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The effects of ambient temperature and heatwaves on daily *Campylobacter* cases in Adelaide, Australia, 1990–2012

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SUMMARY

Campylobacter spp. is a commonly reported food-borne disease with major consequences for morbidity. In conjunction with predicted increases in temperature, proliferation in the survival of microorganisms in hotter environments is expected. This is likely to lead, in turn, to an increase in contamination of food and water and a rise in numbers of cases of infectious gastroenteritis. This study assessed the relationship of *Campylobacter* spp. with temperature and heatwaves, in Adelaide, South Australia.

We estimated the effect of (i) maximum temperature and (ii) heatwaves on daily *Campylobacter* cases during the warm seasons (1 October to 31 March) from 1990 to 2012 using Poisson regression models.

There was no evidence of a substantive effect of maximum temperature per 1 °C rise (incidence rate ratio (IRR) 0·995, 95% confidence interval (95% CI) 0·993–0·997) nor heatwaves (IRR 0·906, 95% CI 0·800–1·026) on *Campylobacter* cases. In relation to heatwave intensity, which is the daily maximum temperature during a heatwave, notifications decreased by 19% within a temperature range of 39–40·9 °C (IRR 0·811, 95% CI 0·692–0·952). We found little evidence of an increase in risk and lack of association between *Campylobacter* cases and temperature or heatwaves in the warm seasons. Heatwave intensity may play a role in that notifications decreased with higher temperatures. Further examination of the role of behavioural and environmental factors in an effort to reduce the risk of increased *Campylobacter* cases is warranted.

Key words: *Campylobacter*, climate, impact of food-borne infections, infectious disease epidemiology.

INTRODUCTION

Changes in climatic conditions, such as warmer ambient temperature and increased frequency in heatwaves, are considered to be contributing factors to the emergence and re-emergence of infectious diseases [1]. Infectious gastrointestinal diseases, including those that are food-borne, are influenced by weather conditions, and this has been evident with increased cases and outbreaks of salmonellosis linked to elevated ambient temperature [2–5]. While the relationship with ambient warmer temperature and increased numbers of cases of *Salmonella* infection has been established, *Campylobacter* infections

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have a less clear relationship with temperature and climate variability. Some studies report a positive association of increasing temperature and incidence of cases [6–13] and others an inverse or no relationship [14–16]. Of the studies, most have been reported from Europe, England and Wales [8–11, 13, 15], the USA and Canada [6, 7, 12]. Two studies have examined data from Australasia: one was a multi-city study [15], and the other compared Adelaide (capital city of South Australia (SA)) and Brisbane (capital city of Queensland) [14].

Human infection with *Campylobacter* spp. is an important cause of food-borne illness, with major consequences for morbidity in individuals and populations [17]. In 2010, of the 550 million infectious gastroenteritis cases reported in the world, 96 million were attributed to *Campylobacter* spp. the most common bacterial infectious agent [17]. In Australia, of the estimated 16·6 million cases of acute gastroenteritis, 4·7% of cases was caused by *Campylobacter* spp. [18]. In SA, around 2000 *Campylobacter* notifications occur each year. Heatwaves, with longer and higher temperatures, may increase the development pace of the pathogen, affect the reservoirs and also impact people's behaviour, including food storage and transportation. With temperature and the frequency of heatwaves predicted to rise, it is important to assess whether these climatic variables have an impact on *Campylobacter* notifications.

The aim of this study was to assess the relationship between reported cases of *Campylobacter* spp., and temperature and heatwaves. The findings will inform the design of public health messages and interventions aimed at improvements in food safety, prevention and control in an effort to reduce burden associated with warmer ambient temperature due to climate change.

METHODS

Adelaide experiences a Mediterranean climate with cool wet winters and hot dry summers with high temperatures in the warm season and heatwaves becoming more frequent, intense and of longer duration [19]. The average ambient temperature in SA has increased by 0·96 °C in the last 95 years. This increase in maximum temperature is occurring more rapidly than that observed nationally [20].

Data collection

Notifiable cases

Laboratory-confirmed *Campylobacter* cases notified to the Communicable Disease Control Branch (CDCB), SA Department for Health and Ageing between 1 January 1990 and 31 December 2012 were obtained from their notifiable disease surveillance system. Cases were included if they were a resident of metropolitan Adelaide. Information on demographic characteristics and illness was extracted for each case.

