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# Attributing heatwave-related mortality to climate change: a case study of the 2009 Victorian heatwave in Australia

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## Abstract

Determining the influence of climate change behind human mortality is of interest to many sectors. However, it is a fledgling field where studies have centered on northern hemisphere events. This study presents the first attribution assessment on the mortality burden of an Australian heatwave to climate change. We focus on excess heatwave- (defined by climatological definitions) related mortality in the state of Victoria that occurred during the 2009 southeast Australian heatwave. An epidemiological model derived from well-established methods defining the relationship between observed heatwave temperatures (95th, 97.5th and 99th percentiles) and mortality is applied to heatwaves in simulations that either include or omit anthropogenic climate forcing from eight climate models. Across all models, the frequency of a heatwave-related mortality event similar to the 2009 Victorian event has, on average, doubled under factual conditions relative to counterfactual conditions. Moreover, on average, around $6 \pm 3$–4 extra individuals out of 31 (an increase of 20%) died as a direct result of extreme temperatures due to anthropogenic influence on the climate. Despite the small total number of attributable deaths as per the epidemiological model, six out of eight climate models predicted a statistically significant anthropogenic influence, indicating that climate change increased the heatwave-related mortality impact of this event. We make clear that, in line with previous Australian-based studies, the focus on mortality relative to the top 5% of temperatures logically infers a smaller mortality signal relative to the top 50% of temperatures, as would be defined by a more general temperature-related epidemiological model. As research, planning and policy interest in the role of climate change behind the burden health—and other adverse impacts of weather and climate extremes—continues to grow, it is vital that interdisciplinary collaborations are nurtured, so that the resulting science is of high-quality rigour, and policy relevance.

## 1. Introduction

Heatwaves are a specific type of extreme temperature event, where anomalously warm conditions persist for multiple days. Whilst there is no universal heatwave definition, in climate-based studies the threshold over which temperatures are classified as extremely hot is generally relative to the local climate, where such temperatures must occur over at least three consecutive days (Perkins and Alexander 2013, Domeisen *et al* 2023). Studies concerned with heatwave impacts, including human health, may employ different definitions to match vulnerabilities. Impact relevant heatwave definitions might include thresholds that determine extreme heat and/or a different minimum event length (e.g. Tong *et al* 2014, Wang *et al* 2015, Hanna *et al* 2016, Jegasothy *et al* 2017, Ebi *et al* 2021) or consider humidity and the physiological response to its combined effect with heat (Vanos *et al* 2020, 2023, Baldwin *et al* 2023). As outlined in section 2, we consider a heatwave definition where the 3-day maximum temperature is above the 95th percentile. The averaging window of our definition accommodates for the lagged response of the adverse health impacts of heatwaves, while the 95th percentile allows for the identification of days where the temperature is climatologically extreme.

Since at least the 1950s, heatwave intensity, frequency and duration have increased over almost every region of the globe, with trends accelerating over recent decades (Perkins-Kirkpatrick and Lewis 2020). Australia is no exception to these trends, particularly in the southeast (Trancoso *et al* 2020, Reddy *et al* 2021), where heatwaves are occurring earlier in the season with increased maximal temperatures (Reddy *et al* 2021). The main driver of heatwave trends across all regions is anthropogenic emissions of greenhouse gases (Seneviratne *et al* 2021). As climate change intensifies in the future, further increases in heatwave length, intensity and frequency are expected (Cowan *et al* 2014). Projected increases in heatwaves will be felt in the myriad of related health impacts such as increased risk of human morbidity and mortality, changes in ecosystems, and altering human infrastructure (e.g. Ebi *et al* 2021, Beggs *et al* 2022, Franklin *et al* 2023, Lüthi *et al* 2023).

In the 2009 summer, one of Australia's most prolonged and intense periods of high temperatures occurred over the southeast of Australia (National Climate Centre 2009), (27 January–8 February; figure 1), with two periods of exceptional daytime heat (>44 °C) during 28–31 January and 6–8 February. Of notable concern were the high daily minimum (night-time) temperatures concurrent with the high daily maxima (daytime), which resulted in the city of Melbourne exceeding an average daily temperature of 35 °C for the first time on record (10.9 °C above average). Across the affected region, maximum daily temperatures were between 12 °C–15 °C warmer than normal over the 28–31 January. While conditions eased slightly on the 1 February, the worst heat over the state of Victoria occurred on the 7 February. Record high temperatures were set over 87% of the state, with Melbourne setting its highest temperature across 150 years of record-keeping. On this day, the maximum temperature was 46.4 °C, surpassing the previous record by almost 1 °C. As of 2024, this record remains unbroken.

The impacts of the heatwave were severe and wide-reaching (McEvoy *et al* 2012, Steffen *et al* 2014). There was also an extensive impact on human health across southeast Australia for the duration of the event (Lindstrom *et al* 2013, Nairn and Fawcett 2015, Nitschke *et al* 2016, Coates *et al* 2022). In Victoria, over the 26 January–1 February 2009 ambulance callouts increased by up to 45%, the number of deaths on arrival increased by 70% and the Chief Health Officer reported 374 excess deaths (ED), an increase of 62% (DHS 2009). This estimate was derived by comparing the number of deaths across the event against what is typically expected at that time of year over the previous five years (DHS 2009). Other methods to estimate the mortality burden also exist, which consider the *long-term* local association between temperature and mortality (e.g. temperature-related or heat-related mortality), where historical mortality rates over many years are related to corresponding observed daily temperatures, thus determining how temperature can predict mortality at a given location (see Vicedo-Cabrera *et al* 2021, Masselot *et al* 2023). Such methods are well-established in the epidemiological literature (e.g. Gasparrini *et al* 2015, Guo *et al* 2018, Zhao *et al* 2019). (Vicedo-Cabrera *et al* 2021, 2023) and have been previously applied to other Australian regions (e.g. Nitschke *et al* 2007, Tong *et al* 2014), as well as being employed in previous heat-related impact attribution assessments (Vicedo *et al* 2023, Stuart-Smith *et al* 2024). Epidemiological models are required for impact attribution assessments, as the observed relationship between the causal event (e.g. heatwave) and the impact (e.g. mortality) is necessary to estimate the impact of interest within factual and counterfactual physical climate model simulations. It is important to emphasize that while epidemiological models are important in predicting temperature-related mortality, they can differ from what occurs during an individual event when other influences are at play, for example, whether appropriate mitigation strategies are accessible and the overall preparedness of the community. This is known within the epidemiological community and is an active area of research. Moreover, it is equally important to make clear that epidemiological models only predict the influence of temperatures (in particular to our study, heatwave-only temperatures) on increases in

