Quantifying the contributions of climate change and adaptation to mortality from unprecedented extreme heat events

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Understanding the mortality effects of the most extreme heat events is central to climate change risk analysis and adaptation decision-making. Accurate representation of these impacts requires accounting for the effects of prolonged sequences of hot days on mortality, the change in that mortality due to anthropogenic forcing, and the potential compensating effects of adaptation to heat. Here, we revisit the August 2003 heat wave in France, a canonical event in a region with rich climate and mortality data, to understand these influences. We find that standard heat mortality exposure-response functions underpredict excess deaths in August 2003 by 60%, but that accounting for the temporally compounding effects of hot days better matches observed mortality. After accounting for compounding effects and applying a machine learning approach to single-event climate attribution, we attribute 6,038 deaths in August 2003 to climate change, ten times higher than previous estimates. Finally, we show that recent adaptation to heat has reduced the projected death tolls of future 2003-like events by more than 80%.

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Understanding the contribution of climate change to human mortality from unprecedented extreme heat events is a critical priority (1). Unprecedented extreme climate events can stress infrastructure or adaptation measures benchmarked to recent experience, posing challenges for societal resilience (2). These record-breaking extreme events are increasing due to anthropogenic forcing (3–5), with future climate change likely to generate even more extreme events than those that have been recently witnessed (6). With respect to historical events, attributing the observed health impacts of extreme heat to climate change (7–10) has the potential to inform ongoing climate litigation (11, 12) and loss and damage compensation (13). With respect to the future, evaluating the impacts of previously unseen events made newly possible by global warming is essential to understanding the health risks of future global temperature levels (14, 15), especially given the potential for individual hot years or events even under rapid decarbonization (16).

At the same time, people have a well-documented ability to adapt to extreme weather, often leading to reductions over time in the effect of heat exposure on mortality (17–21). If the conditions that generated historical extreme heat events recur at present or future levels of warming (15), they may occur not only in a different climate context but also against the backdrop of an evolving temperature-mortality relationship. As a result, accurately quantifying the past and future health risks of extreme heat requires evaluating the competing influences of climate warming and adaptation on mortality.

However, characterizing the impacts of unprecedented extreme events poses specific analytical challenges. Empirically derived exposure-response functions are a standard tool to quantify the health impacts of climate change, but are estimated using data that by definition do not include unprecedented future events. Further, these models tend to treat hot days as additively separable predictors of mortality, neglecting potential compounding effects of multiple days in sequence. Multiple hot days may result in heat accumulation in both the built environment and human bodies (22), and mortality during previous unprecedented events may have been driven by sequences of warm nights that prevented people from cooling themselves after hot days (23). On the other hand, many statistical studies find little additional effect of the sequencing of hot days above their independent effects (24–26). As a result, it remains unclear whether standard statistical models estimated on the full historical distribution of temperatures are suitable for quantifying the effects of unprecedented sequences of very hot temperatures.

Here we revisit the August 2003 heat wave in France, an event which for multiple reasons offers the potential for insight into the contributions of climate change and adaptation to extreme heat mortality. First, 2003 was the hottest summer in Europe in at least 500 years (27, 28), partly due to climate change (29), making it a useful test case for events that are out-of-sample relative to recent experience. Second, mortality in France appeared to be uniquely sensitive to heat prior to 2003 (18), yielding severe mortality during this event (30). Third, France collects detailed daily mortality data, enabling robust statistical analysis. Finally, France adopted a series of adaptation measures immediately following the 2003 event, including the expansion of air conditioning in vulnerable locations such as nursing homes (17) and heat action plans that include educational messaging and proactive visits to isolated people during hot periods (31). Comparing the temperature-mortality relationship before and after 2003 thus offers a simple way to assess the effectiveness of these adaptation measures (17–21).

We take five key steps in our analysis. First, we evaluate the skill of standard exposure-response functions when applied out-of-sample to the 2003 event, to determine whether empirically derived functions can skillfully represent the impacts of unseen events. Second, we develop new empirical response functions that explicitly incorporate temporally compounding heat. Third, we combine these exposure-response functions with a new machine learning-based approach to extreme climate event attribution (32) to quantify the contribution of climate change to mortality in August 2003. Fourth, we evaluate the change in exposure-response functions before and after 2003 and quantify the effect of adaptation on temperature-related mortality in the recent period. Finally, using the same machine learning-based approach to project the intensity of 2003-like events if such events were to recur in a warmer climate, we compare the extent to which recent adaptation can offset the mortality impacts of increasingly intense future heat events.

Mortality during August 2003

The first two weeks of August 2003 were characterized by extreme temperatures centered on France, Germany, and Spain (Fig. 1a), a high-pressure system centered north of France (Fig. 1b), and dry soils across much of the continent (Fig. 1c). Temperatures across France peaked at the end of the first week of August and through the second week, with a maximum population-weighted spatial average of 28.6 °C (daily mean) on 12 August (Fig. 1d).

To understand the death toll of this event, we first derive standard exposure-response functions that relate daily temperatures in French administrative regions (*"departements"*) to mortality in those regions. We find a strong nonlinear relationship between temperature and elevated mortality, where mortality increases at both cold and hot temperatures (Fig. 1e). For example, a 30 °C day is associated with a 50% increase in mortality relative to a day at 20 °C, cumulatively across that day and the 10 days following it. This finding matches the responses found in many previous studies, including those using two-stage pooled time series models (33), and is similar when we use different numbers of lags, polynomial orders, fixed effects, or temperature variables (Fig. S1).

To assess the "true" death toll in August 2003, we also calculate excess deaths relative to region- and time-specific baselines (Methods). Excess deaths are a standard epidemiological approach to quantify elevated mortality without specifying a cause of death or para-

