

Heat-related mortality by comorbidity and cause of death: a time-stratified case-crossover study of 3.7 million deaths in Brazil, 2014–2025

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Research Article

Keywords: Hot Temperature, Mortality, Dementia, Drowning, External Causes, Cross-Over Studies

Posted Date: October 22nd, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-11455254/v1>

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Additional Declarations: The authors declare no competing interests.

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Abstract

The literature on heat vulnerability has emphasised cardiometabolic conditions — obesity, diabetes and cardiovascular disease. We evaluated these candidates and alternatives within a single design, over 3,672,003 deaths from the Mortality Information System (12 Brazilian states, January to May, 2014–2025), using a time-stratified case-crossover analysis, defining case days as those in the top decile of daily maximum temperature within each month, based on measurements from the 318 automatic weather stations located in those states, with lags of 0 to 3 days. Comorbidity was obtained from all lines of the death certificate. Two findings stood out, each within its own analytical category and in a different quantity, not on a common scale: effect modification among the comorbidities, and cause-specific relative risk among the external causes. **Dementia** showed the strongest evidence of greater heat susceptibility among the ten conditions tested: an effect-modification ratio of 1.044 (95%CI 1.029–1.058; $p = 1.1 \times 10^{-9}$), and 1.035 (1.020–1.050) in an analysis stratified by ten-year age bands among individuals aged 65 years or older; Alzheimer's disease as the underlying cause had a relative risk of 1.093 (1.073–1.114), the highest among the specific natural causes examined. **Drowning** was the largest external-cause effect: 1.161 (1.117–1.208; $p = 8.8 \times 10^{-14}$), with a gradient across all deciles analysed, from 0.798 in the coldest decile of the month to 1.256 in the hottest, and a log-linear trend of 1.046 per decile ($p = 1.4 \times 10^{-45}$), peaking on the hot day itself. Adjustment for precipitation did not change the estimate (1.154; 1.104–1.206). Most conventional cardiometabolic conditions showed little or no evidence of greater susceptibility: in the primary analysis, obesity (1.000; 0.976–1.024) and diabetes (1.003; 0.995–1.011) had estimates close to the null, in a study with 80% power, at $\alpha = 0.05$, to detect modifications of 3.4% and 1.1% or greater. Because deaths without a given condition differ in cause composition from deaths with it, each condition was also compared with deaths carrying another of the studied conditions: the dementia estimate strengthened to 1.050 (1.036–1.065), and cerebrovascular disease reached 1.018 (1.009–1.027), a smaller but statistically robust positive modification. The ICD-10 code for exposure to excessive natural heat (X30) was recorded as the underlying cause in only ten deaths, and anywhere on the death certificate in twelve, among 3.7 million. The patterns observed suggest that behavioural mechanisms may contribute to heat vulnerability alongside the physiological mechanisms traditionally considered.

Keywords: Hot Temperature; Mortality; Dementia; Drowning; External Causes; Cross-Over Studies.

Introduction

High temperatures and extreme heat episodes are associated with increased mortality, and identifying vulnerable groups is essential for translating impact estimates into public health action. The heat vulnerability literature has strongly emphasised advanced age and cardiometabolic and respiratory conditions, frequently interpreted through mechanisms of thermoregulation and physiological reserve¹ — impaired heat dissipation, dehydration, circulatory overload, drugs that interfere with thermal control.

This emphasis has two rarely examined limitations. The first is that it is almost always derived from aggregated underlying-cause series, in which comorbidity is not observed. The second is that these interpretations are rarely tested against alternative mechanisms: excess is measured in cardiorespiratory causes and the cardiorespiratory mechanism is inferred, without evaluating competing mechanisms within the same analytical framework.

This work compares these candidates within a single design. One design, one exposure definition, and the same estimator are applied to three sets of questions: (i) **effect modification** by ten conditions recorded on any line of the death certificate; (ii) **effect by chapter and by specific cause**, including the entire external-causes chapter broken down into the seventeen groups with enough deaths for estimation; and (iii) **internal structure** of the positive findings — the temperature–response relationship across the whole temperature distribution of the month, and day-by-day lag.

The question that organises the text is not whether there is an association between heat and mortality, already widely documented, but rather **which patterns of heat vulnerability a mortality database is able to distinguish**.

Methods

Design

The design is equivalent to a time-stratified case-crossover, in which comparisons are made within strata defined by state, year, month and day of the week. Implementation is by daily counts aggregated within these strata, with conditional Poisson likelihood. Since no comparison crosses state, month or day of the week, fixed characteristics of the state, between-month seasonality, secular trend and coding drift across years are controlled by design.

A lag window of 0 to 3 days was adopted on the basis of previous multicountry evidence that the association between heat and mortality appears acutely and lasts three to four days.²

Mortality data

We obtained microdata from the Brazilian Mortality Information System³ for 12 states (federative units: AL, BA, CE, MG, MS, PI, PR, RJ, RN, RR, RS, SC), based on state of residence, covering January through May from 2014 to 2025: **3,672,003 records**, of which 3,275,741 were from natural causes and 396,262 from chapter XX. Data for 2025 are preliminary and are identified as such.

Comorbidity ascertainment from multiple causes of death

The ten conditions examined are those the heat-vulnerability literature most often names as candidates — the cardiometabolic group and chronic kidney disease¹ — together with dementia as the neurological alternative that motivated the comparison, and neoplasm as an internal comparator. No condition was dropped after the estimates were seen: all ten are reported in Table 2. The same applies to the two cause-based analyses, which were not screened selectively — all sixteen ICD-10 chapters appear in Table 1, and all seventeen external-cause groups with enough deaths for estimation appear in Table 4.

Each condition was considered present when the corresponding ICD-10 code appeared **either as the underlying cause of death or anywhere among the multiple causes recorded on the death certificate** (lines A to D and part II), following the multiple-cause-of-death approach.⁴ Codes: obesity

E65–E68; diabetes E10–E14; cardiovascular disease I00–I99, with the subgroups ischaemic I20–I25, cerebrovascular I60–I69 and hypertensive I10–I15; heart failure I50; dementia F00–F03 and G30; neoplasm C00–C97; chronic kidney disease N18–N19.

Code X30, exposure to excessive natural heat, was counted in both ways: as underlying cause and on any line of the certificate, under the same multiple-cause rule applied to the comorbidities.

Heat exposure

Hourly data from the network of automatic weather stations of the National Institute of Meteorology,⁵ 2014 to 2025; 318 of these stations lie in the 12 states analysed and are the ones that enter the state means. The daily maximum of each station underwent quality control: an hourly reading is accepted only if it does not exceed by more than 6 °C the highest dry-bulb value between the same hour and the preceding one. Across the full national extraction the criterion rejected 1,105 of 46.4 million readings (0.0024%); within the states and months analysed, 172 of 11.1 million.

A case day is one in the **top decile of the state-level mean daily maximum temperature within the same month**, by state, with lags of 0 to 3 days, across the 699 state-months that have at least 20 days of valid temperature data, of the 720 in the design; the 21 excluded are gaps in the series for Rio Grande do Norte and Roraima. The definition is relative to the month, and therefore measures the response to comparatively hot days, not to an absolute threshold. Exposure does not depend on the episode having been officially declared.

Precipitation analysis

Hourly precipitation was extracted from the same stations, summed by station and local day and averaged by state: 115,102 state-days. The threshold separating dry from rainy days is the **median of state precipitation computed among the days classified as hot** (0.46 mm), then applied to all days: the median was taken among hot days in order to produce precipitation strata with comparable sample support precisely within the exposure set of interest. The sensitivity of the estimate to more conventional meteorological thresholds (1 mm, 5 mm, three bands and quartiles) is reported in the Results.

Two analyses, with different denominators and different purposes:

Adjustment. The precipitation category enters the stratum definition, which becomes state × year × month × day of the week × rain category. The hot-versus-non-hot comparison therefore occurs within days of comparable precipitation, and the resulting estimate is the effect of temperature **adjusted for rain**.

Effect modification. The same contrast is estimated separately within dry days and within rainy days — hot dry day versus non-hot dry day, and hot rainy day versus non-hot rainy day — and the ratio between the two is reported. Also reported, as a reference, is the contrast between rainy and dry days restricted to days of intermediate temperature (deciles 4 to 7).

Estimated quantities

For specific causes, we estimated the **relative risk** between case days and control days. For comorbidity analyses, the quantity of interest is not the RR among decedents with the condition, since this RR may increase simply because overall mortality increases, but rather the ratio between the hot-day association in decedents with and without the condition recorded:

effect-modification ratio = $RR(\text{with the condition}) / RR(\text{without the condition})$

The two groups share the same days and the same strata, so that temporal components common to both tend to cancel out in the ratio, although differential within-month trends — ones that would differentially affect the prevalence of the condition among deaths and the probability of a hot day — are not necessarily controlled. The comparison by comorbidity is restricted to natural causes, because comorbidities are mentioned in 0.3% to 5.1% of external-cause deaths against 10.8% of external causes overall, which would enrich the comparison group with external causes and shift every effect-modification ratio downwards.

