within event thermoregulatory adaptation that partially buffers health stress (Deshayes and Périard, 2023; Mol et al., 2024; Tyler et al., 2016; Vu et al., 2019; Yurkevicius et al., 2021). In contrast, short-duration extremes may terminate before such responses can be initiated or completed, and their episodic nature may reduce opportunities for avoidance or organised mitigation. Repeated exposure to short-duration ETEs may therefore generate cumulative physiological load and behavioural disruption with chronic implications that differ from those of sustained events.

Third, understanding pathways is essential for prevention. The cumulative effects of long-term ETE exposure may not arise solely from direct exposure induced physiological load. Lifestyle behaviours such as physical activity, sleep, and sedentary time are established determinants of blood pressure and may be shaped by ETE exposure, creating indirect behavioural pathways. ETE exposure may also interact with these behaviours, amplifying or buffering hypertension risk. However, evidence that jointly characterises direct effects, mediation, and interaction within a unified counterfactual framework remains limited for long-term ETE exposure and chronic cardiovascular risk.

This study examines the cumulative effects of long-term exposure to four temporally exclusive ETE categories on hypertension risk and quantifies behavioural pathways linking these exposures to hypertension risk. Using a nationwide prospective cohort from the China Health and Nutrition Survey (CHNS), we followed 8,147 adults free of hypertension at baseline who contributed one or more non-

overlapping observation periods. To obtain interpretable time-substitution estimates with a coherent non-ETE reference, we argue that at least four conceptually related yet temporally exclusive ETEs, namely cold spells, short-duration cold extremes (SD-CEs), heatwaves, and short-duration heat extremes (SD-HEs), should be examined simultaneously. We then apply four-way decomposition to quantify controlled direct effects, interaction effects, mediated interaction, and pure indirect effects with respect to key lifestyle behaviours. By integrating a necessary identification condition with an expanded ETE typology and pathway decomposition, our study strengthens inference on long-term ETE exposure and informs targeted adaptation and behaviour-sensitive prevention under increasing climate extremes.

2 Methods

2.1 Data

We conducted a prospective cohort study using data from the CHNS, a nationally representative longitudinal project established in 1989. To date, CHNS has collected ten waves of data from around 7,200 households and over 30,000 individuals across 15 provincial-level units, including eight provinces with county-level geographic identifiers (Shandong, Guangxi, Jiangsu, Henan, Hubei, Hunan, Guizhou, Liaoning) and three municipalities (Shanghai, Beijing, Chongqing), using a multistage random cluster sampling design. These long-term, large-scale, and rigorously sampled data ensure the representativeness of our study and the validity of our findings.

Five waves of CHNS data from 2004, 2006, 2009, 2011, and 2015 were used to establish the prospective cohort, as in these years, key ETE exposure variables were consistently available under the same specification. We constructed multiple observation periods for each participant, each defined by a pair of consecutive waves, with the earlier wave serving as baseline and the later wave as follow-up. Participants were included if they were free of hypertension at baseline. Further inclusion criteria

were: (1) age ≥ 18 years at all eligible survey waves; (2) complete data on hypertension and lifestyle behaviours; and (3) participation in at least two waves. Participants could contribute more than one non-overlapping period; for instance, individuals free of hypertension in 2004, 2006, and 2009 contributed periods from 2004 to 2006 and from 2006 to 2009. Once diagnosed with hypertension, subsequent observations for that participant were excluded.

2.2 Hypertension

Trained medical staff measured blood pressure on the right upper arm using a mercury sphygmomanometer. After five minutes of seated rest, three consecutive measurements of systolic blood pressure (SBP) and diastolic blood pressure (DBP) were taken at one to two minute intervals. Hypertension was defined according to the 2024 Revised Chinese Guidelines for the Prevention and Treatment of Hypertension, as SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg. A hypertension case was identified if at least two valid readings met the threshold. Self-reported physician diagnosis and antihypertensive medication use were then considered as supplementary criteria, particularly for participants without valid blood pressure measurements and for those who did not meet the measurement-based criterion but reported taking antihypertensive medication. The survey date of the first hypertension identification was used as the event time. Self-reported diagnosis duration was not used to back-calculate onset time, as it may be affected by recall bias and was not consistently available.

2.3 Lifestyle behaviours

We considered three lifestyle behaviours measured as daily duration: physical activity, sleep, and sedentary behaviour. Physical activity duration was calculated as total time accumulated across domestic, leisure, and transport domains using detailed CHNS activity modules. Specifically, domestic activities included, for example, food shopping, cooking, and house cleaning; leisure activities encompassed, for example, sports, dancing, and Tai Chi; and transport activity was assessed from

individuals' reported commuting to and from work or school by active and public transport. These activity classifications follow the CHNS questionnaires, which are available online as open-access resources. Sleep duration was based on self-reported hours usually slept in a 24-hour period, including daytime and nighttime sleep. Sedentary behaviour duration was derived from reported time spent in sedentary activities during a typical day, such as screen-based media, computer use, reading, and other sedentary pursuits.

2.4 Covariates

Our analyses adjusted for sex, educational attainment, working environment, air conditioning ownership, net individual income adjusted to 2015 values, urbanisation index (Jones-Smith and Popkin, 2010), body mass index (BMI), history of diabetes, smoking status, and alcohol intake. We also adjusted for fine particulate matter (PM_{2.5}) and ozone (O₃), which have been widely recognised as important risk factors for hypertension (Krittanawong et al., 2023; Tang et al., 2024; Yin et al., 2020). For each observation period, air pollution exposure was defined as the number of days with excessive concentrations of PM_{2.5} and O₃. This metric captures episodic high-pollution conditions that may coincide or cluster temporally with ETEs, as supported by previous evidence on the co-occurrence of O₃ and PM_{2.5} exceedance days with ETEs (Gao et al., 2023; Huang et al., 2025b; Wang et al., 2022). Air pollution data were obtained from the China High Air Pollutants dataset (Wei et al., 2020; Wei et al., 2022). Excessive PM_{2.5} days were defined as days with daily mean concentration at or above 75 µg/m³, and excessive O₃ days were defined as days with daily 8-hour maximum at or above 100 µg/m³.

2.5 A new identification strategy and long-term exposure to ETEs

We argue that exhaustively accounting for a complete set of temporally exclusive ETEs within the exposure window can help preserve a clearly defined reference and meaningful interpretation of

the cumulative cardiovascular effects of long-term ETE exposure. Here, a ‘complete set’ is defined as the aggregate of temporally exclusive ETE categories that collectively occupy the entire duration outside of genuinely non-ETE conditions. Understanding such effects inherently requires a structural perspective: the interpretability and validity of any estimates rest fundamentally on the existence of a meaningful reference – namely, relative to genuinely non-ETE conditions – rather than solely on the estimates themselves. However, when the considered ETEs fail to constitute such a complete set due to the omission of any categories, and where these omitted ETEs are associated with cardiovascular outcomes, the estimated cardiovascular risk per unit of exposure for the considered ETEs may be biased, and its interpretation relative to predefined non-ETE conditions may become less clear. The identification problem arises because regression models inherently operate under a *ceteris paribus* assumption: the estimated coefficient for the focused ETE category reflects the change in risk associated with a unit of change in the duration of that ETE, with durations for all other modelled ETE categories held constant. If any ETEs are omitted and are associated with cardiovascular outcomes, it becomes ambiguous what an additional unit of exposure to a given ETE is actually substituting for: it may arise from changes in the duration of omitted ETEs or from changes in the duration of genuinely non-ETE conditions. In short, the reference group is contaminated. As shown by the derivation in **Appendix A**, this contamination corresponds to an additional term in Eq. A.10, whose magnitude depends jointly on the health effects of the omitted ETE categories and their covariance with the residual component of the target ETE exposure. With this contamination term present, the estimated coefficient no longer represents the contrast between the target ETE category and the predefined genuinely non-ETE reference condition. This, in turn, may structurally undermine the validity and interpretability of the estimated effect.

