

Association between daily maximum temperature and immediate-to-delayed gout flare hospitalisations: a population-level time series study in metropolitan Perth, Australia

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Abstract

Background

Ambient temperature may alter the risk of gout flare. We assessed the association between daily maximum temperature on the immediate-to-delayed (*lag*) gout flare hospitalisations at the population-level.

Methods

Data were extracted from the Western Australian Hospital Morbidity Data Collection for this time series study for metropolitan Perth (Australia) from 1980–2014, with meteorological data obtained from the Scientific Information for Land Owners dataset. We examined the association (relative risk [RR] and 95% confidence intervals [CI]) between gout flare hospitalisations, daily maximum temperature and lag days since exposure using quasi-Poisson regression and the *distributed lag non-linear model*.

Results

Average daily maximum temperature was 24°C (5th percentile=17°C; 95th percentile=36°C). There were 6.2 and 6.8 gout flare hospitalisations per 1,000,000 population, from lag day 0 to lag day 21 following exposure to maximum temperatures of 17°C and 36°C, respectively. Risk of gout flare hospitalisations (reference=24°C) with maximum temperature was modified by sex and age. Males aged ≥75 years had higher risk of gout flare hospitalisations following hotter days (36°C), beginning immediately on lag day 0 (RR=1.39; 95% CI: 1.02–1.88) and from lag day 7 (RR=1.11; 95% CI: 1.01–1.21) to lag day 10 after colder days (≤15°C). Females aged ≥75 years had higher risk from lag day 3 (RR=1.12; 95% CI: 1.02–1.23) to lag day 4 after a 35°C day. Males aged <75 years had higher risks from lag day 6 (RR=1.06; 95% CI: 1.01–1.12) to lag day 8 after 35°C days but lower risk from lag day 7 (RR=0.96; 95% CI: 0.92–0.99) to lag day 8 following colder days (17°C).

Conclusion

Our study shows associations between extreme temperatures, both hot and cold, and immediate-to-delayed gout flare hospitalisations. Our findings will inform and guide public health measures and health system preparedness during the impending extremes of temperature with particular attention to older people.

Keywords

hospital data; meteorological data; gout flare; cold temperature/*adverse effects; hot temperature/*adverse effects; sex differences; age differences; environmental exposure; epidemiologic methods; risk assessment/methods; statistical models: Australia

Highlights

- Theorised biological mechanisms and small-scale observational studies suggest that temperature extremes can increase the risk of gout flares.
- Using population-level data, we show that the risk of gout flare hospitalisations was modified by sex and age, with older (≥75 years) and younger (<75 years) males and females showing different trends.
- Increased risk of gout flare hospitalisations can occur immediately or between 3 to 10 days after exposure to temperature extremes.
- Following days with maximum temperatures ≥35°C, males aged ≥75 years, females aged ≥75 years and males aged <75 years had 39%, 12% and 6% increased risk of gout flare hospitalisations relative to daily maximum temperatures of 24°C, respectively.
- Following colder days (maximum temperature ≤17°C), only males aged ≥75 years experienced an increased risk (11%) of gout flare hospitalisations relative to 24°C, and this occurred 7 to 10 days after exposure.
- Our findings will inform and guide public health measures and health system preparedness to mitigate the impacts of the anticipated increases in intensity, frequency and duration of temperature extremes associated with climate change, with particular attention to older people, such as those aged ≥75 years.

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Introduction

The prevalence and incidence of gout has increased in many developed countries over the last few decades due to increasing longevity, increased use of medicines that can trigger gout, dietary trends, greater obesity and increased survival from many inflammatory-related comorbidities [1, 2]. The number of people experiencing gout flares is expected to further increase due to the impact of climate change where extremes of temperature, both hot and cold, are expected to increase in intensity, frequency and duration [3]. This is supported by theorised biological mechanisms which propose that the reduced solubility of urate crystals at cold temperatures could result in micro-crystal precipitation which would trigger an inflammatory response [4, 5]. Higher temperatures can cause volume depletion and contribute to metabolic acidosis, both of which can decrease renal urate excretion and increase serum urate. Evidence from observational studies supports the impact of temperature on gout flare. A study based on self-reports from 632 participants in the United States (US) found that higher temperatures were associated with a 40% increased risk of gout flare compared to moderate temperatures [6]. In a smaller analysis of hospital records for 82 patients in Israel, high temperatures (i.e. temperature above mean monthly temperature) in the preceding four days were associated with hospitalisation for gout flare [7]. Studies on seasonality have been inconclusive with higher risks observed during the summer months in England and Wales [8] and during the spring months in Italy [9]. Meanwhile a study of new prescriptions for urate lowering medicines to treat asymptomatic hyperuricemia or gout in Japan (where these medicines are approved for these indications) found higher prescription rates in summer and autumn compared to winter [10].