Within natural causes the group without a given condition is still not homogeneous in cause composition: it is enriched for causes with high heat relative risks. After observing differences in cause composition between the comparison groups, a post hoc **homogeneous-comparator** analysis was performed, in which each condition is compared only with deaths carrying at least one of the other nine studied conditions. The primary definition — with the condition against without it — is retained, because it is the quantity a reader expects; the homogeneous comparator shows how much of each estimate depends on the composition of the comparison group.

Structure tests

Two tests that the relative risk alone does not provide:

Temperature–response pattern. Each day of the month receives its temperature decile by station (1 = coldest, 10 = hottest). **A separate contrast is then estimated for each decile of interest:** each contrast includes only the decile being evaluated, as exposure, and deciles 4 to 7, as reference; all other deciles are removed from the stratum. Thus, each decile-specific estimate is obtained independently against the same intermediate reference band. These analyses used lag 0, so that each day was classified according to its own temperature decile. A gradient across the distribution would reinforce the interpretation of an association with temperature, whereas an effect restricted to the cut-point would be more consistent with sensitivity to the threshold definition.

Isolated lag. For each k , case days are exactly the hot days plus k , and the other lag days are removed from the stratum. This separates the same-day response from the cumulative response. The primary analysis examined lags 0 to 3; lags 4 to 7 were examined additionally, to look for the compensatory deficit that short-term mortality displacement would produce. Because a lagged estimate cannot be read without knowing the temperature of the lagged day itself, the mean temperature decile at each lag, and of the control days, is reported alongside.

Trend. The categorical presentation of the deciles describes the ordering but does not test it. A log-linear trend in the decile was also tested, without reference aggregation: within each stratum, under the null hypothesis deaths are distributed uniformly across days, and with x_d the decile of the day the score statistic $T = \sum n_d \cdot x_d$ has expectation $\sum N_s \cdot \text{mean}_s(x)$ and variance $\sum N_s \cdot \text{var}_s(x)$. The effect size is $\exp(\beta)$, with β the solution of $\sum_s [T_s - N_s \cdot \sum x e^{\beta x}] / \sum e^{\beta x} = 0$, and reads as a relative risk **per decile** of temperature. For effect modification, the ratio between the trends of the two groups is reported as an effect-modification ratio per decile.

Estimated excess

The expected number under the null hypothesis is $E(1) = \sum_s N_s \cdot E_s / (E_s + U_s)$, with N_s the total deaths of stratum s , E_s the case days and U_s the control days; the observed excess is $A - E(1)$, with A the deaths on case days.

For a condition that modifies the effect, the **excess associated with the estimated effect modification** is $A - E(\theta_0)$, with θ_0 the relative risk estimated in the group without the condition and $E(\theta) = \sum_s N_s \cdot \theta E_s / (\theta E_s + U_s)$. These are the deaths above what would be expected had the observed effect equalled the one estimated among deaths without the condition, and the number is necessarily smaller than the total excess, which includes the portion common to both groups.

Both quantities are **estimated excesses under a relative definition of hot day**, and not counts of deaths causally attributable to heat.

Internal comparators and sensitivity analyses

Internal comparator: neoplasms, generally characterised by a more prolonged clinical trajectory and with no specific expected mechanism of acute increase in heat susceptibility; an effect-modification ratio close to or below 1 is expected. **Direction contrast:** the coldest decile of the month, where the effect should reverse or disappear. Neither is a negative control in the formal sense — people with neoplasms may be vulnerable to heat, and cold has its own effect on mortality — and they are used as internal comparators. **Null comparator:** congenital malformations, for which no acute response to ambient temperature is expected.

Holiday control. Five intervals were removed from the stratum: Carnival, from two days before Carnival Saturday to two days after Ash Wednesday (nine days); Holy Week, from Maundy Thursday to the following Thursday (eight days); and New Year, 21 April and 1 May, each with the same lag of 0 to 3 days applied to the exposure (four days each). That is 29 calendar days by construction; in practice the annual total ranges from 25 to 29, a mean of 27.9 of the 151 days from January to May, because in years when Easter falls on 20 or 21 April the Holy Week window overlaps with that of 21 April.

Robustness analyses

Death-certificate completeness. If certificates completed on hot days carried fewer codes, every effect-modification ratio would be displaced downwards for reasons that have nothing to do with heat. The number of distinct ICD-10 codes was therefore counted for every natural-cause death, and the same contrast applied to the total code count and to the death count; the ratio between the two is the change in codes per certificate on hot days.

Within-month trend. A time-stratified case-crossover is unbiased when exposure has no trend within the stratum. Temperature does trend within a calendar month, so three narrower stratifications were run with the exposure definition unchanged: half-month and ten-day blocks crossed with day of the week, and a bidirectional matched design in which each case day is compared only with the non-exposed days at ± 7 and ± 14 days, which share its day of the week by construction.

Place of occurrence. Deaths were classified by place of occurrence — health service, home, public place or other — and the same contrast applied, as an exploratory examination of whether the circumstances surrounding death modify the association.

Finer exposure scale. To test the state-level exposure directly, each death was reassigned to the automatic station nearest the seat of its municipality of residence, within 50 km and within 100 km — a finer unit than the microregion. The same analysis was repeated with the municipality of occurrence in place of residence, which addresses the concern that a drowning may happen away from home.

Estimation

Conditional Poisson likelihood,⁶ with score-based p values. Overdispersion was assessed as the Pearson χ^2 of the fitted conditional model over its degrees of freedom, with cells defined by state and date within each stratum; where ϕ exceeded 1, standard errors were scaled by $\sqrt{\phi}$ and the resulting confidence intervals, p values and RRmin are reported alongside the unscaled ones. Alongside each null result, the smallest **detectable** modification with 80% power and $\alpha = 0.05$ (RRmin) is reported — it is a measure of the study's power under the specifications used, and not an equivalence bound nor the limit of the confidence interval. Analyses in Python 3.10. All numbers in the manuscript are generated by `cc_manuscrito.py` from the panels, with no manual transfer.

Ethical aspects

Public, de-identified microdata from the Mortality Information System were used, of unrestricted access in the repository of the Department of Informatics of the Unified Health System and without variables allowing direct identification of individuals. As this is publicly accessible and public domain information, aggregated and without the possibility of individual identification, the research does not fall among those that must be registered and evaluated by the CEP/CONEP system, under items II, III and V of the sole paragraph of article 1 of National Health Council Resolution no. 510/2016, observing the general principles of National Health Council Resolution no. 466/2012.

Results

1. Circulatory and respiratory causes do not rank among the largest chapter-level associations

Table 1. Relative risk on hot days, by ICD-10 chapter, all causes.

chapter	deaths on case days	RR	95%CI	p
VI Nervous system (G00–G99)	32,885	1.072	1.057–1.086	1.5×10^{-23}
I Infectious and parasitic (A00–B99)	93,566	1.052	1.043–1.060	2.6×10^{-35}
XII Skin and subcutaneous tissue (L00–L99)	5,304	1.046	1.012–1.082	0.0084
XX External causes (V01–Y98)	109,151	1.046	1.038–1.053	7.3×10^{-32}
XVIII Symptoms and ill-defined findings	59,167	1.045	1.034–1.055	1.3×10^{-17}
XIII Musculoskeletal and connective tissue	4,370	1.038	1.000–1.077	0.048
IV Endocrine and metabolic (E00–E90)	59,063	1.030	1.019–1.040	1.5×10^{-8}
X Respiratory system (J00–J99)	106,892	1.027	1.020–1.035	1.8×10^{-12}
V Mental disorders (F00–F99)	11,739	1.027	1.004–1.051	0.019
XIV Genitourinary system (N00–N99)	32,029	1.021	1.007–1.035	0.0032
IX Circulatory system (I00–I99)	245,927	1.018	1.013–1.023	1.1×10^{-12}
II Neoplasms (C00–D48)	163,610	1.009	1.002–1.015	0.006
XVI Perinatal (P00–P96)	12,830	1.001	0.980–1.023	0.93

XI Digestive system (K00–K93)	45,691	0.998	0.987–1.010	0.79
III Blood and blood-forming organs	5,059	0.990	0.957–1.025	0.57
XVII Congenital malformations (Q00–Q99)	6,694	0.985	0.957–1.015	0.33

With this sample size, even associations of small magnitude reached statistical significance; hence the magnitude and the ranking of the effects are particularly informative. Circulatory and respiratory causes, which feature prominently in the heat–mortality literature,¹ ranked in the **lower half** of the table, with 1.018 and 1.027. Higher estimates were observed for the nervous system (1.072) and external causes (1.046). This ranking guided the rest of the analysis.