In addition, sustained events alone do not constitute a complete ETE typology if short-duration extremes are omitted. As emphasised in Introduction, short-duration and sustained ETEs may differ in

salience, behavioural response, and physiological adaptation, implying potentially distinct cumulative health effects. Therefore, the omission of short-duration ETEs can directly contribute to the identification problem, for two reasons: (i) short-duration and sustained ETEs collectively constitute the complete set of temporally exclusive ETEs, and (ii) the impacts of long-term exposure to short-duration ETEs may differ from those associated with exposure to sustained ETEs. We argue that at least four conceptually related yet temporally exclusive categories of ETEs, namely heatwaves, short-duration heat extremes (SD-HEs), cold spells, and short-duration cold extremes (SD-CEs), should be considered simultaneously in the estimation, irrespective of the target ETE, as illustrated in **Eq. (A.3)**.

We identified ETEs using high-resolution (0.1°) daily minimum and maximum temperature data from the China Daily Near-Surface Air Temperature (CDAT) dataset, which integrates satellite observations, reanalysis products, and ground-based measurements, providing robust spatial accuracy across China, with R^2 values ranging from 0.95 to 0.99 (Fang et al., 2022). Daily temperatures were assigned based on residential locations using prefecture level identifiers for Shanghai, Beijing, and Chongqing, and county level identifiers for the remaining provinces. For each observation period, exposure was measured over the exact interval between the baseline and follow-up survey dates for each individual. Heatwaves were defined as at least three consecutive days with daily maximum temperature exceeding the observation-specific 90th, 95th, or 97.5th percentile threshold. Thresholds were calculated for each observation period using local daily maximum temperatures during the warm season from May to September over the three years preceding the baseline date. Cold spells were defined analogously using daily minimum temperatures during the cold season from November to March and the 10th, 5th, or 2.5th percentiles. SD-HEs and SD-CEs were defined as short-duration ETEs that reached the respective thresholds but did not meet the duration criterion for heatwaves or cold spells (i.e., lasting fewer than three consecutive days, specifically only one or two days). A three-year reference window was used to define ETEs relative to recent local temperature conditions and to

reduce the potential influence of long-term climate warming trends on ETE identification. For each observation interval, the total number of days meeting each ETE definition was annualised to represent the average yearly exposure burden and improve comparability across intervals of unequal length.

2.6 Statistical analysis

We applied semi-parametric Cox proportional hazards models to estimate the association between long-term ETE exposure and hypertension risk under the proposed identification strategy. The main exposure variables were the annualised cumulative days of four temporally exclusive ETE categories within each observation period. Models were estimated separately for matched threshold sets, pairing heat-related thresholds at the 90th, 95th, or 97.5th percentiles with cold-related thresholds at the 10th, 5th, or 2.5th percentiles. Under this identification strategy, the coefficient for each ETE category is interpretable as the marginal change in hypertension risk associated with one additional day of that category relative to non-ETE days. It should be noted that the non-ETE reference condition is not assumed to be physiologically homogeneous or risk-free. Rather, it represents a predefined ETE-free reference category within the exposure window.

Survival time was defined on the age scale to flexibly account for age-related confounding (Thiébaud and Bénichou, 2004). We clustered standard errors at the participant level to account for within-individual correlation across observation periods. Covariates were measured at follow-up for each observation period. We first fitted crude Cox models including only ETE exposures as unadjusted references, followed by adjusted models with different covariates. Because lifestyle behaviours may function as both confounders and exposure responsive mediators within the same interval, we therefore specified two versions of the model with different covariate structures: one adjusted for lifestyle behaviours and one without such adjustment. Adjustment for lifestyle behaviours helps control confounding due to behavioural differences, but may underestimate the impact of ETE exposure by

absorbing part of the mediation into the lifestyle behaviours variables. By contrast, models without this adjustment capture the combined influence of ETE exposure and its behavioural mediation more effectively but may introduce endogeneity bias. We assessed multicollinearity using variance inflation factors and tested the proportional hazards (PH) assumption using Schoenfeld residuals, finding no evidence of collinearity or violation. Stratified analyses examined potential effect modification by sex, age, educational attainment, working environment, residence type, and region type. Region type was further interpreted as a proxy indicator of residential heating zone, with northern and southern regions representing traditional heating and non-heating zones, respectively. Differences between subgroups were assessed using Z-tests.

We then applied a four-way decomposition approach to examine how the interplay between long-term ETE exposure and less-healthy lifestyle behaviours contribute to hypertension risk. Recent applications of this approach have demonstrated its utility in assessing the joint contributions of environmental exposures and behavioural factors to chronic health outcomes (Zhang et al., 2023; Zhang et al., 2024). The strength of this approach lies in its counterfactual framework within a unified specification, which disentangles the net effects of four pathways of interplay (VanderWeele, 2014): the controlled direct effect, reference interaction effect, mediated interaction effect, and pure indirect effect (see **Fig. S1** for mathematical representation of these pathways).

The controlled direct effect captures the direct cumulative effects of ETE exposure, independent of its interplay with less healthy lifestyle behaviours. The reference interaction effect reflects changes in risk arising from the interaction between ETE exposure and less healthy lifestyle behaviours, beyond the sum of their individual effects. The mediated interaction effect represents the risk that occurs when ETE exposure induces changes in less healthy lifestyle behaviours, and these changes further interact with the exposure to influence hypertension risk. The pure indirect effect captures risk mediated solely through changes in less healthy lifestyle behaviours resulting from ETE exposure. The four-way

decomposition utilised less healthy lifestyle behaviours recorded at the follow-up wave of each observation period. From a standpoint of the data-generating process (i.e., the underlying causal mechanism; DGP), these observed behaviours contain a mixture of different components. They incorporate a responsive component, reflecting the potential change caused by ETE exposure during the observation period, while they also contain a component that is not affected by ETE exposure, representing a long-term natural pattern manifested during the observation period. The characteristics of these behaviour variables, together with the four-way decomposition approach designed to partition the overlapping influences between pathways, therefore, enable a coherent and comprehensive analysis.

The four-way decomposition was conducted using the *med4way* command, which allows estimation of the four-way effect decomposition using parametric or semi-parametric regression models and is applicable to cohort studies with survival outcomes (Discacciati et al., 2019). The command fits two models: an outcome model as a function of the exposure, mediator, their interaction, and confounders, and a mediator model as a function of the exposure and confounders. The outcome model supports several regression forms, including, for example, accelerated failure time and Cox models, whereas the mediator model can be specified as linear or logistic regression. In this study, we specified a Cox regression model for hypertension risk and a linear regression model for the lifestyle mediator. The exposure change was defined as an increase in long-term ETE exposure from the 25th to the 75th percentile, with covariates fixed at their mean values. Less-healthy lifestyle behaviours were considered as mediators, including low-level physical activity, short sleep duration, and long sedentary time. These mediator levels were set at the 25th percentile of physical activity, the 25th percentile of sleep duration, and the 75th percentile of sedentary time, respectively. The decomposed effects were reported on the excess relative risk (ERR) scale.