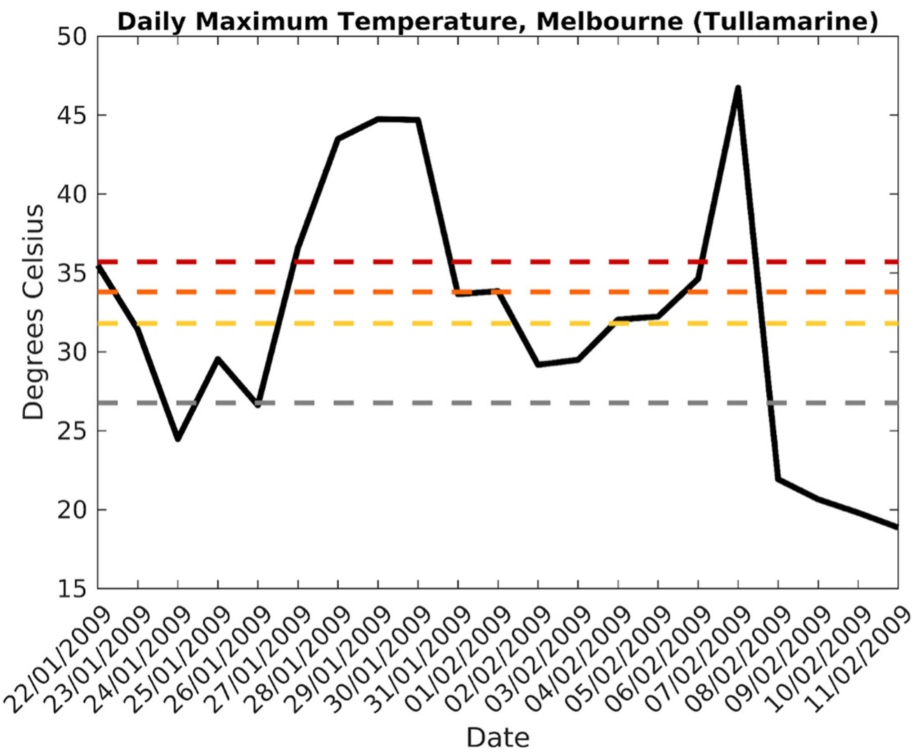


**Figure 1.** Time series of daily maximum temperature over the location of the Tullamarine station (Melbourne Airport) during the 2009 heatwave. Data is from the Australian Water Availability Project/Australian Climate Gridded Dataset (AWAP/AGCD) (Jones *et al* 2009), which is used to assess heatwaves climatologically in this study and is predominantly underpinned by the Tullamarine station in this location. Grey dashed line indicates the summer mean temperature over 1996–2015, and the yellow, orange and red dashed lines are the respective annual 95th, 97.5th and 99th percentiles for the same time period.

mortality. They do not predict all-cause mortality, nor total deaths, and as such, cannot be directly compared to reports of all-cause mortality. We re-visit these points and their relevance to our findings in section 4.

By employing well-established methods across two disciplines, this paper seeks to assess the role of anthropogenic climate change behind the predicted human mortality impact of the 2009 southeast Australian heatwave. Integral to a study of this nature is interdisciplinary collaboration across the climate and epidemiological disciplines. Here we refer to interdisciplinarity as per Aboelela *et al* (2007), where resulting research generally includes a conceptual model that links frameworks from each discipline, employees methodologies that are not limited to any one field, and requires the perspectives and skills of the involved disciplines throughout multiple phases of the research process. Similar approaches have been adopted overseas (e.g. Mitchell *et al* 2016, Vicedo-Cabrera *et al* 2023, Stuart-Smith *et al* 2024), but this study is the first of its kind for Australia, paving the way for future developments in interdisciplinary research to determine how climate change is affecting the health of the Australian community.

## 2. Methods

We utilize a similar approach to an impact attribution assessment as Perkins-Kirkpatrick *et al* (2022). Specifically, we establish the relationship between daily maximum temperature and human mortality from observed temperature and recorded mortality data. The epidemiological model employed in our analysis determines excess heatwave-related mortality for Victoria, that is, the increased mortality occurring due to the temperature on heatwave-defined days (see sections 2.2.2 and 2.2.3) applied to output from simulations of physical climate models to derive an estimate of the mortality impact of heatwave temperatures across all simulated events where anthropogenic influence on the climate is included, and where it is omitted. We form an impact attribution assessment based on comparing the change in the estimated heatwave-related mortality burden between these experiments. Figure 2 provides an overview of the methods employed in this study. Note that the heatwave-related mortality and the physical heatwave attribution are undertaken separately, despite being underpinned by the same climatological data and overarching definition (Perkins-Kirkpatrick *et al* 2022).

The author team drew from expertise in climate modeling, epidemiology, and social science (see figure 2 for a schematic displaying the methodological steps). This enabled climate model simulations to be processed

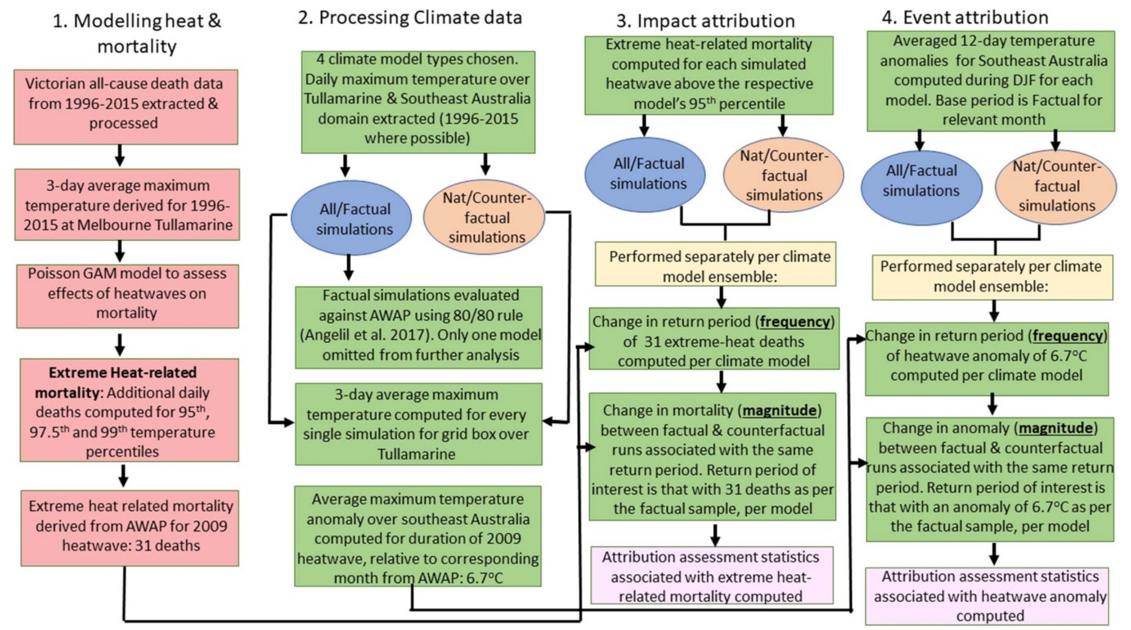


**Figure 2.** Schematic displaying the methodological steps in this study. Despite health (Column 1) and climate (Column 2) methods requiring different expertise, they should be conducted concurrently so that concerns with misuse or misrepresentation are eliminated, whilst also ensuring the study is relevant across disciplines. This is important for any impact attributions assessment, where the output of the two methods is brought together (Column 3).

and evaluated, the mortality data to be sourced, cleaned and analyzed, and for heatwaves and health impacts to be contextualized. In doing so, the study design provides a more complex setting for the research to be conducted and for the results to be interpreted. Because of this interdisciplinary focus, we include an event attribution assessment of the climatological heatwave and the corresponding methods (Column 4 in figure 2) in the supplementary material as it may be of interest to some readers, however, it is not core to the attribution of the mortality response to heatwave-specific temperatures. Moreover, while the health and climate methods are initially segregated (Columns 1 and 2 in figure 2), they are both integral to performing impact attribution (Column 3 in figure 2), highlighting the interdisciplinarity of a study like this. Therefore, constant interaction between the disciplines while the methods are being developed and performed is key.