2. The comorbidities, ordered by strength of evidence

Table 2. Effect modification by recorded condition, natural causes, all ages. EM ratio = RR with the condition / RR without the condition.

condition	deaths on case days	RR with	RR without	EM ratio	95%CI	p	RRmin
dementia	32,898	1.067	1.022	1.044	1.029–1.058	1.1×10^{-9}	1.020
cerebrovascular	95,924	1.034	1.023	1.011	1.003–1.020	0.010	1.012
diabetes	108,903	1.027	1.024	1.003	0.995–1.011	0.46	1.011
obesity	10,618	1.024	1.024	1.000	0.976–1.024	0.99	1.034
hypertensive disease	203,867	1.021	1.025	0.996	0.990–1.002	0.24	1.009
chronic kidney disease	50,030	1.016	1.025	0.991	0.980–1.002	0.13	1.016
heart failure	74,442	1.012	1.025	0.988	0.978–0.997	0.0083	1.013
cardiovascular (I00–I99)	407,692	1.020	1.028	0.992	0.987–0.997	0.0019	1.007
neoplasm (internal comparator)	171,807	1.010	1.028	0.982	0.976–0.989	9.5×10^{-8}	1.009
ischaemic heart disease	95,360	1.006	1.026	0.980	0.972–0.989	3.6×10^{-6}	1.012

Six of the ten conditions reached significance at 5%, and what matters is not which ones did, but in which direction and with what magnitude. **Two estimates were above 1.** Dementia, at 1.044, was the largest in magnitude and the only estimate **above 1** to survive Bonferroni correction for ten conditions ($\alpha = 0.005$). **Cerebrovascular disease** was also above 1, but at 1.011 (1.003–1.020) and $p = 0.010$: the estimated modification was substantially smaller than that observed for dementia, and did not survive the correction. This may warrant attention, but does not provide robust evidence after correction for multiple testing.

Four estimates were below 1; three survived Bonferroni correction: ischaemic heart disease (0.980), neoplasm (0.982) and cardiovascular disease as a whole (0.992). Heart failure also showed an estimate below 1 (0.988), but did not survive the correction. Neoplasm, the internal comparator, showed the expected inverse direction, and the remaining inverse estimates were similar.

In the remaining four there was no statistical evidence of effect modification, and the study's statistical power makes these null estimates informative. Obesity gave exactly 1.000 in a study with 80% power to detect an aggravation of 3.4% or greater; diabetes gave 1.003, with power to detect 1.1% or more; hypertensive disease 0.996, with power for 0.9%; chronic kidney disease 0.991, with power for 1.6%. **None showed robust evidence of greater susceptibility after accounting for multiplicity**, and low statistical power alone is unlikely to explain the absence of effects of these magnitudes at $\alpha = 0.05$.

When the analysis was restricted to 65 years or older, dementia retained 1.041 (1.026–1.055; $p = 2.5 \times 10^{-8}$), which makes it unlikely that the finding is explained by differences between individuals aged under 65 years and those aged 65 years or older — 98.2% of deaths with recorded dementia fall in that band. The restriction, however, **did not fix age within the group**: the proportion of deaths with recorded dementia ranged from 1.6% between 65 and 74 years to 5.4% between 75 and 84 and 9.5% above 85. The effect-modification ratio was then estimated within ten-year bands.

Table 2b. Effect-modification ratio for dementia within narrow age bands, natural causes.

band	deaths on case days	RR with	RR without	EM ratio	95%CI	p	RRmin
65–74	3,116	1.044	1.016	1.028	0.983–1.074	0.22	1.065
75–84	11,928	1.062	1.022	1.039	1.015–1.063	0.0011	1.034
85+	17,244	1.075	1.040	1.033	1.013–1.054	0.0011	1.029
pooled within band	32,288	—	—	1.035	1.020–1.050	2.2×10^{-6}	—

Stratification by 10-year age bands **attenuated the estimate from 1.044 to 1.035** — part of the crude effect-modification ratio is explained by the age distribution — but did not eliminate it, and the point estimates were similar across the three bands ($I^2 = 0\%$). The 65 to 74 band, with 3,116 deaths, did not reach significance on its own and did not contradict the others.

The comparison group of Table 2 is not neutral in cause composition. Deaths carrying none of the ten conditions had a higher hot-day relative risk (1.035; 1.031–1.040) than deaths carrying at least one (1.019; 1.016–1.022), a ratio of 0.984 (0.979–0.990; $p = 2.1 \times 10^{-8}$). This is composition and not certification: the number of codes per certificate did not change on hot days (section 8). Table 2c repeats every estimate against a comparator matched on that composition.

Table 2c. Effect modification under a homogeneous comparator: each condition against deaths carrying at least one of the other nine studied conditions, natural causes.

condition	deaths	primary	95%CI	homogeneous	95%CI	p
dementia	32,898	1.044	1.029–1.058	1.050	1.036–1.065	5.5×10^{-12}
cerebrovascular	95,924	1.011	1.003–1.020	1.018	1.009–1.027	6.1×10^{-5}
diabetes	108,903	1.003	0.995–1.011	1.009	1.001–1.018	0.025
obesity	10,618	1.000	0.976–1.024	1.005	0.981–1.029	0.68
hypertensive disease	203,867	0.996	0.990–1.002	1.003	0.997–1.010	0.32
chronic kidney disease	50,030	0.991	0.980–1.002	0.997	0.985–1.008	0.55
heart failure	74,442	0.988	0.978–0.997	0.993	0.983–1.002	0.13
cardiovascular (I00–I99)	407,692	0.992	0.987–0.997	1.002	0.995–1.009	0.59
neoplasm (internal comparator)	171,807	0.982	0.976–0.989	0.987	0.980–0.994	2.3×10^{-4}
ischaemic heart disease	95,360	0.980	0.972–0.989	0.985	0.977–0.994	6.5×10^{-4}

Dementia strengthens, from 1.044 to **1.050 (1.036–1.065; $p = 5.5 \times 10^{-12}$)**: the principal finding does not depend on the choice of comparison group. Cerebrovascular disease moves from 1.011 to **1.018 (1.009–1.027; $p = 6.1 \times 10^{-5}$)** and survives Bonferroni correction for ten conditions. Three of the four estimates below 1 do not survive the change — cardiovascular disease as a whole moves to 1.002 and heart failure to 0.993, both null — and only neoplasm (0.987) and ischaemic heart disease

(0.985) remain below 1. Diabetes gives a small positive estimate (1.009; 1.001–1.018) that does not survive correction. Obesity is null under both comparators.

3. Dementia: structure of the association

Table 3. Effect-modification ratio for dementia by temperature decile of the same month (reference: deciles 4 to 7, lag 0) and by isolated lag.

decile	deaths	RR with	RR without	EM ratio	95%CI	p
D10 (hottest)	10,214	1.068	1.036	1.031	1.004–1.058	0.024
D9	10,356	1.061	1.026	1.035	1.008–1.062	0.012
D8	10,472	1.084	1.021	1.061	1.034–1.090	1.0×10^{-5}
D3	9,926	1.003	0.995	1.009	0.982–1.036	0.53
D2	9,889	0.976	0.991	0.985	0.959–1.012	0.27
D1 (coldest)	11,847	0.976	0.984	0.991	0.968–1.016	0.48

lag	deaths	EM ratio	95%CI	p
lag 0	12,407	1.031	1.009–1.053	0.0052
lag 1	12,175	1.060	1.037–1.083	1.5×10^{-7}
lag 2	11,667	1.047	1.024–1.070	5.2×10^{-5}
lag 3	11,004	1.035	1.012–1.059	0.0028

The categorical pattern was more of a **step than a gradient**: no consistent evidence of greater susceptibility in the cold half of the month (0.99 across the three coldest deciles), whereas effect-modification estimates were consistently above 1 across the three hottest deciles (1.03–1.06), without rising from the eighth to the tenth decile. The log-linear trend test applied to the ratio between the two groups was nevertheless significant — **1.005 per decile (1.003–1.007; $p = 2.8 \times 10^{-6}$)** — so that the two interpretations coexist: there was a trend along the scale, but almost all of the contrast lay between the cold half and the hot half, rather than within the hot half. The **association remained statistically detectable across all four lags examined**, with the peak at lag 1 under state-level exposure. The position of the peak within the first two days is not stable: when exposure is assigned by the station nearest the municipal seat, the peak moves to lag 0 (1.097 against 1.086 at lag 1), with almost entirely overlapping intervals (Table S6). What the data support is that the dementia association is spread across the four days, not that it is delayed by exactly one. Extending the isolated-lag analysis beyond the primary analysis window found no compensatory deficit: the dementia relative risk was 1.051 at lag 4, 1.021 at lag 5, 1.045 at lag 6 and 1.034 at lag 7, and the ratio to deaths without dementia stayed between 1.021 and 1.050 across those days (Table S4). Days 4 to 7 after a hot day are not warm — their mean temperature decile is 4.6 to 5.3, against 4.8 for the control days of that analysis and 5.5 for all days of the month — so the persistence is not explained by the temperature of the later days themselves.