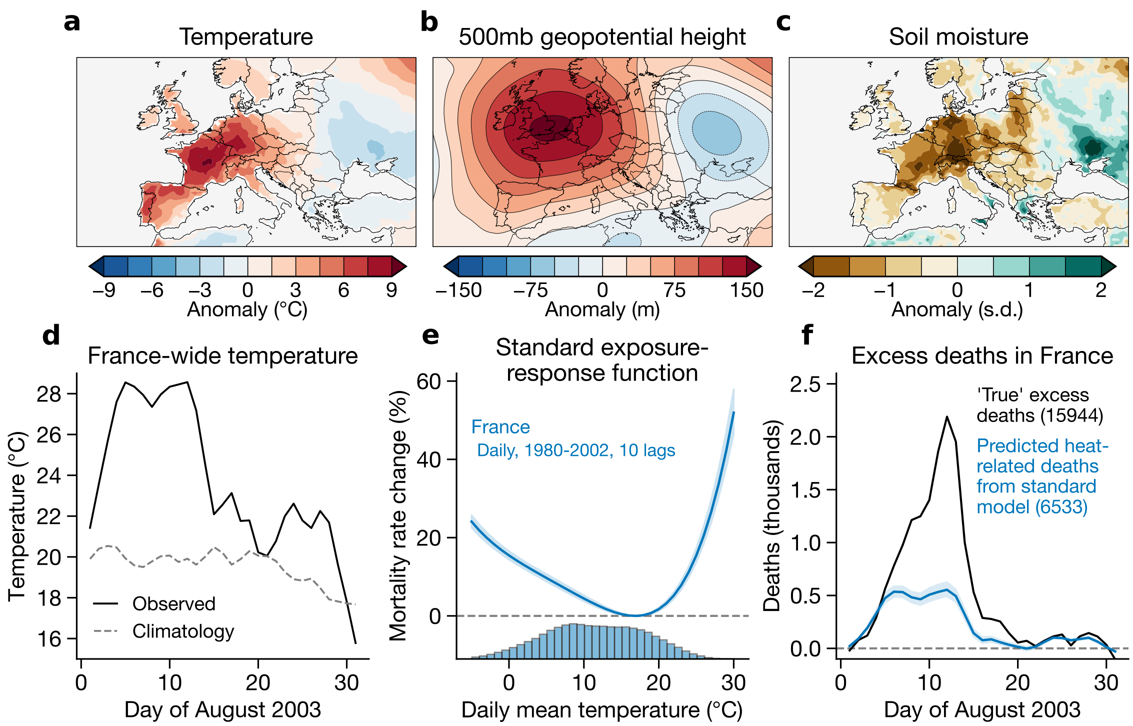

Figure 1: Physical and epidemiological characteristics of the August 2003 heat wave in Europe. a, b, c) Anomalies of temperature (a), 500-mb geopotential height (b), and soil moisture (c) averaged over 1-14 August 2003. d) Population-weighted average temperature across French departements in August 2003 (black), along with the 1980-2002 mean (gray). e) Exposure-response function relating daily mean temperature to mortality rates over 1980-2002, using a fourth-order polynomial and 10 lags of temperature (Methods). Lower histogram shows distribution of daily temperatures in the sample. f) Excess deaths (black) and heat-related deaths (blue) across France. Excess deaths are based on deviations relative to averages and heat-related deaths are based on the exposure-response function in panel (e). In (e) and (f), shading shows 95% bootstrapped confidence intervals.

metric exposure-response function. We estimate $\sim$15,900 excess deaths in France across all of August (Fig. 1f), which aligns well with other estimates (34). We note that hereafter, we use "excess deaths" to refer to estimates of total elevated mortality relative to averages and "heat-related deaths" to refer to mortality specifically predicted by a temperature exposure-response function.

The exposure-response model shown in Fig. 1e is estimated using data from 1980 through 2002, but not including 2003, so that we can perform an out-of-sample prediction of heat-related mortality during August 2003 and compare it to our estimate of excess deaths. Using the 1980-2002 temperature-mortality association (Fig. 1e) to predict the August 2003 death toll underestimates total mortality by 60%: 6,533 heat-related deaths (95% confidence interval [CI]: 5,807 - 7,378) compared to 15,944 excess deaths (Fig. 1f). This underestimate is not a unique feature of our specification; alternative polynomials, fixed effects, lag lengths, and temperature exposures yield similar results (Fig. S2).

Because excess deaths do not specify a cause or exposure-response function, an alternative interpretation of this result is that there were only $\sim$6,500 heat-related deaths and some other unrelated cause explains the remaining $>$9,000. However, there is no other known cause concurrent with the heat wave that would explain such a large number of excess deaths (35), and the magnitude of the mortality increase far exceeded typical variation from other causes (34). We thus interpret the gap between excess deaths and heat-related deaths as an underestimate from the standard exposure-response function rather than an overestimate from the excess deaths calculation.

Temporally compounding heat-related mortality

We hypothesize that neglecting the unique effects of multiple hot days in a row may contribute to the underestimate from the standard model, which does not consider the order of hot days within our 10 days of lags. To incorporate temporal compounding, we modify our regression approach to distinguish between hot days that occur in sequence and hot days that occur after a relatively typical or cool day. Specifically, again using the pre-2003 data, we estimate exposure-response functions that are interacted with a function of the previous days' temperature anomaly, meaning the effect of current-day temperatures on mortality are allowed to vary according to the temperature anomaly of the previous day. In our main approach, we use a binned function of the percentile of the previous day's temperature anomaly, allowing flexible, non-parametric variation in the effect of current-day temperatures depending on the previous day (Methods).

We find that in the pre-2003 period, sequencing a hot day after another hot day can double the mortality effect of that hot day relative to it occurring in isolation (Fig. 2a, 2b). Specifically, a 30 °C day increases mortality by 52% relative to a 20 °C day when it follows a day with a typical temperature (i.e., in the middle quintile of temperature anomalies). However, when a 30 °C