## 2.1. Population and study setting

The population of the state of Victoria, Australia in 2009 was 5.44 million with 73% residing in the capital city, Melbourne (Australian Bureau of Statistics 2010). The population has been increasing 1.8% on average per year since 2004 with a median age of 36.9 years and where children under 15 make up 18.5% of the population. Two-thirds of the population were of working age in 2009 and it has a high proportion of residents born overseas (22.2% of the Australian population in 2006). Most of the State is classified as Oceanic (Cfb) according to the Köppen climate classification system (including the area of highest population density, Melbourne) Remaining areas of Victoria are classified as Warm summer mediterranean (*Csb*) and Hot summer mediterranean (*Csa)* (Peel *et al* 2007).

## 2.2. Mortality estimation

### *2.2.1. Mortality data*

Daily counts of all-cause death data of Victorian residents from 1 January 1996 to 31 December 2015 (inclusive) are obtained from the Australian Bureau of Statistics (ABS 2010). The data obtained from ABS were de-identified and restricted to statistical division of usual residence by gender and grouped into five age categories (i.e. 0–15, 16–64, 65–74, 75–84 and ⩾85). Daily data on maximum temperature and rainfall were obtained from Tullamarine Airport station, a meteorological observation station near the center of the most populous area in the State of Victoria, Melbourne, for the study period (1 January 1996 to 31 December 2015). The maximum temperature is the highest temperature recorded in the location over a given 24 h period from 9 am to 9 am local time. These data were accessed from the Australian Bureau of Meteorology (BoM). For the same study period, the quarterly population data of the State of Victoria were obtained from the ABS.

**Table 1.** Excess mortality associated with heatwaves in Victoria during 1996–2015. The additional deaths per day (column 4) is used to estimate heatwave-related mortality from physical climate model simulations (see sections 2.3.3 and 2.4). Column 1 refers to the percentiles of the daily maximum temperature distribution in the study area during the study period. RR stands for relative risk, and eCI refers to the empirical confidence interval. Column 5 displays how many days fall in to each percentile during 1996–2016, and column 6 displays the total deaths for all heatwave days for each percentile across the 20 year period.

| Heatwave threshold (%) | RR | Baseline deaths | Additional deaths per day (RR-1)*Baseline death) (95% eCI) | Number of Heatwave days (during 1996–2015) | Total additional deaths over 1996–2015 (=Number of heatwave days* Number of additional deaths) |
|---|---|---|---|---|---|
| 95th | 1.045 | 92 | 4 (2,5) | 163 | 652 (372,902) |
| 97.5th | 1.036 | 92 | 3 (2,5) | 59 | 177 (109,284) |
| 99th | 1.086 | 92 | 7 (5, 9) | 24 | 168 (139,216) |

*2.2.2. Heatwave definition for epidemiological assessment*

In this study, we used daily maximum temperature to estimate the public health impact of heatwaves, as has been done previously in other temperature-mortality studies over Australia (e.g. Nitschke *et al* 2007, Wilson *et al* 2013, Tong *et al* 2014). Heatwaves were defined as periods exceeding specific thresholds based on a three-day moving average of daily maximum temperatures. We adopted three thresholds, the 95th, 97.5th and 99th percentiles of the distribution of the daily maximum temperatures during 1996–2015, respectively (table 1), based on previous Australian studies (Wilson *et al* 2013, Tong *et al* 2014) and consistent with multiple climatological heatwave metrics (Perkins and Alexander 2013, Domeisen *et al* 2023). A summary of the absolute temperature and occurrence of these thresholds are described in table S1 of the Supplementary Material.

*2.2.3. Heatwave-mortality association*

Before giving details of our epidemiological assessment, we must first stress multiple key points so readers are clear on what our study intends to achieve. Firstly, our study is only assessing excess heatwave-related mortality, that is, the mortality associated with days that fall under the heatwave definition described in section 2.2.2. While not an unprecedented approach (Nitschke *et al* 2007, Wilson *et al* 2013, Tong *et al* 2014), it differs from some other temperature or heat-related mortality estimates (e.g. Gasparrini *et al* 2015, Vicedo-Cabrera *et al* 2021, 2023), where mortality is associated with temperatures other than those in the top 5% over three consecutive days. Secondly, an epidemiological model is based on the long-term relationship between temperature (heatwave temperature in our case) and assess the effect of **only (heatwave) temperature** on deaths. Because of its footing in timeseries analysis (Bhaskaran *et al* 2013, Tong *et al* 2014, Gasparrini *et al* 2015, Zhao *et al* 2019), an epidemiological model yields the **overall predictive relationship** between (heatwave) temperature and in mortality. Thirdly, our epidemiological model estimates the **increased risk** of temperature changes on mortality. Based on these two points, it is important not to directly compare the predicted excess mortality from an epidemiological model with total all-cause mortality that is often reported soon after an event occurs. While epidemiological models are computed from historical daily mortality counts (e.g. see Gasparrini *et al* 2015, Vicedo-Cabrera *et al* 2021, 2023), measuring total all-cause mortality is a different entity to increased mortality risk due to (heatwave) temperature. These two mortality estimates are reporting different yet equally important entities and are underpinned by different, yet well-established methods. Fourthly, current epidemiological models do not account for either the occurrence or lack of appropriate adaptation and mitigation strategies (e.g. public education, early warning systems behavioral changes, access to cooling). Since they estimate (heatwave) temperature mortality over many years, included individual events could feasibly have different adaptation and mitigation responses, thus resulting in range (i.e. not just a linear) of event-specific temperature-mortality responses on which the model is based. Accounting for adaptation is an on-going area of research in epidemiology. Despite this caveat, epidemiological models such as the one produced in our study remain a gold-standard approach in predicting temperature-related mortality and are regularly employed in interdisciplinary research to understand how both standard and extreme temperatures can increase mortality over many locations across the world (Bhaskaran *et al* 2013, Tong *et al* 2014, Gasparrini *et al* 2015, Guo *et al* 2018, Zhao *et al* 2019) as well as previously being employed to estimate how climate change has influenced temperature-related mortality (e.g. Vicedo-Cabrera *et al* 2021, 2023, Stuart-Smith *et al* 2024). Should readers like to further understand epidemiological methodologies detecting how temperature can increase mortality research, we refer them to previous research such as Bhaskaran *et al* (2013), Tong *et al* (2014), Gasparrini *et al* (2015), Guo *et al* (2018), Zhao *et al* (2019) and Vicedo-Cabrera *et al* (2021), Vicedo-Cabrera *et al* (2023)).

We use a quasi-Poisson generalized additive model (GAM) to explore the effect of heatwave temperatures on mortality and morbidity for the State of Victoria. The GAM model has been widely used in time-series regression studies of mortality and temperature (Bhaskaran *et al* 2013, Tong *et al* 2014, Gasparrini *et al* 2015, Zhao *et al* 2019), and we use a smoothing function (Bhaskaran *et al* 2013) to capture patterns within the time-series data, while carefully accounting for various confounding factors. Seasonality is controlled for by using a natural cubic spline of the day of the year of the study period with 5 degrees of freedom per year (Bhaskaran *et al* 2013). The long-term trend is controlled for by using a natural cubic spline of day of the year and one degree of freedom allocated per ten years of the study period. Population size is included as an offset while the day of the week is treated as a categorical variable with weekends serving as reference category (Gasparrini *et al* 2015, Zhao *et al* 2019). To control for the confounding effect of influenza-related deaths, our analysis excludes each annual season coinciding with peak influenza activity in Australia (June to August) (Walker *et al* 2006). The distributed lag effect of the heatwaves on mortality were captured following the parameterization established in earlier studies (Guo *et al* 2018, Zhao *et al* 2019). A 10 day lag period was considered to capture delayed effects (Guo *et al* 2018) and mortality displacement, where deaths are shifted forward by a few days. The lag-response function was modeled with a natural cubic spline with four degrees of freedom.