The association was robust across all sensitivity analyses: without holidays 1.051; 95th percentile 1.045; 80th percentile 1.034; excluding 2020–2022 1.045; 2014–2019 only 1.052; 2023–2025 only 1.037. None fell below 1.03 or lost statistical significance.

Table 3b. Consistency with underlying-cause analyses: chapters V and VI broken down.

group	deaths on case days	RR	95%CI	p
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G30 Alzheimer's disease	16,849	1.093	1.073–1.114	2.8×10^{-20}
F00–F03 other dementias	2,160	1.065	1.010–1.123	0.020
G40–G41 epilepsy	2,608	1.057	1.007–1.108	0.024
rest of chapter VI	9,082	1.056	1.029–1.084	3.1×10^{-5}
G20 Parkinson's disease	3,373	1.046	1.003–1.091	0.036
G00–G09 infectious diseases of the nervous system	973	0.983	0.909–1.062	0.66
rest of chapter V (F, except dementia)	9,579	1.019	0.994–1.045	0.13

The chapter with the largest effect in Table 1 was the nervous system, and within it the largest effect was Alzheimer's disease, at 1.093 — the highest relative risk among the specific natural causes examined in these analyses. Mental disorders that are **not** dementia showed no statistically detectable association. The two analyses converged on the same condition through **two distinct operationalisations of the information recorded on the death certificate**. They do not constitute independent evidence — G30 is part of the very definition of dementia in the multiple-cause analysis — but one uses mention on any line and the other only the underlying cause, and the second does not depend on the number of additional conditions recorded on the remaining lines of the certificate.

4. Among external causes, drowning stands out

Table 4. Relative risk on hot days, chapter XX broken down into groups.

group	deaths on case days	RR	95%CI	p	RRmin
drowning (W65–W74)	4,010	1.161	1.117–1.208	8.8×10^{-14}	1.058
other forces of nature (X31–X39)	331	1.147	1.000–1.316	0.050	1.217
contact with animals and plants (X20–X29)	218	1.149	0.973–1.357	0.10	1.269
other accidents (X58–X59)	1,666	1.104	1.039–1.174	0.0014	1.091
electric current, temperature, pressure	1,146	1.088	1.011–1.170	0.024	1.110
complications of medical care (Y40–Y84)	1,422	1.081	1.012–1.154	0.020	1.098
mechanical forces (W20–W64)	956	1.077	0.995–1.166	0.066	1.120
sequelae of external causes (Y85–Y89)	393	1.077	0.951–1.219	0.24	1.194
fire and flames (X00–X19)	750	1.059	0.968–1.159	0.21	1.138
accidental poisoning (X40–X49)	684	1.056	0.961–1.160	0.26	1.144
assault (X85–Y09)	38,080	1.046	1.033–1.059	2.8×10^{-12}	1.018
suicide (X60–X84)	10,372	1.042	1.017–1.068	0.00074	1.035
transport (V01–V99)	24,019	1.041	1.024–1.057	7.5×10^{-7}	1.023
falls (W00–W19)	10,735	1.027	1.004–1.052	0.024	1.034
undetermined intent (Y10–Y34)	9,290	1.025	0.999–1.051	0.058	1.037
suffocation (W75–W84)	3,293	1.018	0.976–1.063	0.40	1.063
legal intervention (Y35–Y36)	1,774	0.992	0.936–1.051	0.78	1.086

Bonferroni correction for 17 groups requires $\alpha = 0.0029$, and five groups remained significant. Among them, **drowning had the highest relative risk (1.161), followed by other accidents (1.104); assault,**

suicide and transport had smaller effects, between 1.041 and 1.046. Three groups had high point estimates but did not survive the correction — other forces of nature (1.147), contact with animals and plants (1.149) and electric current, temperature and pressure (1.088) — all with fewer than 1,200 deaths on case days and wide confidence intervals.

ICD-10 code X30, "exposure to excessive natural heat", was recorded as the underlying cause in ten deaths and anywhere on the death certificate in twelve deaths among 3,672,003 records. This contrast illustrates how much of the mortality temporally associated with hot days is not explicitly coded as heat exposure on the death certificate.

5. Drowning: structure of the association

Table 5. Drowning by temperature decile of the same month and by isolated lag.

decile	deaths	RR	95%CI	p
D10 (hottest)	1,377	1.256	1.168–1.351	6.9×10^{-10}
D9	1,403	1.180	1.098–1.268	6.9×10^{-6}
D8	1,214	1.073	0.995–1.158	0.068
D4–D7	reference	1	—	—
D3	1,049	0.904	0.835–0.978	0.012
D2	1,040	0.902	0.834–0.977	0.011
D1 (coldest)	1,148	0.798	0.741–0.859	1.8×10^{-9}

lag	deaths	RR	95%CI	p
lag 0	1,725	1.341	1.266–1.422	3.2×10^{-23}
lag 1	1,448	1.158	1.088–1.232	3.8×10^{-6}
lag 2	1,331	1.110	1.041–1.184	0.0015
lag 3	1,219	1.051	0.983–1.124	0.14

The gradient was monotonic across the deciles analysed, from 0.798 to 1.256: on the coldest days of the month the relative risk of drowning was about 20% lower, and on the hottest days 26% higher, relative to deciles 4 to 7. The eighth decile, on its own, did not reach significance ($p = 0.068$), and deciles 4 to 7 were aggregated as reference; the formal log-linear trend test, which does not depend on that aggregation, yielded **1.046 per decile (1.039–1.052; $z = 14.2$; $p = 1.4 \times 10^{-45}$)**. The association was strongest **on the day itself** and was no longer statistically detectable by the third day (lag 3: 1.051; 0.983–1.124). Beyond the primary analysis window the estimate was 1.008 at lag 4, and at lags 5 to 7 the estimates fell below 1 — 0.956, 0.980 and 0.950. The lagged days were themselves cooler than average: at lag 7 the mean decile is 4.64 against 5.50 for all days of the month, and a trend of 1.046 per decile predicts 0.962 against the 0.950 observed (Table S4). These later deficits therefore do not provide clear evidence of mortality displacement.

Table 5b. Breakdown of drowning.

stratum	deaths	RR	95%CI	p
W69 natural water	2,042	1.151	1.089–1.217	5.5×10^{-7}
W74 unspecified	1,537	1.170	1.098–1.247	1.4×10^{-6}
W67 swimming pool	81	1.001	0.766–1.308	0.99
0–14 years	561	1.112	1.002–1.234	0.046

15–39 years	1,840	1.238	1.167–1.312	1.1×10^{-12}
40–59 years	1,110	1.145	1.062–1.233	0.00038
60–79 years	419	1.035	0.918–1.167	0.58
80+ years	49	0.915	0.648–1.292	0.62
male sex	3,530	1.162	1.114–1.212	2.2×10^{-12}
female sex	475	1.157	1.032–1.296	0.012
death between 12h and 17h	1,685	1.163	1.095–1.236	1.1×10^{-6}
death between 18h and 23h	608	1.225	1.107–1.356	8.9×10^{-5}
death between 06h and 11h	745	1.058	0.967–1.158	0.22
death between 00h and 05h	170	1.025	0.849–1.236	0.80
January	1,091	1.228	1.138–1.324	9.2×10^{-8}
February	961	1.181	1.089–1.281	5.9×10^{-5}
March	779	1.198	1.096–1.309	6.9×10^{-5}
April	695	1.062	0.967–1.167	0.21
May	484	1.080	0.964–1.209	0.18

Higher point estimates were observed for drownings in **natural waters than in swimming pools**, among younger than older age groups, during the afternoon and early evening rather than overnight, and from January through March rather than April or May; these are descriptive contrasts, not formal tests of heterogeneity. Without holidays the relative risk rose to 1.182; weekend (1.173) and weekday (1.153) estimates did not differ; every period subset reproduced the finding, including 2023–2025 (1.185).

In a specificity analysis, intentional drowning (X71) showed no similar pattern — 0.927 (0.747–1.150) — although the small number of events ($n = 122$) limits this comparison, which does not allow misclassification between the two categories to be ruled out.