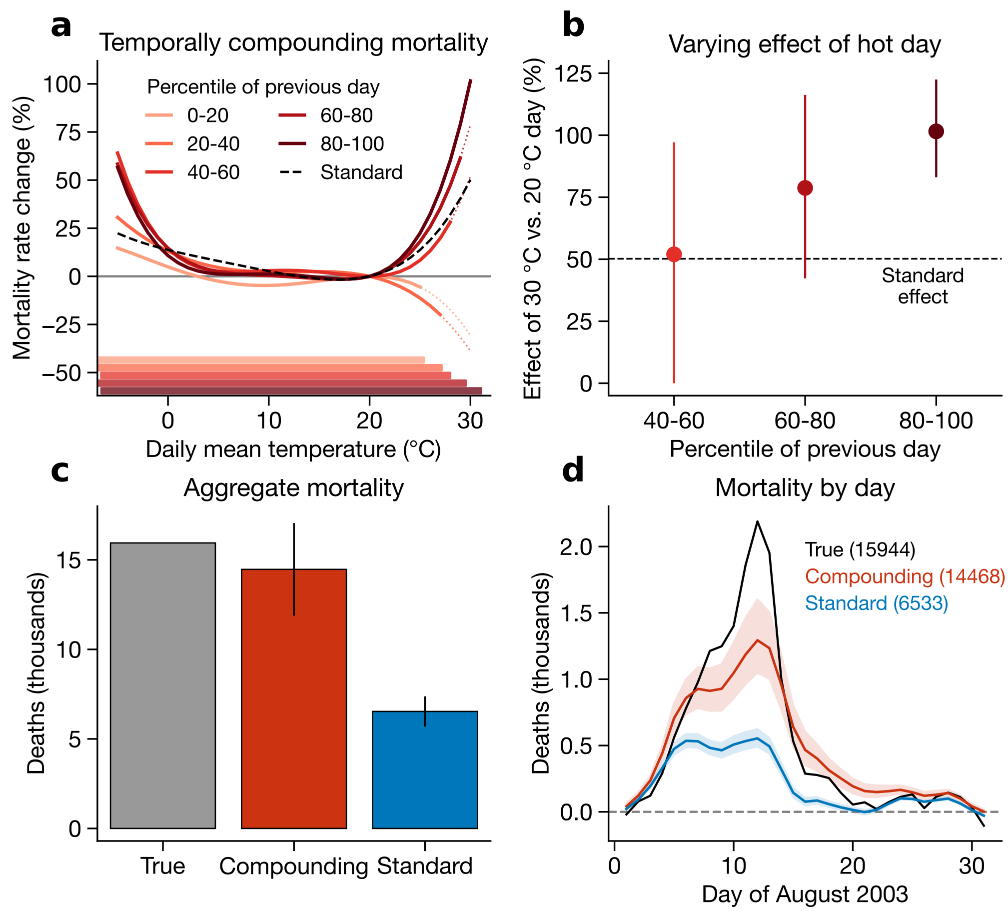

Figure 2: Temporal compounding of heat mortality. a) Mortality exposure-response functions binned by quintiles of temperature anomalies on the previous day, using pre-2003 data. For example, the darkest red curve shows the effect on temperature on mortality on day t if day $t-1$ was in the hottest 20% of days for its location and season. Black dashed curve indicates the "standard" exposure-response function from Fig. 1e. Bottom colored bars indicate the ranges of current-day temperatures experienced in each bin of the previous day, and the curves are shown with dotted lines where they extend beyond these ranges (Methods). b) Effect of hot day according to previous day's temperature. Specifically, we show the effect of a 30 °C day relative to a 20 °C day across the top three bins of the previous day's temperature. Dashed black line shows the effect inferred from the standard exposure-response curve without compounding. c) Heat-related deaths in August 2003 in France from both the compounding and standard models, compared to excess deaths. Bar height shows average prediction and black line shows 95% range. d) Predicted mortality from the heat wave by day. Solid line shows mean and shading shows 95% range.

day follows a relatively hot day (one that was in the top 20% of temperatures), it increases mortality by 100%, relative to it being a 20 °C day (Fig. 2b).

The strong differentiation between the responses conditional on previous days suggests an important role for temporal compounding in shaping heat wave mortality. Indeed, predicting August 2003 mortality using the compounding model shown in Fig. 2a yields a death toll that is much closer to the excess deaths total than the standard model (Fig. 2c). The mean prediction from the compounding model is 14,468 heat-related deaths (CI: 12,093 - 17,052) compared to the mean of 6,533 from the standard model, and the confidence intervals from the compounding model include the 15,944 total excess deaths value. We also test alternative functional forms for the interaction between current and previous days, finding that each yields significantly greater mortality than the standard model (Methods).

While neither the standard nor compounding model captures the magnitude of peak excess deaths in the second week of August (Fig. 2d), it is possible that this peak may not reflect the actual timing of deaths; reported death dates during the heat wave may be uncertain, since the French health system was stressed and many of the deceased were found days or even weeks after they had presumably died (35). As such, we judge the total number of deaths (Fig. 2c) to be a better metric of model skill than their exact date (Fig. 2d).

Anthropogenic contributions to heat-related mortality

An improved representation of the underlying heat-mortality response allows us to return to the question of how many heat-related deaths in August 2003 were due to anthropogenic climate change. We use convolutional neural networks trained on global climate models to simulate counterfactual August 2003 temperatures at 0 °C of global mean temperature change, rather than the $\sim$0.8 °C observed at the time of the event (Methods). We find that climate change increased temperatures across France by an average of 1.4 °C in the first two weeks of August 2003 (Fig. 3a).

Applying our new exposure-response functions to these observed and counterfactual temperatures, we find that the counterfactual event would have caused 8,429 excess deaths, rather than the 14,468 we estimate occurred with observed temperatures (Fig. 3b). As a result, we attribute 6,038 heat-related deaths to climate change (CI: 4,475 - 7,874), 41% of the mortality from the event and more than double the analogous result from the standard model that does not account for temporal compounding (Fig. 3c). Climate change-driven mortality

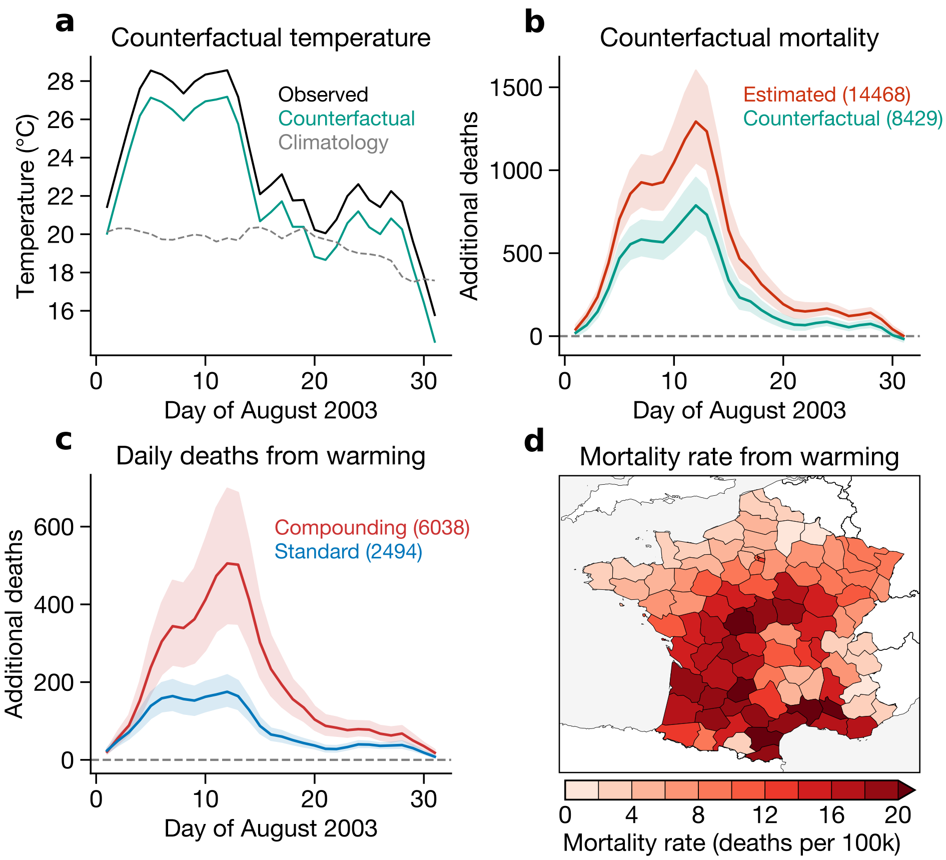

Figure 3: Mortality attributable to climate change in August 2003. a) Observed (black), counterfactual (green), and climatological (gray) temperatures across France in August 2003. b) Mortality in August 2003 under observed conditions (red, as in Fig. 2d) and under counterfactual conditions (green). c) Mortality attributable to climate change, calculated as the predictions from observed conditions minus the predictions from counterfactual conditions. Blue curve shows the analogous calculation using the standard model. In (b) and (c), solid line shows the mean prediction and shading shows 95% range. d) Mortality rate due to climate change in each French departement, defined as deaths per 100,000 population.

was substantial across all of France, but concentrated in the central and southern regions (Fig. 3d), with >20 deaths per 100,000 population contributed by global warming in some southern departements.

The contribution of climate change to mortality witnessed in 2003 raises the question of the magnitude of mortality from a similar event if it occurred in the near future. On the other hand, France may have adapted to heat extremes following 2003 by adopting measures such as heat action plans (31), potentially altering the future death toll of a physically similar event (17). To test this question, we re-estimate the exposure-response function using data from 2004-2019, under the assumption that shifts in the response over time indicate adaptation.

