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Non-optimal temperature and ischemic heart disease burden in Hangzhou, China mortality, years of life lost, and subtype-specific patterns

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Abstract

Background

Mortality counts do not reflect age at death, and it remains unclear whether mortality and years of life lost (YLL) identify the same high-risk temperatures, lag windows, and ischemic heart disease (IHD) subtypes. We examined these patterns for overall IHD, acute myocardial infarction (AMI), and chronic IHD.

Methods

We conducted a population-based time-series study in Hangzhou, China, during 2014–2021. Distributed lag nonlinear models were used to estimate temperature associations with daily mortality and YLL. Attributable burden was partitioned into moderate and extreme cold and heat.

Results

Across 2,922 days, 37,919 IHD deaths and 566,166.3 YLL were recorded. Cold effects were delayed and accumulated over longer lags, whereas heat effects were concentrated within the first week. For overall IHD, extreme-cold relative risks were 1.97 (95% CI 1.42–2.73) for mortality and 1.93 (1.33–2.79) for YLL over lag 0–21 days; corresponding extreme-heat estimates over lag 0–7 days were 1.36 (1.22–1.52) and 1.33 (1.17–1.52). AMI and chronic IHD showed similar lag patterns. Temperatures below the minimum-risk temperature accounted for 17.46% of IHD deaths, and moderate cold contributed about 95% of the cold-attributable mortality estimate.

Conclusions

Mortality and YLL provided complementary perspectives on temperature-related IHD burden. Although thermal extremes had the highest relative risks, frequent moderate cold accounted for most cold-attributable mortality, supporting distinct prevention windows for heat and cold.

Keywords

Ischemic heart disease; ambient temperature; years of life lost; distributed lag nonlinear model; attributable burden

Introduction

Ischemic heart disease (IHD) remains a major cause of premature death and health loss worldwide [1]. Beyond established metabolic and behavioral risk factors, ambient temperature is an important environmental determinant of cardiovascular health [2, 3]. Both heat and cold have been associated with increased cardiovascular mortality, and climate change is altering the frequency, intensity, and population distribution of thermal exposures. Importantly, population burden is not determined only by the relative risk on extreme-temperature days: moderately non-optimal temperatures occur far more often and may therefore account for a substantial share of temperature-attributable mortality [4, 5].

The basic temperature-IHD relationship is already well established. Heat-related cardiovascular effects generally occur rapidly, whereas cold-related effects may persist over longer lag periods [6]. Studies in China and other settings have also reported a greater mortality burden from cold

than from heat [7, 8], and previous work in Hangzhou identified short-term heat effects and more sustained cold-related increases in IHD mortality [9]. Accordingly, an important remaining question is not simply whether temperature is associated with IHD mortality, but how that association translates into premature life loss, whether it is expressed similarly across major IHD manifestations, and whether the temperatures associated with the highest relative risks are also those responsible for the greatest population burden.

Mortality counts and years of life lost (YLL) capture related but different dimensions of disease burden. Death counts weight each fatal event equally, whereas YLL incorporates remaining standard life expectancy at the age of death and therefore reflects premature mortality [10]. Incorporating YLL may reveal dimensions of temperature-related IHD burden that are not apparent from mortality counts alone [11]. This distinction may be particularly relevant because IHD includes clinically distinct manifestations, especially acute myocardial infarction (AMI) and chronic IHD, which may differ in their vulnerability and temporal response to thermal stress [12]. Although temperature has been linked to acute coronary events and chronic cardiovascular mortality [12], few studies have evaluated mortality and YLL in parallel while also examining major IHD subtypes, their exposure-lag profiles, and the distribution of attributable burden across moderate and extreme cold and heat. Such an integrated approach can determine whether conventional mortality counts and premature life loss lead to the same prevention priorities.

Using eight years of population-based mortality surveillance in Hangzhou, a subtropical city with substantial seasonal temperature variation, we therefore addressed three linked questions: whether mortality and YLL show concordant temperature-response and lag patterns; whether

those patterns are shared by AMI and chronic IHD; and whether the parts of the temperature distribution associated with the highest relative risks coincide with those generating the greatest attributable burden. We estimated nonlinear and delayed associations using distributed lag nonlinear models, quantified mortality and YLL attributable to moderate and extreme cold and heat, and explored effect modification. By framing the analysis around premature life loss, disease subtype, temporal window, and population burden, the study was designed to extend existing evidence beyond conventional mortality counts and extreme-event comparisons.

Methods

Mortality data

Daily mortality records for registered residents of Hangzhou were obtained from the Mortality Surveillance System of the Hangzhou Center for Disease Control and Prevention (CDC) for January 1, 2014, through December 31, 2021. Deaths were classified according to the underlying cause of death coded using the International Classification of Diseases, 10th Revision (ICD-10). Death reports underwent routine review by the reporting medical institution and county- and municipal-level CDC staff, together with linkage and underreporting checks used in the local mortality-surveillance system.

The primary outcome was IHD (ICD-10 I20–I25). Subtype analyses focused on AMI (I21–I22) and chronic ischemic heart disease (I25). Other acute IHD codes (I20 and I24) remained included in the overall IHD endpoint but were not modeled as a separate subtype. Individual

records contained sex, age at death, date of death, residential administrative area, educational level, place of death, and underlying-cause ICD-10 information. Daily counts and daily YLL were aggregated separately for IHD, AMI, and chronic IHD.

Meteorological and air pollution data

Meteorological data were obtained from the Hangzhou Meteorological Bureau and included daily mean, maximum, and minimum temperature and daily mean relative humidity. Air pollution data were obtained from the Hangzhou Environmental Monitoring Center and represented citywide daily concentrations from the national monitoring network, including fine particulate matter ($PM_{2.5}$), inhalable particulate matter (PM_{10}), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), carbon monoxide (CO), and daily maximum 8-hour ozone (O_3). Calendar variables included day of week and public holidays. Daily mortality, meteorological, pollution, and calendar series were aligned by date for the complete 2014–2021 study period.