To the best of our knowledge, no study has examined the association between ambient temperature and gout flare hospitalisations at the population level. In this study, we used population-level data to assess the association between daily maximum temperature on the immediate-to-delayed risk of gout flare hospitalisations in a metropolitan city. We also determined if this association was modified by sex and/or age because males and females of different ages are known to have different risks for gout flare [11–13]. Findings from this study will inform and guide public health measures and health system preparedness during the impending extremes of temperature associated with climate change.

Methods

Data sources

We used the Western Australian Rheumatic Disease Epidemiological Registry (WARDER) to access unit-record linked data from the Western Australian Hospital Morbidity Data Collection (HMDC). This data collection is maintained by the Western Australian Department of Health and linked through the Western Australian Data Linkage System (WADLS). WARDER contains all public and private hospital admissions between 1980 and 2014 for patients with principal

and secondary discharge diagnosis of systemic autoimmune rheumatic disease, gout, or osteoarthritis [14].

Study setting

Metropolitan Perth in Western Australia (WA) is bounded by the Indian Ocean on the west and the Darling Range on the east. It is situated on relatively flat land spanning (at the time of writing) approximately 150km from north to south and up to approximately 45km, from east to west. This region experiences a hot-summer Mediterranean climate and cool to mild wet winters [15].

Hospitalisation data

For this time series study, we used the HMDC dataset to identify daily hospitalisations to public and private hospitals where the principal diagnosis was gout (International Classification of Diseases, Ninth Revision, Clinical Modification [ICD-9-CM] code 274 and the International Classification of Diseases Tenth Revision, Australian Modification [ICD-10-AM] code M10) among residents of the Perth metropolitan area (identified from residential postcode) between 1 January 1980 and 31 December 2014 (observation period). We excluded patient transfers where the interval between discharge and subsequent admission for the patient was within one day consistent with our previous work [16, 17]. During this period, the estimated resident population was 1.02 million in 1980 and increased to 2.01 million in 2014.

Meteorological data

We obtained daily maximum temperature data for the Perth Regional Office station (latitude=31.96°S; longitude=115.87°E; elevation=19.0 m above mean sea level) from the Scientific Information for Land Owners (SILO) dataset in Australia, hosted by the Science and Technology Division of Queensland Government's Department of Environment and Science [18]. This was the only station in the metropolitan area with temperature data from 1980 to 2014. Data available from this weather station included daily maximum/minimum temperatures, rainfall, vapour pressure, vapour pressure deficit, evaporation, solar radiation, relative humidity at maximum temperature, relative humidity at minimum temperature, mean sea level pressure.

Other data

Data on WA public holidays were obtained from Nager.Date [19] and used to control for public holidays in the regression model. Estimated resident population data for population denominators were obtained from the Australian Bureau of Statistics [20].

Statistical analyses

We calculated descriptive statistics for daily maximum temperature (mean, standard deviation [SD], median, 1st, 5th, 95th, 99th percentiles) and number of daily gout flare hospitalisations for the observation period. Colder days are defined as those $\leq 5^{\text{th}}$ percentile of daily maximum

temperatures, while hotter days are those $\geq 95^{\text{th}}$ percentile of daily maximum temperatures.

In addition, for each day during our observation period with a particular temperature value (e.g. 16°C) we counted the number of gout flare hospitalisations per 1,000,000 population over lag days 0-21. We then averaged these counts/1,000,000 population for each temperature value.

Risk of immediate-to-delayed gout flare hospitalisations by daily maximum temperature

We used a distributed lag non-linear model (DLNM) [21] to investigate the relative risk of gout flare hospitalisations with maximum daily temperature, allowing for potential non-linear associations as well as lagged effects. This model was selected to account for the potential lag period between temperature exposure and gout flare hospitalisations [7]. Specifically, a cross-basis function, derived as a special tensor product of natural cubic splines, was used to jointly model maximum daily temperature ($^{\circ}\text{C}$) and the lag of the number of days since that exposure. Spline parameters were consistent with another Australian study [22]. Spline 1 of the cross-basis function was specified using three internal knots placed at equally spaced values of temperature, and spline 2 using an intercept plus three internal knots placed at equally spaced values of the logged lag days with a maximum lag of 21 days [22]. We used the mean of the daily maximum temperatures across all years 1980-2014 as the reference temperature (24°C) for calculating relative risks (RRs). Furthermore, a spline term was included to control for changes with time during the study period by modelling day number as an integer, from 1 (1 January 1980) to '12784' (31 December 2014) using a natural spline with 6 degrees of freedom (df) per year [22]. Three additional model terms were included: daily humidity (%), modelled using a natural spline with 4 df; weekend day (yes or no); public holiday (yes or no). This was fitted as a quasi-Poisson Generalized Linear Model (GLM) with a log link function. We adjusted for weekends and public holidays to account for differences in health service utilisation and patient behaviours on these days [23].