Temperature data

Recordings of daily maximum temperature (T_{max}) in degrees Celsius (°C) from 1990 to 2012 were obtained from an Australian Bureau of Meteorology (BOM) weather monitoring station close to the Adelaide city centre. We used T_{max} as our exposure variable because it is considered to be a better index of exposure than average or minimum temperature [3, 21]. This was also in keeping with previous studies in Adelaide, in which T_{max} was used as a predictor of health outcomes related to heat exposure [22, 23].

Heatwave definition

In this study, a heatwave was defined to have occurred when the daily T_{max} reached or exceeded 35 °C for 3 or more consecutive days in a given period. This definition has been applied in previous heat-health studies conducted in Adelaide [4, 22–25]. Because of the uncertainty about the characteristics that make heatwaves hazardous to health [26], we assessed the role of intensity (T_{max} of $\geqslant$35 °C during a heatwave event), duration (the length of a heatwave, in number of days) and timing (occurrence of a heatwave according to timing within the season, e.g. first, second and so on) on daily *Campylobacter* notifications.

ANALYSIS

Temperature effects

A time-series Poisson regression model was fit to estimate the effect of T_{max} on daily *Campylobacter* cases. The analysis dataset was restricted to the warm season from 1 October to 31 March, to control for the potential confounding effects of seasonal fluctuations [27].

We used Spearman's correlation coefficient to examine the relationship between T_{max} and the daily number of *Campylobacter* notifications in the warm season. To identify any delayed effects of T_{max} on daily *Campylobacter* notifications, we performed cross-correlation analyses and examined different lags in time. Sensitivity analyses of different lag times up to

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28 days were conducted based on the cross-correlation results and were taken into account in the regression models. We controlled for autocorrelation (AC) of daily *Campylobacter* notifications based on the autocorrelation function (ACF) and partial ACF so as to identify the most appropriate autoregressive (AR) order.

To take into account potential confounders of temperature effects, we included day of the week (as a categorical variable with Sunday as the reference day), public holidays (as an indicator variable), and linear and quadratic terms for year to adjust for long-term trends. Lag values were also included to estimate the delayed effects of temperature on daily *Campylobacter* notifications. In the case of overdispersion, a negative binomial model was fitted. Goodness-of-fit tests were used to assess model fit.

Different temperature thresholds were examined to ascertain if a differential relationship across the temperature spectrum existed for a number of *Campylobacter* cases. A lowess smoother at a bandwidth of 0·8 was used to assess the shape of the exposure–response relationship between T_{max} and counts of *Campylobacter*. Piecewise linear regression models were fitted with a single breakpoint at the identified temperature thresholds using the 'hockey-stick' nl command in Stata [28].

Heatwave effects

Poison regression models were used to examine the effect of heatwaves on daily *Campylobacter* cases. Generalized estimating equations were used to account for the clustering of observations within a heatwave. We accepted an exchangeable correlation structure within each cluster of heatwave days and used the quasi-likelihood under the independence model criterion to select the best working correlation structure. Similar to the models we used to assess the effect of T_{max} on daily *Campylobacter* notifications, we included day of the week, public holidays and year and year^2 in our statistical models.

Heatwave characteristics

As well as examining each of the three heatwave characteristics (intensity, duration and timing, as described earlier), we also estimated the overall effects of heatwaves on daily notifications by including a binary variable (heatwave and non-heatwave days). Separate models were fitted to examine the effects of each heatwave characteristic on number of daily *Campylobacter* cases. Heatwave day (e.g. days 3, 4 or 5) was used to examine the day which produced a greater risk of *Campylobacter* infection. Intensity was defined as daily T_{max} recorded within heatwaves with four temperature ranges (35–36·9, 37–38·9, 39–40·9 and $\geqslant$41 °C) included in the model. We examined duration by length of 3, 4 and 5 or more days. We also considered whether the duration was short (3 days) compared with long (4 or more days). We considered two different characteristics of timing. We defined timing by the first, second and third heatwave events within each warm season denoting the order of occurrence. We then examined whether timing differed by the occurrence of a heatwave event in the early part of the warm season (October to December) or later (January to March).