Calculation of years of life lost

YLL was calculated using the Global Burden of Disease (GBD) 2019 standard reference life table. Deaths were classified into 21 age groups. For each calendar day t, daily YLL was calculated as:

$$YLL_t = \sum_s \sum_a D_{t,a,s} * L_a$$

Let $D_{t,a,s}$ denote the number of deaths occurring on day t within age group a and sex stratum s, and let L_a represent the GBD 2019 standard remaining life expectancy corresponding to age group a. The sex-specific daily YLL were computed by aggregating the age-specific YLL values

within each sex category. A uniform, sex-neutral GBD 2019 reference life table was utilized for both males and females; sex-specific life tables were not employed in the analysis. Furthermore, neither age weighting nor time discounting procedures were applied.

Statistical analysis

Daily mortality, daily YLL, and mean temperature were summarized using means, standard deviations (SDs), ranges, and quartiles. Total deaths, total YLL, and YLL per death were additionally summarized for overall IHD and major subtypes.

Associations between temperature and IHD outcomes were estimated using distributed lag nonlinear models (DLNMs) within generalized linear models. Daily deaths were modeled using quasi-Poisson regression with a log link. Because daily YLL is a non-negative weighted outcome rather than an integer count, we analyzed it using the same quasi-Poisson log-link framework as a quasi-likelihood mean model to estimate multiplicative changes in expected daily YLL while allowing overdispersion, without requiring YLL to follow a Poisson distribution. The core model was:

$$Y_t \sim \text{quasi-Poisson}(\mu_t, \varphi)$$

$$\log(\mu_t) = \alpha + cb(T_t, l) + ns(time_t, 7\ df/year) + ns(RH_t, 3\ df) + DOW_t + Holiday_t$$

Here, Y_t denotes the daily death count or daily YLL on day t; μ_t is its expected value; φ is the quasi-Poisson dispersion parameter; $cb(T_t, l)$ is the two-dimensional cross-basis for daily mean temperature T_t and lag l; ns() denotes a natural cubic spline; RH_t is daily mean relative humidity; DOW_t is a seven-level day-of-week indicator; and $Holiday_t$ is a public-holiday indicator. Calendar time was modeled with 7 degrees of freedom (df) per year and relative humidity with

3 df.

The exposure dimension of the cross-basis used a natural cubic spline with internal knots at 6.3, 25.2, and 29.2 °C, corresponding to the 10th, 75th, and 90th percentiles of the observed temperature distribution. The lag dimension included an intercept and a natural cubic spline with internal knots at 5.25, 10.50, and 15.75 days. The maximum lag was 21 days to capture both short-lag heat associations and delayed cold associations. For each disease endpoint and outcome type, the minimum-risk temperature (MMT) was identified as the temperature associated with the minimum overall cumulative relative risk (RR) over lag 0–21 days, searched on a 0.1°C grid between the 1st and 99th percentiles of the observed temperature distribution. Extreme cold and extreme heat were defined as the 1st percentile (P1, 0.8°C) and the 99th percentile (P99, 33.0°C) of the observed daily mean temperature distribution, respectively. Cumulative RRs were estimated over lag 0–4, 0–7, 0–14, and 0–21 days from the same 21-day cross-basis model. Lag 0–21 was used as the principal summary window for cold, whereas lag 0–7 was used for heat.

Separate DLNMs were fitted for AMI and chronic IHD. Because each disease-endpoint model used its own MMT, comparisons of subtype-specific RR magnitudes were interpreted descriptively, with emphasis on the consistency of temperature–response and lag patterns rather than formal ranking of susceptibility.

Attributable burden was estimated from the fitted DLNM using the backward-attribution framework of Gasparrini [13]. For each endpoint, all cold was defined as temperatures below the endpoint-specific MMT and all heat as temperatures above the MMT. Extreme cold covered

171 the observed minimum through P1; moderate cold covered P1 through the MMT; moderate heat
172 covered the MMT through P99; and extreme heat covered P99 through the observed maximum.
173 Attributable numbers (deaths or YLL) and attributable fractions (AFs) were estimated, with 95%
174 confidence intervals (CIs) obtained by Monte Carlo simulation of the model coefficients.

175 Effect modification for overall IHD was evaluated for age (<75 vs ⩾75 years), sex, and
176 geographic grouping (core urban districts vs other districts). Subgroup-specific DLNMs were
177 used to obtain descriptive cold lag 0–21 and heat lag 0–7 estimates. Formal effect modification
178 was assessed in joint models containing subgroup-by-cross-basis interaction terms, using a joint
179 Wald F test with day-clustered robust covariance. Because subgroup-specific models used
180 subgroup-specific MMTs, subgroup RR contrasts were treated as descriptive and the interaction
181 test was considered the primary inferential assessment.

182 Three sets of sensitivity analyses were conducted. First, we evaluated the robustness of the
183 DLNM specification by varying the maximum lag to 14 and 28 days; using 4, 5, or 6 df for the
184 temperature dimension; using 3, 4, or 6 df for the lag dimension; changing calendar-time control
185 from the primary 7 df/year to 6 or 8 df/year; increasing the relative-humidity spline from 3 to
186 4 df; and replacing daily mean temperature with daily maximum or daily minimum temperature.
187 Second, six single-pollutant models separately adjusted for $PM_{2.5}$, PM_{10}, CO, SO_2, NO_2, or O_3
188 using natural cubic splines with 3 df, and selected two-pollutant models included $PM_{2.5}$ + O_3,
189 PM_{10} + O_3, or NO_2 + O_3. Third, YLL associations were re-estimated using a Tweedie
190 generalized additive model with a log link, which accommodates zero values and positive
191 continuous outcomes. Analyses were performed in R version 4.2.2 using the dlnm, mgcv,

FluMoDL, and sandwich packages. Two-sided $P < 0.05$ was considered statistically significant.

Ethics approval

The study was approved by the Ethics Committee of Hangzhou Center for Disease Control and Prevention on December 8, 2022. The requirement for informed consent was waived because the analysis used de-identified mortality-surveillance data aggregated to daily outcomes.