The response of mortality to temperature in 2004-2019 is milder than in 1980-2002 (Fig.

4a); in the later period, a 30 °C day vs. a 20 °C day increases mortality by 18%, compared to 50% in the earlier period. And while there remains differentiation between the effects of hot days following hot, mild, and cool days, the 2004-2019 standard model is nearly identical to the 2004-2019 response when the previous day was hot (compare black and dark red curves in Fig. 4a). This result does not mean that temporal compounding has no effect after 2003, but it does suggest that the post-2003 standard model captures nearly all of this effect, potentially because previous- and current-day temperatures are highly correlated. As a result, explicitly modeling temporal compounding appears to add less additional information about extreme heat events after 2003.

Our counterfactual simulations are based on projecting the same meteorological conditions (Fig. 1a, b, c), but at higher levels of annual global mean temperature (GMT; Methods). We use annual GMT anomaly values of 1.5 °C and 2 °C for present and near-future conditions, respectively, emphasizing that these values refer to *annual* GMT rather than long-term global warming levels (16, 36). (For reference, the annual global mean temperature anomaly was ∼0.8 °C in 2003 and ∼1.5 °C in 2024.)

If a 2003-like event were to occur at global temperature levels of 1.5 °C or 2 °C but with the pre-2003 exposure-response function, we estimate heat-related mortality of 22,039 and 30,361 deaths, respectively (Fig. 3b). That is, if the temperature-mortality relationship had not changed following 2003, we estimate that near-term warming could approximately double

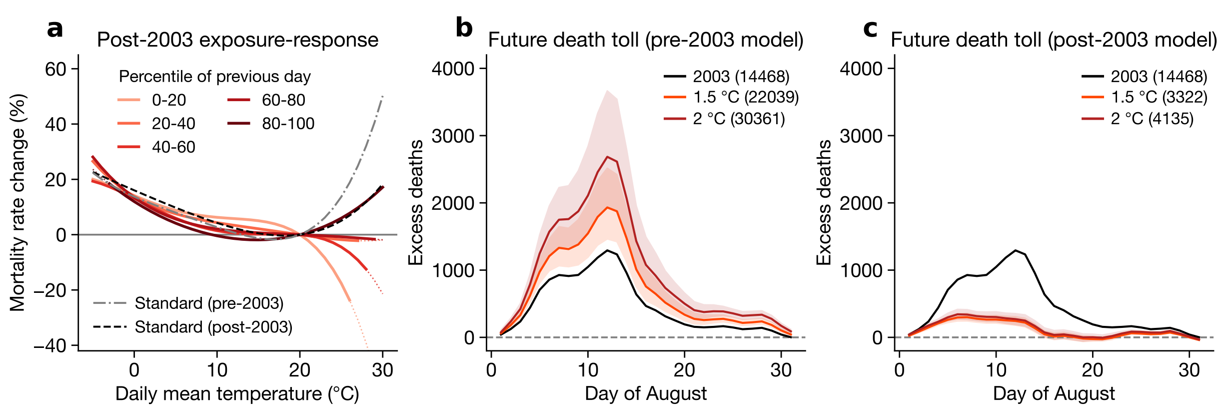

Figure 4: Projected mortality from 2003-like events in the near future. a) Exposure-response functions as in Fig. 2 using the standard approach (black dashed line) and accounting for temporal compounding (red lines), fitted to data from 2004-2019 instead of 1980-2002. The gray dash-dotted line shows the standard model from the 1980-2002 sample (as in Fig. 2a). b) Excess deaths for a 2003-like event at 1.5 °C and 2 °C of annual mean temperature using the 1980-2002 (i.e., pre-2003-event) exposure-response functions. c) As in (b), but using the 2004-2019 exposure-response functions. In (b) and (c), solid line indicates mean projection and shading shows 95% range.

the death toll witnessed in 2003. On the other hand, incorporating post-2003 changes in the response function reduces the projected death toll by nearly an order of magnitude (Fig. 3c), with excess deaths of 3,322 and 4,135 at 1.5 °C and 2 °C. In other words, France's apparent adaptation to extreme heat has reduced the mortality consequences of a future 2003-like event by 85%.

However, we emphasize that even with this adaptation, a 2003-like event at 1.5 °C annual global temperatures would, at its peak, increase daily mortality by 22% above its average daily rate. This is substantially lower than the 94% increase estimated during the peak of the 2003 event, but nevertheless highlights the magnitude of the health impacts of extreme heat, even after substantial adaptation.

Discussion and Conclusion

Our results demonstrate that during at least one widely studied extreme event, the sequencing of multiple extreme hot days played a key role in driving heat-related mortality. This finding differs from other work arguing that hot days have an additively separable effect on health (24–26). However, given that temporal compounding appears to play a much smaller role following 2003, our results may not generalize to all events, especially since pre-2003 France appeared to be an outlier in its heat sensitivity even relative to the rest of Europe (18). Still, given projected future increases in record-shattering heat events (6), understanding the impacts of sequences of hot days in other regions remains a research priority.

Our estimate of ∼6,000 deaths attributable to climate change in August 2003 in France is more than ten times larger than the previous estimate by Mitchell et al. (7) of 506. There are several potential reasons for this. We calculate mortality across all of France, rather than just in Paris. Mitchell et al. also use the fraction of attributable risk to quantify human influence on mortality, a metric which is not necessarily appropriate for impact attribution (10). Additionally, however, the exposure-response function used in Mitchell et al. may underestimate heat-related mortality. Specifically, they use an exposure-response function that estimates a peak of 5 heat-related deaths per 100,000 population in Paris. By contrast, our excess deaths calculation (Fig. 1f) shows a peak of nearly 14 deaths per 100,000 in Paris (Fig. S3). While we previously noted that the timing of peak mortality may be uncertain, Mitchell et al. also report 34 cumulative heat-related deaths per 100,000 population in Paris across June-August 2003, but we calculate 51 excess deaths per 100,000 population in Paris across the same time period, approximately 50% larger. Together, these results suggest that if Mitchell et al.'s exposure-response curve were extended to the rest of France, it would underestimate true excess mortality.

This discussion illustrates that a key contribution of our work is to derive and use an exposure-response function that is more appropriate for the event in question than a standard function. Future work on the impacts of extreme climate events should take care to ensure that exposure-response functions estimated from a full distribution of climate variables are skillful at representing particular extreme events of interest.

On the other hand, we find that the post-2003 response function is very different from the pre-2003 response (Fig. 4a), which may reflect adaptations undertaken in response to the death toll in 2003. Indeed, when we simulate the same heat event with two different response functions (Fig. 3b, c), we find that the milder response function generates a ten-fold reduction in mortality, suggesting large health benefits from these adaptations. Unfortunately, many other countries have not adopted the same measures as France (37), and adaptation to extreme heat appears limited on a global scale (18). So, progress in France may not indicate widespread adaptation elsewhere, though it does help to quantify the potential benefits of adaptation in context of intensifying heat extremes.