We initially analysed all cases and then stratified by biological sex (hereafter sex) and age (<75 years, ≥ 75 years). We chose this age cut-off as it is commonly used in age-related analyses [8, 24, 25]. Furthermore, we explored using 54 and 64 years as age cut-offs, however there was only a small number of total hospitalisations in females aged 54-64 years (<90 hospitalisations) to produce a stable model fit and estimates. However, there were sufficient numbers of total hospitalisations for both males and females in the <75 -year and ≥ 75 -year age groups for the statistical modelling and convergence. Due to the small number of days with hot and cold extremes ($<1\%$ each), temperatures $<15^{\circ}\text{C}$ were recoded to 15°C while those $>40^{\circ}\text{C}$ were recoded to 40°C .

We used Stata for data handling and preparation and R for DLNM and GLM. DLNM was fitted using the *dlnm* package in R [26].

Sensitivity analyses

In sensitivity analyses to determine the robustness of our results, we increased the number of internal knots for Splines 1

and 2 from three to four. We also increased the df for 'changes with time' from 6 to 7 df per year, and for 'daily humidity' from 4 to 5 df.

Ethics

Approval to conduct this study was obtained from the Human Research Ethics Committee of WA Department of Health (approval no. 2016/24) where a waiver of consent was granted as the research met the criteria outlined in the National Statement on Ethical Conduct in Human Research. Analyses were conducted according to relevant local and national guidelines and regulations.

Results

Descriptive statistics

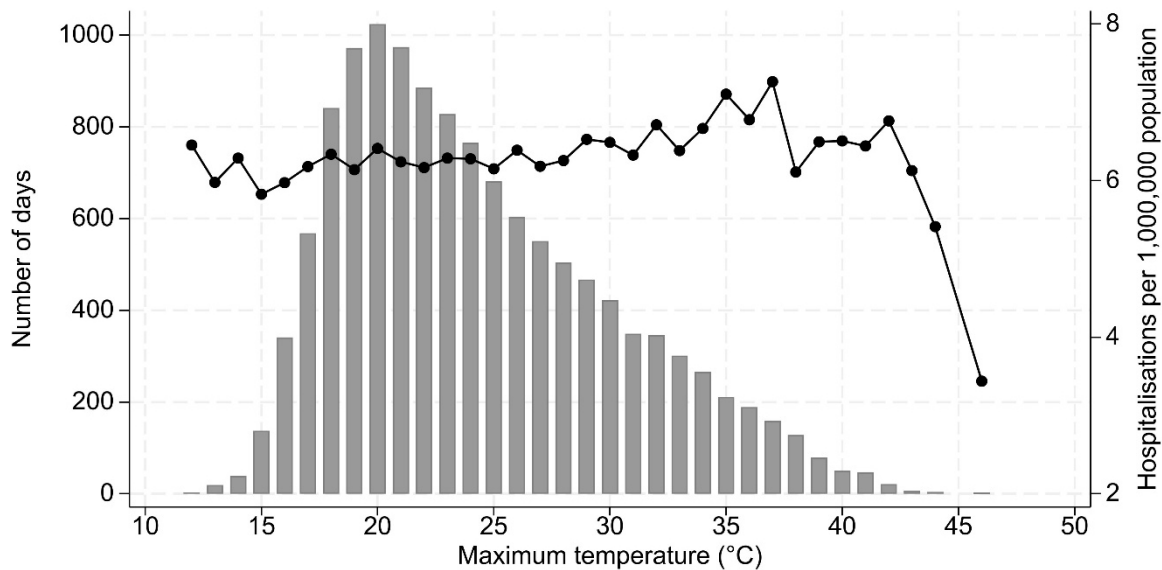
During the study observation period of 12,784 days, daily maximum temperatures ranged from 12°C to 46°C , with mean and median of 24°C ($\text{SD}=5.9$) and 23°C , respectively (Figure 1). First, 5^{th} , 95^{th} and 99^{th} percentile daily maximum temperatures were 15°C , 17°C , 36°C and 40°C respectively. Hence in this study, colder and hotter days are when daily maximum temperatures are $\leq 17^{\circ}\text{C}$ and $\geq 36^{\circ}\text{C}$, respectively.