Results

Study characteristics and premature-mortality burden

The study covered 2,922 consecutive days from January 1, 2014, through December 31, 2021. A total of 37,919 IHD deaths were recorded, corresponding to 566,166.3 YLL and a mean of 14.93 YLL per death (Supplementary Table S1). AMI accounted for 13,292 deaths and 249,179.8 YLL (18.75 YLL per death), whereas chronic IHD accounted for 24,024 deaths and 304,025.7 YLL (12.66 YLL per death). A further 598 deaths (12,858.6 YLL) were coded as other acute IHD (I20/I24) and remained included in the overall IHD endpoint. On average, there were 12.98 IHD deaths and 193.76 IHD YLL per day. Corresponding daily means were 4.55 deaths and 85.28 YLL for AMI, and 8.22 deaths and 104.05 YLL for chronic IHD (Table 1). Mean daily temperature was 18.12°C (SD 8.59°C), ranging from -5.0°C to 35.6°C.

Overall IHD temperature‑response associations and differential lag patterns

The MMT was approximately 27.9°C for IHD mortality and 26.9°C for IHD YLL. Cumulative temperature–response curves showed increasing risk toward low temperatures, with the cold

association becoming progressively stronger as the cumulative lag window lengthened. In contrast, the high-temperature association was most evident over shorter lag windows (Figure 1). Mortality and YLL curves were closely parallel. For extreme cold (P1 = 0.8°C), the cumulative mortality RR increased from 1.41 (95% CI 1.19–1.67) over lag 0–7 days to 1.62 (1.27–2.07) over lag 0–14 days and 1.97 (1.42–2.73) over lag 0–21 days. The corresponding YLL RRs were 1.50 (1.23–1.82), 1.64 (1.24–2.16), and 1.93 (1.33–2.79), respectively (Table 2). Lag-specific curves indicated a delayed cold association, with the highest point estimates at approximately lag 5 for both mortality and YLL (Figure 2). For extreme heat (P99 = 33.0°C), the strongest lag-specific association occurred on or immediately after the day of exposure (Figure 2). The cumulative mortality RR was 1.27 (1.15–1.41) over lag 0–4 days and 1.36 (1.22–1.52) over lag 0–7 days; the corresponding YLL RRs were 1.26 (1.12–1.42) and 1.33 (1.17–1.52). Unlike cold, extending the cumulative window beyond the first week did not strengthen the heat association.

Subtype-specific associations for AMI and chronic IHD

AMI and chronic IHD generally reproduced the overall IHD pattern of stronger long-lag cold associations and shorter-lag heat associations (Figure 3; Supplementary Figures S1–S4). For AMI mortality, the RR for extreme cold was 1.96 (1.12–3.42) over lag 0–21 days, while the RR for extreme heat was 1.33 (1.11–1.60) over lag 0–7 days. For AMI YLL, extreme cold was associated with an RR of 2.12 (1.31–3.43) over lag 0–21 days, whereas the lag 0–7 heat estimate was 1.24 (0.96–1.61) and was less precise (Table 2). For chronic IHD, the extreme-cold lag 0–21 RR was 1.91 (1.27–2.88) for mortality and 1.91 (1.20–3.02) for YLL. Extreme heat over lag

0–7 was associated with an RR of 1.34 (1.17–1.55) for mortality and 1.30 (1.11–1.52) for YLL. The lag-specific curves for chronic IHD showed cold peaks around lag 5–6 and heat associations concentrated near lag 0, consistent with the overall IHD temporal profile (Supplementary Figures S3 and S4). The AMI YLL cumulative curve had a lower endpoint-specific minimum-risk temperature and wider uncertainty than the other disease-endpoint curves (Supplementary Figure S1).

Temperature-attributable mortality and YLL

For overall IHD mortality, an estimated 6,621 deaths (95% CI 848 to 11,298) were attributable to temperatures below the MMT, corresponding to an AF of 17.46% (2.24% to 29.79%) (Supplementary Table S2). Most of this burden arose from moderate cold: 6,295 attributable deaths, AF 16.60% (1.57% to 28.89%). Extreme cold accounted for 408 deaths, AF 1.08% (0.60% to 1.52%). Thus, moderate cold represented approximately 95% of the point estimate of cold-attributable IHD deaths. The all-heat mortality burden was smaller, with 423 attributable deaths and an AF of 1.12% (0.44% to 1.70%). For IHD YLL, the point estimate for all-cold burden was 65,256 YLL (AF 11.53%), although the 95% CI included the null (-6.58% to 25.76%). Moderate cold accounted for 60,290 YLL (AF 10.65%), whereas extreme cold accounted for 5,965 YLL (AF 1.05%, 0.51% to 1.52%). Heat accounted for an estimated 6,460 YLL (AF 1.14%, 0.22% to 1.97%). The stacked attributable-fraction profiles confirmed that moderate cold was the dominant component for overall IHD and chronic IHD mortality, whereas component estimates for AMI YLL were less stable because its endpoint-specific MMT was lower (Figure 4). Subtype-specific cold-attributable estimates were less precise for

AMI: the AF was 9.06% (-23.38% to 31.21%) for mortality and 10.21% (-2.59% to 20.05%) for YLL. Chronic IHD showed a larger cold-attributable mortality fraction of 20.25% (1.21% to 34.85%); the corresponding YLL AF was 16.62% (-5.98% to 33.33%) (Supplementary Table S2).

Effect modification

Age modified the overall temperature–lag association when participants were divided at 75 years. The joint interaction test was statistically significant for both IHD mortality (P = 0.0246) and IHD YLL (P = 0.0316) (Supplementary Table S3). Descriptively, the extreme-cold lag 0–21 RR was 2.47 versus 1.99 for mortality and 2.29 versus 2.10 for YLL among those aged <75 versus ≥75 years, respectively. Heat lag 0–7 RRs were 1.28 versus 1.34 for mortality and 1.22 versus 1.32 for YLL. There was no statistically significant evidence of effect modification by sex (P = 0.160 for mortality and P = 0.196 for YLL). Geographic grouping showed evidence of heterogeneity for mortality (P = 0.033) but not for YLL (P = 0.111).