As per previous epidemiological studies, we calculate the relative risk (RR) of mortality on heatwave days compared non-heatwave days. Across the whole period (1996–2015), excess deaths are calculated for three heatwave thresholds: (a) at the 95th percentile and above but less than 97.5th percentile; (b) at the 97.5th percentile and above but below 99th percentile; and (c) 99th percentile and above. These percentiles computed for the entire 1996–2015 period, corresponding to additional daily deaths of 4, 3 and 7, respectively (see table 1). In in other words, heatwave days as per our definition in section 2.2.2 are separated from all other days for our time period. Using the GAM model, we then compute the increased risk of mortality associated with each of the three percentiles relative to all non-heatwave days. In this study, we have translated this increased risk to extra absolute deaths (see table 1), and then add up all daily extra absolute deaths for each discrete heatwave duration. We calculated empirical CIs (95% eCIs) using Monte Carlo simulations (500 samples) to quantify the uncertainty in estimating excess deaths (see table 1). We acknowledge that additional deaths associated with the 97.5th percentile are smaller than that of the 95th percentile, likely due to the small sample size associated with each percentile. Note that the maximum daily mortality rate is 7, however, most extreme heat events last for longer than one day, thereby resulting in a higher overall excess mortality.

The excess deaths due to heatwave events was calculated as follows:

$$ED = (\mathrm{RR} - 1) \times N \times D$$

where RR is the relative risk derived from the model. $D$ is the daily average of baseline deaths during the heatwave period. $N$ is the number of heatwave days during the study period. This formula was modified from previous studies that modeled the burden of mortality due to extreme temperatures (Guo *et al* 2018, Zhao *et al* 2021).

Note that, based on the methods described above, our study focuses on *heatwave*-related mortality. Other studies have mainly focused on temperature- or heat-related mortality, where mortality is associated with all temperatures above a local threshold from which an increase in deaths may be expected (e.g. Gasparrini *et al* 2015, Vicedo-Cabrera *et al* 2021, 2023). However, there is evidence of previous Australian epidemiological assessments focusing on heatwave-related mortality only (e.g. Nitschke *et al* 2007). We employ a similar statistical model as these studies to estimate mortality from local temperatures, but only focus on deaths occurring at temperatures at or above the 95th percentile for at least consecutive days, that is, deaths that occur due to extreme temperatures only during a climatologically-defined heatwave. We chose this approach since the vast majority of climatological heatwave definitions only consider a heatwave to occur when the temperature is at least as warm as the 95th percentile over a period of consecutive days (see Domeisen *et al* 2023). We therefore explore the mortality signal of these days only, and not those of slightly lower (but in some cases, likely still relatively extreme) temperatures.

### 2.3. Climate methods

#### *2.3.1. Climate models*

Following recent protocols (e.g. Philip *et al* 2020, Swain *et al* 2020, Van Oldenborgh *et al* 2021), we employ a range of physical climate models with different configurations and resolutions to perform our attribution assessment. These models provide two types of simulations; the first (factual simulations) includes the effect of historical anthropogenic emissions, and the second (counterfactual simulations) omits them, thus simulating an alternative, 'natural' climate in the absence of human interference. Three types of climate model configurations were employed for our main analysis:

1. Atmosphere only;
2. fully-coupled ocean and atmosphere; and
3. Weather@Home.

Models in group 1 simulate the atmospheric response to prescribed sea surface temperatures (SSTs). The observed SSTs are used for the factual simulations, and either an estimate of SST warming due to human influence (CAM5.1–1degree) or the observed long-term warming (d4PDF-G, ECHAM5.4) is removed from the observed SSTs to produce the counterfactual simulations. Group 2 models are fully-coupled in their ocean and atmosphere, and provide multiple factual simulations. Their respective counterfactual climates are derived from 1851–1900 in the historical simulations where there is little anthropogenic influence on the climate (Gillett *et al* 2016). While group 3 (weather@home) is technically an atmosphere-only model, each weather@home simulation spans only 2009, such that there are thousands of simulations overall for both factual and counterfactual experiments (Black *et al* 2016). See table S2 in the supplementary material for model-specific details including the number and length of each simulation, time period extracted, resolution, and modeling center details. Note that we also originally employed all available climate models participating in the 'detection and attribution model intercomparison project', however these simulations could not be used since they did not provide sufficient sample sizes of daily data for precise characterization of extreme events on daily timescales with return periods larger than a few years.

*2.3.2. Climate model evaluation*

The factual simulations of all climate models are compared to observations to assess whether each model is fit for purpose. Our evaluation method is based on Angélil *et al* (2017) and models that compare poorly are removed from further analysis based on author judgment since they do not appropriately simulate events greater than the 80th percentile, our (arbitrary) threshold for extremes (see below). For each individual climate model, we pool daily maximum temperature values from all factual simulations, compute a cumulative distribution function, and compare to the observed cumulative distribution of daily maximum temperature of the Australian Water Availability Project/Australian Gridded Climate Dataset (AWAP/AGCD) (Jones *et al* 2009) for the time period 1996–2014 (1996–2011 for d4PDF). Our climate model base periods do not perfectly align with that used in the epidemiological methods because of restrictions in data availability. Since the true signature of the heatwave encompassed a region much greater than Victoria, we evaluate each model across the entire affected domain (28°S–45°S, 129°E–155°E). However, since we are only interested in *extreme* temperatures, we compare model and AWAP/AGCD data above their relative 80th percentiles. In order to reduce the inherent temperature bias in each climate model we follow the procedure of Angélil *et al* (2017). Here, we bias correct AWAP/AGCD against each individual model ensemble, by adjusting for the difference in the mean above the 80th percentile between AWAP/AGCD and each respective model. While this infers a shift in the observed temperature distribution, the distributional shape stays the same, which means that the exceedance of relative thresholds (i.e. percentiles) remains consistent, which is important for heatwave calculations (see section 2.2.3). To be deemed 'fit-for-purpose', each model must have 80% of the AWAP/AGCD temperature values above the 80th percentile fall within the respective model's 5th–95th confidence band computed by the model's own factual simulations. Since the set-up of Weather@Home provides thousands of runs for 2009 only, model evaluation was based on separately provided climatological simulations that spanned 1996–2014. Since there was not a consistent number of simulations per calendar year in Weather@Home, each calendar year was bootstrapped 10 000 times to provide a robust and evenly-distributed sample for evaluation against AWAP/AGCD. This method of evaluation is intended to roughly assess the overall tail behavior without being too specific, because the relevant property of the tail that is important for us will differ between the factual and counterfactual scenarios; like Angélil *et al* (2017) we find this method an effective means of distinguishing climate models with clearly inappropriate tail behavior.

*2.3.3. Heatwave calculations in climate models*

For the mortality burden analysis, heatwaves in the climate models are derived using the same methodology as per the epidemiological model in section 2.2.2. In every simulation, a moving 3 day average of maximum temperature is derived. The 95th, 97.5th and 99th percentiles are computed for the period 1996–2014 from the factual simulations (in the case of Weather @ Home, a 120-member climatology ensemble for 1996–2014 is used, and in the case of d4PDF, the base period truncated at 2011). We use the factual-based percentiles across both experiments (i.e. factual and counterfactual) to detect heatwaves in the corresponding model since this is a similar period and climate setting (i.e. anthropogenic influence on the climate was already occurring) from which the mortality and extreme heat relationship described in section 2.2.3 is derived. If any of the three percentiles were exceeded, the given day is flagged as a heatwave day, meaning that both single- and multiple-day events were included in assessing extreme heat-related mortality.