Ours is an analysis of opportunity in some respects. France makes local daily mortality publicly available, but many other governments either do not collect or do not share this data, posing a challenge for researchers. Further, our machine-learning-based attribution approach has been shown to skillfully simulate temperature during the August 2003 event, but other heat waves such as the 2021 Pacific Northwest event have proven difficult to simulate both by our method (32) and others (38, 39). Alongside more sophisticated exposure-response functions, additional advances in physical event attribution may be necessary to understand the climate change contribution to mortality during more recent unprecedented events.

Our analysis uses novel econometric and machine learning tools to reveal several important insights about the influences of climate change and adaptation on mortality during a very extreme heat event. After accounting for the unique effects of multiple hot days in sequence, climate change can be linked to around 40% of the mortality this event, even with GMT anomalies only about half of their current value (0.8 °C in 2003 vs. 1.5 °C in 2024). Even if global temperatures are stabilized near their current levels, this additional warmth may contribute more than half of mortality during future similar extreme heat events (15). However, we also reveal significant potential to reduce these harms if strong adaptation actions are taken. Widespread adoption of policies similar to those undertaken in France following 2003 may be necessary to avert mass mortality from future extreme heat.

Methods

Data

Our primary climate data is from the E-OBS station-based data product (40), which we use in the regression models and mortality prediction. E-OBS data is aggregated to the level of French *departements*, weighting by the population of each grid grid cell within the departement. To plot the maps in Fig. 1, we use ERA5 reanalysis data (41) averaged over 1-14 August, with anomalies defined relative to grid cell and day of year.

Daily mortality data spanning 1980-2019 on the universe of deaths in France are made available by INSEE, the French statistical agency (`https://www.insee.fr/fr/information/4769950`). These data are provided at the commune level, a relatively fine geographic resolution, but we aggregate them to departements for comparison with climate data and to merge them with population data. We drop overseas territories from this analysis and focus only on the 94 departements in continental France.

Excess deaths

Excess deaths calculations are a standard way to assess deviations in mortality from expected conditions. The procedure is twofold: (1) model mortality as a function of spatial and temporal baseline factors; and (2) subtract these baseline values from observed mortality during some time period of interest.

We model mortality over 1980-2019 as a function of departement-specific day-of-year averages and departement-specific annual averages, meaning we allow each departement to have its own seasonal cycle and long-term trend. Specifically, we estimate an Ordinary Least Squares model for the log of the mortality rate (M) in departement i as a function of day-of-year d and year y:

$$log(M)_{idy} = \mu_{iy} + \delta_{id} + \epsilon_{idy} \tag{1}$$

For each departement-day in August 2003, we then subtract the predicted values using this equation from the observed mortality rate to calculate excess mortality.

Standard temperature-mortality exposure-response function

The goal of an exposure-response function is to describe a relationship between an exposure (e.g., temperature) and an outcome of interest (e.g., mortality rates). A standard approach is to regress mortality rates on a function of temperature, usually nonlinear, as well as non-parametric controls for all region- and time-specific average factors that could confound this relationship ("fixed effects"). For example, our approach models log mortality rates as a function of local daily temperature, departement-by-day-of-year fixed effects, and departement-by-year fixed effects:

$$log(M)_{idy} = \sum_{j=0}^{L} \left[f(T_{i(d-j)y}) \right] + \mu_{iy} + \delta_{id} + \epsilon_{idy} \tag{2}$$

We note two features of this equation. First, we include L lags of daily temperature to account for the delayed effects of heat and cold. In our main analysis we use 10 lags, since the effect of heat appears to occur primarily in the first several days and decay well before the tenth day later (Fig. S4), but extend the lags to 31 in sensitivity analyses (Fig. S1). Second, the fixed effects ($\mu_{iy} + \delta_{id}$) are precisely the same as those used in the excess deaths estimation (Eqn. 1). In effect, then, this approach seeks to isolate the temperature-driven component of excess mortality.

In our main analysis, we use a fourth-order polynomial for $f(\cdot)$ and daily mean temperature for the exposure variable, both following other work (18, 42). Daily mean temperature, relative to daily maximum or minimum, has the advantage of balancing the effects of both hot days and warm nights, which aligns with our interest in the effects of heat accumulation. In Fig. S1, we show results using a cubic model or daily maximum or minimum temperature, which are qualitatively similar to our preferred specification.

Incorporating temporal compounding

As with most other work in this domain, the exposure-response function in Eqn. 2 treats temperatures on different days as linearly additive. Two hot days have the same effect on mortality if they occur on $d-8$ and d or if they occur on $d-1$ and d. Our approach to temporal compounding is to relax this assumption by interacting the temperature on each day with the temperature anomaly on the previous day. This approach asks the question: Does a hot day have the same effect when it occurs after another hot day as when it occurs after an average or cold day?

Specifically, we estimate several variations of the following model:

$$log(M)_{idy} = \sum_{j=0}^{L} \left[f(T_{i(d-j)y} + f(T_{i(d-j)y}) \times g(T^{a}_{i(d-j-1)y}) \right] + \mu_{iy} + \delta_{id} + \epsilon_{idy} \quad (3)$$

This model adds the interaction with temperatures on day d with temperature anomalies T^a on day $d-1$. For lagged days $d-j$, we interact that day's temperature with the temperature anomaly on day $d-j-1$. For example, temperatures on day $d-2$ are interacted with temperature anomalies on day $d-3$.

We use the previous day's temperature anomalies (T^a), rather than absolute temperatures (T), in this interaction. Anomalies are calculated relative to each departement and day of year separately. Understanding this choice requires some explanation of the role of interaction terms in fixed effects models. Typically, the role of fixed effects in statistical models is to remove variation that the researcher wants to exclude from estimation. For example, in Eqn. 2, the departement-by-day-of-year fixed effects remove the average temperature in each departement separately for each calendar day. This is desirable because there may be factors that vary seasonally that confound the temperature-mortality relationship, such as increased respiratory viruses in winter.

Importantly, however, when interactions are included in fixed effects models (Eqn. 3), the interaction term is estimated before the fixed effects are removed rather than after it (43). This means that estimating the interaction between current and previous days in Eqn. 3 would leverage variation across departements, seasons, and years; for example, it would ask whether the previous day occurred in the summer or the winter rather than solely whether it was an unseasonably warm day for that location and time of year. Using temperature anomalies rather than absolute temperatures allows us to remove the same unit- and seasonally-varying factors prior to estimating the interaction. For further details on interactions and nonlinearities in fixed effects models, we refer the reader to McIntosh and Schlenker (43).

The function $g(\cdot)$ can take many forms, and there is no strong theoretical prior for the functional form of this across-day interaction. In our main analysis, we use a flexible binned approach to avoid placing parametric assumptions on the interactions between days. Specifically, we interact each day with a binary variable ("dummy variable") that denotes whether the *previous* day was in each of five quintiles of temperature anomalies (i.e., the 0-20th percentiles, 20-40th percentiles, and so on). Estimating the exposure-response functions with this interaction results in five freely varying exposure-response functions that correspond to each bin (Fig. 2a).