Sensitivity analyses

Adjustment for individual pollutants and selected two-pollutant combinations did not materially change the principal IHD estimates (Supplementary Figure S5). For cold lag 0–21, pollutant-adjusted RRs remained close to the primary estimates for both mortality and YLL; similarly, heat lag 0–7 estimates changed little after adjustment for $PM_{2.5}$, PM_{10}, CO, SO_2, NO_2, O_3, or selected pollutant pairs. The principal temperature associations were also robust to alternative model specifications and temperature metrics (Supplementary Figure S6). Estimates remained broadly consistent when the maximum lag was changed to 14 or 28 days; when the

degrees of freedom for the temperature, lag, calendar-time, or humidity terms were varied; and when daily maximum or minimum temperature replaced daily mean temperature. The Tweedie sensitivity model likewise closely reproduced the quasi-Poisson YLL results (Supplementary Figure S7). For extreme cold over lag 0–21, the quasi-Poisson and Tweedie RRs were both approximately 1.93 with nearly identical confidence intervals, while for extreme heat over lag 0–7 both models yielded an RR of approximately 1.33. Together, these analyses support the robustness of the main findings to the selected DLNM parameterization, exposure metric, co-pollutant adjustment, and the assumed mean–variance relationship for YLL.

Discussion

Rather than simply confirming the established association between ambient temperature and IHD mortality, this study adds three linked perspectives: premature life loss, disease subtype, and population-attributable burden. Mortality and YLL showed broadly concordant exposure-lag patterns, with heat-related associations concentrated in the first week and cold-related associations accumulating over a longer period. AMI and chronic IHD showed similar temporal profiles, while AMI generated substantially more YLL per death. At the population level, the highest relative risks occurred at temperature extremes, but approximately 95% of the point estimate of cold-attributable IHD deaths arose from moderate cold. Together, these findings show that the public-health importance of non-optimal temperature depends not only on the magnitude of relative risk, but also on when deaths occur, how much life is lost, and how frequently a given exposure occurs.

The parallel analysis of mortality and YLL is important because the two endpoints answer different questions. Death counts quantify the number of fatal events, whereas YLL incorporates age at death and therefore captures premature mortality[10]. In our data, AMI accounted for 13,292 deaths compared with 24,024 chronic IHD deaths, yet the mean YLL per death was 18.75 years for AMI and 12.66 years for chronic IHD. Thus, a lower number of deaths did not translate directly into a proportionately smaller premature-mortality burden. Temperature-related relative risks were generally similar for mortality and YLL, but the attributable YLL estimates were less precise: for all cold, the YLL attributable fraction was 11.53% and its 95% CI included the null. The contrasting lag structures also have practical implications. Heat effects in our study were strongest on or immediately after exposure and were largely captured within lag 0-7 days, whereas cold associations strengthened as the cumulative window extended to 21 days. This pattern is consistent with the broader temperature-cardiovascular literature [14, 15], and with studies showing that heat-related cardiovascular events are concentrated at short lags while cold effects may persist for weeks [16, 17]. The contribution of the present analysis is therefore not the observation that heat is acute and cold is delayed per se, but the demonstration that the same temporal distinction is evident for both death counts and premature life loss in IHD. From a prevention perspective, the results support different response windows: immediate protection during hot weather and continued cardiovascular vigilance after cold exposure.

The different time courses are biologically plausible. Heat can induce cutaneous vasodilation, sweating, dehydration, hemoconcentration, electrolyte disturbance, tachycardia, and increased

317 myocardial oxygen demand [18, 19]. These changes may precipitate myocardial ischemia or
318 acute coronary events within hours or days [20]. Cold exposure activates sympathetic responses
319 and peripheral vasoconstriction, raises blood pressure, and may increase blood viscosity,
320 fibrinogen, platelet activity, and thrombogenicity; coronary vasoconstriction may be
321 particularly relevant in atherosclerotic vessels [21]. Cold-season respiratory infections may
322 further contribute to delayed coronary risk, consistent with evidence of increased acute
323 myocardial infarction risk following influenza infection [22]. These mechanisms are consistent
324 with, but do not by themselves prove, the causal pathways underlying the observed lag profiles.

325 AMI and chronic IHD showed broadly similar heat- and cold-related lag patterns despite their
326 different clinical manifestations. This suggests that temperature-related hemodynamic,
327 autonomic, inflammatory, and thrombotic stresses may act across the ischemic heart disease
328 spectrum [18, 21]. Environmental thermal variation has also been associated with myocardial
329 infarction incidence in individual-level analyses [23]. At the same time, our subtype results
330 should not be read as proof of equal susceptibility. Each endpoint was modeled against its own
331 minimum-risk temperature, so cross-subtype relative-risk magnitudes are appropriately
332 interpreted descriptively rather than as formal rankings. This distinction also clarifies how the
333 present study differs from earlier work in Hangzhou that focused on temperature variability and
334 IHD mortality [9]: the current analysis extends the local evidence to daily mean temperature,
335 YLL, major IHD subtypes, lag structure, and attributable burden.

336 A second key distinction is between risk intensity and population burden. Extreme cold was
337 associated with the largest cumulative relative risks, but moderate cold accounted for 6,295 of

the estimated 6,621 cold-attributable IHD deaths—about 95% of the point estimate. This pattern is consistent with previous temperature-attributable mortality studies showing that frequent moderately non-optimal temperatures may account for a larger population burden than rare extremes [2, 24, 25]. For public health, this means that strategies triggered only by severe cold spells may miss a large share of preventable IHD burden. Heat remains important because of its rapid onset and because warming may increase high-temperature exposure [12]. The practical implication is therefore not to replace heat preparedness with cold preparedness, but to combine rapid heat-health responses with broader protection during ordinary cold weather and a longer post-cold surveillance window [26, 27].