There were 5,731 gout flare hospitalisations, and in general, the number of admissions increased with higher temperatures (Figure 1). For example, there were, on average, 5.8, 6.2, 6.8 and 6.5 hospitalisations per 1,000,000 population over lag days 0-21 (i.e. from lag day 0 to lag day 21) following exposure to daily maximum temperatures of 15°C , 17°C , 36°C and 40°C respectively. Similarly, the number of gout flare hospitalisations increased with higher temperatures amongst older (≥ 75 years) and younger (<75 years) males and females (Supplementary Table 1). However, these are crude counts and do not consider the complex exposure-time-outcome relationship. Findings hereafter are based on DLNM which provide a comprehensive picture of the time-course of the exposure-outcome relationship [27] and considers possible effect modification due to sex and age.

Risk of immediate-to-delayed gout flare hospitalisations by daily maximum temperature

Significant exposure-time-outcome associations were observed for the risk of gout flare hospitalisations with daily maximum temperature (i.e., relative to the reference temperature of 24°C) in all data, and separately by sex and age group (<75 years, ≥ 75 years) (Figure 2). At lag day 7, (i.e. 7 days following temperature exposure), hotter days (36°C) were associated with increased risk of gout flare hospitalisations in all data ($\text{RR}=1.04$; 95% CI: 1.01-1.08), with a similar RR estimated for males only ($\text{RR}=1.08$; 95% CI: 1.01-1.15) but no significant association estimated for females. People aged ≥ 75 years had higher estimated RRs following both colder ($\leq 15^{\circ}\text{C}$: $\text{RR}=1.08$; 95% CI: 1.01-1.16) and hotter days (36°C : $\text{RR}=1.07$; 95% CI: 1.02-1.12), relative to the reference temperature (Figure 2). However, those younger than 75 years had higher but non-significant estimated RRs on lag day 7 on hotter days (36°C : $\text{RR}=1.03$; 95% CI:

Figure 1: Distribution of daily maximum temperatures (bar) and gout flare hospitalisations per 1,000,000 population (line) over lag days 0-21 from 1980 to 2014



0.99-1.07), relative to the reference temperature. Given these findings, results presented hereafter are stratified by sex and age (i.e. males aged ≥ 75 years [1,465 hospitalisations], females aged ≥ 75 years [869 hospitalisations], males aged < 75 years [2,936 hospitalisations] and females aged < 75 years [461 hospitalisations]).

Males aged ≥ 75 years had higher risk of gout flare hospitalisations (Figure 3, Supplementary Figure 1, Supplementary Table 2) following hotter days (36°C) beginning immediately on lag day 0 ($\text{RR}=1.39$; 95% CI: 1.02-1.88) and again from lag day 4 ($\text{RR}=1.10$; 95% CI: 1.02-1.18) to lag day 8 ($\text{RR}=1.06$; 95% CI: 1.01-1.12). They also experienced higher risk of gout flare hospitalisations from lag day 7 ($\text{RR}=1.11$; 95% CI: 1.01-1.21) to lag day 10 ($\text{RR}=1.09$; 95% CI: 1.01-1.18) after colder days ($\leq 15^{\circ}\text{C}$). Females aged ≥ 75 years did not experience increased risk of gout flare hospitalisations following hotter ($\geq 36^{\circ}\text{C}$) days but had higher risk at 35°C from lag day 3 ($\text{RR}=1.12$; 95% CI: 1.02-1.23) to lag day 4 ($\text{RR}=1.11$; 95% CI: 1.01-1.22).

Males aged < 75 years (Figure 4, Supplementary Figure 2, Supplementary Table 2) did not experience increased risk after hotter days ($\geq 36^{\circ}\text{C}$) but had higher risks due to 35°C days from lag day 6 ($\text{RR}=1.06$; 95% CI: 1.01-1.12) to lag day 8 ($\text{RR}=1.05$; 95% CI: 1.01-1.09). They had higher but non-significant risks following colder days (17°C) from lag day 1 ($\text{RR}=1.04$; 95% CI: 0.96-1.13) to lag day 3 ($\text{RR}=1.01$; 95% CI: 0.95-1.06), but significant lower risk from lag day 7 ($\text{RR}=0.96$; 95% CI: 0.92-0.99) to lag day 8 ($\text{RR}=0.96$; 95% CI: 0.93-0.99). Females aged < 75 years did not experience higher risk of gout flare hospitalisations after hotter or colder days over lag day 0 to lag day 21, relative to the reference temperature.