We report incidence rate ratios (IRRs) with 95% confidence intervals (CIs) with results interpreted as per cent (%) change in the number of daily *Campylobacter* counts per °C increase in T_{max} and during heatwave periods compared with non-heatwave periods. A significance level of 0·05 was used for all statistical tests. Analyses were conducted using StataSE 13 (StataCorp LP, College Station, Texas, USA).

Ethics approval

Ethics approval was given by the Human Research Ethics Committees of the University of Adelaide (H-202–2011) and the SA Department for Health and Ageing (463/07/2014).

RESULTS

Descriptive statistics

In Adelaide, from 1990 to 2012, 35 601 *Campylobacter* cases were notified, with 18 570 (52%) reporting onset of illness in the warm season. During this period, there were no outbreaks detected, and hence no records were excluded from the analyses. Figure 1 shows the temporal distribution of daily *Campylobacter* spp. notifications over the entire study period with no obvious peaks occurring in the warmer months. Temperature summary statistics for the entire study period, by season, and by heatwaves are displayed in Table 1. The mean daily T_{max} during the warm seasons was 26·5 °C (standard deviation (S.D.) = 6·1) and 38·4 °C (S.D. = 2·2) during heatwaves. Over the study period, 213 heatwave days across 50 distinct episodes were recorded. The length of heatwaves ranged from 3 to 15 days with a mean of 3·17 (S.D. = 2·40)

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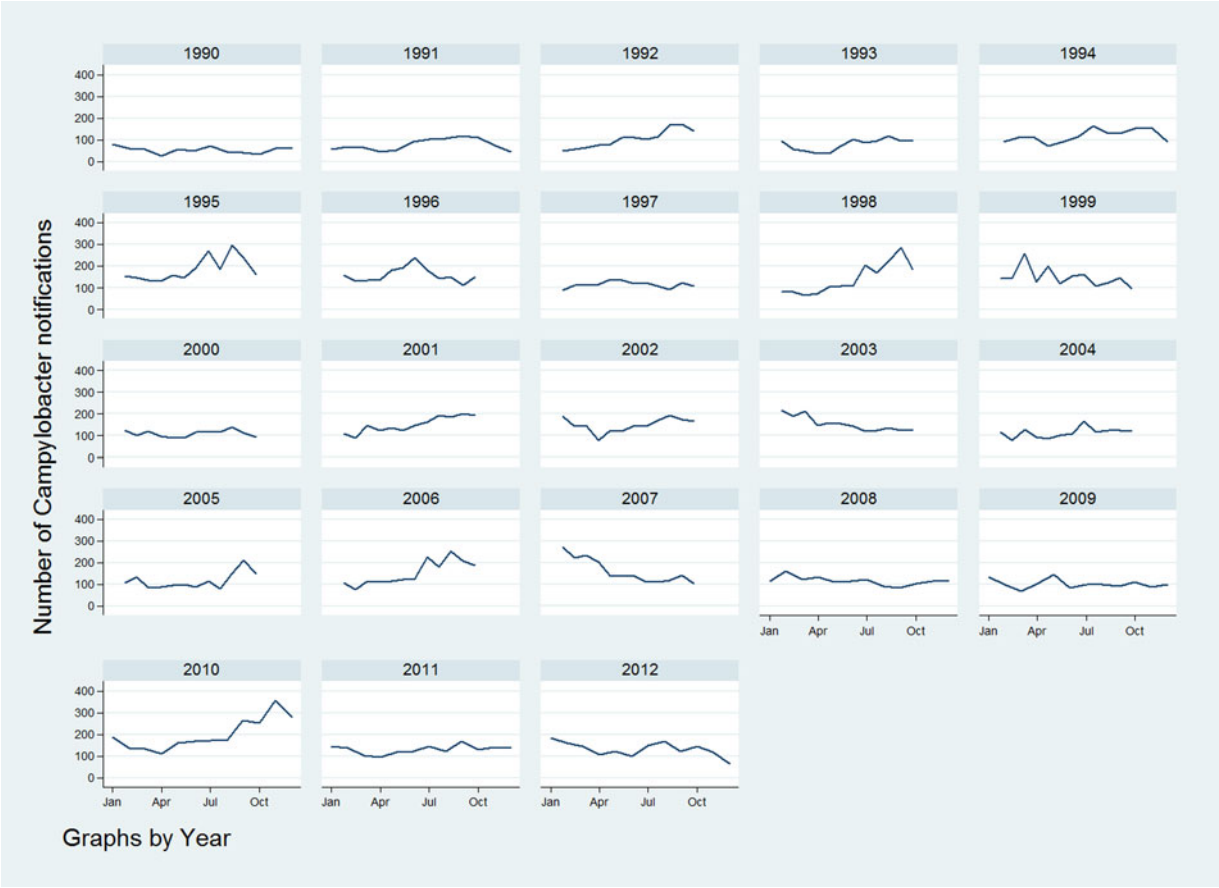