Adjusting for precipitation did not change the estimate. With the rain category included in the stratum, so that the comparison occurred between days of similar precipitation, drowning gave **1.154 (1.104–1.206; $p = 2.2 \times 10^{-10}$)**, compared with 1.161 (1.117–1.208) in the unadjusted analysis. Adjustment for precipitation produced little evidence of material confounding at this exposure resolution.

Estimated separately within each category, the association was **1.040 (0.945–1.144)** on dry days and **1.187 (1.130–1.248)** on rainy days, a ratio of 0.876 (0.786–0.975; $p = 0.02$). **There was no evidence that the excess was greater on hot dry days**; if anything, the observed difference was in the opposite direction to that predicted by the fair-weather hypothesis. Estimates in the same direction for other outcomes analysed using the same contrast — nervous system 0.961 (0.928–0.996), falls 0.944 (0.887–1.003), transport 1.008 (0.967–1.050) — reduce the specificity of this finding for drowning and warrant caution in its interpretation.

The estimate ranged from 1.141 to 1.160 across different parameterisations of precipitation, against 1.161 without adjustment (Table S1).

As a reference, in the contrast restricted to days of intermediate temperature (deciles 4 to 7), rainy days showed **0.918 (0.830–1.014)** of drowning relative to dry days — an estimate in the direction of lower risk, with an interval that includes unity.

6. Null and inverse findings

These results are presented together because they help characterise the relative specificity of the main findings. The interpretation of a null result depends, among other aspects, on the power to detect effects of clinically relevant magnitude; the RR_{min} column states which effect would have been detected with 80% power, at $\alpha = 0.05$.

Table 6. Null and inverse findings, with the corresponding statistical power.

test	estimate	95%CI	would detect
obesity — effect modification	1.000	0.976–1.024	3.4%
obesity — modification in 65+	0.967	0.930–1.006	5.7%
diabetes — effect modification	1.003	0.995–1.011	1.1%
hypertensive disease — modification	0.996	0.990–1.002	0.9%
chronic kidney disease — modification	0.991	0.980–1.002	1.6%
cardiovascular disease — modification	0.992	0.987–0.997	0.7%
ischaemic heart disease — modification	0.980	0.972–0.989	1.2%
heart failure — modification	0.988	0.978–0.997	1.3%
neoplasm — modification (internal comparator)	0.982	0.976–0.989	0.9%
mental disorders except dementia — RR	1.019	0.994–1.045	3.6%
infectious diseases of the nervous system — RR	0.983	0.909–1.062	11.8%
suffocation — RR	1.018	0.976–1.063	6.3%
legal intervention — RR	0.992	0.936–1.051	8.6%
fire and flames — RR	1.059	0.968–1.159	13.8%
accidental poisoning — RR	1.056	0.961–1.160	14.4%
undetermined intent — RR	1.025	0.999–1.051	3.7%
swimming-pool drowning — RR	1.001	0.766–1.308	46.6%
suicide by drowning (X71) — RR	0.927	0.747–1.150	36.1%
coldest decile, drowning (direction contrast)	0.798	0.741–0.859	—
coldest decile, dementia (direction contrast)	0.991	0.968–1.016	—
digestive system — RR	0.998	0.987–1.010	1.6%
perinatal — RR	1.001	0.980–1.023	3.1%
congenital malformations — RR	0.985	0.957–1.015	4.3%

In the primary analysis, **obesity, diabetes, hypertensive disease and chronic kidney disease showed no evidence of greater effect modification**, in a study with 80% power, at $\alpha = 0.05$, to detect modifications of 3.4%, 1.1%, 0.9% and 1.6% or greater. Under the homogeneous comparator, diabetes showed a small positive estimate that did not survive correction for multiple testing, whereas obesity remained null (Table 2c). The low-power null findings — swimming pool, X71, fire — are identified as such and do not support any conclusion.

7. Estimated excess deaths

Over the 12 years and 12 states, from January to May:

- **Drowning: 4,010 deaths observed on case days against 3,647.6 expected, +362.**
- **Among decedents with recorded dementia: 32,898 deaths were observed on case days against 31,547.7 expected, +1,350 — of which +892 are deaths above what would be expected had the observed effect equalled the one estimated among deaths without dementia, by the formula A**

– $E(\theta_0)$ given in the Methods. This is the excess associated with the estimated effect-modification ratio.

Both values are **estimated excesses under a relative definition of hot day**, conditional on the design, and not counts of deaths causally attributable to heat. For drowning in particular, residual confounding by precipitation remains possible, given the state-level and daily resolution of the measure, and above all by mobility and behavioural exposure, as discussed below. No compensatory deficit was observed through lag 7 for dementia. For drowning, later estimates below 1 coincided with cooler lagged days and were compatible with the observed temperature–response relationship, providing no clear evidence of short-term mortality displacement (Table S4).

8. Robustness of the two main findings

Death-certificate completeness does not change with temperature. Natural-cause deaths rose 2.41% on case days (1.0241; 1.0214–1.0267) and the total number of ICD-10 codes recorded rose 2.44% (1.0244; 1.0230–1.0259), a ratio of **1.0004 (0.9974–1.0033; $p = 0.82$)**. Certificates completed on hot days carry the same amount of information as those completed on control days, which removes the most direct artefactual explanation for a uniform displacement of the effect-modification ratios.

Overdispersion is present and small. ϕ was 1.03 for the nervous system chapter, 1.06 for dementia, 1.10 for the circulatory chapter, 1.13 for drowning and 1.29 for the external-causes chapter. The largest estimate, $\phi = 1.54$, was observed for all natural-cause mortality; scaling by ϕ did not materially alter the corresponding inference. Scaling standard errors by $\sqrt{\phi}$ leaves every interval and every conclusion intact: the dementia effect-modification ratio goes from 1.029–1.058 to 1.029–1.059, drowning from 1.117–1.208 to 1.114–1.211, and the RRmin of the null conditions rises in the third decimal place — obesity from 1.034 to 1.035, diabetes from 1.011 to 1.012 (Table S2).

Neither finding depends on within-month trend. Under bidirectional matching, in which each case day is compared only with the non-exposed days at ± 7 and ± 14 , drowning gives 1.149 (1.108–1.192) against the published 1.161, and the dementia effect-modification ratio gives 1.042 (1.029–1.056) against 1.044, on more informative strata than the primary design (5,150 against 3,938). Half-month strata reproduce both. Ten-day strata attenuate both — drowning 1.112, dementia 1.027 — but retain only half the drowning deaths, so that attenuation mixes loss of information with control of trend (Table S3). The circulatory chapter moves in the opposite direction, from 1.018 to 1.023 (1.019–1.028) under matching, so the published estimate for that chapter is if anything slightly attenuated by within-month trend; the ordering of Table 1 is unchanged.

A finer exposure scale does not raise the estimates. Reassigning each death to the automatic station nearest the seat of its municipality left the ranking and the statistical significance of every finding unchanged, while lowering each estimate slightly: drowning from 1.161 to 1.131 within 50 km and 1.123 within 100 km, Alzheimer's disease from 1.093 to 1.064, and the dementia effect-modification ratio from 1.044 to 1.037. Congenital malformations, used as a null comparator, remained compatible with 1 at every scale (0.985, 1.019, 1.010), and repeating the analysis by municipality of occurrence instead of residence moved drowning in the third decimal place (Table S6).

Place of occurrence. Heat-associated mortality was greater for deaths occurring in public places or other non-healthcare settings (1.056; 1.039–1.073) than for deaths occurring in health services

(1.022; 1.019–1.025) — a ratio of 1.031 (1.014–1.048; $p = 2.5 \times 10^{-4}$) relative to all natural causes — with deaths at home between the two (1.030; 1.024–1.036). This analysis is exploratory.

Discussion

The two main signals are consistent with distinct behavioural mechanisms

Dementia and drowning appear unrelated. Both, however, show patterns consistent with the importance of behaviour in the face of heat. The caveat applies from the outset: this design identifies associations and temporal signatures, and does not measure behaviour, hydration, thermoregulation, cognition or exposure to water.

For dementia, the pattern is consistent with impaired behavioural self-regulation — recognising the need to drink, seeking shade, removing clothing, asking for help — and with the excess of hospitalisations and post-discharge deaths from Alzheimer's disease described during heatwaves.⁷ A pharmacological contribution is plausible for anticholinergics, whose interference with thermoregulation is documented in meta-analysis; for antipsychotics, the same review found no evidence.⁸ The observed pattern suggests that impaired cardiovascular reserve alone is unlikely to provide a sufficient explanation: heart failure, a condition in which limited cardiovascular reserve would be particularly plausible, did not show greater susceptibility, whereas dementia did.