An alternative, more parsimonious approach is to linearly interact the temperatures of the current and previous days. This approach yields even more improved predictions of 2003 mortality than the binned approach, predicting 16,385 heat-related deaths across August 2003 and showing greater peak mortality than the binned approach (Fig. S5). However, this model imposes a strong parametric (i.e., linear) assumption on the interactions between days. An intermediate approach is to estimate a natural cubic spline with a knot at 0 °C in the previous day's anomaly, which yields separate nonlinear interactions for cold previous days and hot previous days. This approach, on the other hand, overestimates mortality during the event, predicting 21,682 excess deaths (Fig. S5). Regardless, given that both of these alternative approaches predict greater mortality than our preferred binned specification, our main results can be considered conservative.

The temperatures of a day and the day preceding it are not independent, statistically or physically. Fig. S6 shows a clear correlation between the temperature anomaly on day $t-1$ and the temperature on day t. This relationship is not necessarily a problem for our analysis; as long as there is variation in one quantity conditional on the other, we can statistically identify their interaction. However, it does affect the interpretation of the exposure-response curves in Fig. 2a. The red bars on the bottom of Fig. 2a show the ranges of current-day temperatures conditional on the bins of previous days. For example, when the previous day is extremely hot for a given location and season (dark red bar), current-day temperatures can exceed 30 °C. However, when the previous day is cold for that location and season (lightest red bar), current-day temperatures do not exceed ∼25 °C. The dotted lines in Fig. 2a show where we extrapolate beyond the ranges of the bins.

In all regressions, we sample uncertainty in exposure-responses with bootstrap resampling. We sample departements with replacement 500 times, keeping observations from the same departement together to account for within-unit autocorrelation (akin to clustering standard errors by departement). We present the mean and 95% confidence interval (2.5th - 97.5th percentiles) of these bootstrap samples.

Machine learning predictions

We use the machine-learning-based extreme event attribution method developed by Trok et al. (32). This approach trains a convolutional neural network (CNN) on an ensemble of climate model simulations to predict local daily temperature from the global mean temperature, meteorological conditions including geopotential height, surface pressure, and soil moisture, and calendar day. The trained networks are then applied to ERA5 reanalysis in an out-of-sample prediction to predict local temperatures as a function of observed meteorological conditions. In our counterfactual predictions, we maintain the observed meteorology and day of year, but vary the global mean temperature to create dynamically consistent events that would have occurred had the global temperature been different.

The target for the predictions is the average temperature over a region in southern and central Europe that encompasses France, exactly as in Trok et al. (32). Again as in Trok et al., we train CNNs separately on two climate models, CanESM5 and UKESM1-0-LL, that have sufficient daily data for training. For each climate model, we train the CNNs 10 times, with each CNN using a different random seed, to account for randomness in the training process. The one change we make to the method developed in Trok et al. is to use daily mean temperature as the predictand instead of daily maximum temperature.

We use a simple delta-method bias correction before applying these predictions to our observational data. First, for each day in August 2003, we take the difference between the predictions at the counterfactual global temperatures (0, 1.5, and 2 °C) and the predictions at the observed global temperature (0.8 °C in 2003). We then apply the region-wide "delta" for each day uniformly to each departement's temperature for that day to create counterfactual temperature time series for each departement.

Calculating heat-related mortality

We calculate heat-related mortality by applying exposure-response functions to observed or counterfactual time series and climatological baselines. Because the dependent variable in the regressions is log mortality, comparing the function of two different temperature values yields percent changes in mortality. We then multiply these percent changes in mortality by baseline numbers of deaths to calculate additional deaths due to heat.

When calculating mortality from the 2003 event, we compare observed temperatures in 2003 to the 1980-2002 average for each corresponding calendar day, and multiply the resulting percent differences by the 1980-2002 average number of deaths for each calendar day.

For calculations of counterfactual heat-related mortality, we combine the 500 bootstrap iterations of the regression coefficients and the 20 different CNNs (10 CNNs with different random seeds, each for two climate models), yielding 10,000 total estimates. In our main analysis, we simply pool all CNNs trained on the two different climate models, but we note that the CNNs trained on UKESM1-0-LL yield slightly lower counterfactual mortality and thus greater attributed mortality than CanESM5 (Fig. S7). Our main results fall in between the totals yielded by each distinct model.