Sensitivity analyses

In sensitivity analyses to determine robustness of our results, we observed similar shapes of exposure-time-outcome associations when we modified the number of knots for Splines

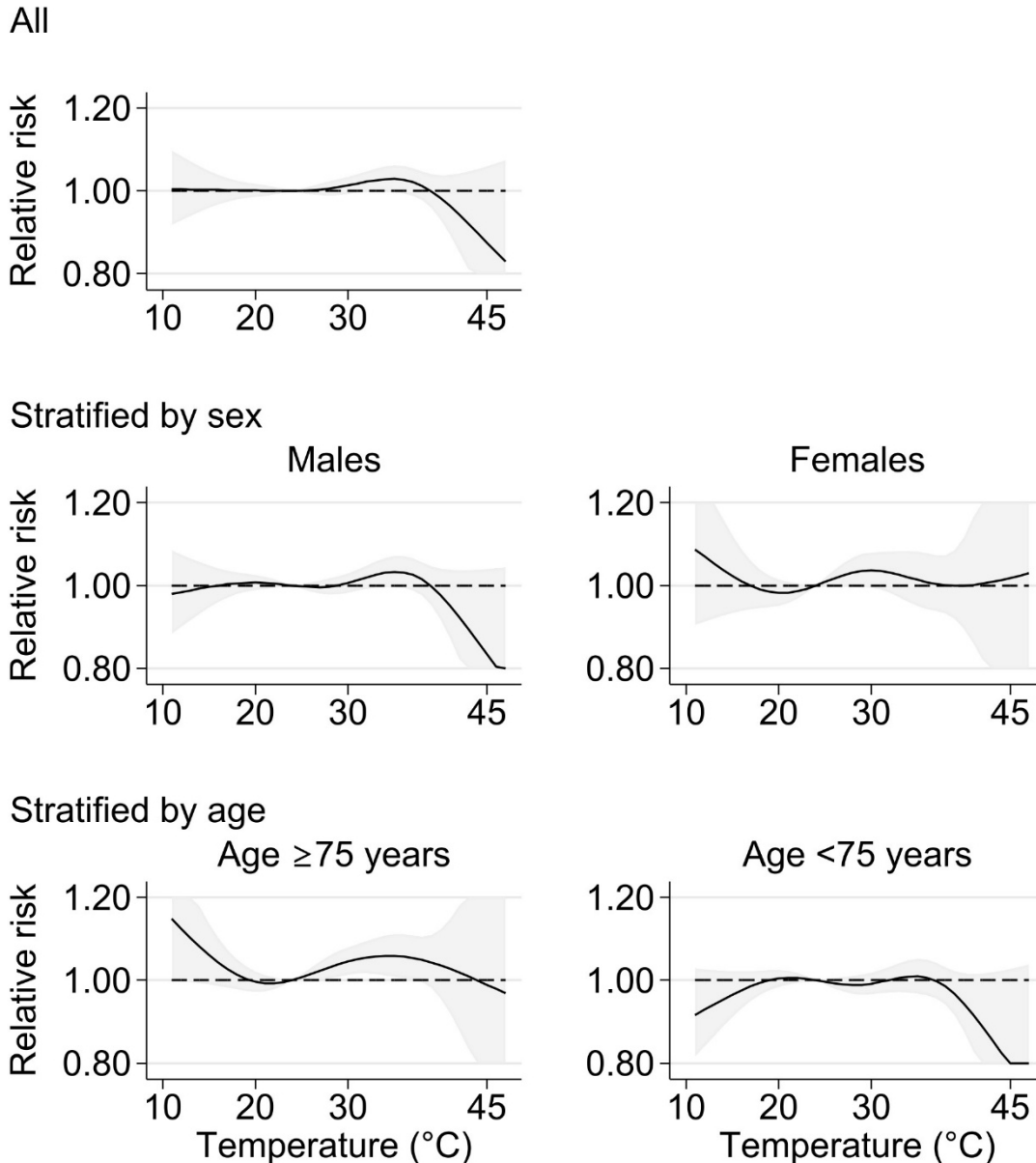
1 and 2 and df for 'changes with time' and 'daily humidity' (Supplementary Figures 3-6).

Discussion

In this first of its kind population-level study of gout flare hospitalisations following exposure to temperature extremes, our findings align with theorised biological mechanisms and earlier small-scale observational studies indicating hot [6, 7] and cold [4, 5] temperatures are associated with increased risk of gout flares. We found that the risk of gout flare hospitalisations was modified by the sex and age of admitted patients, with older (≥ 75 years) and younger (< 75 years) males and females showing different trends. The risk of gout flare hospitalisations was 39%, 12% and 6% higher following days with maximum temperatures $\geq 35^{\circ}\text{C}$, relative to reference temperature days (daily maximum= 24°C) for males aged ≥ 75 years, females aged ≥ 75 years and males aged < 75 years, respectively. Only males aged ≥ 75 years experienced increased risk (11%) of gout flare hospitalisations after exposure to cold temperatures and this occurred 7 to 10 days after exposure. We also found that increased risk of gout flare hospitalisations can occur immediately or between 3 to 10 days after exposure to temperature extremes. Our study adds to the number of conditions that have increased risks of morbidity during temperature extremes including cardiovascular disease, kidney disease, diabetes and mental health conditions [22, 28–30].