### 2.4. Impact attribution

#### *2.4.1. Extreme heat-related mortality in climate model simulations*

Similar to Perkins-Kirkpatrick *et al* (2022), we estimate the impact—in this case, the excess heatwave-related mortality—during the simulated factual and counterfactual heatwaves defined by section 2.2.3. If a heatwave is flagged, then the corresponding absolute excess mortality rate in column 5 of table 2 is fitted. That is, for days above the 95th percentile but below the 97.5th percentile, an excess mortality of 4 is used, for days between the 97.5th and 99th percentile, an excess mortality of 3 is used, and for days above the 99th percentile, an excess of mortality of 7 is used. Over consecutive days, the excess mortality is summed across all consecutive days above the 95th percentile to obtain the total extreme heat-related mortality for that event. The result is a large sample of estimated extreme heat-related excess mortality across all factual and counterfactual heatwaves per physical climate model simulation. Because the observed excess heatwave-related mortality across Victoria is defined in section 2.1 by using the Tullamarine weather station, the closest land grid box from each model simulation to this location is used for the model-based Victorian excess extreme heat-related mortality estimation. In the Supplementary Material, we provide an attribution assessment of the causal heatwave. However since the meteorological signature of the heatwave encompassed all of Southeast Australia, the corresponding assessments are based on this much larger spatial domain.

#### *2.4.2. Performing impact attribution*

To determine the fraction of excess heatwave-related mortality to be attributed to anthropogenic influence, we re-fit the epidemiological model defined in section 2.2.3 back to the AWAP/AGCD daily maximum temperature data. Using AWAP/AGCD daily maximum temperature from the grid box encompassing the Tullamarine weather station over the main period of the 2009 heatwave (28 January–8 February), a total of 31 excess heatwave-related deaths occurred. Therefore, the attribution analysis below is based on 31 excess deaths due to the most extreme temperatures, and how such a mortality rate has changed in frequency and intensity due to anthropogenic climate change. We note that this figure is substantially different than the 374 excess deaths reported by the Chief Health Officer (DHS 2009) and Coates *et al* (2022). As stated in section 2.2.3, these figures are examining mortality differently—our calculation is representing *excess deaths above the non-heatwave baseline which are directly due to the heatwave conditions only*, whereas the latter figure is representing the total excess deaths during the two-week event compared to what is expected, on average, at that time of year. Despite elevated temperatures persisting for around two weeks, only three days were deemed heatwaves as per the definition in section 2.2.1. We discuss the difference between excess heatwave-related mortality and total all-cause mortality further in section 4.

Frequency changes in excess heatwave-related mortality of 31 are computed by comparing how often this mortality rate occurs in the factual and counterfactual simulations within the same model ensemble. Magnitude changes are computed by comparing the mortality rates associated with the same return period in the factual and counterfactual simulations within the same model ensemble. Here, the return period of 31 heatwave-related deaths was first determined from a given model's factual simulations based on the Weibull formula (Kızılersü *et al* 2018). Next, the mortality rate corresponding to the same return time in the counterfactual simulations was derived and subtracted from 31. Note that because each physical climate model has its own evolving climate governed by the models' individual physical setup, both attribution steps (i.e. frequency and magnitude) must be performed separately per model before overall ensemble results across all can be yielded. Similar to Perkins-Kirkpatrick *et al* (2022), we present the lower (factual 5th percentile—counterfactual 95th percentile) and upper bounds (factual 95th percentile—counterfactual 5th percentile) as well as the median (factual original model sample—counterfactual original model sample) of this signal, per individual model ensemble.

In section 3, we compare the attribution results across each model ensemble to provide some quantitative as well as qualitative results on how climate change impacted the excess heatwave-related mortality during the 2009 Victorian case study.

## 3. Results

Figure 3 displays return period plots of heatwave-related mortality for each set of factual and counterfactual experiments across the physical climate models deemed suitable by evaluation. With the exception of two models, CAM5-1-1degree (figure 3(a)) and Weather@Home (figure 3(d)), there is a clear statistically

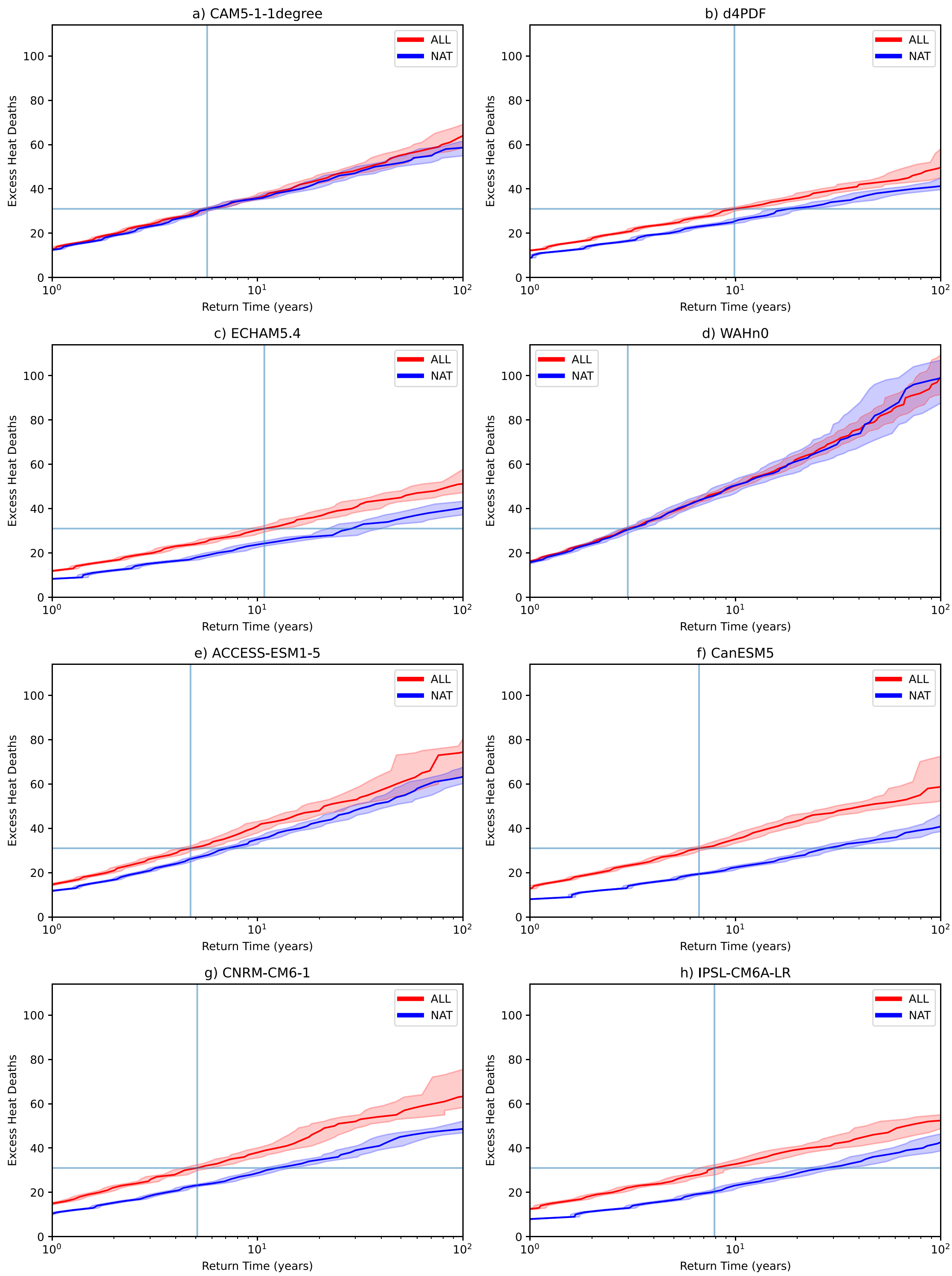


**Figure 3.** Return period plots of extreme heat-related mortality based on the Factual (or ALL, red) and counterfactual (or NAT, blue) simulations for each respective climate model. The light blue horizontal line indicates the mortality rate of 31 excess deaths, whereas the light blue vertical line indicates the corresponding return period for the specific climate model.