For drowning, the pattern observed is particularly consistent with a **behavioural increase in exposure to water** on hot days⁹: young adults, in the afternoon and early evening, in natural waters and not in swimming pools. Physiological mechanisms that may contribute to drowning on a hot day — dehydration, syncope, fatigue, interaction with alcohol — are not measured by this database and are not excluded. Precipitation, by contrast, was measured, and adjusting for it did not materially change the finding (Results).

The contrast becomes explicit in the temporal signatures, which this design separates. **Drowning peaked sharply on the hot day itself (1.341) and was no longer statistically detectable by the third day; for dementia the association was spread across all four lags examined, with the peak in the first two days but its exact position not stable across exposure scales.** Extending the isolated-lag analysis beyond the primary analysis window sharpens both readings. No compensatory mortality deficit was observed through lag 7 for dementia. For drowning, the estimates below 1 at lags 5 to 7 coincided with lagged days that were themselves cooler than average, and were close to what the temperature–response relationship predicts for such days, so they do not provide clear evidence of mortality displacement. For dementia, the association remained elevated through lag 7, even when temperatures on those later days had returned to approximately the level of control days; we note the persistence without interpreting it further, since it could reflect the original exposure, correlated intermediate exposures, or meteorological structure that the decile of a single day does not capture. The temporal patterns are consistent with distinct processes: for drowning, a predominantly immediate response to exposure; for dementia, an association spread over several days rather than concentrated on one. A behavioural interpretation thus offers an integrating hypothesis for two findings with distinct epidemiological signatures, without excluding concomitant physiological mechanisms.

What can be inferred from the null and inverse findings

Interpreted in isolation, the two main signals could represent findings arising from multiple comparisons. What reinforces the interpretation of the two main signals is their relative specificity within a set of conditions analysed **in the same design, on the same days, with the same estimator** and with declared power, together with the structure by temperature and lag and the sensitivity analyses. Both main findings were reproduced under bidirectional matching at ± 7 and ± 14 days, reducing concern that within-month temperature trends generated the associations.

For the four null estimates in Table 2, the study had 80% power to detect effect modifications ranging from 0.9% to 3.4%, and the estimate for diabetes is particularly informative regarding the absence of modification of relevant magnitude: 1.003 over 108,903 deaths, with 80% power to detect 1.1% at $\alpha = 0.05$. Lack of statistical power alone is therefore unlikely to explain the absence of a modification of that magnitude, although misclassification arising from death-certificate ascertainment remains possible.

The composition of the comparison group matters, and it was examined rather than assumed. Deaths carrying none of the ten conditions are enriched for causes with high heat relative risks, and their hot-day relative risk (1.035) exceeds that of deaths carrying at least one (1.019). This is not a difference in how the certificate was completed: the number of codes per certificate did not change on hot days (1.0004; 0.997–1.003). Under a comparator matched on that composition the dementia estimate strengthened, from 1.044 to 1.050, so the principal finding does not depend on the choice of comparison group. Three of the four estimates below 1 do not survive the same change — cardiovascular disease as a whole moves to 1.002 and heart failure to 0.993 — and only neoplasm (0.987) and ischaemic heart disease (0.985) remain below 1. The reading that conditions with a more predictable clinical course and a larger component of in-hospital mortality are systematically less sensitive to acute displacement is therefore weaker than Table 2 alone suggests, and we do not press it.

Two estimates move upwards under the homogeneous comparator. **Cerebrovascular disease** goes from 1.011 ($p = 0.010$) to 1.018 (1.009–1.027; $p = 6.1 \times 10^{-5}$), surviving Bonferroni correction for ten conditions: a smaller but statistically robust positive modification, and the one conventional cardiometabolic candidate for which the evidence does point to greater susceptibility. Diabetes shows a small positive estimate under the homogeneous comparator (1.009; 1.001–1.018), which did not survive correction for multiple testing. Obesity remains null under both comparators (1.000 and 1.005; 0.981–1.029).

This does not say that obesity and diabetes are not heat vulnerability factors. It says that the **mention of these conditions on the death certificate does not identify a substantially more susceptible subgroup**, and that low statistical power alone is unlikely to explain the absence of effects of these magnitudes at $\alpha = 0.05$. For obesity, substantial under-ascertainment is evident: the condition appears on only 1.1% of certificates, a proportion incompatible with population prevalence, and misclassification is differential. For diabetes, which was recorded in 12.4% of natural-cause deaths, under-ascertainment provides a less obvious explanation.

In an exploratory analysis, heat-associated mortality was greater for deaths occurring in public places or other non-healthcare settings than for deaths occurring in health services — a pattern consistent with, but not sufficient to establish, the hypothesis that the circumstances surrounding death modify

acute heat-related risk. Place of occurrence is not a measure of clinical trajectory, and we read it no further than that.

Chapter-level results provide a consistent pattern

Circulatory and respiratory causes, which occupy a central position in the literature on heat-related mortality,¹ ranked in the lower half of Table 1: 1.018 and 1.027, against 1.072 for the nervous system and 1.046 for external causes. This does not imply an absence of association; rather, the magnitude was comparatively **small and non-specific**, close to the estimate for all natural causes.

And within the nervous system chapter, the association was concentrated in Alzheimer's disease (1.093), whereas mental disorders that are not dementia showed no statistically detectable association (1.019, $p = 0.13$), and neither did infectious diseases of the nervous system (0.983). The convergence between the analysis of recorded comorbidity and that of the underlying cause, which use different information from the same certificate, provides evidence of internal consistency of the work in favour of dementia.

Drowning: remaining sources of uncertainty

Drowning showed one of the most marked patterns of internal consistency in the study — a monotonic gradient across the deciles analysed, with a formal trend of 1.046 per decile, a peak at lag 0, and distinctive patterns by type of water body, age band and time of day. Even so, two sources of uncertainty remain.

The first is precipitation, which was explicitly examined. Drowning studies that model several meteorological factors simultaneously show that the respective contributions of temperature and precipitation cannot be distinguished from descriptive patterns alone.⁹ In these data the association is strong: in the coldest decile of each month the mean state precipitation was 9.4 mm and 84.4% of days lay above the adopted rain cut-off, against 1.8 mm and 49.8% in the hottest decile, with a monotonic gradient between the two extremes. Thus, the unadjusted contrast could partly reflect a comparison between hot dry days and cool rainy days rather than between hot and mild days. Precipitation from the same stations allowed this possibility to be examined, and the results did not support it at the spatial and temporal resolution available. With rain included in the stratum, the estimate changed from 1.161 to 1.154, practically unchanged. And, in the estimates obtained separately by category, there was no evidence that the excess was greater on hot dry days; on days of intermediate temperature, rain showed an estimate in the direction of lower drowning risk (0.918), and even so the excess persisted on hot rainy days.

Stratification by precipitation therefore **did not support the hypothesis that the finding was explained by the occurrence of dry days**. This weakens a competing explanation, but does not amount to eliminating confounding by precipitation in the strong sense: categorisation does not eliminate residual confounding within bands, and the daily state-level measure does not distinguish intensity, timing or spatial distribution of precipitation.

The second is mobility. Even after adjustment for the precipitation category, the excess may be an increase in exposure — more people in the water — and not an increase in risk per hour of exposure. The simultaneous excess in transport accidents (1.041) is consistent with a broader increase in mobility on hot days. Two considerations limit this interpretation for drowning: the relative magnitude of the association is approximately four times greater (16.1% against 4.1%) and the trend

per decile is about three times steeper (1.046 against 1.014), and the excess is in natural waters and not in swimming pools. But distinguishing an increase in exposure from an increase in risk per unit of exposure requires data that the Mortality Information System does not have.

This mechanistic uncertainty does not negate the potential public health implications. **The results provide epidemiological justification for evaluating the inclusion of drowning-prevention messages in heat alerts, with attention to young adults and to exposure to natural waters,** in the afternoon of the hot day itself. Such preventive messaging could remain relevant even if the association primarily reflects increased exposure rather than increased risk per unit of exposure.

What code X30 does not capture in these data

X30 — "exposure to excessive natural heat" — was recorded as the underlying cause in ten deaths out of the 3.7 million records over twelve years and twelve states, and on any line of the certificate, as underlying or associated cause, in twelve. **The X30 count identifies only a very small fraction of the deaths that make up the excesses temporally associated with hot days in this design.** The estimated excesses were far larger than the number explicitly coded with X30, and it is worth keeping in mind that the numbers are not of the same nature: approximately **1,350 excess deaths among people with recorded dementia** — a quantity that includes the effect of heat on that group as a whole, and of which **892** correspond to the portion associated with the estimated effect modification — and **362 excess drowning deaths**, which is the excess of the outcome itself. This illustrates the value of designs based on excess mortality, rather than relying exclusively on explicitly attributed causes.