Fig. 1. Annual distribution of monthly notifications of *Campylobacter* infection, 1990–2012, Adelaide, South Australia.

days. During the study period, there were no recorded heatwaves in 1990, 1996 and 2005, and none occurred outside of the warm season. The proportion of days during the warm season with a recorded T_{max} over 40 °C was 1%.

The final time-series Poisson regression model included an AR structure of order three and daily T_{max}, based on the maximum correlation coefficients. We examined the data for overdispersion, and as there was none, negative binominal models were not fitted.

Effects of maximum temperature on *Campylobacter* infections

Correlation of T_{max} with *Campylobacter* notifications was negligible. There was no lagged effect of T_{max} on the number of daily *Campylobacter* cases. A third-order AC of the number of campylobacteriosis notifications was detected (IRR 1·037, 95% CI 1·032–1·042, $P = <0·01$). There was no substantiative effect of T_{max} per 1 °C rise (IRR 0·995, 95% CI 0·993–0·997, $P = <0·01$) on *Campylobacter* cases.

Temperature thresholds

Figure 2 demonstrates the exposure–response relationship between T_{max} and daily *Campylobacter* cases during the warm season. The relationship between temperature and *Campylobacter* notifications changed across the observed temperature range – as T_{max} increased, the number of cases decreased. However, a clear temperature threshold was not detected.

Effect of heatwaves and heatwave characteristics on *Campylobacter* notifications

As illustrated in Table 2, no association between heatwaves and an overall increase in daily *Campylobacter* cases was identified (IRR 0·906, 95% CI 0·800–1·026, $P = 0·126$). When examining heatwave characteristics, a 3-day heatwave compared with a heatwave with a duration of 4 and 5 days decreased the risk of infection on daily *Campylobacter* counts by 21% (IRR 0·795, 95% CI 0·689–0·918, $P = 0·002$). A 19% decrease in cases (IRR 0·818, 95% CI 0·679–0·987, $P = 0·036$) was estimated with the first heatwave in the season,

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Table 1. *Daily maximum temperature (T_{max}) by season, 1990 to 2012, Adelaide, South Australia*

Time period	Mean	Minimum	25th percentile	50th percentile	75th percentile	90th percentile
1990 – 2012[a]	22·3°C	9·9	17°C	21·1°C	26·6°C	32·3°C
Cool season[b]	18·2°C	9·9	15·3°C	17·2°C	20·2°C	24·1°C
Warm season[c]	26·5°C	13	21·9°C	25·7°C	31°C	35·3°C
Heatwaves[d]	38·4°C	35	36·6°C	38·3°C	39·8°C	41·5°C

[a] *Campylobacter* cases (n=35,601) notified in the study period.
[b] *Campylobacter* cases (n=17,031) notified in the cool season (April to September).
[c] *Campylobacter* cases (n=18,570) notified in the warm season (October to March).
[d] *Campylobacter* cases (n=908) notified during heatwaves (within the warm season).