significant separation between the factual and counterfactual simulations. That is, according to these models, more heatwave-related excess deaths occurred over Victoria circa 2009 due to anthropogenic forcing on the climate. CAM5-1-1degree (figure 3(a)) and Weather@Home (figure 3(d)) show considerable overlap of the predicted return times of all excess extreme heat-related mortality values between the factual and counterfactual simulations. This means that, according to these two models, excess heatwave-related mortality around 2009 over Victoria neither increased nor decreased due to anthropogenic forcing on the climate. This is likely due to the spatial scale on which mortality was estimated (see section 2.4). Since the extreme heat-related mortality was assessed relative to a single temperature station, the corresponding

**Table 2.** Return periods and the change in frequency of an extreme heat-related mortality event of 31 excess deaths across Victoria per model ensemble, expressed in years. Numbers in brackets indicate the 5th–95th percentiles (i.e. the lower and upper bounds) of each ensemble, whereas the number outside of brackets is the respective ensemble's median. Results in column 4 are computed by dividing the respective counterfactual return period by the factual return period. Therefore a change in frequency of 2 indicates that the event occurs, on average, twice more often in factual climate simulations compared to counterfactual simulations. See table S2 in the supplementary material for the details of each model, and section 2 about how excess mortality was computed from the factual and counterfactual climate model experiments.

| Model | Return period counterfactual (years) | Return period factual (years) | Change in average frequency |
|---|---|---|---|
| CAM5-1-1degree | 5.8 (5.3–6.3) | 5.7 (5.2–6.2) | 1.0 |
| d4PDF | 18.2 (15.5–21.8) | 9.9 (8.7–11.3) | 1.8 |
| ECHAM5.4 | 28.8 (22.1–40) | 10.8 (9.2–13) | 2.7 |
| Weather@home | 3.1 (2.8–3.5) | 3.0 (2.8–3.1) | 1.0 |
| ACCESS-ESM1-5 | 7.1 (6.5–7.9) | 4.7 (4.2–5.4) | 1.5 |
| CanESM5 | 27.7 (23.8–33.6) | 6.6 (5.9–7.6) | 4.2 |
| CNRM-CM6-1 | 12.4 (10.9–14.4) | 5.1 (4.4–5.9) | 2.4 |
| IPSL-CM6A-LR | 27.2 (22.7–34) | 7.9 (6.7–9.6) | 3.4 |
| Ensemble | 16.3 (12.4–18.9) | 6.7 (5.64–7.5) | 2.3 |

attribution needs to be performed on a comparable spatial scale, i.e. a single model gridbox encompassing the station. This scale differs to that on which the causal event should be attributed, which should encompass the entire domain that experienced intense heat during the meteorological event. At smaller spatial scales, variability becomes a relatively higher influence, particularly on the extreme events that can cause adverse impacts (Sillmann *et al* 2017), therefore inhibiting the detection of the forced (i.e. due to climate change) signal. Moreover, it is important to remember that there is a non-linear relationship between the causal weather or climate event and the corresponding impact (Perkins-Kirkpatrick *et al* 2022), inclusive of temperature and mortality. While all climate models analyzed determine that climate change has increased the likelihood of the causal heatwave (see figure S3 in the supplementary material), this does not automatically infer a similar, if any, change in mortality. Moreover, to preserve its meteorological signature, the spatial and temporal scale of the causal heatwave is very different to that of the more local-scale mortality we assess, further reducing the comparibility of the anthropogenic signal between the larger-scale heatwave and local-scale mortality. While it is curious that only two of the eight model ensembles employed were significantly influenced by scale in the heatwave-related mortality attribution, this is an issue best addressed by future research in the climate attribution community. This result also highlights the need for employing multiple climate models in impact attribution, as results can vary—even for sound physical reason—across different physical models.

For each climate model, table 2 presents the return periods in the counterfactual and factual simulations of 31 heatwave-related deaths, as well as the change in frequency across the two simulations (i.e. the counterfactual return period divided by the factual return period). It is worth noting that there is considerable variance across the models in terms of their respective return periods in the factual and counterfactual simulations. For example, ECHAM5.4 and CanESM5 project a median return period of close to 1-in-30 years in their counterfactual simulations, whereas weather@home projects a 1-in-3 year return period. This once again highlights the importance of not restricting an (impact) attribution assessment to just one physical climate model, as they can produce varying quantitative results. Most models simulate a median return period of less than 1-in-10 years in their respective factual simulations, yet weather@home and CAM5 do not project a change in return period between the counterfactual and factual simulations. Across the six models that project a change in the return period of 31 excess heatwave-related deaths, the range in the change in frequency is between 1.8–4.2. That is, according to these six models, 31 excess deaths due to heatwave conditions now occurs 1.8–4.2 times more often due to anthropogenic influence on the climate. When treating all examined models equally, the overall median estimate is 2.3 times. This means that a Victorian heatwave-related mortality event of 31 extra deaths occurs on average 2.3 times more often than what it previously would have without anthropogenic influence on the climate.

Based on the above results, there is strong evidence that anthropogenic climate change has increased excess heatwave-related mortality similar what occurred over Victoria in 2009. Additionally, by comparing the difference in the mortality rate for the same return period across the counterfactual and factual simulations within each model, any change in the number of excess deaths can also be attributed to anthropogenic climate change (see section 2.3.2). The results presented in table 3 demonstrate that six out of eight climate models employed indicate an increase of between five to twelve more deaths due to heatwave conditions for the corresponding return period in the factual simulations. That is, according to these models, between five (16.1%) to twelve (38.7%) fewer individuals than the 31 excess deaths that our epidemiological

**Table 3.** Difference in the magnitude of an extreme heat-related mortality event (factual minus counterfactual) for each physical climate model. Here, the return period of 31 excess heatwave-related deaths is computed from the factual simulations. Then, the magnitude of excess heatwave-related deaths associated with the same return period on the respective counterfactual simulations is computed. The table presents the difference in excess heatwave-related mortality between the factual and counterfactual simulations, both in absolute values and as a percentage increase relative to 31 excess heatwave-related deaths.