2.7 Sensitivity analyses

We conducted sensitivity analyses to assess the robustness of our findings. First, we extended the duration criteria for ETEs from three consecutive days to four and five days. Second, lifestyle behaviours may themselves act as mediators in the relationship between long-term ETE exposure and hypertension. To account for this, our main analyses estimated two specifications, one with and one without adjustment for lifestyle behaviours, thereby providing complementary perspectives on the role of ETE exposure in affecting hypertension risks. As an additional alternative, we conducted sensitivity analyses using marginal structural Cox models with stabilised inverse probability weights (SIPW) (Karim et al., 2014; Lee et al., 2016). This approach provides another potential solution by explicitly addressing exposure-confounder feedback, whereby lifestyle behaviours can be shaped by exposure within the same observation interval while also relating to hypertension risk. While such models enhance robustness by retaining confounding control without blocking indirect pathways, the SIPW approach can yield less precise estimates, and is therefore reported as a complementary sensitivity analysis rather than a replacement for the main analyses. Third, to evaluate the potential influence of outcome classification and event-time assignment based on supplementary self-reported information, we conducted an additional sensitivity analysis excluding participants whose hypertension status was determined using self-reported physician diagnosis. Fourth, to assess the robustness of our findings to alternative ETE threshold definitions, we redefined ETEs using 5-year and 10-year historical reference periods and re-estimated the Cox models. Fifth, to assess sensitivity to alternative air-pollution exposure metrics, we replaced annual $PM_{2.5}$ and O_3 exceedance days with annual mean concentrations in the main Cox models. Sixth, to assess sensitivity to differences in background temperature across residential areas, we additionally adjusted for mean temperature during each participant-period observation. However, it should be emphasized that this model was not intended to replace the main model, because adjustment for mean temperature may alter the interpretation of ETE coefficients.

Since mean temperature is derived from the same daily temperature distribution used to define ETEs, holding it constant while estimating the effect of ETE days implies compensatory changes in the temperatures of the non-ETE reference period. The mean-temperature-adjusted model therefore represents a conditional contrast under a constrained temperature background, rather than the original contrast against the predefined non-ETE reference structure. Seventh, to visualise potential non-linear associations between ETE exposure and hypertension risk, we used restricted cubic splines with four knots in Cox models under the 90th and 10th percentile threshold definitions, with the median value of each ETE exposure as the reference value. Eighth, for the four-way decomposition, we varied the change in ETE exposure from the 25th to 75th percentile to the 10th to 90th percentile range.

3 Results

3.1 Study population characteristics

After applying the exclusion criteria, 8,147 participants were included (**Fig. 2**). Baseline characteristics are summarised in **Table 1**. The mean age was 47.5 years, and the cohort was balanced by sex (49.1% male, 50.9% female). In terms of education, 13.3% had no formal schooling, 69.3% completed primary or secondary education, and 17.4% held post-secondary qualifications, including technical or vocational degrees. For occupational and living conditions, 61.2% worked outdoors and 39.1% had access to air conditioning. 1.7% reported a history of diabetes, and the mean BMI was 23.2. 14.5% reported alcohol consumption above the recommended limit, and 30.9% were current or former smokers. Regarding lifestyle behaviour, participants engaged in about 1.8 hours of physical activity, slept for an average of 8 hours, and spent around 3.0 hours in sedentary behaviour per day. During follow-up, 1,062 incident hypertension cases were identified. Participants who developed hypertension were slightly more likely to be female, older, and to have higher BMI. They more frequently worked outdoors, lacked air conditioning and reported excessive alcohol consumption. They also tended to live

in areas with more days exceeding PM_{2.5} and O₃ standards and had lower personal income.

Descriptive statistics of annualised ETE exposure are shown in **Table 2**. Under the 90th percentile heat threshold, participants experienced on average 9.55 heatwave days and 6.61 SD-HE days per year. Under the corresponding 10th percentile cold threshold, participants experienced 11.34 cold spell days and 6.43 SD-CE days per year. As percentile thresholds became more stringent (95th and 97.5th for heatwaves; 5th and 2.5th for cold spells), the average number of exposure days declined accordingly. In most cases, the number of exposure days related to cold extremes exceeded that related to heat extremes. In addition, low to moderate correlations between sustained and short-duration ETEs were observed in the Spearman correlation matrix (**Fig. S2**).

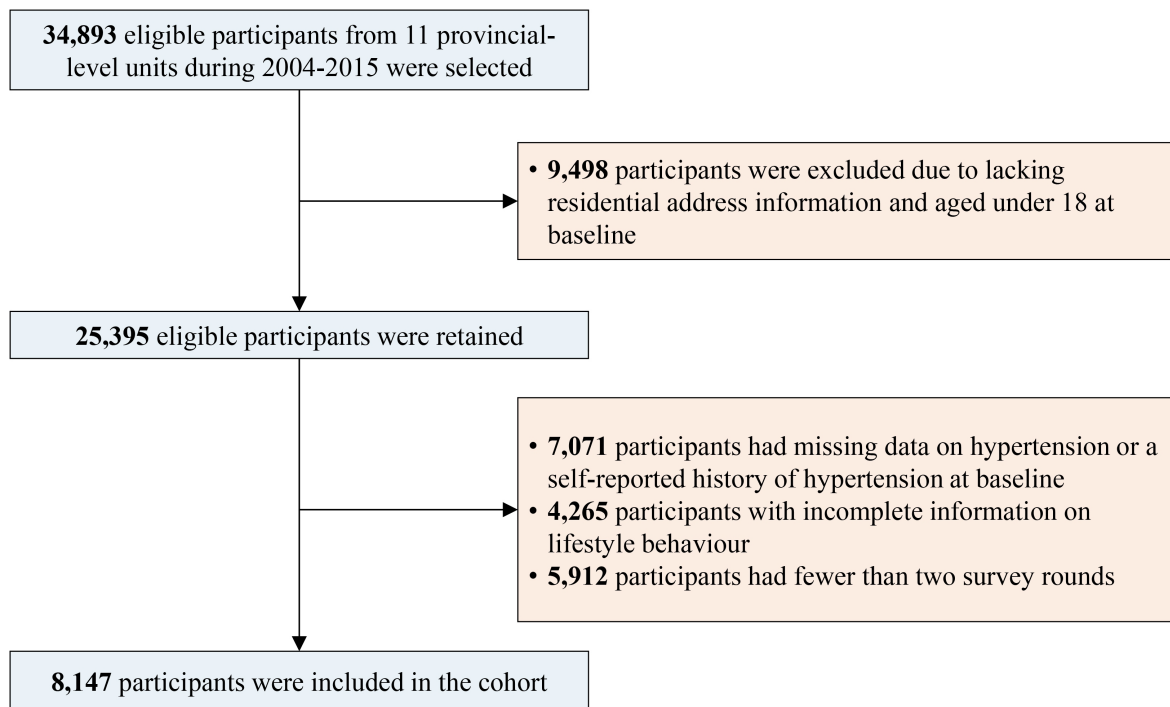


Fig. 2 Flow diagram of exclusion criteria for the participants.

Table 1 Baseline characteristics for all study participants.