Supplementary Figures

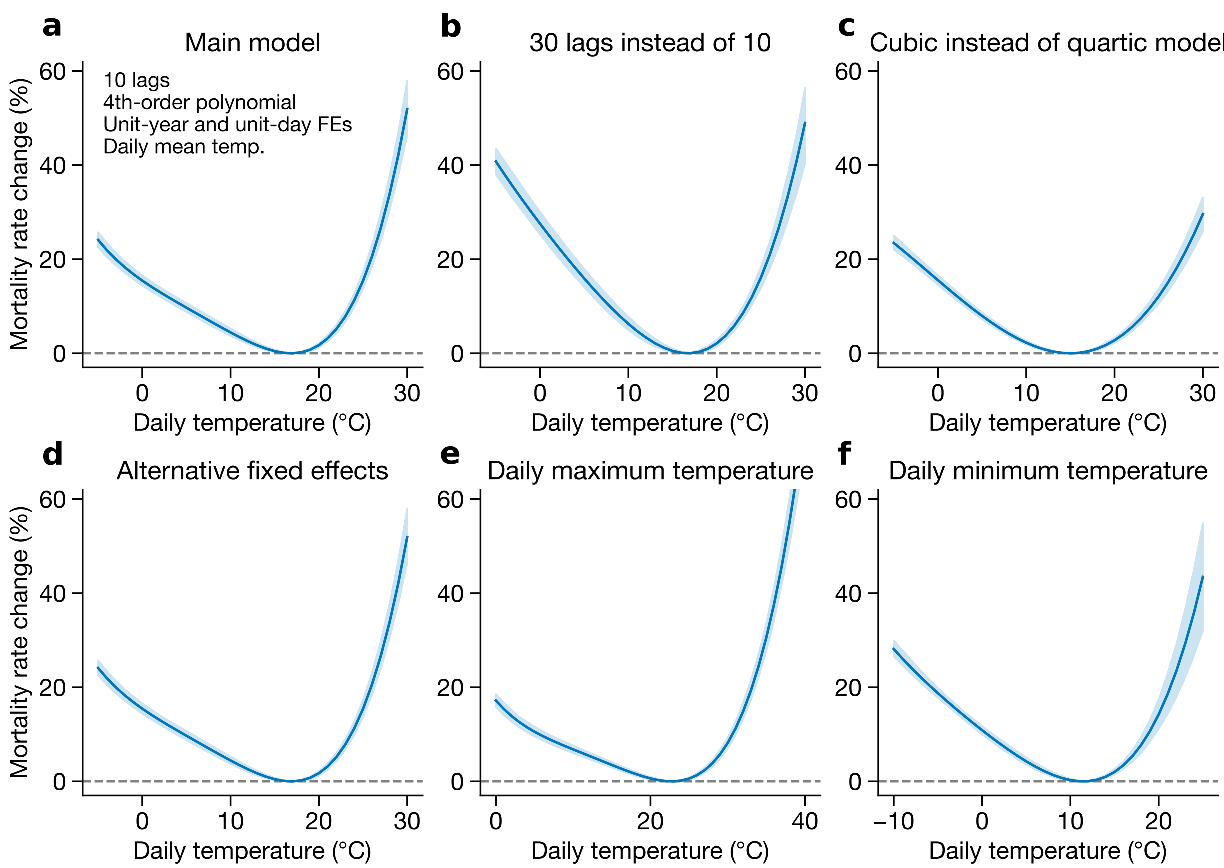

Figure S1: Alternative exposure-response specifications. Each panel shows the response of mortality to temperature using a different regression specification. Panel (a) shows our main model using 10 lags, a 4th-order polynomial, unit-year and unit-day-of-year fixed effects, and daily mean temperature. Panel (b) increases the number of lags of 30, panel (c) uses a cubic (3rd-order) instead of quartic model (4th-order), panel (d) uses separate unit, year, and day-of-year fixed effects, panel (e) uses daily maximum instead of mean temperature, and panel (f) uses daily minimum temperature. Note the different x-axis ranges in (e) and (f) due to the different underlying ranges of the corresponding variables.

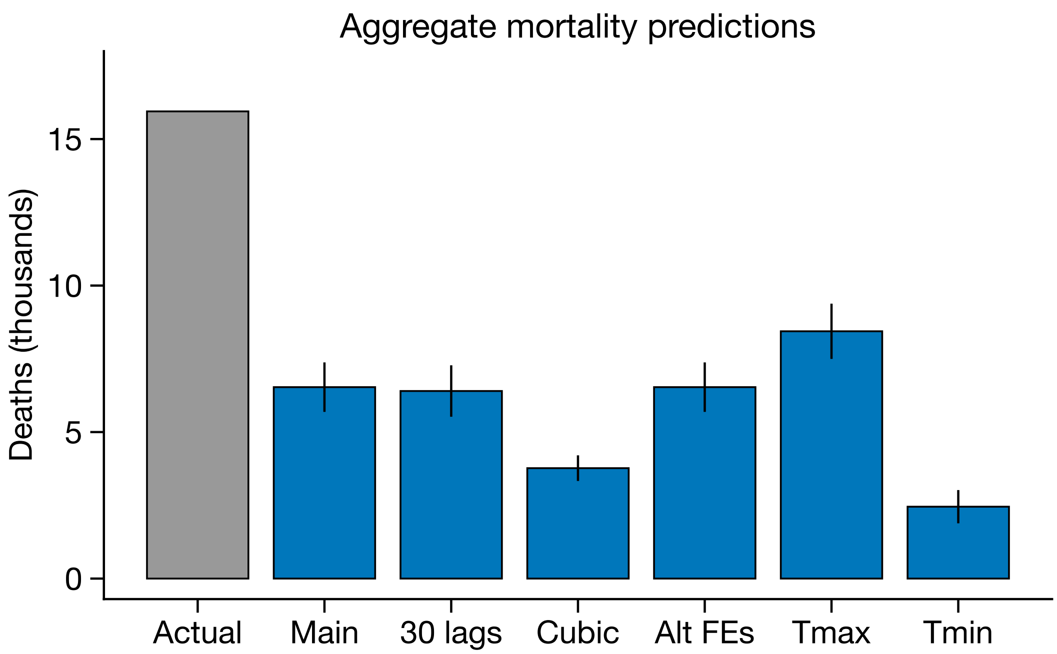

Figure S2: Alternative mortality predictions for August 2003. As in Fig. 2c, but for the range of alternative specifications shown in Fig. S1.

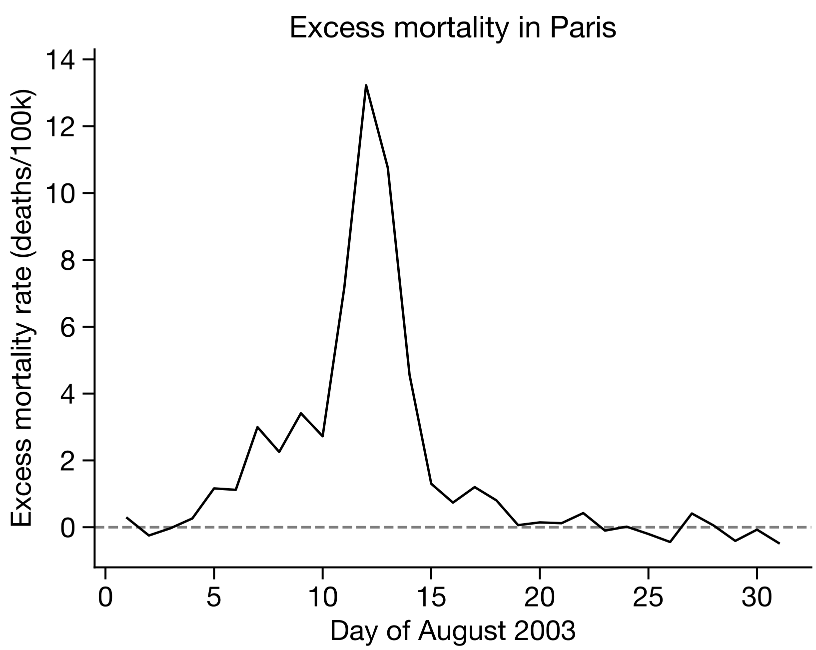

Figure S3: Excess mortality rate in Paris in August 2003. Excess deaths, per 100,000 population, in the Paris departement. Excess deaths are calculated as in Fig. 1f, meaning observed total deaths minus region- and day-of-year-specific baseline averages. A peak of $\sim$13 on 12 August in this plot can be interpreted as: Paris experienced 13 more deaths per 100,000 people on 12 August 2003 than the average 12 August across all years.

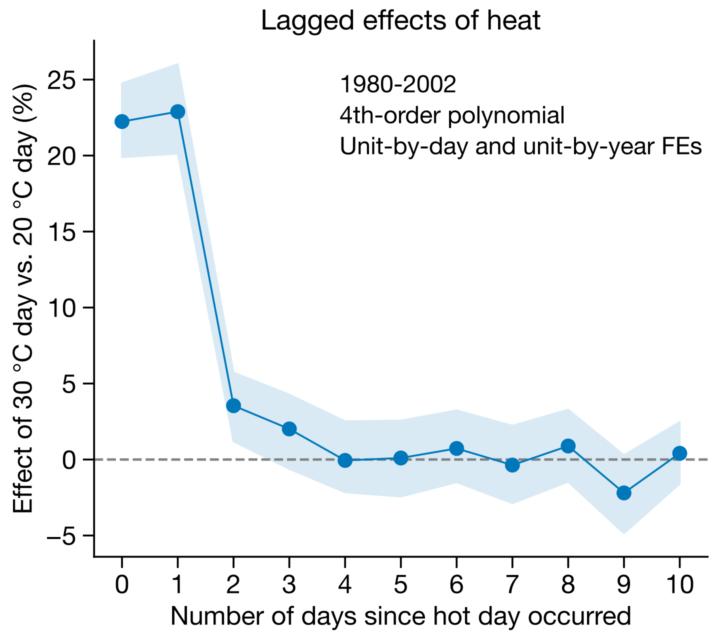

Figure S4: Lagged effects of heat over time. Effect on mortality of a "hot day," defined here as a 30 °C day relative to a 20 °C day, on the contemporaneous day and the 10 days following it. Dots show average across 500 bootstraps and shading shows 95% confidence interval.