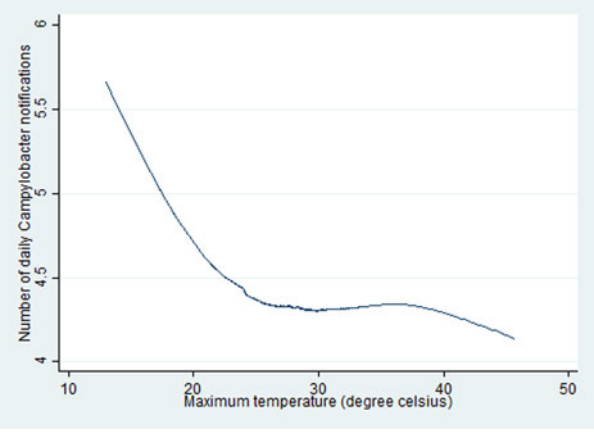

Fig. 2. Exposure–response relationship between maximum temperature and daily *Campylobacter* notifications, reported in the warm season (October to March), 1990–2012, Adelaide, South Australia.

thus reducing the risk compared with subsequent heatwaves in the season. The number of cases was lower in the early months of the warm seasons compared with the later months. Heatwave intensity within a temperature range of 39–40·9 °C on daily cases decreased the risk of infection (IRR 0·811, 95% CI 0·692–0·952, $P = 0·010$) by 19%.

DISCUSSION

Our study found that there is no substantiative effect of T_{max} on daily *Campylobacter* cases in the warm seasons. When examining the effect of heatwaves on daily *Campylobacter* notifications, there was little evidence of an effect of an increased risk of infection. These findings indicate that *Campylobacter* incidence in Adelaide may not be affected by temperature in the warm seasons or during heatwaves.

Few studies have examined the relationship between temperature and *Campylobacter* spp. in the warm seasons and none so far have considered the effects of heatwaves on cases. Among those studies that have been conducted, a positive association of increasing temperature and incidence of cases have been reported in studies from Europe [10, 13], the UK [8, 9, 11], the USA [12] and Canada [6, 7].

Our findings that temperature and heatwaves did not increase the risk of infection concur with a study in Australia comparing temperature effects and *Campylobacter* cases in Adelaide that has a temperate climate, with Brisbane a sub-tropical climate [14]. The study in Adelaide found an inverse relationship with temperature and *Campylobacter* cases, yet in Brisbane the effect was positive [14]. It is postulated that this difference could be associated with weather conditions specific to that area, which could have an impact on animal reservoirs or proccesses along the food chain [14]. Likewise a multi-jurisdictional study that compared the effects of temperature on *Campylobacter* infection across multiple continents of Europe, Canada, Australia and New Zealand did not find a strong effect of temperature on *Campylobacter* cases [15]. This suggests that the impact of temperature on cases varies within and between geographical regions, thereby affecting disease transmission and environmental routes.

We found no lagged effect of temperature in the warm seasons on cases. Contrary to this, previous studies found lags ranged from 1 to 6 weeks. A positive association was found between *Campylobacter* and temperature in the current and previous weeks in a UK study [8]. Studies that identified long lags of 8 weeks or more were those that had a null effect of temperature on *Campylobacter* cases [14–16]. Bi *et al.* reported no discernible lag effect of temperature on *Campylobacter* cases in Adelaide but a lag of up to 6 weeks on cases in Brisbane [14]. The lack of a lag effect in Bi *et al.* and our study suggests that the main route of

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Table 2. *Effect estimates of heatwave characteristics on daily Campylobacter cases*

Heatwave characteristic	IRR	(95% CI)	*P*-value
Heatwave	0·906	0·800–1·026	0·126
Day of heatwave			
Day 3	0·916	0·805–1·042	0·184
Day 4	0·882	0·723–1·077	0·221
Day 5	0·850	0·656–1·101	0·219
Duration			
Short	0·916	0·805–1·042	0·184
Long	0·873	0·724–1·053	0·157
Duration by length			
Day 3	0·795	0·689–0·918	0·002
Day 4	1·113	0·917–1·351	0·278
Day 5	0·842	0·641–1·108	0·221
Timing by order of occurrence in a season			
First heatwave	0·818	0·679–0·987	0·036
Second heatwave	0·884	0·731–1·069	0·207
Third heatwave	1·019	0·811–1·280	0·871
Timing by months in season			
Early	1·145	1·099–1·192	<0·001
Late (referent)	1		
Intensity by temperature range			
35–36·9 °C	0·938	0·803–1·095	0·421
37–38·9 °C	0·919	0·804–1·052	0·223
39–40·9 °C	0·811	0·692–0·952	0·010
⩾41 °C	0·949	0·745–1·209	0·676