Variables	Total (<i>n</i> = 8,147)	Hypertension at the end of follow-up (<i>n</i> = 1,062)	No hypertension at the end of follow-up (<i>n</i> = 7,085)
Sex			
Male	4,003 (49.1%)	502 (47.3%)	3,501 (49.4%)
Female	4,144 (50.9%)	560 (52.7%)	3,584 (50.6%)
Age	47.5 ± 13.9	57.1 ± 11.2	46.1 ± 13.7
Educational attainment			
No formal education	1,081 (13.3%)	242 (22.8%)	839 (11.8%)
Primary or secondary education	5,649 (69.3%)	715 (67.3%)	4,934 (69.6%)
Post-secondary qualification	1,417 (17.4%)	105 (9.9%)	1,312 (18.5%)
Working environment			
Outdoor	4,983 (61.2%)	677 (63.7%)	4,306 (60.8%)
Indoor	2,886 (35.4%)	364 (34.3%)	2,522 (35.6%)
Others	278 (3.4%)	21 (2.0%)	257 (3.6%)
Air conditioning ownership			
Yes	3,188 (39.1%)	384 (36.2%)	2,804 (39.6%)
No	4,959 (60.9%)	678 (63.8%)	4,281 (60.4%)
Net individual income, mean ± std	18,390.7 ± 29,774.2	13,481.6 ± 19,822.7	19,126.6 ± 30,925.7
Urbanisation index, mean ± std	69.2 ± 20.2	69.0 ± 20.1	69.2 ± 20.2
Body mass index (BMI), mean ± std	23.2 ± 3.4	24.6 ± 3.5	23.0 ± 3.3
History of diabetes			
Yes	136 (1.7%)	44 (4.1%)	92 (1.3%)
No	8,011 (98.3%)	1,018 (95.9%)	6,993 (98.7%)
Smoking status			
Current or ever smoking	2,519 (30.9%)	325 (30.6%)	2,194 (31.0%)
Non-smoking	5,628 (69.1%)	737 (69.4%)	4,891 (69.0%)
Alcohol intake			
Above the recommended limit	1,184 (14.5%)	189 (17.8%)	995 (14.0%)
Within the recommended limit	6,963 (85.5%)	873 (82.2%)	6,090 (86.0%)
Days with excessive PM _{2.5} , mean ± std	92.2 ± 56.7	93.1 ± 55.1	92.0 ± 56.9
Days with excessive O ₃ , mean ± std	115.5 ± 36.2	119.7 ± 36.6	114.9 ± 36.1
Physical activity (hours/day), mean ± std	1.8 ± 1.7	1.7 ± 1.5	1.8 ± 1.7
Sleep duration (hours/day), mean ± std	8.0 ± 1.2	7.9 ± 1.3	8.1 ± 1.2
Sedentary time (hours/day), mean ± std	3.0 ± 1.9	2.8 ± 1.8	3.1 ± 2.0

348 Note. The recommended limit for alcohol intake was defined as 15 grams per day, which is in line with

349 the Chinese Dietary Guidelines (Chinese Nutrition Society, 2022).

Table 2 Descriptive statistics of average ETE exposures (days) across all participant–period observations.

Variables	Min	Mean	Max	Std
Heatwave				
P90	0.00	9.55	31.50	5.78
P95	0.00	4.14	24.00	3.65
P97.5	0.00	1.75	13.50	2.14
SD-HE				
P90	1.33	6.61	16.50	2.64
P95	0.00	4.12	11.50	1.88
P97.5	0.00	2.63	9.50	1.83
Cold spell				
P10	0.00	11.34	36.00	7.38
P5	0.00	4.64	26.00	4.69
P2.5	0.00	2.19	21.50	3.14
SD-CE				
P10	2.00	6.43	15.00	2.52
P5	0.67	4.77	15.50	2.45
P2.5	0.00	2.77	12.50	1.95

Notes. P2.5, P5, P10, P90, P95, and P97.5 represent ETEs defined under the 2.5th, 5th, 10th, 90th, 95th, and 97.5th percentile temperature thresholds, respectively. SD-HE, short-duration heat extreme; SD-CE, short-duration cold extreme.

3.2 Long-term ETE exposure and hypertension risk

Fig. 3 and Table S1 present the estimated hazard ratios (HRs) for hypertension associated with each additional day of exposure to the four ETE categories, relative to non-ETE days. Results were highly consistent with and without adjustment for lifestyle behaviours. Across all temperature thresholds defining ETEs, higher SD-CE exposure was associated with increased hypertension risk under the linear specification (HR range: 1.05–1.06). Restricted cubic spline analysis further showed a significant non-linear association for SD-CE under the 10th-percentile threshold definition (P for non-linearity = 0.002). The exposure-response curve showed a slight decline in hypertension risk at

SD-CE exposures below approximately 6 days, followed by a progressive increase in risk at higher exposure levels (**Fig. S3**). For cold spells, exposure to additional days was associated with higher risk only when defined at the 10th percentile threshold (HR = 1.02; 95% CI: 1.01–1.03), where cold spells were more frequent. Regardless of the temperature threshold, exposure to SD-CEs was associated with a higher risk of hypertension than exposure to cold spells. For SD-HEs, the risk of hypertension increased with each additional day of exposure when defined by the 95th and 97.5th percentile temperature thresholds (HR range: 1.04–1.05). No statistically significant association between exposure to heatwaves and hypertension risk was found at the 0.05 level.

In stratified analyses (**Fig. 4 and Tables S7-S9**), associations for cold-related events were broadly consistent across subgroups. Specifically, long-term exposure to cold spell and SD-CEs defined at the 10th, 5th, and 2.5th percentiles similarly elevated risks of hypertension as those identified in the main analysis, with no statistically significant differences detected between strata, including the north–south heating-zone proxy. For SD-HE exposure, stronger associations were observed among males (HR = 1.08, 95% CI: 1.03–1.14) and participants living in the southern region (HR range: 1.07–1.11), with statistically significant between subgroup differences.

3.3 Four-way effect decomposition results

Fig. 5, Figs. S4-S5 and Tables S10-S12 present the results of the four-way decomposition. Across different specifications, the direction and significance of total effects were consistent with Cox model estimates. This alignment between the two methods further supports the robustness of our findings. Pathway-specific results indicated that total effects were mainly reflected in the controlled direct effect component, a pattern that may be compatible with cumulative physiological burden associated with long-term ETE exposure.

For cold spell exposure, the controlled direct effect consistently showed the largest magnitude,

and was statistically greater than zero under the 10th percentile threshold, independent of its decomposition with less healthy lifestyle behaviours. This suggests that the association between cold spell exposure and hypertension risk was mainly reflected in the direct component, rather than in pathways mediated by the measured lifestyle behaviours. Under these temperature thresholds, the reference interaction effects suggested heterogeneity by behaviour type. Low-level physical activity showed a negative reference interaction that partially offset the controlled direct effect, while short sleep duration showed a positive reference interaction effect, suggesting the joint pattern of cold spell exposure and short sleep may correspond to excess hypertension risk beyond their separate components. For sedentary behaviour, reference interaction effects were limited for cold spells. Mediated interaction and pure indirect effects were generally small and not statistically significant.

For SD-CE exposure, pathway patterns were similar to those observed for cold spells. For SD-HE exposure, pathway patterns were also similar, with the additional feature that reference interaction with long sedentary time was positive at the 95th and 97.5th percentile temperature thresholds. Although not statistically significant, its magnitude amounted to 17% – 44% of the controlled direct effect, suggesting a potentially meaningful but uncertain interaction-related component between SD-HE exposure and long sedentary time.