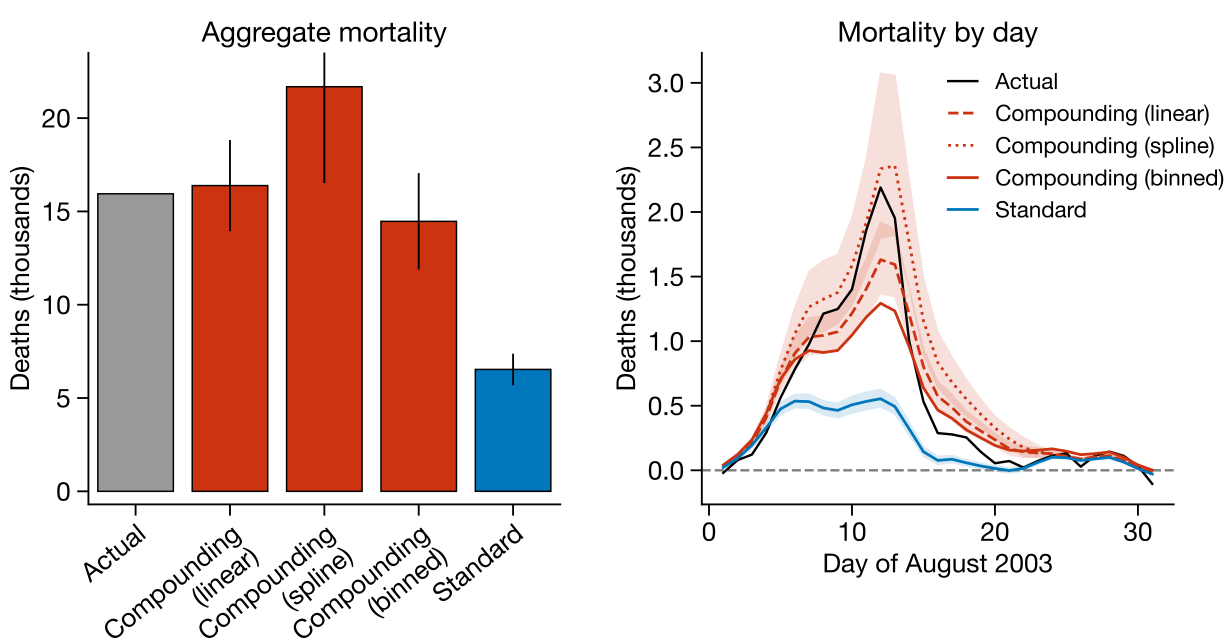

Figure S5: Mortality predicted using alternative function forms for interaction. As in Fig. 2c and 2d, but adding results with a model that interacts current-day temperatures with the previous day's temperature anomaly using either a linear interaction or a natural spline interaction.

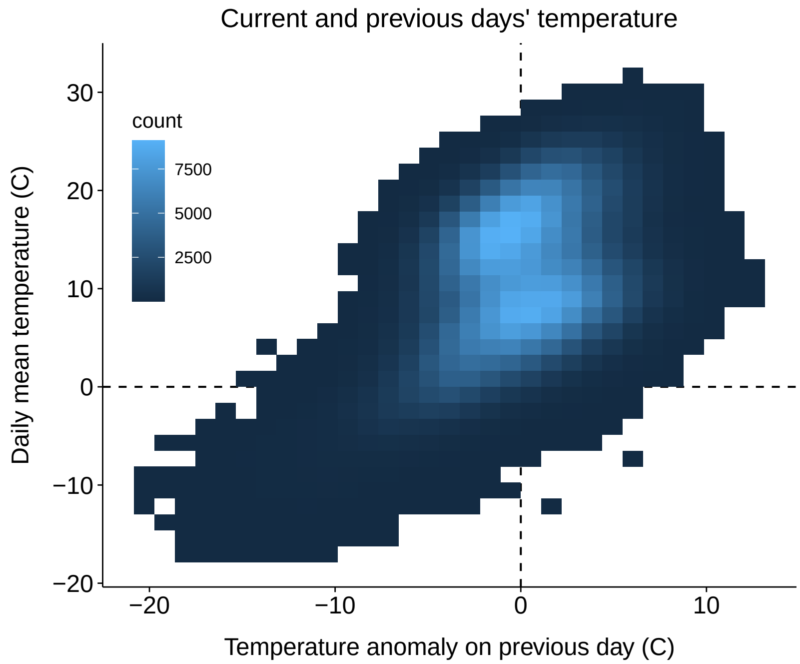

Figure S6: Bivariate density of current and previous days' temperatures. Relationship between temperature anomalies on the previous day (x-axis) and tmeperature on the current day (y-axis). Coloring shows the number of observations in each two-dimensional bin.

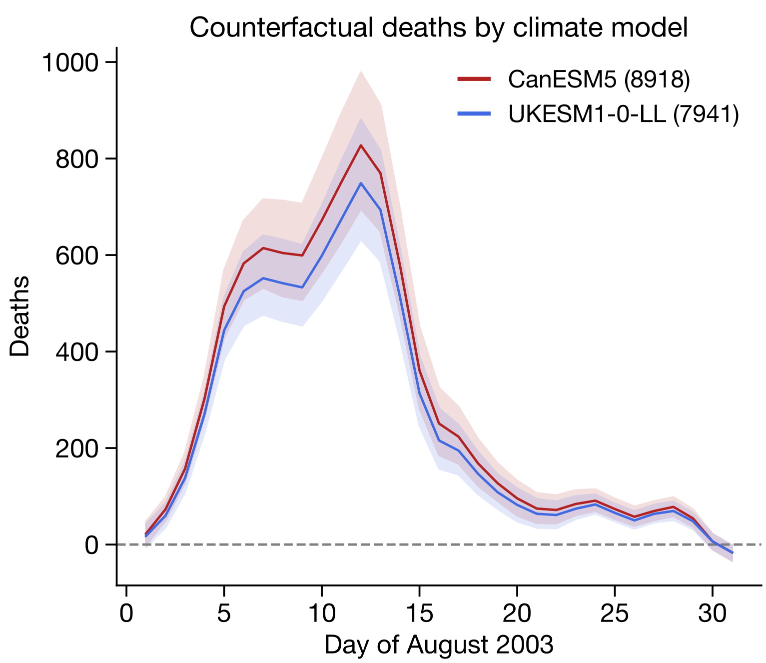

Figure S7: Counterfactual mortality from two distinct climate models. As in Fig. 3b, but when separating the CNNs trained on the two different climate models (CanESM5 and UKESM1-0-LL). Subtracting the totals shown in the legend from 14,468 yields climate change-attributable mortality, 5,550 for CanESM5 and 6,527 for UKESM1-0-LL.

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