| Model | Lower bound | Median | Upper bound |
|---|---|---|---|
| CAM5-1-1degree | −2(−6.4%) | 0 (0%) | 2 (6.4%) |
| d4PDF | 3 (9.7%) | 6 (19.3%) | 8 (25.8%) |
| ECHAM5.4 | 4 (13%) | 7 (22.3%) | 9 (29%) |
| Weather@home | −2 (−6.4%) | 1 (3.2%) | 2 (6.4%) |
| ACCESS-ESM1-5 | 3 (9.7%) | 5 (16.1%) | 7 (22.3%) |
| CanESM5 | 10 (32.3%) | 12 (38.7%) | 13 (41.9%) |
| CNRM-CM6-1 | 6 (19.3%) | 8 (25.8%) | 9 (29%) |
| IPSL-CM6A-LR | 6 (19.3%) | 11 (35.5%) | 12 (38.7%) |
| Ensemble | 2.7 (8.7%) | 6.3 (20.3%) | 9.8 (31.6%) |

model predicted for the 2009 event would have been killed under conditions where anthropogenic influence is absent. The exceptions are CAM5-1-1degree and Weather@Home, which also yielded no detectable anthropogenic signal in the return period of 31 excess heatwave-related deaths (figure 3, table 2). Across all eight models employed, the average median difference in the magnitude of the observed excess heatwave-related mortality event is 6.3 ± 3.6 deaths. In other words, a heatwave-related mortality event occurring in the absence of climate change with the same return period as an extra 31 deaths under anthropogenic conditions would have resulted in around an extra 25 deaths, meaning that anthropogenic influence on the climate has therefore increased the excess heatwave-related mortality in 2009 by 20%.

## 4. Discussion

This study is the first to assess how anthropogenic climate change influenced the health impacts of an Australian heatwave. Specifically, we focus on excess heatwave-related mortality in Victoria during the 2009 southeast Australian heatwave, determined from 20 years of temperature and mortality data. The extra deaths above three extreme temperature percentiles (95th, 97.5th and 99th) were computed. This epidemiological model was applied to eight physical climate models to determine the simulated excess heatwave-related mortality in simulations with and without anthropogenic emissions of greenhouse gases. The epidemiological model was also fitted to Melbourne-based daily maximum temperature during the 2009 Victorian heatwave so that the excess heatwave-related mortality during this event could be quantified. It is the resulting mortality rate—i.e. 31 excess heatwave-related deaths—that formed the basis of our impact attribution assessment.

Analyzing many thousands of years of data from physical climate models, this study found strong evidence that anthropogenic climate change very likely increased the frequency and magnitude of excess heatwave-related mortality events like that experienced in Victoria during the 2009 southeast Australian heatwave. The frequency of an excess heatwave-related mortality event over Victoria similar to 2009 is doubled—that is, it occurs, on average, twice more often in the factual simulations compared to the counterfactual simulations. Moreover, the analysis determines that an extra six to seven people died (an increase of roughly 20%) during the heatwave conditions because of anthropogenic influence on the climate. Our assessment is in line with multiple studies from other regions that demonstrate the mortality impact of temperature has measurably increased because of climate change (e.g. Mitchell *et al* 2016, Ebi *et al* 2021, Vicedo-Cabrera *et al* 2021, 2023, Stuart-Smith *et al* 2024). Moreover, our assessment is strengthened by employing eight, fit-for purpose climate models of varying structural and physical setups.

In the remainder of this section, we discuss the importance and implications of some of the methodological considerations of this study. These include using multiple climate models; estimating excess heatwave-related mortality; and the critical importance of interdisciplinary collaboration.

### 4.1. Using multiple climate models

A significant influence on the spread of the strength of the anthropogenic signals across the climate models presented in section 3 is the different structural and physical characteristics of each individual climate model. For example, the different climate sensitivities (e.g. Nijsse *et al* 2020, Zelinka *et al* 2020) among the models may influence the strength of the attributable signal, as would their representation of underpinning physical mechanisms such as the location and persistence of high-pressure systems that induce heatwave conditions over the region (Pezza *et al* 2012, Parker *et al* 2013, Purich *et al* 2014). Yet since all eight models evaluated well against baseline observations, there is not enough evidence to select just one or a smaller subset of models. Additionally, it is possible that the range among the attributed changes in mortality magnitudes and return periods could be widened if more available well-evaluating climate models were included in the study. Where possible, numerous climate models should be included (subject to evaluation), such that known but also plausible structural, physical and dynamical uncertainties across different climate models are sampled as best as possible. This is now common practice in extreme event attribution studies (Philip *et al* 2020, Swain *et al* 2020, Van Oldenborgh *et al* 2021) and should also be the case for impacts attribution.

### 4.2. Choice of mortality estimation

In this study, the excess mortality response to heatwave conditions is computed only for temperatures at or above the 95th percentile persisting for at least three days. This has resulted in an arguably modest excess mortality rate of 31 deaths that, based on the method described in section 2.2, occurred over just four days of the 12 days meteorological event (see supplementary material). This excess heatwave-related mortality is just a fraction of the 374 total excess deaths reported for the 26 January–1 February 2009, though it is worth noting that this amount of total excess deaths was a 62% increase from the previous 5 year average (DHS 2009). While we make clear in section 2.2.1 that these two figures are not directly comparable, we discuss some of their differences as it is likely of interest to some readers. At first pass, the difference between these mortality estimates could call into question why our assessment yielded a small excess heatwave-related mortality signal. One explanation for such discrepancies is the specific type of mortality being estimated, and the underpinning methods. The 374 excess deaths (62% increase) reported by DHS (2009) was all excess deaths across a week, relative to a short-term baseline, yet the epidemiological model compares mortality counts on heatwave days to all other non-heatwave days over a 20 year period. Importantly, the total excess mortality rate includes deaths not directly associated with a physiological response to extremely hot temperatures—for example, these could hypothetically include a drowning at a beach, or another death indirectly caused by the heat—as well as deaths that would occur at lower, technically non-heatwave temperatures. However, the epidemiological model used in this study instead only considers how many *extra* people die due to an adverse physiological response brought on by very extreme temperatures.

A strength of employing an epidemiological model is that many data points underpin the resulting relationship; in this study a total of 5465 days contributed, 276 of which were heatwave days (see table 1) as per section 2.2.1. Similarly, epidemiological models serve as an appropriate impact function necessary to undertake impact attribution (Perkins-Kirkpatrick *et al* 2022). Impact functions are required so that samples of the impact (i.e. excess heatwave-related mortality) can be created from the many individual casual events (i.e. heatwaves) within the physical climate model simulations using a method. In our study, we have been as consistent as possible in ensuring our heatwave definition is the same in defining the casual heatwaves and the excess heatwave-related mortality. This consistency—over both event and impact and across a 20 year period—could not be achieved with a singular value such as total excess mortality, further complicated by underpinning moving baselines.

It is also worth discussing the appearingly low excess mortality for each heatwave threshold. While each threshold is associated with less than ten extra excess deaths per day, this somewhat low heatwave-only mortality rate is in line with a similar study over the Australian city of Adelaide (Nitschke *et al* 2007). In this case, no extra deaths were associated with the extreme temperatures of heatwaves, likely due to an adaptation effect discussed below (Nitschke *et al* 2007). This suggests that oftentimes during heatwaves, some Australian communities are able to undertake appropriate measure to reduce mortality impacts. Moreover, similar epidemiological models have more broadly examined how heat-related mortality (Vicedo-Cabrera *et al* 2021, 2023) and temperature related mortality (Gasparrini *et al* 2015), where the main difference is the specific temperatures the mortality data is compared against. In the case of temperature-related mortality, the relationship between deaths and all temperatures at a given location is defined (Gasparrini *et al* 2015), whereas for heat-related mortality, the relationship is generally restricted to the local warmer season (Vicedo-Cabrera *et al* 2021, 2023). Because of the larger temperature and mortality samples these approaches require, they will also yield mortality estimates that are almost always certainly larger than *heatwave*-related mortality, which is based on a smaller subsample. By no means does this infer that either temperature, heat, extreme-heat related mortality, or even some other method is the single best way to assess the effect of (heatwave) temperatures on human mortality. Moreover, the choice of which exact method to use ultimately comes down to the original framing of the attribution assessment, similar to event attribution assessments by climate scientists (Otto *et al* 2012, Perkins-Kirkpatrick *et al* 2024). As defined in sections 1 and 2, we saught to assess the anthropogenic signal behind any excess heatwave-related mortality during the 2009 Victorian event, and therefore chose the most appropriate method available to define the impact.