Limitations

Exposure measured at the state level — the most serious limitation of the design. Temperature is the mean of the automatic stations of the state, and the death is assigned by the state of residence. In large, climatically heterogeneous states — Bahia, Minas Gerais, Mato Grosso do Sul, Piauí, Rio Grande do Sul — the state mean may be determined by stations hundreds of kilometres from the place of death. This misclassification would, in principle, tend to be predominantly non-differential and to **attenuate** the estimates, although it cannot be ruled out that it varies across outcomes: drowning is concentrated where there is a river, reservoir or coastline, which are not necessarily the regions that dominate the state mean. This error may therefore not be purely non-differential across outcomes, especially for external causes associated with travel. For drowning there is the added fact that the unit of analysis is **residence**, and the event may occur while travelling — a weekend trip, tourism — so that residence and place of event diverge more often than in natural mortality. Both of these were tested rather than left as conjecture. When each death was reassigned to the automatic station nearest the seat of its municipality — a finer unit than the microregion — every estimate fell rather than rose: drowning from 1.161 to 1.131 within 50 km and 1.123 within 100 km, Alzheimer's disease as underlying cause from 1.093 to 1.064, and the dementia effect-modification ratio from 1.044 to 1.037 (Table S6). These are the same deaths under a different exposure classification, with widely overlapping intervals and no formal contrast between the scales, so this does not establish that the finer scale is the better measurement; what it does show is that the attenuation expected from state-level measurement is not supported by the data. Assigning drowning by municipality of occurrence rather than residence changed the estimate in the third decimal place (1.164 against 1.161 at state level, 1.121 against 1.123 within 100 km), so divergence between residence and place

of event does not appear to drive the finding. Exposure misclassification remains a limitation; its direction is simply not the one anticipated.

Exposure relative to the month. The decile is defined within each month, which measures the response to comparatively hot days and not to an absolute threshold. An effect that manifests only above a fixed temperature would be underestimated.

Precipitation was examined through stratum adjustment and stratified sensitivity analyses; humidity was not considered.

Air pollution was not included in the analysis. Ozone and particulate matter may covary with temperature and contribute to short-term mortality associations; residual confounding by air pollution therefore cannot be excluded.

Comorbidity ascertainment relied on death-certificate reporting. Mention on the death certificate depends on how fully the certificate was completed and on what the cause of death was; misclassification is differential, and its direction is not determinable a priori. How fully the certificate was completed does not, however, vary with temperature (section 8), so this misclassification is not itself a function of the exposure. This applies both to the null findings and to dementia — with the difference that, for dementia, consistency with the underlying-cause analysis provides a second route of verification, which is unavailable for the null findings.

Partial coverage. Twelve states and the months of January to May. The restriction of months excludes winter, when heatwaves of relative anomaly occur in the Southeast and South, and also excludes the December peak of drowning.

Preliminary data in 2025. The composition by cause is not mature in the preliminary database; the subsets restricted to 2014–2019 reproduce both main findings.

Multiplicity. Ten conditions, seventeen external-cause groups and sixteen chapters were tested. Dementia and drowning survive Bonferroni within their respective sets; the breakdowns by decile, lag, age, place, time of day and precipitation category are exploratory and descriptive, without correction, and their p values should not be interpreted as independent confirmatory tests. The homogeneous-comparator analysis, the lags beyond three days and the place-of-occurrence analysis were run after the primary results and are reported as such.

Drift in intent coding. The fraction of chapter XX coded as undetermined intent goes from 6.2% in 2014 to 23.5% in 2025. The comparison is always within the same month and year, which protects the relative risk. However, analyses of individual external-cause groups remain susceptible to this coding drift.

Conclusion

In a single design applied to 3.7 million deaths, the patterns of heat vulnerability differed substantially across the conditions examined. Among the comorbidities evaluated, dementia showed the strongest evidence of greater susceptibility, and that association strengthened when each condition was compared with deaths carrying another of the studied conditions. Most conventional cardiometabolic conditions showed little or no evidence of greater susceptibility — obesity and diabetes had estimates close to the null, in a study with 80% power to detect modifications of 3.4% and 1.1% or greater. Cerebrovascular disease was the exception, with a smaller but statistically

robust positive modification under the homogeneous comparator. In the ordering by chapter, the circulatory chapter showed an estimate lower than those observed for the nervous system and external causes chapters.

Two findings stand out, each within its own analytical category. Among the ten comorbidities, **dementia** gave an effect-modification ratio of 1.044, or 1.035 in an analysis stratified by ten-year bands among individuals aged 65 years or older, convergent with a relative risk of 1.093 for Alzheimer's disease as the underlying cause. Among the seventeen external-cause groups, **drowning** gave a relative risk of 1.161, with a gradient from 0.798 to 1.256 across the deciles, a trend of 1.046 per decile and a peak on the hot day itself. These are different quantities — a ratio of two relative risks, and a relative risk — and we do not place them on a common scale or rank one against the other. What they share is that each is the most prominent result of its own comparison. Both are compatible with a behavioural interpretation, which remains a hypothesis: the design measures neither behaviour nor exposure to water. Their contrasting temporal signatures — a sharp peak on the hot day itself against an association spread across the four lags — are consistent with distinct processes.

The findings have potential implications for prevention and provide epidemiological justification for evaluating the inclusion of dementia and drowning risk in heat alert communication, while further studies are needed to refine the separation between temperature and precipitation at higher spatial and temporal resolution, to measure behavioural exposure, and to resolve exposure below the state level. The patterns most prominent in these data involve **people with dementia, who may depend on others to recognise and respond adequately to the demands of heat, and drownings occurring mainly among young adults and in natural waters, in the afternoon of the hot day itself**. The drowning excess is of similar magnitude in men and women — 1.162 and 1.157, ratio 1.005 (0.890–1.134) — so that the male predominance among the deaths (88%) does not appear to arise from greater relative modification of the heat–mortality association; baseline differences in exposure remain a plausible explanation. Whether and to what extent these groups currently feature in Brazilian heat-related risk communication strategies is a question this study did not evaluate and one that deserves an assessment of its own.

The contribution of this work is not the two findings on their own but the comparison that produced them: ten recorded conditions, sixteen chapters and seventeen external-cause groups examined on the same days, with the same estimator and with declared power, so that what stands out can be read against what does not. That comparison is also what qualifies the null results. Finding little or no effect modification for obesity, diabetes or cardiovascular disease says that their mention on the death certificate does not identify a substantially more susceptible subgroup in this design; it does not say that these conditions carry no heat-related risk, and it does not displace them from clinical attention. The wider implication is methodological: vulnerabilities that fall outside the conditions most often examined — a neurological diagnosis, an external cause — are visible only to analyses broad enough to look for them.

Data availability

The aggregated daily counts that the analyses consume, the quality-controlled temperature and precipitation series, and the complete code that produces every table in this manuscript are deposited at Zenodo: <https://doi.org/10.5281/zenodo.22235538> — data under CC BY 4.0, code

under the MIT licence. That is the concept DOI, which always resolves to the most recent version of the deposit.

Individual mortality microdata are not redistributed. They are public and obtained directly from the Department of Informatics of the Unified Health System, and the deposit lists every source file with its size and SHA-256 checksum, so that any reproduction can be verified to have started from the same origins.

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Supplementary material

Table S1. Drowning on hot days, sensitivity of the adjusted estimate to the definition of the precipitation categories. In every row the rain category is part of the stratum, alongside state, year, month and day of the week.

precipitation categorisation	deaths	RR	95%CI	p
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no rain adjustment, restricted to the precipitation panel	3,995	1.160	1.115–1.207	1.4×10^{-13}
median of hot days (0.46 mm)	3,458	1.154	1.104–1.206	2.2×10^{-10}
meteorological threshold ≥ 1 mm	3,389	1.155	1.104–1.209	3.7×10^{-10}
threshold ≥ 5 mm	3,474	1.160	1.109–1.213	5.7×10^{-11}
three bands: < 1 , $1-5$, ≥ 5 mm	2,818	1.141	1.084–1.200	3.8×10^{-7}
quartiles of the distribution itself (0.13 / 1.48 / 5.40 mm)	2,454	1.157	1.094–1.223	2.7×10^{-7}

The first row does not exactly reproduce the estimate of the main analysis (4,010 deaths, RR 1.161; 95%CI 1.117–1.208) because it **uses only the state-days with available precipitation data**, so as to be comparable to the other rows: 209 of the 21,119 state-days of the panel have temperature but not precipitation, and 15 drowning deaths occurred in them on case days. The difference between the two estimates is in the third decimal place.