The joint temperature-lag association differed by age for both mortality and YLL and by geographic grouping for mortality, whereas sex-related heterogeneity was not statistically evident. The apparently stronger relative cold estimates among adults aged <75 years should not be interpreted as showing that younger adults have greater absolute vulnerability. Differences in baseline risk, occupational or outdoor exposure, housing, heating and cooling access, urban form, healthcare access, and the greater remaining life expectancy assigned to deaths occurring at younger ages in the YLL metric could all contribute [28, 29]. Replication in larger multicity datasets would be needed before translating these subgroup patterns into targeted recommendations.

The robustness analyses strengthen confidence in the main exposure-lag patterns. Estimates were little changed after adjustment for individual or selected pairs of air pollutants, alternative lag lengths and spline specifications, different temperature metrics, or changes in time and

humidity control. For YLL, the Tweedie model closely reproduced the quasi-Poisson estimates, supporting the use of the quasi-Poisson model as a mean-structure approach rather than a literal Poisson distributional assumption. Methodologically, the value of the study lies in combining mortality, YLL, IHD subtype, lag structure, and burden partitioning in one analytic framework. This combination allows three questions to be separated: which temperatures produce the largest relative risks, when the associated burden occurs, and which exposures account for the greatest loss of life at the population level.

This study has several limitations. First, the time-series design captures population-level temporal associations and cannot establish individual-level causal effects. Second, citywide outdoor temperature does not measure personal exposure, particularly indoor temperature, occupational exposure, or heating and air-conditioning use. Third, individual cardiovascular risk factors, comorbidities, medication use, socioeconomic circumstances, and adaptive behaviors were unavailable, leaving potential residual confounding and limited ability to explain subgroup heterogeneity. Fourth, IHD subtypes were defined from the underlying cause of death and may be affected by diagnostic or coding error. Fifth, the moderate/extreme burden partition depends on the estimated minimum-risk temperature; this is especially important for YLL models with relatively flat or uncertain minima, and it contributes to uncertainty in some attributable estimates. Finally, the study was conducted in a single subtropical city. Although this setting provides a well-characterized population with substantial seasonal temperature variation, the magnitude of risk, minimum-risk temperature, and balance between cold and heat burden may differ in populations with other climates, infrastructures, and adaptive capacities.

Conclusion

Non-optimal temperature was associated with both fatal IHD events and premature life loss, but the public-health picture depended on more than the magnitude of the relative risk. Heat-related associations were rapid, cold-related associations accumulated over a longer period, and these temporal profiles were broadly shared by AMI and chronic IHD. Mortality and YLL were complementary rather than interchangeable: AMI produced substantially more YLL per death, while some YLL-attributable estimates remained imprecise. Most importantly, the greatest cold-related population burden came from frequent moderate cold rather than the most extreme cold days. These findings support climate-sensitive cardiovascular prevention that distinguishes high-risk from high-burden exposures and pairs immediate heat protection with broader, sustained cold-weather risk management.

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Additional files

Additional file 1

File format: PDF

Title: Supplementary tables and figures

Description: Supplementary Tables S1–S3 and Supplementary Figures S1–S7, including descriptive characteristics, temperature-attributable burden, effect-modification analyses, subtype-specific temperature–response and lag-response associations, pollutant-adjusted analyses, alternative model specifications and temperature metrics, and Tweedie sensitivity analyses.

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Authors' contributions

Tiewei Zhang was responsible for formal data analysis, visualization, and writing the original draft. Yan Zhang, Caixia Jiang, Bing Liu, Kang Qin, and Biao Li contributed to data collection

and quality control. Jue Xu was responsible for project administration and supervision, critically reviewed and revised the manuscript, and served as the corresponding author. All authors reviewed and approved the final version of the manuscript.

Availability of data and materials

The datasets analyzed in this study were obtained from the Mortality Surveillance System of the Hangzhou Center for Disease Control and Prevention. Because these data contain sensitive mortality information and are subject to institutional data-use restrictions, they cannot be made publicly available. Qualified researchers may request access to the data, subject to approval by the data custodian and compliance with relevant confidentiality and data-protection requirements. Requests for further information regarding data access should be directed to the corresponding author, Jue Xu, at xu.jue@outlook.com.

Declarations

Ethics approval and consent to participate

This study was conducted as part of a broader mortality research project approved by the Ethics Committee of Hangzhou Center for Disease Control and Prevention (Ethics Approval No. 2023-KS03) on December 8, 2022, and registered in the National Medical Research Registration System of China (Registration No. MR-33-23-049014). The requirement for informed consent was waived by the Ethics Committee because the study used de-identified mortality-surveillance data aggregated to daily outcomes. All procedures were conducted in

accordance with relevant institutional guidelines and regulations.

Consent for publication

Not applicable. The study was based on secondary analysis of de-identified mortality-surveillance data and does not contain any identifiable individual-level information requiring consent for publication.

Competing interests

The authors declare no competing interests.

Table 1. Daily ischemic heart disease mortality, years of life lost, and ambient temperature

Variable	Mean	Standard deviation	Minimum	P25	Median	P75	Maximum
IHD mortality	12.98	4.84	2.00	10.00	12.00	16.00	33.00
IHD YLL	193.76	76.49	19.98	137.72	186.33	240.94	654.03
AMI mortality	4.55	2.40	0.00	3.00	4.00	6.00	15.00
AMI YLL	85.28	51.35	0.00	46.51	79.16	116.81	326.86
Chronic IHD mortality	8.22	3.71	0.00	6.00	8.00	10.00	22.00
Chronic IHD YLL	104.05	48.62	0.00	68.27	97.91	132.83	311.92
Mean temperature (°C)	18.12	8.59	-5.00	10.60	19.00	25.20	35.60

Abbreviations: IHD, ischemic heart disease; AMI, acute myocardial infarction; YLL, years of life lost.