Our observations from this population dataset can be explained by physiological, lifestyle and social factors. Older people have a greater number of risk factors for gout flares during temperature extremes including higher levels of serum uric acid, poorer thermoregulation and more likely to consume alcohol daily (e.g. 12.6% of those aged ≥ 70 years consumed alcohol daily compared to 7.3% aged 50-59 years) than younger people [12, 31, 32]. We observed that males aged ≥ 75 years experienced a 39% increased risk of gout flare hospitalisations following hotter days (daily maximum

Figure 2: Relative risk of gout flare hospitalisations plotted against maximum temperature



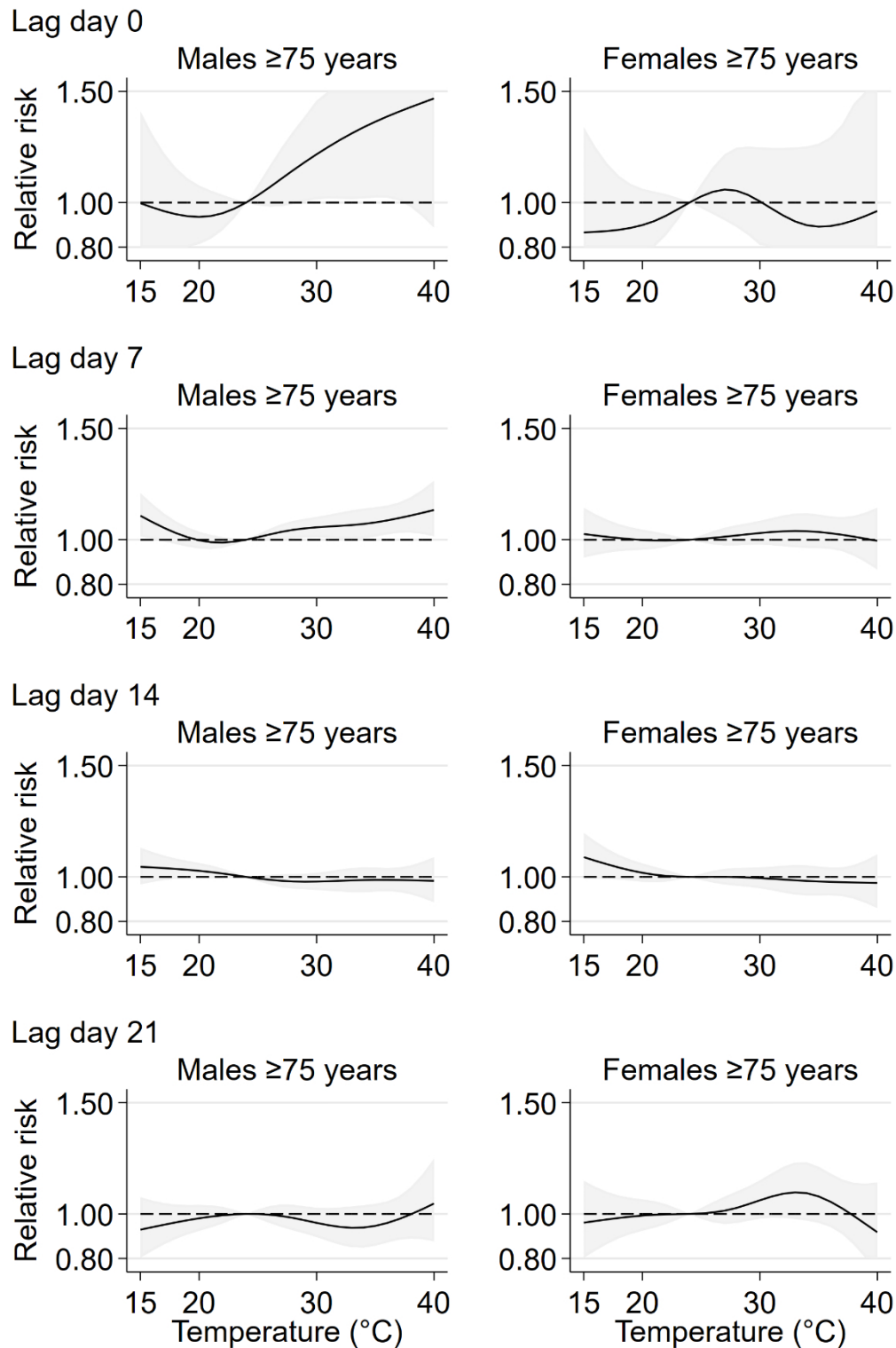
Plots are for lag day 7 and presented for all cases and stratified by sex and age. Reference temperature= 24°C . For ease of display, relative risks and 95% confidence intervals (grey shading) have been capped at 0.80 and 1.20.