The number of deaths varies across the other rows because strata with no discordance between case days and control days do not contribute to the conditional likelihood, and the finer partition produces more strata in that condition. The table therefore compares specifications over the informative strata available under each parameterisation, and not over a fixed population; the stability of the estimates across the rows should be read with that caveat. Reproduced by `microdados/cc_precip.py`.

Table S2. Overdispersion. ϕ is the Pearson χ^2 of the fitted conditional model over its degrees of freedom, with cells defined by state and date within each stratum.

outcome	deaths	ϕ	RR	95%CI published	95%CI with ϕ	p published	p with ϕ
all natural causes	886,332	1.536	1.024	1.021–1.027	1.021–1.027	2.5×10^{-72}	1.0×10^{-47}
VI nervous system	32,885	1.028	1.072	1.057–1.086	1.057–1.086	1.5×10^{-23}	5.9×10^{-23}
IX circulatory	245,927	1.095	1.018	1.013–1.023	1.013–1.023	1.1×10^{-12}	1.0×10^{-11}
X respiratory	106,892	1.137	1.027	1.020–1.035	1.019–1.035	1.8×10^{-12}	3.8×10^{-11}
XX external causes	109,151	1.293	1.045	1.038–1.053	1.037–1.054	7.3×10^{-32}	5.1×10^{-25}
dementia (mention)	32,898	1.059	1.067	1.053–1.082	1.052–1.082	4.2×10^{-21}	5.2×10^{-20}
drowning	4,010	1.126	1.161	1.117–1.208	1.114–1.211	8.8×10^{-14}	2.1×10^{-12}
assault	38,080	1.197	1.046	1.033–1.059	1.032–1.060	2.8×10^{-12}	1.7×10^{-10}
transport	24,019	1.197	1.041	1.024–1.057	1.023–1.059	7.5×10^{-7}	6.1×10^{-6}

Effect-modification ratios under the same scaling: dementia 1.044 (1.029–1.059), cerebrovascular 1.011 (1.002–1.020), diabetes 1.003 (0.995–1.012), obesity 1.000 (0.976–1.024), hypertensive disease 0.996 (0.990–1.003), chronic kidney disease 0.991 (0.980–1.003), neoplasm 0.982 (0.975–0.989). The RRmin values rise in the third decimal place: obesity 1.034 to 1.035, diabetes 1.011 to 1.012, hypertensive disease 1.009 to 1.010, chronic kidney disease 1.016 to 1.017.

Table S3. Sensitivity to within-month trend. The exposure definition is unchanged in every column; only the stratum changes.

outcome	month (published)	half-month	ten-day	matched ± 7 and ± 14
drowning	1.161 (1.117–1.208)	1.143 (1.090–1.199)	1.112 (1.045–1.184)	1.149 (1.108–1.192)

VI nervous system	1.072 (1.057–1.086)	1.079 (1.061–1.097)	1.077 (1.053–1.100)	1.075 (1.061–1.089)
IX circulatory	1.018 (1.013–1.023)	1.023 (1.017–1.029)	1.028 (1.020–1.037)	1.023 (1.019–1.028)
all natural causes	1.024 (1.021–1.027)	1.026 (1.023–1.029)	1.024 (1.020–1.029)	1.026 (1.024–1.029)
dementia, effect modification	1.044 (1.029–1.058)	1.043 (1.026–1.061)	1.027 (1.005–1.050)	1.042 (1.029–1.056)

The matched design retains 4,007 of the 4,010 drowning deaths and yields 5,150 informative strata against 3,938 in the primary design. The ten-day blocks retain 2,074 drowning deaths and 2,463 strata, so the attenuation in that column mixes loss of information with control of trend.

Table S4. Isolated lag from 0 to 7, with the temperature of the lagged day. Lags 0 to 3 differ slightly from Tables 3 and 5 because here the stratum excludes lags 0 to 7 rather than 0 to 3, so the control set is not the same.

lag	mean decile of the day	dementia RR	95%CI	dementia / no dementia	drowning RR	95%CI
0	9.92	1.076	1.052–1.100	1.037	1.332	1.251–1.418
1	7.73	1.098	1.074–1.123	1.061	1.142	1.070–1.218
2	6.26	1.073	1.049–1.097	1.049	1.120	1.048–1.196
3	5.49	1.050	1.026–1.074	1.040	1.065	0.995–1.141
4	5.25	1.051	1.027–1.075	1.050	1.008	0.939–1.081
5	5.14	1.021	0.997–1.046	1.021	0.956	0.890–1.028
6	4.86	1.045	1.020–1.070	1.050	0.980	0.912–1.053
7	4.64	1.034	1.008–1.061	1.036	0.950	0.878–1.027

Mean decile of the control days of this analysis, 4.78; of the control days of the primary design, 4.72; of all days of the month, 5.50 by construction.

Table S5. Cumulative excess with a widening window of 0 to K days.

K	% of days exposed	dementia	without dementia	all natural causes	drowning
3	27.1%	+1,350	+12,189	+13,540	+362
5	36.1%	+1,307	+10,254	+11,561	+324
7	43.5%	+1,295	+7,650	+8,945	+235
10	52.6%	+1,154	+5,398	+6,552	+115
14	61.7%	+767	+3,268	+4,035	–80

This table is not a test of mortality displacement, and the decline across rows should not be read as one. As K widens, the exposed set absorbs days that are cooler than the month average (Table S4), so the contrast is progressively diluted and finally reversed; at K = 14, 61.7% of days are classified as exposed and the estimand is no longer the one of interest. Only the relative comparison is informative: between K = 3 and K = 14 the dementia excess falls to 57% of its initial value while the excess for all natural causes falls to 30%. The evidence on displacement is in the isolated lags of Table S4: no compensatory deficit for dementia through lag 7, and for drowning later estimates below 1 that coincide with lagged days cooler than average.

Table S6. Exposure at a finer scale: each death reassigned to the automatic station nearest the seat of its municipality, within 50 km and within 100 km. The state column reproduces the primary analysis, from an independent extraction and independent code.

By municipality of residence:

outcome	state	≤50 km	≤100 km
all deaths	1.026 (1.024–1.029)	1.026 (1.023–1.029)	1.026 (1.024–1.029)
dementia, relative risk	1.067 (1.053–1.082)	1.062 (1.047–1.077)	1.061 (1.047–1.076)
dementia, effect-modification ratio	1.044 (1.029–1.058)	1.038 (1.023–1.053)	1.037 (1.024–1.052)
dementia, effect modification, 65+	1.041 (1.026–1.055)	1.033 (1.018–1.048)	1.034 (1.019–1.048)
G30 Alzheimer's disease	1.093 (1.073–1.114)	1.065 (1.044–1.087)	1.064 (1.044–1.084)
drowning	1.161 (1.117–1.208)	1.131 (1.083–1.181)	1.123 (1.080–1.168)
other external causes	1.041 (1.034–1.049)	1.037 (1.029–1.045)	1.038 (1.030–1.046)
neoplasm (internal comparator)	1.009 (1.004–1.015)	1.010 (1.004–1.016)	1.011 (1.005–1.017)
congenital malformations (null comparator)	0.985 (0.957–1.015)	1.019 (0.988–1.052)	1.010 (0.981–1.040)

By municipality of occurrence:

outcome	state	≤50 km	≤100 km
drowning	1.164 (1.119–1.211)	1.127 (1.079–1.177)	1.121 (1.078–1.165)
dementia, effect-modification ratio	1.043 (1.029–1.058)	1.035 (1.021–1.050)	1.036 (1.022–1.050)

Isolated lag, relative risks:

lag	drowning, state	drowning, ≤100 km	dementia, state	dementia, ≤100 km
0	1.341 (1.266–1.422)	1.271 (1.197–1.348)	1.070 (1.048–1.093)	1.097 (1.074–1.119)
1	1.158 (1.088–1.232)	1.136 (1.067–1.209)	1.097 (1.074–1.121)	1.086 (1.063–1.109)
2	1.110 (1.041–1.184)	1.064 (0.998–1.134)	1.071 (1.049–1.095)	1.071 (1.048–1.094)
3	1.051 (0.983–1.124)	1.027 (0.961–1.097)	1.045 (1.022–1.069)	1.036 (1.014–1.060)

The dementia columns are relative risks among deaths with dementia recorded, not the effect-modification ratios of Table 3, and the lag stratum here excludes lags 0 to 3, as in Table 3 and Table 5, rather than 0 to 7 as in Table S4. The shape is preserved at both scales — drowning peaks on the hot day and falls to the null by lag 3, dementia is spread across the four days — but the position of the dementia peak moves from lag 1 to lag 0, with intervals that overlap almost entirely.