Table 2. Cumulative risks of extreme cold and heat for IHD mortality and years of life lost

Disease	Exposure/endpoint	Lag 0–4	Lag 0–7	Lag 0–14	Lag 0–21
IHD	Cold, mortality	1.04 (0.90–1.20)	1.41 (1.19–1.67)	1.62 (1.27–2.07)	1.97 (1.42–2.73)
IHD	Cold, YLL	1.11 (0.95–1.30)	1.50 (1.23–1.82)	1.64 (1.24–2.16)	1.93 (1.33–2.79)
IHD	Heat, mortality	1.27 (1.15–1.41)	1.36 (1.22–1.52)	1.27 (1.11–1.46)	1.32 (1.11–1.56)
IHD	Heat, YLL	1.26 (1.12–1.42)	1.33 (1.17–1.52)	1.20 (1.03–1.41)	1.27 (1.06–1.54)
AMI	Cold, mortality	1.17 (0.92–1.48)	1.48 (1.11–1.97)	1.61 (1.06–2.45)	1.96 (1.12–3.42)
AMI	Cold, YLL	1.25 (1.02–1.53)	1.46 (1.14–1.88)	1.63 (1.14–2.33)	2.12 (1.31–3.43)
AMI	Heat, mortality	1.28 (1.08–1.51)	1.33 (1.11–1.60)	1.28 (1.02–1.60)	1.22 (0.92–1.62)
AMI	Heat, YLL	1.25 (0.99–1.58)	1.24 (0.96–1.61)	1.09 (0.79–1.50)	1.30 (0.88–1.90)
Chronic IHD	Cold, mortality	0.96 (0.81–1.15)	1.35 (1.09–1.68)	1.59 (1.17–2.17)	1.91 (1.27–2.88)
Chronic IHD	Cold, YLL	0.99 (0.82–1.21)	1.45 (1.14–1.84)	1.56 (1.10–2.20)	1.91 (1.20–3.02)
Chronic IHD	Heat, mortality	1.25 (1.10–1.43)	1.34 (1.17–1.55)	1.26 (1.06–1.50)	1.35 (1.09–1.66)
Chronic IHD	Heat, YLL	1.24 (1.07–1.43)	1.30 (1.11–1.52)	1.23 (1.02–1.49)	1.34 (1.06–1.70)

Abbreviations: IHD, ischemic heart disease; AMI, acute myocardial infarction; YLL, years of life lost.

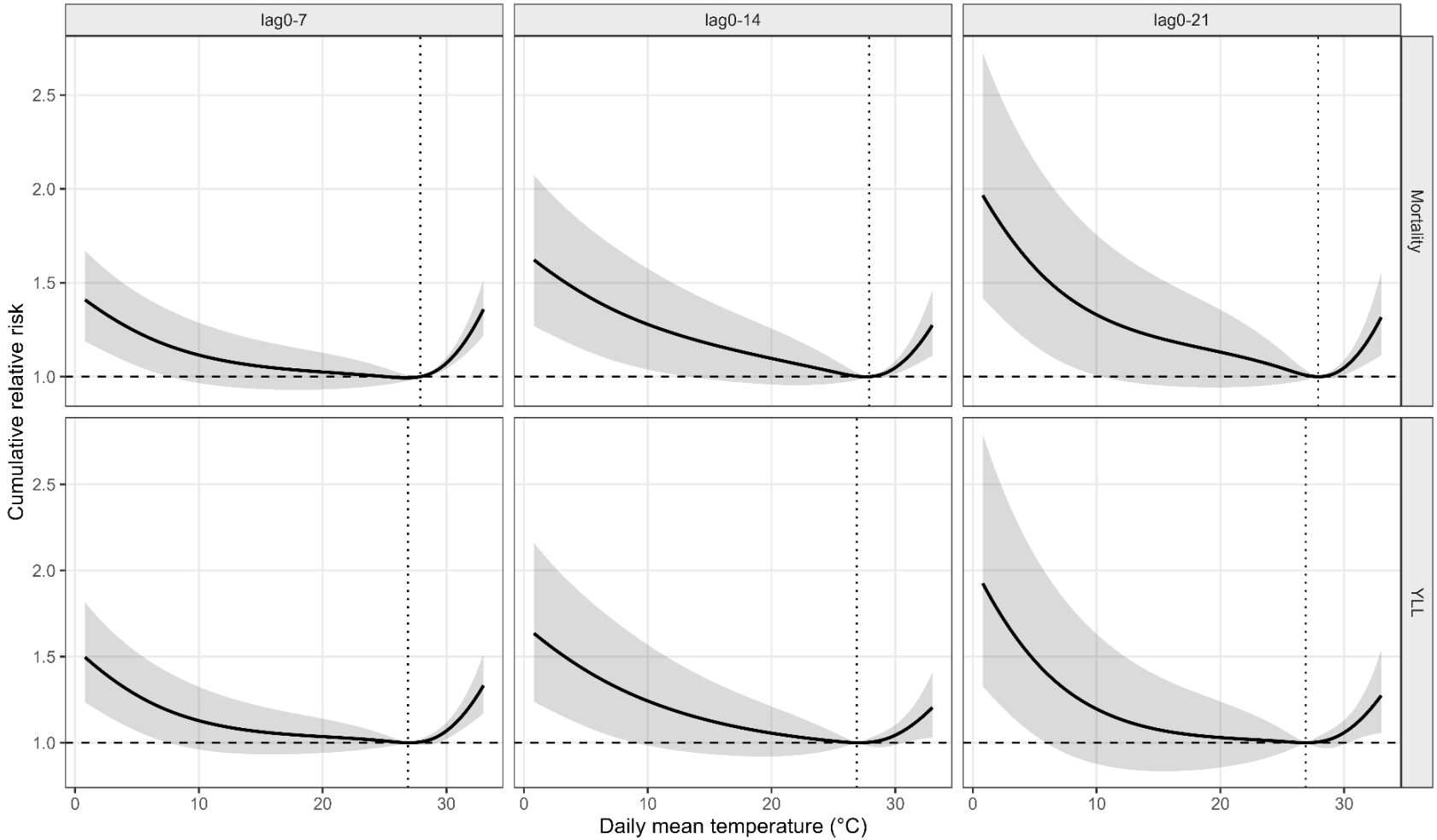


Fig. 1. Cumulative temperature–response associations for IHD mortality and years of life lost