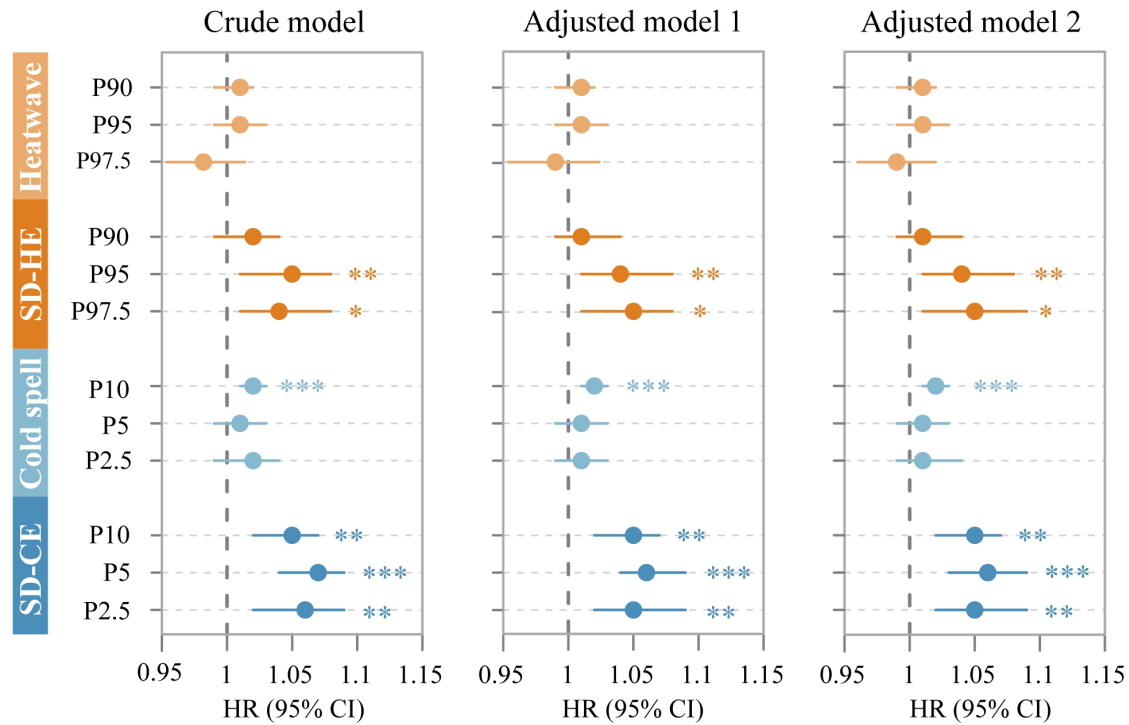


Fig. 3 Estimated HRs for hypertension risk associated with each additional day of ETE exposure compared with non-ETE conditions.

Notes. P2.5, P5, P10, P90, P95, and P97.5 represent ETEs defined under the 2.5th, 5th, 10th, 90th, 95th, and 97.5th percentile temperature thresholds, respectively. SD-HE, short-duration heat extreme; SD-CE, short-duration cold extreme. Model 1 was adjusted for sex, educational attainment, working environment, history of diabetes, air conditioning ownership, body mass index, net individual income, urbanisation index, alcohol intake, smoking status, and the number of days with excessive PM_{2.5} and O₃ concentrations. Model 2 was further adjusted for physical activity, sleep duration and sedentary time. *** $p < 0.001$; ** $p < 0.01$; and * $p < 0.05$.

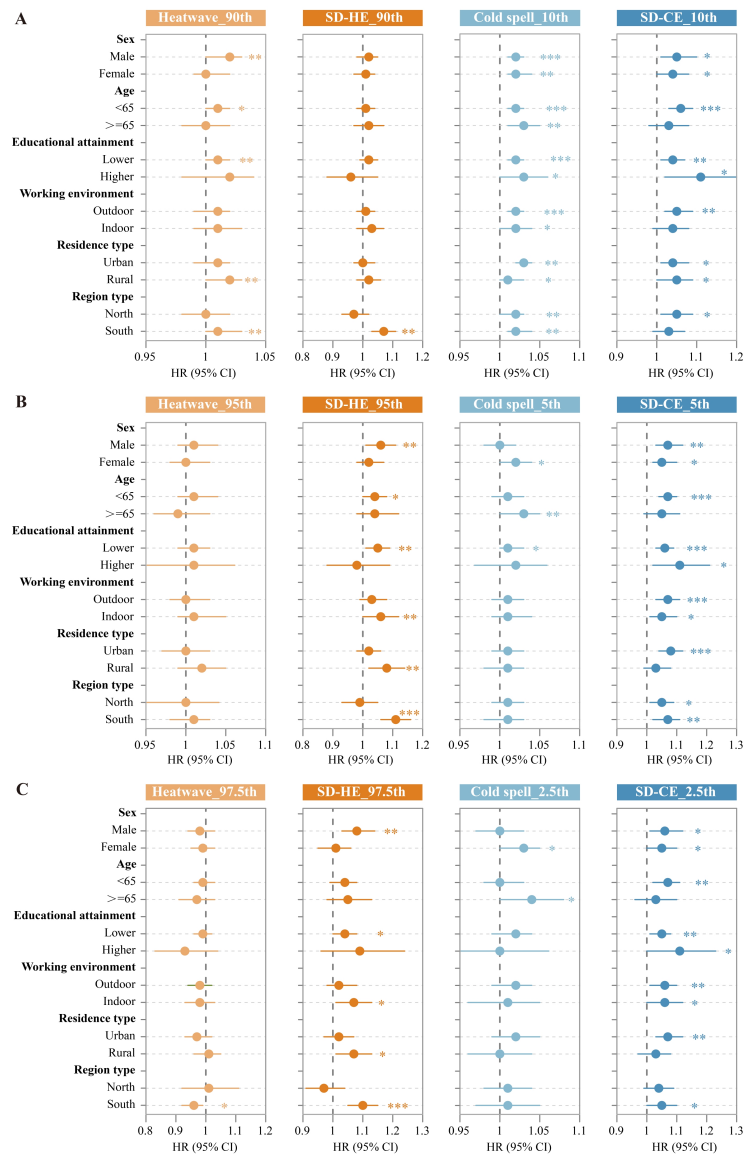


Fig. 4 Stratification analysis for the association between the four categories of ETEs and hypertension.

Notes. Heatwave_90th, 95th, 97.5th and SD-HE_90th, 95th, 97.5th denote heatwaves and short-duration heat extremes defined under the 90th, 95th, 97.5th percentile temperature thresholds, respectively. Cold spell_10th, 5th, 2.5th and SD-CE_10th, 5th, 2.5th denote cold spells and short-duration cold extremes under the 10th, 5th, 2.5th percentile temperature thresholds, respectively. Estimates were fully adjusted for sex, educational attainment, working environment, history of diabetes, air conditioning ownership, body mass index, net individual income, urbanisation index, alcohol intake, smoking status, the number of days with excessive PM_{2.5} and O₃ concentrations, physical activity, sleep duration, and sedentary time. *** $p < 0.001$; ** $p < 0.01$; and * $p < 0.05$.