IRR, incidence rate ratio; CI, confidence interval; *P*-value (0·05 significance level).
Adjusted for long-term trends, day of the week (reference day is Sunday) and public holidays. The reference group are non-heatwave days.

transmission may not be foodborne [14]. Generally, lag effects indicate when and where food contamination could have occurred, with short time lags pointing to food contamination closer to the time of consumption, and long lags indicating effects at the production processing stages [8]. In our study, we found that the number of campylobacteriosis notifications was related to the number occurring in the previous 3 days. As there was little evidence in our study of a lagged effect of temperature on daily counts in our study, we did not further explore this association with heatwaves.

We were unable to discern a temperature threshold, although by visually inspecting the plot, we observed a decrease in cases with a rise in T_{max} above approximately 36 °C. These results need to be interpreted with caution and warrant further investigation. It may be that ambient temperature in the warm seasons and heatwaves are not linked to an increase in the risk of infection as indicated by daily *Campylobacter* notifications as the bacteria is sensitive to high temperature. The relationship between pathogen growth and temperature is non-linear [29], and there could be a temperature above which proliferation and survival of *Campylobacter* spp. in the environment will begin to decline. *Campylobacter* spp. does not multiply at temperatures below 30 °C; hence, the bacteria do not increase in foods kept at usual room temperatures in temperate regions [30].

Limitations in this study are similar to those reported in our related work concerned with effects of temperature and heatwaves on *Salmonella* cases [3, 4]. Passive disease surveillance systems are likely to result in an under-reporting of *Campylobacter* notifications [31], but this is not likely to affect the estimates of the association between temperature and heatwaves in the warm seasons and the risk of *Campylobacter* infection. We did not exclude cases that travelled prior to becoming unwell because of the incomplete data recorded in the disease notification surveillance system. Only a small proportion of cases are expected to travel, and inclusion of cases that travelled is unlikely to affect the results [32].

It remains unclear as to why *Campylobacter* infections have a less obvious relationship than *Salmonella* with

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temperature and climate variability. Transmission of campylobacteriosis is complex, as there are many routes and exposure pathways including environmental paths [33, 34]. The seasonality with campylobacteriosis peaking in spring is not fully understood, but the environment is credited to play a role in this [30].

Campylobacter spp. is ubiquitous in the environment, and hosts include wild domestic animals and birds. It can be found in the gastrointestinal tract of cattle, sheep, goats, dogs, rabbits, cats, chickens, turkey, duck and pigs [35]. It is hypothesised that flies can act as a vector for the transmission of *Campylobacter* spp. to humans [36]. Food-borne transmission is another route as the primary source for *Campylobacter* infection in humans is the consumption of poultry meat [37]. A case–control study in Australia identified chicken consumption as the main risk factor for *Campylobacter* infection, with a population-attributable risk estimate of >50 000 cases each year [38]. Although *Campylobacter* spp. is sensitive to high temperatures and dry environments, the bacteria survive well in poultry processing production stages [35] further supporting poultry meat as a high-risk food for *Campylobacter* transmission to humans.

Environmental transmission of *Campylobacter* infection via birds, farm and other wild animals to humans is multi-factorial. These many routes of transmission, some which may not be temperature-dependent, are multi-faceted. From our study, as well as those conducted previously, we know that varying environmental and climatic conditions prevail between continents, countries and regions, and may have an impact on disease transmission and case incidence [14]. This calls for further studies from different countries with different climatic conditions to ascertain a truer picture of the role of temperature in the incidence of *Campylobacter* infection.

Our study suggests that temperature has a limited role in increased incidence of *Campylobacter* notifications in the warm season, and we also found little evidence for an effect of heatwaves on cases. Limited understanding about the reservoirs and transmission routes for *Campylobacter* infection make it difficult to determine the role of ambient temperature in the warm season on disease incidence. Notwithstanding this, previous studies have established that there is a relationship with temperature and consequently the role of the environment, especially warmer temperature should not be ignored with the emerging evidence of climate change and predicted increase in the frequency of warmer days.

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DECLARATION OF INTEREST

We confirm that there is no conflict of interest.

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