temperature $\geq 36^{\circ}\text{C}$) and with a shorter lag time (on lag day 0 vs lag day 3, respectively) following exposure than females in the same age group, relative to the reference temperature (24°C). Additionally, elderly males but not elderly females had increased risk of gout flare hospitalisations following colder days (daily maximum temperature $\leq 17^{\circ}\text{C}$) relative to the reference temperature. It is possible that during our study period, elderly males were more likely to be involved in outdoor activities (e.g. gardening, fishing) [33–35] during the hot and cold temperatures than elderly females, thus increasing exposure which may predispose them to a higher risk of gout flare. In contrast, younger (< 75 years) males, but not females, experienced increased risk of gout flare hospitalisations only following hotter days, relative to the reference temperature. Males < 75 years are perhaps more likely to be involved in outdoor occupations (e.g. farming, construction work) [36, 37] and consume alcohol daily (e.g. 8.8% of males aged 50–59

years consumed alcohol daily compared to 5.8% of females aged 50–59 years) [32] than females in this age group. We also observed a small (4%) but statistically significant decrease in risk of gout flare hospitalisations on lag day 7 to lag day 8 following colder days for males aged < 75 years relative to the reference temperature. This suggests possible ‘harvesting’, a phenomenon where outcomes are only brought forward by a brief period of time by the effect of exposure. Here, the non-significant 1–4% increases in risk of hospitalisations on lag day 1 to lag day 3 resulted in later decreases in risk from lag day 7 to lag day 8.

Whilst our findings appear consistent with earlier papers, there are some notable differences apart from different populations and climate zones. A study in the US using a time-stratified case-crossover study design found that hot but not cold temperatures increased this risk of gout flares [6]. Unlike our population-level study, their participants were respondents

Figure 3: Relative risk of gout flare hospitalisations plotted against maximum temperature for lag days 0, 7, 14 and 21, for people aged ≥ 75 years