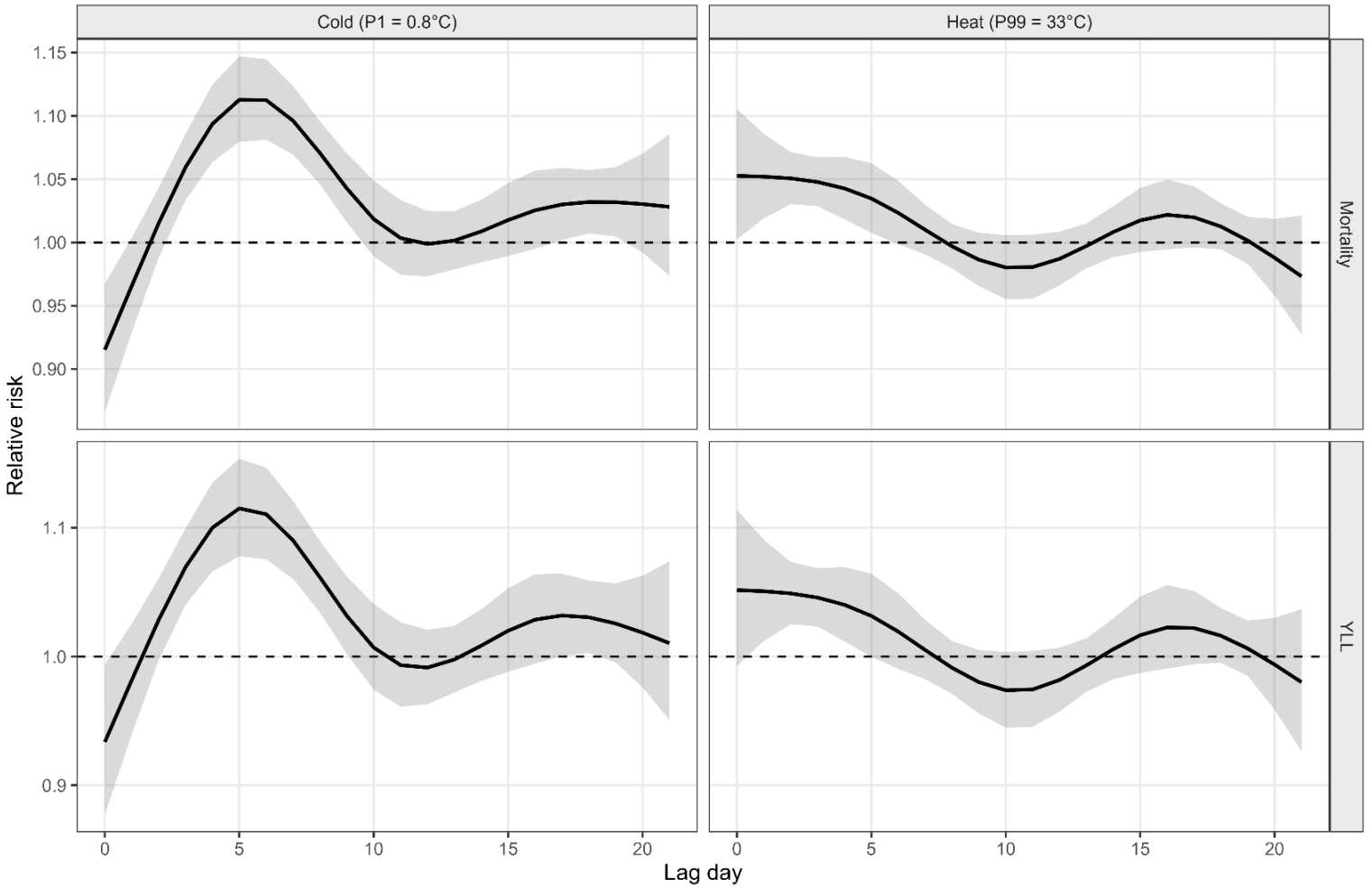


Fig. 2. Lag-specific effects of extreme cold and heat on IHD mortality and years of life lost