As discussed in section 4.1, using multiple climate models is now considered protocol in climate-based extreme event attribution assessments. Impact attribution is still in its infancy (at least relative to climate-focused extreme event attribution) and the employment of multiple impact methods in attribution assessments remains to be explored. As noted above, there may well be instances when only one impact method is suitable for the study at hand. However, there will ultimately be instances where the impact of interest could be appropriately estimated in a number of ways, plausibly resulting in different quantitative impact estimates. Where appropriate, future research should explore what effect multiple impact methods might have on both the strength and robustness on the derived anthropogenic signal. Indeed, the range of the anthropogenic signal may increase when multiple impact methods are employed, however, similar to the use of multiple physical climate models, the robustness of the signal could be increased and the confidence in at least a qualitative attribution assessment enhanced. Although beyond the scope of our study, a first attempt could explore flipping the approach used here, that is, employing one physical climate model but multiple temperature/mortality methods. This would see the study instead performed through a more health/impact-focused lens. Further work could combine multiple climate and health/impacts methods to achieve both a thorough and comprehensive impact attribution assessment across each participating discipline.

### 4.3. Strong, on-going and supported interdisciplinary collaboration

This study was made possible through consistent and iterative engagement between epidemiologists and climate scientists, necessary for all health and climate attribution assessments (e.g. see Vicedo-Cabrera *et al* 2021, 2023, Stuart-Smith *et al* 2024). All impact attribution research is ultimately an interdisciplinary endeavor (see Harris *et al* 2024). Whilst impact attribution cannot exist without collaboration, challenges ranging from mis-matching data and different knowledge and skill sets to a misinterpretation of the other discipline's methods, can make the process complex. Because the future of impact attribution heavily relies on the successful collaboration across disciplines, the research should be underpinned by co-ordinated programs targeted at providing robust assessments that build on previous knowledge. While impact attribution assessments are increasing worldwide, different methods exist across the climate and impact fields, particularly when working with minimal or no consultation. In order for impact attribution to grow into an established and potentially operational field, greater, on-going knowledge-sharing is essential so that incorrect assumptions across fields are not replicated, all data and methods are employed appropriately, and assessments are not conducted by a single discipline in isolation. Our recommendations are not just unique to our study, and are echoed by Carlson *et al* (2024), who strongly advocate for consistent interdisciplinary collaboration and investment to ensure health impact attribution is performed with a high research integrity.

Future research on attributing health burdens to climate change over Australia should also explore the effect of adaptation. Recent research over Europe demonstrated that anthropogenic temperature increases over the last 5 decades resulted in over 1700 deaths, however, another 700 were avoided due to changes in the exposure and vulnerability to extreme heat over the same period (Stuart-Smith *et al* 2024). Soon after the 2009 southeast Australian heatwave, the BoM released its operational heatwave forecast service, providing multi-day forecasts of heatwave severity across the country (Nairn and Fawcett 2015). All states and territories have devised heatwave management plans, and the dangers of extreme heat and how to alleviate them are widely broadcast when a heatwave is forecast. While there is strength in including as many data points as possible in an epidemiological model, the exposure-response relationship among the individual events can vary considerably, particularly if adaptation and mitigation measures are either accessible or not. While our epidemiological model provided a respectable fit to the 20 years of data overall (see figure S2 in the supplementary material) there was a heatwave within the time series (January 2014) where a reduction in mortality occurred, likely because of appropriate adaptation and mitigation. Similarly, the 2009 heatwave caught the community unawares (DHS 2009) and likely resulting in much higher mortality values than otherwise expected, even under heatwave conditions. The condrum here is that, as discussed above, impact functions that cross between the event and impact space are necessary (Perkins-Kirkpatrick *et al* 2022), yet individual events can buck the historically-defined long-term relationship due to non-physical factors, a possibility for the 2009 Victorian event. While it is well outside the scope of this paper to redefine well-established and best-practice epidemiological models we highlight this caveat as an avenue for future interdisciplinary research.

An additional avenue for future interdisciplinary research is assessing how anthropogenic climate change may increase the likelihood of system-breaking heat-related mortality events may become in the future. As outlined both here and in Perkins-Kirkpatrick *et al* (2022), the impacts of extreme events are non-linear to the causal event, as well as a given systems response to an impact also being non-linear, for example, the ability of a hospital or ambulance network to operate effectively during high-impact heatwaves. Since the analysis focused on how climate change influenced the extreme heat-related mortality of a specific event, a systems-breaking assessment was outside of scope. However, such an analysis would be invaluable in planning for effective mitigation of health impacts of extreme temperature events, and would be another product of the deep and sustained interdisciplinary collaboration the authors of this study highly recommend. Moreover, attribution could be employed to analyze how an ageing population would alter the overall population vulnerability to extreme heat over time, concurrent with increasing anthropogenic influence on the climate. In short, there are many avenues to which an attribution methodology can be applied to better understand the various influences on human health from extreme heat, and indeed, from other weather and climate extremes.

## 5. Conclusion

This study is the first to assess the influence of anthropogenic climate change behind human mortality during an Australian heatwave. It demonstrated a higher and more frequent heatwave-associated excess mortality rate due to human influence on the climate. This adds to the growing body of literature that climate change is causing more heat-related deaths across the world, with future rates projected to increase rapidly, especially in the absence of appropriate adaptation strategies (Lüthi *et al* 2023). While the 2009 event was one of Australia's deadliest heatwave to date (Coates *et al* 2022), rising trends in heatwave frequency, intensity and duration (Cowan *et al* 2014, Reddy *et al* 2021) could increase the likelihood of higher heat-related mortality events in the future, particularly when mitigation strategies are not accessible. Although the focal event and impact assessed here occurred more than a decade before this study was published, it has laid important interdisciplinary foundations and knowledge from which future Australian heat/health impacts attribution research—as well as other Australian impact attribution studies—will greatly benefit.

We stress that interdisciplinary studies such as ours are only possible with deep, iterative and co-ordinated collaboration. This is vital to guarantee all researchers fully understand the appropriate use of and uncertainty associated with all underpinning methods, as well as restrictions and properties of the data employed. Deep collaboration also ensures that the conclusions and any associated implications of impact attribution assessments are properly interpreted and communicated to audiences in the respective disciplines, as well as to broader audiences in general. In the not-to-distant future, there will likely be an increased interest in understanding how climate change has impacted human morbidity and mortality via extreme weather and climate events. It is important that these studies properly embrace interdisciplinary collaboration (Harris *et al* 2024), so that the resulting impact attribution assessments are robust and performed to the highest standard possible.

## Data availability statement

The data cannot be made publicly available upon publication because no suitable repository exists for hosting data in this field of study. The data that support the findings of this study are available upon reasonable request from the authors.

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