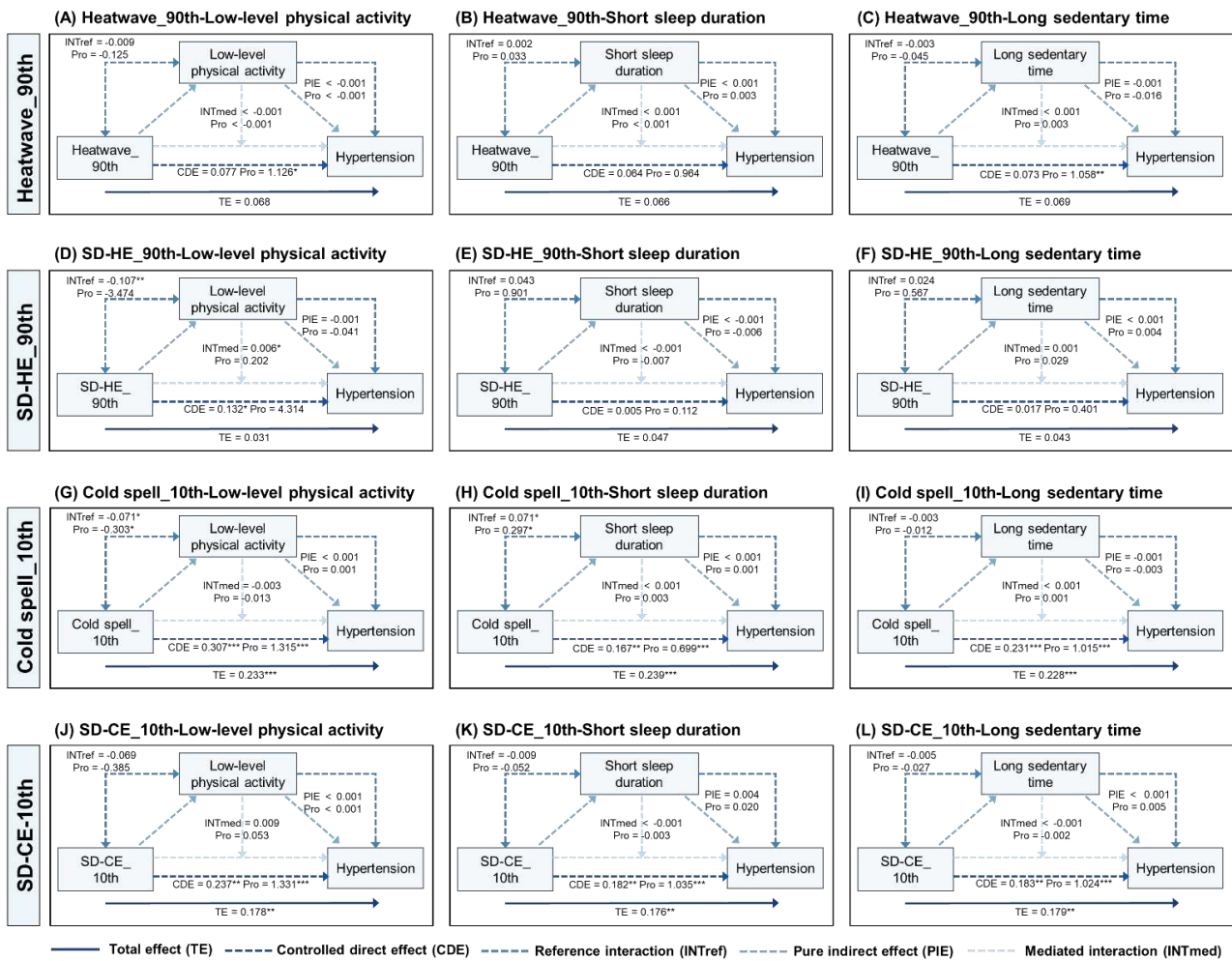


Fig. 5 Four-way decomposition capturing the joint impacts of ETE exposure defined under the 90th/10th percentile temperature thresholds and three lifestyle behaviours on hypertension.

Notes. The decomposition effects shown in the figure represent excess relative risk. Pro indicates the proportion of the effect attributable to each significant pathway. Estimates were adjusted for sex, educational attainment, working environment, history of diabetes, air conditioning ownership, body mass index, net individual income, and urbanisation index, alcohol intake, smoking status, days with excessive PM_{2.5} concentrations and days with excessive O₃, and, for each model, the other two lifestyle behaviours. Heatwave_90th and SD-HE_90th denote heatwaves and short-duration heat extremes defined under the 90th percentile temperature thresholds, respectively. Cold spell_10th and SD-CE_10th denote cold spells and short-duration cold extremes under the 10th percentile temperature thresholds, respectively. *** $p < 0.001$; ** $p < 0.01$; and * $p < 0.05$.

3.4 Sensitivity analyses

First, we extended the definition of ETEs from three to four and five consecutive days (Table S1). Our findings derived from the Cox models remained similar to the main analysis. However, some minor, threshold-specific differences emerged for the effects of cold spell and SD-HE exposures. Under the specification using a four-day criterion to define sustained ETEs, the effect of SD-HE exposure became significant at the 90th percentile threshold. When the criterion was extended to five consecutive days, the estimated effects of SD-HE exposure were significantly greater than zero across all percentile thresholds (90th, 95th, and 97.5th), and so was the 97.5th percentile cold-spell exposure. Second, we applied marginal structural Cox models with stabilised inverse probability weights to account for potential bias arising from the dual role of lifestyle behaviours as confounders and mediators in the relationship between ETE exposure and hypertension. The results also remained highly consistent with the main analyses (Table S2). Third, after excluding participants whose hypertension status was identified on the basis of self-reported physician diagnosis, the results were generally consistent with the main analysis (Table S3). Fourth, results based on 5-year and 10-year threshold reference periods suggested greater sensitivity for heat-related events, while the overall patterns remained broadly consistent with the main analysis (Table S4). Fifth, when the numbers of PM_{2.5} and O₃ exceedance days were replaced with their annual mean concentrations, the results remained consistent with the main analysis (Table S5). Similar results were also obtained after additional adjustment for mean temperature during each participant-period observation (Table S6). Sixth, we redefined the change in ETE exposure in the four-way decomposition from the 25th – 75th to the 10th – 90th percentile range. The results from the sensitivity analyses remained highly consistent in terms of the direction and significance of the estimates, which confirmed the robustness under this alternative

specification (Tables S13–S15).

4 Discussion

In this nationwide prospective cohort study, we examined the cumulative effects of long-term exposure to four temporally exclusive ETE categories, namely heatwaves, SD-HEs, cold spells, and SD-CEs, on hypertension risk relative to non-ETE conditions. Moving beyond the predominant focus on short-term or concurrent exposures, this study extends the existing literature on the cardiovascular impacts of ETEs by explicitly addressing the cumulative implications of long-term exposure for chronic cardiovascular outcomes. The study contributes both empirically and methodologically, and provides insights relevant to understanding and mitigating the adverse effects of ETE exposure on chronic cardiovascular outcomes, with particular relevance to hypertension.

Regarding our empirical findings, we observed that greater long-term exposure to cold spells was associated with an increased risk of hypertension only when cold spells were defined using higher, rather than lower, temperature thresholds. This pattern may reflect cumulative effects of repeated cold exposure, including sustained sympathetic activation, endothelial dysfunction, and gradual vascular remodeling, which together may contribute to the development of hypertension (Yoshimoto et al., 2025). Moreover, compared to colder and rarer cold spells defined by lower temperature thresholds, cold spells defined at higher thresholds may include days that are insufficiently severe to be perceived and socially recognised as weather emergencies. As a result, such exposures may be less likely to trigger behavioural adjustments or organised preventive measures, leaving individuals more frequently exposed in an unmitigated physiological state. In contrast to cold spells, we found no statistically significant association between heatwave exposure and hypertension risk. This finding may reflect context specific adaptation, behavioural avoidance, or infrastructural protection, and it underscores that chronic impacts of sustained heat

exposure may be more heterogeneous than those of cold exposure across settings and definitions.

A central empirical finding is that long-term exposure to short-duration ETEs, namely SD-CEs and SD-HEs, was associated with increased risks of hypertension across all or most temperature thresholds. In particular, the restricted cubic spline analysis further revealed a significant non-linear association for SD-CE exposure, with the risk increasing more clearly at higher cumulative exposure levels. This pattern may reflect the cumulative physiological effects of recurrent cold exposure. Repeated cold exposure may elevate blood pressure and promote inflammatory responses, potentially placing greater strain on the cardiovascular system as exposure accumulates over time (Cheng and Su, 2010). Our findings further suggest that, for long-term cumulative exposure, the temporal structure of extreme events may matter in addition to their thermal intensity. Compared with sustained events, short-duration extremes may impose repeated acute stress episodes with less opportunity for effective behavioural or physiological adjustment, thereby contributing more strongly to cumulative cardiovascular strain over time (Leppäluoto et al., 2001). Taken together, these results suggest that short-duration ETEs constitute a meaningful, but often overlooked, dimension of climate-related exposure relevant to chronic cardiovascular risk.