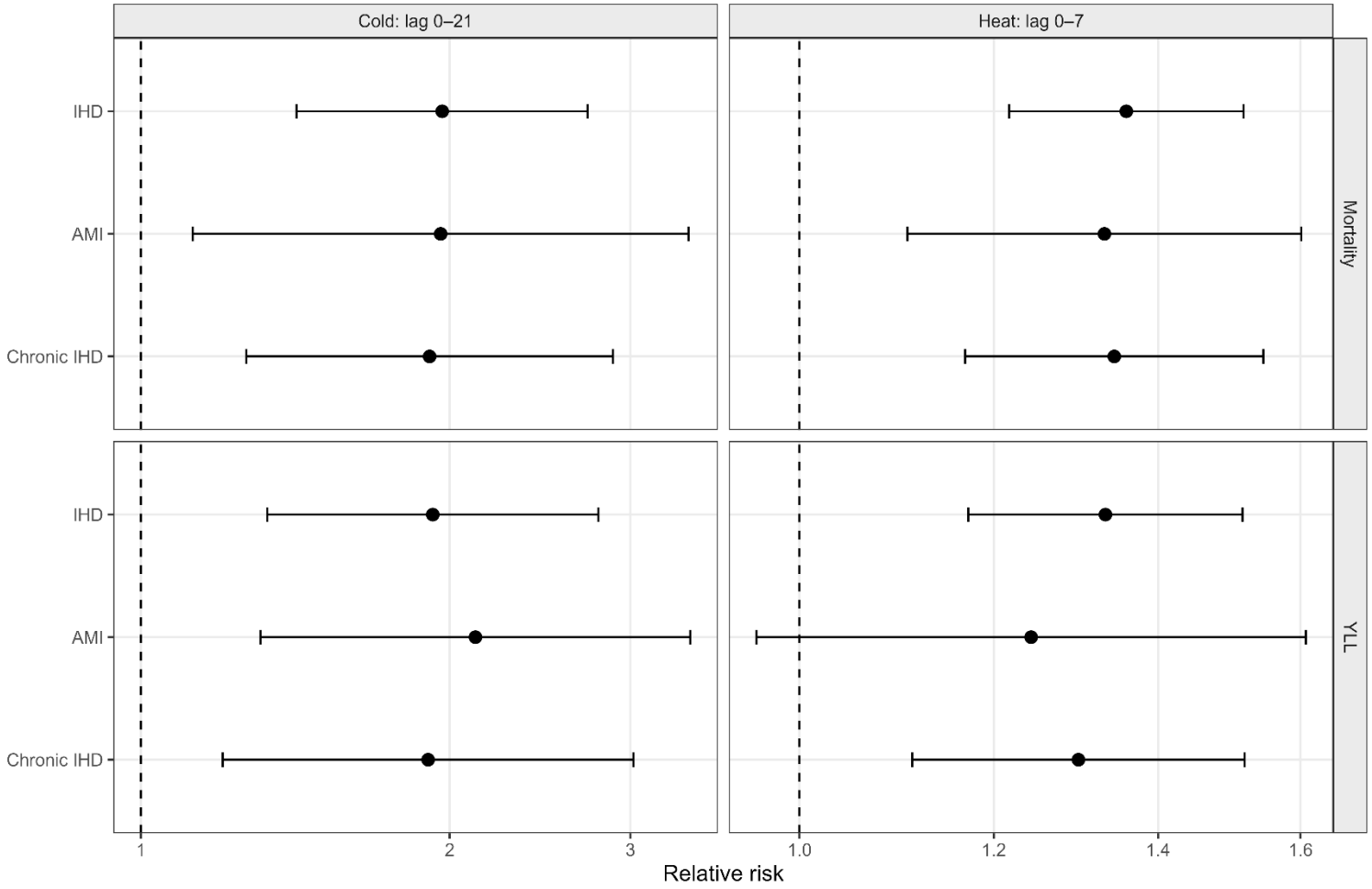


Fig. 3. Cumulative effects of extreme cold and heat on mortality and YLL across IHD subtypes

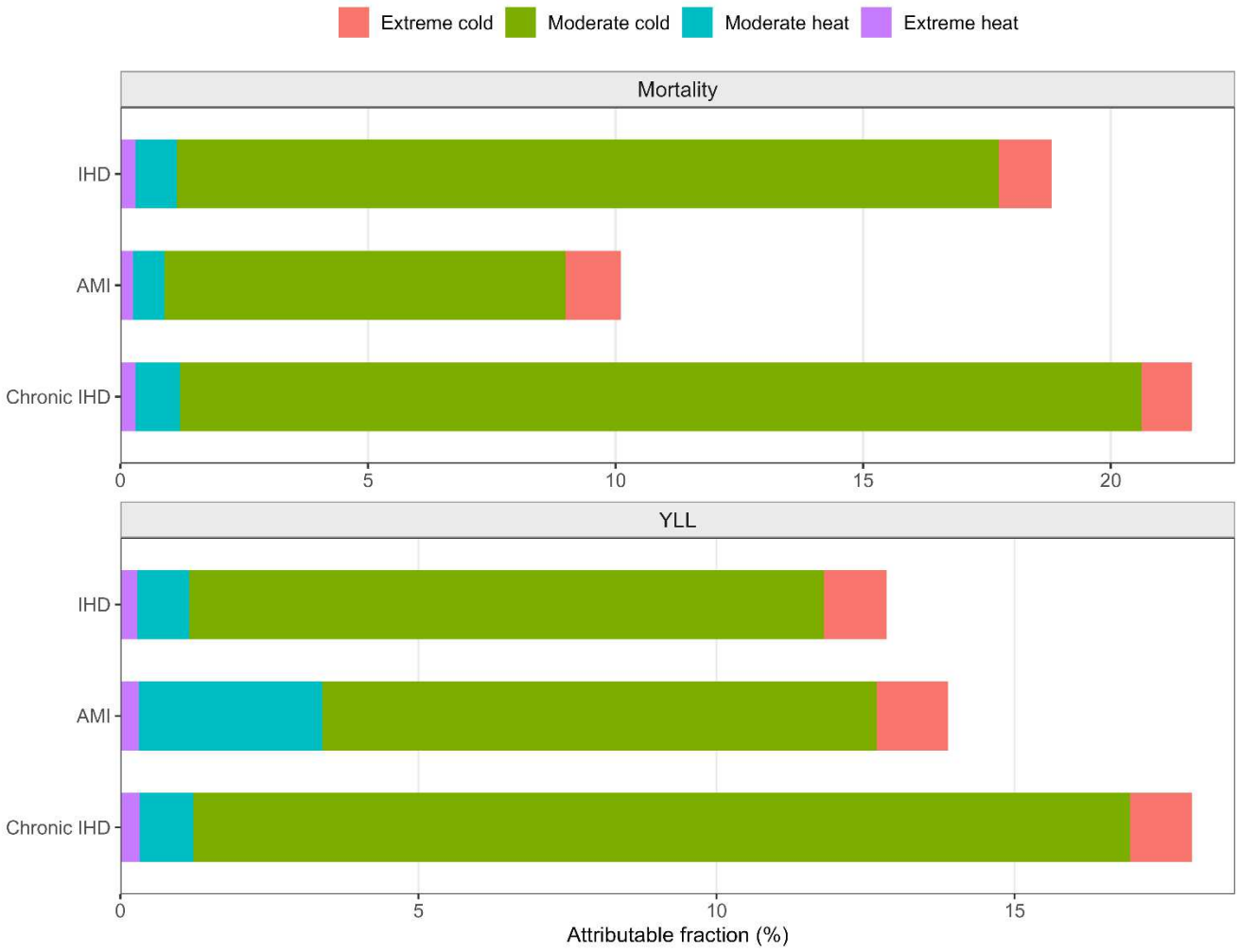


Fig. 4. Temperature-attributable fractions of mortality and years of life lost across ischemic heart disease subtype

Supplementary Files

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