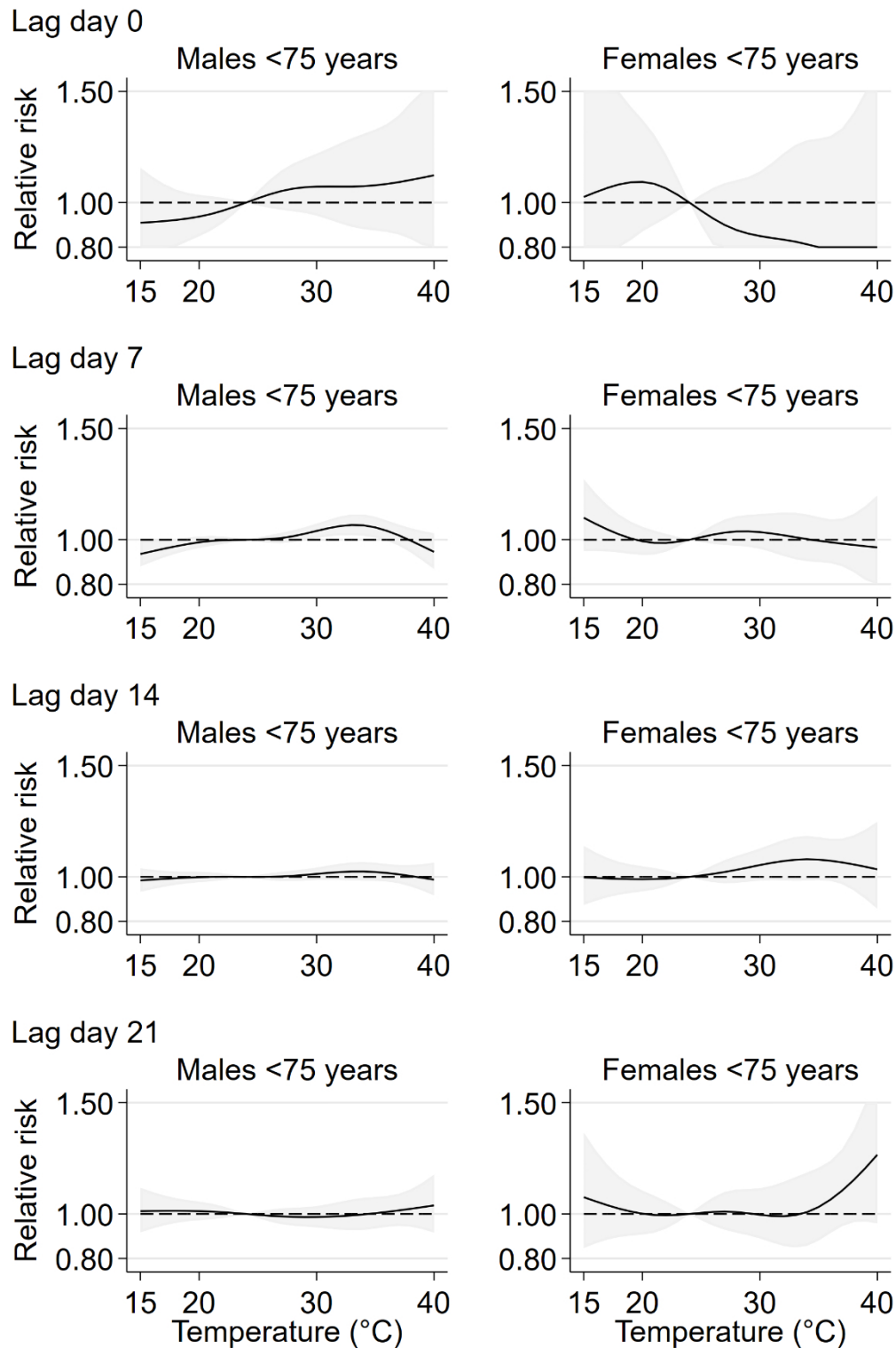


Plots are stratified by sex and age. Reference temperature=24°C. For ease of display, relative risks and 95% confidence intervals (grey shading) have been capped at 0.80 and 1.50.

to a search engine identifying the search term “gout” and were directed to the study website to provide self-reported information on their gout condition. It is likely that their study excluded many elderly patients who were less likely to regularly use the internet [38]. Furthermore, that study did not

stratify by sex and age, as was performed in the current study. Without stratification, we found that cold temperatures were not associated with the risk of hospitalisation for gout flare suggesting that failure to stratify by sex and age will mask the effect modification.

Figure 4: Relative risk of gout flare hospitalisations plotted against maximum temperature for lag days 0, 7, 14 and 21, for people aged <75 years



Plots are stratified by sex and age. Reference temperature=24°C. For ease of display, relative risks and 95% confidence intervals (grey shading) have been capped at 0.80 and 1.50.

The strength of this study was its use of de-identified individual-level hospital data for an entire metropolitan population, which allowed systematic ascertainment of all public and private hospital admissions for gout flare ensuring

complete identification of cases. This reduces issues with selection bias as well as reporting or recall bias in terms of experiencing a gout flare, as potentially experienced with other research [39]. There are several limitations to this study.

Firstly, we only used hospitalisation data and did not have primary healthcare data which would have captured less severe gout flares that did not require hospitalisation. Although we did not use mortality data, we do not expect this to influence our findings as deaths related to gout represented 0.3% of all deaths in Australia [40]. Secondly, we used temperature data from the Perth Regional Office station, which is higher than coastal areas and lower than more inland regions. However, a previous time-series modelling study showed that using temperature data from a single site produced similar temperature-health estimates compared to models that used averaged temperature or spatio-temporal data [41]. Thirdly, our findings are from a single Australian region with distinct topography and maximum temperatures (range 12-46°C) and may not be generalisable to other jurisdictions. Each jurisdiction will need to evaluate the risk separately and our study using population-level data in conjunction with DLNM offers a viable approach. Fourthly, due to small case numbers, we were unable to stratify by smaller age groups. Fifthly, whilst we did not adjust for air quality, earlier studies have shown minimal confounding by air pollution [42, 43]. Moreover, adjustment for air quality in temperature studies is usually not warranted unless the causal inference assumption is firstly established [44]. Finally, our data are up to the end of 2014 and findings may be different with more recent data where more extremes in temperature may have occurred in more recent years. Although there are now new treatments for gout [45] and modern buildings are designed to accommodate temperature fluctuations, the number of hospital admissions for gout flare in Australia has increased modestly from 25 per 100,000 population in 2011/12 to 27 per 100,000 population in 2021/22 [40].

In conclusion, our study shows associations between extreme ambient temperatures, both hot and cold, and gout flare hospitalisations in metropolitan Perth. Our findings will inform and guide public health measures and health system preparedness during the impending extremes of temperature associated with climate change with particular attention to the older population, such as males and females aged ≥ 75 years.

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Author contributions

All authors were involved in drafting the article or revising it critically for important intellectual content, and all authors approved the final version to be published. Derrick Lopez had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study conception and design. Lopez

Analysis and interpretation of data. Lopez, Marriott

Acquisition of data. Nossent, Preen, Keen, Inderjeeth

Data sharing statement

The datasets generated and/or analysed during the current study are not publicly available due to the terms of the ethics approval granted by the Western Australian Department of Health Human Research Ethics Committee (WADOH HREC) and data disclosure policies of the Data Providers. The datasets may be available from the corresponding author upon request and subject to approval from the WADOH HREC and relevant custodians.

Conflicts of interest

DL and DBP are on the Editorial Board of this journal and had no role in the editorial process of this manuscript. All other authors have no conflicts of interest to report.

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