Our results also differ from much of the previous long-term literature, which has reported broadly robust adverse cumulative effects of sustained ETE exposure on chronic cardiovascular outcomes, including hypertension (Peng et al., 2024; Tang et al., 2025; Tuerdi et al., 2025; Zhou et al., 2023). While contextual factors and other sources of heterogeneity may partly account for these differences, the inclusion of short-duration ETE exposures in our study may also play an important role. As emphasised throughout this research, exhaustively accounting for a complete set of temporally exclusive ETEs within the exposure window is important for maintaining a clearly defined reference and

509 meaningful interpretation of the cumulative cardiovascular effects of long-term ETE
510 exposure. Without this, estimated effects for any given ETE category may partly reflect
511 substitution from omitted ETE categories rather than from non-ETE days alone.

512 Beyond total effects, we examined behavioural pathways through which long-term
513 ETE exposure may be related to hypertension risk. Across specifications, the
514 decomposition results suggested that the cumulative cardiovascular effects of ETE
515 exposure were mainly reflected in controlled direct effect components, a pattern that may
516 be compatible with cumulative physiological burden associated with long-term exposure.
517 Lifestyle behaviours appeared to modify these associations through interaction-related
518 components. Low-level physical activity interacted with these exposures to attenuate risk,
519 whereas short sleep duration and, under SD-HE exposure, long sedentary time appear to
520 show positive interaction-related components. One possible explanation for the attenuating
521 interaction with low-level physical activity is reduced outdoor exposure time, which may
522 lower actual contact with extreme temperatures (Ho et al., 2023). In contrast, insufficient
523 or irregular sleep can disrupt autonomic regulation by reducing parasympathetic activity
524 and increasing cardiovascular reactivity. When combined with ETE-induced sympathetic
525 activation, this may amplify blood pressure responses beyond what would be expected from
526 either factor alone (Crandall and Wilson, 2011; Fink et al., 2018). For SD-HE exposure,
527 long sedentary time may further intensify heat-related strain by impairing thermoregulatory
528 efficiency, promoting venous pooling and low endothelial shear stress, and limiting heat
529 dissipation (Crandall and González-Alonso, 2010; Stanford et al., 2024; Thosar et al., 2015).
530 Importantly, sedentary behaviour and low physical activity capture distinct behavioural
531 dimensions. Individuals can meet activity recommendations while still spending long
532 periods sitting, which may independently increase vulnerability during heat-related stress.
533 This distinction is consistent with the differential interaction patterns observed.

534 These findings have practical implications for chronic disease oriented climate

adaptation. First, prevention frameworks and early warning systems often prioritise sustained ETEs because of their acute impacts (Huang et al., 2025a; Wang et al., 2025), yet our results suggest that short-duration extremes can also contribute to hypertension risk when exposure accumulates over time. A practical implication is therefore to incorporate short-duration ETEs in routine forecast-based public health advisories. For example, short-duration ETEs sustained 1-2 days could be flagged and paired with brief, actionable protective recommendations, particularly in settings where such events occur frequently. Moreover, our stratified analyses indicate heterogeneity in associations across subgroups and regions, supporting more targeted guidance to populations and areas with larger estimated effects (e.g., males and residents in southern regions) via workplaces, community health services, and primary care (Zhou et al., 2025). Second, our threshold pattern for cold spells indicates that moderately cold but frequent events captured under higher thresholds also warrant attention. Because such events are less likely to be recognised as weather emergencies, they may create a prevention gap despite their cumulative impacts. This highlights the value of complementing severe event alerts with lower-intensity, higher-frequency cold-risk messaging that emphasises everyday protection, such as maintaining adequate indoor warmth, wearing layered clothing, and encouraging blood pressure self-monitoring among susceptible individuals.

Our results also inform climate-sensitive lifestyle guidance. Regular physical activity remains a cornerstone of hypertension prevention and management (Qiao et al., 2025; Thompson et al., 2003; Wu et al., 2025), but its health effects may depend on ambient temperature conditions and exposure timing. This underscores the need for safe activity planning during temperature extremes, such as shifting exercise to more moderate hours and using temperature-controlled settings. Beyond physical activity, our pathway results also point to behaviour-specific recommendations for other modifiable behaviours. For example, encouraging breaks in sedentary time during SD-HE days and prioritising sleep

protection during cold spell days. Taken together, these behaviour-specific recommendations can be integrated into heat or cold health alerts as part of early warning during temperature extremes.

Beyond the empirical findings, our study makes a substantive methodological contribution to the assessment of cumulative cardiovascular effects of long-term ETE exposure. We provide a formal mathematical demonstration that a meaningful and clearly interpretable reference contrast may require exhaustively accounting for a complete set of temporally exclusive ETEs within the exposure window. Failure to meet this condition, a common practice in existing work, may contaminate the reference condition and weaken per day interpretation for any target ETE category. We further highlight that for assessing and understanding such effects, at least four conceptually related yet temporally exclusive ETEs must be examined simultaneously. This is particularly important because individuals do not experience heat or cold extremes in isolation; rather, they may be repeatedly exposed to different types of ETEs across years. It is important to emphasise, however, that we do not claim this strategy to be a definitive or exhaustive solution; rather, it should be regarded as a fundamental methodological strategy for valid inference. In practice, ETE categories may be further disaggregated, for example, by distinguishing day-night compounding ETEs (Liu et al., 2025; Yang et al., 2024) or differentiating ETEs compounded with air pollutants (Chen et al., 2025; Cheng et al., 2024), provided that a complete set of temporally exclusive ETEs can be exhaustively accounted for.

We highlight that, under such an identification strategy, the inclusion of temporally exclusive ETE categories and the corresponding reference condition must be determined ex ante according to the specific research question, so that the included categories together constitute a complete set of ETEs and the remaining exposure window represents a predefined genuinely ETE-free reference condition, rather than being determined ex post by statistical significance. For example, our analysis found no statistically significant

association between long-term exposure to heatwaves and hypertension risk. However, this does not imply that exposure to heatwaves can be arbitrarily omitted in future research. This is not merely because effect estimates may vary across contexts, but more fundamentally for two reasons. First, point estimates for such exposure are not exactly zero, and their exclusion could still introduce bias. Second, retaining such categories helps ensure that the non-ETE condition can be meaningfully conceptualised as ETE-free, thereby providing a clearly defined reference for interpreting the effects of the ETEs under consideration.

This framework may also support policy and practice by providing a clearer interpretation of the estimated exposure contrast. Within a fixed exposure window, the total number of days is constant; therefore, any reduction in ETE exposure necessarily corresponds to more days under non-ETE conditions. Using non-ETE conditions as the reference thus links the estimated coefficient to an exposure-reduction scenario aligned with climate adaptation and public-health practice: reducing population exposure to extreme-temperature conditions and increasing days under comparatively lower-risk non-ETE conditions. This provides a quantitative basis for comparing the long-term health risks of different ETE types, identifying events that may require priority intervention, and establishing a common evaluative standard for assessing whether different adaptation strategies reduce cumulative ETE-related health risks relative to the same non-ETE reference condition.

Our research has, nevertheless, several limitations. First, exposure and outcome timing may have been subject to misclassification. ETE exposure was assessed using county-level data because of privacy restrictions, which may introduce non-differential exposure misclassification, particularly in geographically large counties. In addition, annualising ETE exposure over unequal follow-up intervals may not fully capture inter-annual variability, because the same annualised value may reflect either stable yearly

exposure or ETEs concentrated in specific years. Moreover, we did not incorporate the exact onset time of hypertension, particularly the duration since physician diagnosis, because the exact dates of physician diagnosis or medication initiation were unavailable and self-reported diagnosis duration may be subject to recall error. Although assigning event time to the survey wave at which hypertension was first identified is common in cohort studies, it may still introduce event-time misclassification and bias the estimated associations. Second, lifestyle behaviours were assessed only at the follow-up wave, rather than through precise measurement of long-term behavioural patterns over the observation interval. Although these measures were treated as proxies for habitual behavioural status, they may not fully capture behavioural patterns and changes occurring throughout the interval, which may affect both mediation-related and interaction-related components of the decomposition. This limitation also reflects a broader methodological challenge in cohort studies, where long-term behaviours are often difficult to measure precisely. Addressing this challenge would help distinguish habitual behavioural patterns from exposure-related behavioural changes and improve the interpretation of decomposed behavioural components. Moreover, although we examined the individual interplay between long-term ETE exposure and three unhealthy lifestyle behaviours, we did not assess interactions involving multiple simultaneous behaviours. This remains methodologically challenging because approaches for decomposing pathways involving multiple mediators in a manner comparable to four-way decomposition are still limited. Third, residual confounding may remain. Dietary variables, including sodium intake, were not included in the primary models because of incomplete dietary information in the CHNS dataset, and unmeasured dietary factors could have influenced the estimated associations. Fourth, the north – south regional classification serves only as a proxy for residential heating zone and cannot fully disentangle heating infrastructure from broader climatic adaptation. Future studies with individual-level heating use are needed to better evaluate

whether residential heating modifies the health effects of cold-related ETE exposure.

5 Conclusions

Clear and interpretable estimation of cumulative cardiovascular effects of long-term ETE exposure may require modelling a complete set of temporally exclusive ETEs, including cold spells, SD-CEs, heatwaves, and SD-HEs. Applying this strategy in a nationwide prospective cohort study, we found that long-term exposure to short-duration ETEs (SD-CEs and SD-HEs), which has received limited academic and policy attention, was a more robust risk factor for hypertension than exposure to sustained ETEs. Our findings also indicated that, while the hypertension risk associated with long-term ETE exposure is largely driven by exposure-induced physiological load, interactions with lifestyle behaviours exert an additional influence. These results support greater attention to short-duration ETEs in monitoring and prevention and underscore the value of climate-sensitive behavioural guidance for hypertension risk reduction.

Appendix A

We begin by proving a proposition: in a correctly specified model, in which categories of temporally exclusive ETEs within the exposure window are exhaustively accounted for, the coefficient for exposure to a given category of ETE accurately represents the per-unit risk of exposure to that ETE relative to the genuinely non-ETE condition. To formalise this, we assume the true underlying relationship for the risk of a chronic cardiovascular outcome, r , is a function of the exposure – for example, measured in days – to a complete set of k categories of temporally exclusive ETEs within the exposure window, denoted as $K = (e_1, e_2, \dots, e_k)$, where $k \in \mathbb{N}^+$, as well as of exposure to non-ETE conditions (n). For clarity, other covariates are omitted from the equations, as their exclusion does not affect the validity of the proof.

Such a relationship is presented in its vector form as **Eq. (A.1)**. For each individual i , these exposure durations – $e_1, e_2, \dots, e_k$, and n_i – are subject to the constraint that they sum to the total length of the time window of interest (e.g., total number of days between the baseline and follow-up), as shown in **Eq. (A.2)**. Due to the perfect multicollinearity amongst the exposure vectors, **Eq. (A.1)** cannot be estimated directly. Therefore, the non-ETE exposure is treated as the reference, and we substitute the constraint from **Eq. (A.2)**, which transforms **Eq. (A.1)** into a common, estimable form shown in **Eq. (A.3)**. This estimable model shows that when categories of temporally exclusive ETEs within the exposure window are exhaustively accounted for, i.e., when exposure to ETEs (included in the model) and exposure to the non-ETE condition (not included in the model) collectively span the entire time window of interest, the coefficient for each ETE vector represents the risk from exposure to that ETE relative to exposure to the non-ETE condition. The coefficient for a given e_i , which is $\gamma_i - \theta$, essentially quantifies the additional risk of one day of exposure to ETE type i compared to one day of exposure to the non-ETE condition.

$$\mathbf{r} = \alpha \mathbf{1} + \sum_{i \in K} \gamma_i \mathbf{e}_i + \theta \mathbf{n} + \boldsymbol{\vartheta} \quad (\text{A.1})$$

$$\text{s.t. } \sum_{i \in K} e_i + n_i = T \quad (\text{A.2})$$

$$\mathbf{r} = (\alpha + \theta T) \mathbf{1} + \sum_{i \in K} (\gamma_i - \theta) \mathbf{e}_i + \boldsymbol{\vartheta} \quad (\text{A.3})$$

Now, we consider an incomplete equation (**Eq. (A.4)**), in which the sum of exposure to the considered sets of ETEs $M = (e_1, e_2, \dots, e_m) \subset K$, together with the exposure to non-ETEs conditions, does not span the full-time window of interest, since M is a subset of K :

$$\mathbf{r} = \eta \mathbf{1} + \sum_{i \in M} \delta_i \mathbf{e}_i + \boldsymbol{\varepsilon} \quad (\text{A.4})$$

According to the Frisch-Waugh-Lovell Theorem, δ_i can be determined:

$$\delta_i = \text{Cov}(\mathbf{r}, \mathbf{v}_i) / \text{Var}(\mathbf{v}_i) \quad (\text{A.5})$$

$$\mathbf{e}_i = c_0 \mathbf{1} + \sum_{j \in M, j \neq i} c_j \mathbf{e}_j + \mathbf{v}_i \quad (\text{A.6})$$

where vector $\mathbf{v}_i$ is the residual from the auxiliary regressions defined in **Eq. (A.6)**. To reveal the true composition of the coefficient of δ_i , we substitute the correctly specified model from **Eq. (A.3)** into $\mathbf{r}$ of **Eq. (A.5)**:

$$\delta_i = \text{Cov} \left((\alpha + \theta T) \mathbf{1} + \sum_{j \in M} (\gamma_j - \theta) \mathbf{e}_j + \boldsymbol{\vartheta}, \mathbf{v}_i \right) / \text{Var}(\mathbf{v}_i) \quad (\text{A.7})$$

Since $\mathbf{v}_i$ is uncorrelated with both the intercept vector and the vector $\mathbf{e}_i$ from which it originates, and since the error term vector is uncorrelated with all vectors for explanatory variables, we have:

$$\text{Cov}(\mathbf{r}, \mathbf{v}_i) = (\gamma_i - \theta) \text{Cov}(\mathbf{e}_i, \mathbf{v}_i) + \sum_{j \in K/M} (\gamma_j - \theta) \text{Cov}(\mathbf{e}_j, \mathbf{v}_i) \quad (\text{A.8})$$

where $\mathbf{e}_j$ is an element in K that is not included in M . According to the properties of residuals, the covariance between a variable and its residual component is equal to the variance of that residual. Thus, **Eq. (A.8)** reduces to:

$$\text{Cov}(\mathbf{r}, \mathbf{v}_i) = (\gamma_i - \theta) \text{Var}(\mathbf{e}_i, \mathbf{v}_i) + \sum_{j \in K/M} (\gamma_j - \theta) \text{Cov}(\mathbf{e}_j, \mathbf{v}_i) \quad (\text{A.9})$$

Therefore, δ_i can ultimately be expressed as:

$$\delta_i = \gamma_i - \left[\theta - \frac{\sum_{j \in K/M} (\gamma_j - \theta) \text{Cov}(\mathbf{e}_j, \mathbf{v}_i)}{\text{Var}(\mathbf{v}_i)} \right] \quad (\text{A.10})$$

Eq. (A.10) shows that the coefficient for the exposure to ETE type i cannot be interpreted as the additional risk of one day of exposure to ETE type i relative to one day of exposure to the non-ETE condition. Specifically, the reference for this coefficient is not solely the exposure to the non-ETE condition; it is instead a composite variable that is contaminated by the effects of all other omitted ETEs weighted by their partial correlations.

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