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Heatwaves differentially affect risk of *Salmonella* serotypes

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Summary *Objectives:* Given increasing frequency of heatwaves and growing public health concerns associated with foodborne disease, we examined the relationship between heatwaves and salmonellosis in Adelaide, Australia.
Methods: Poisson regression analysis with Generalised Estimating Equations was used to estimate the effect of heatwaves and the impact of intensity, duration and timing on salmonellosis and specific serotypes notified from 1990 to 2012. Distributed lag non-linear models were applied to assess the non-linear and delayed effects of temperature during heatwaves on *Salmonella* cases.
Results: Salmonella typhimurium PT135 notifications were sensitive to the effects of heatwaves with a twofold (IRR 2.08, 95% CI 1.14—3.79) increase in cases relative to non-heatwave days. Heatwave intensity had a significant effect on daily counts of overall salmonellosis with a 34% increase in risk of infection (IRR 1.34, 95% CI 1.01—1.78) at >41 °C. The effects of temperature during heatwaves on *Salmonella* cases and serotypes were found at lags of up to 14 days.
Conclusion: This study confirms heatwaves have a significant effect on *Salmonella* cases, and for the first time, identifies its impact on specific serotypes and phage types. These findings will contribute to the understanding of the impact of heatwaves on salmonellosis and provide insights that could mitigate their impact.

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Introduction

Human *Salmonella* infection is a significant global public health problem with an estimated 93.8 million cases per year, of which 80.3 million are foodborne.[1] In Australia with a population of approximately 22 million, the incidence of infectious gastroenteritis attributed to foodborne disease is estimated to be 4.1 million cases per year.[2] *Salmonella* is a common cause of foodborne illness in humans and is a frequently reported bacterial aetiological agent in Australia.[3] Bacterial infectious agents such as *Salmonella* that cause foodborne disease are sensitive to temperature variability, and in warmer temperatures the rate of replication is high, with *Salmonella* proliferation occurring more rapidly in animal guts and food.[4]

Adelaide, the capital city of South Australia (SA) experiences a Mediterranean climate with mild winters and hot, dry summers. Heatwaves in SA have increased in frequency since 1950.[5] If the effects of climate change continue to accumulate, coupled with the prediction of increasing heatwave events in Australia and worldwide, then the increase in risk for foodborne disease, in particular salmonellosis is considerable. A relationship between warmer ambient temperature and salmonellosis has been previously studied.[6–17] These studies found increases of 1–15% of cases per 1 °C rise in temperature.[13]

Nonetheless, there are no reported studies on the effect of heatwaves on incidence of *Salmonella* notifications which is important given that disease burden could be greater than that associated with temperature alone, and infection from *Salmonella* is preventable. The aim of this study was to examine if there is a relationship between heatwaves and number of *Salmonella* cases in Adelaide, and to assess the impact of heatwaves on specific *Salmonella* serotypes.

Methods

Data sources

Daily laboratory confirmed salmonellosis cases resident within the Adelaide metropolitan region and notified between 1 January 1990 and 31 December 2012 were obtained from the SA Health Department's notifiable disease surveillance system. Surveillance of people diagnosed with *Salmonella* infection relies on doctors and laboratories to notify suspected and confirmed cases as part of their legal duty. Information extracted for notified cases included date of onset of illness and demographic characteristics. *Salmonella* serotype identified for each case was extracted from the same surveillance system which reports data on *Salmonella* serotypes and phage types. The use of laboratory methods in SA to serotype and phage type *Salmonella* isolates have been consistent over the study period.

Climatic information was obtained from an Australian Bureau of Meteorology (BOM) weather monitoring station close to the Adelaide city centre. Daily maximum temperature (T_{max}) in degrees Celsius (°C) and rainfall in millimetres (mm) from 1990 to 2012 were extracted. Recordings from this station, according to BOM, are representative of weather conditions across the metropolitan region of Adelaide.[16,18–21]

Heatwave definition and characteristics

A heatwave definition was defined as a period where the daily T_{max} reaches or exceeds three or more consecutive days of 35 °C. We selected this definition as it has demonstrated an effect on human health in other SA studies examining the relationship between heatwaves and morbidity and mortality outcomes.[18–20] The analysis dataset was restricted to the warm season from 1 October to 31 March, so as to control for the potential confounding effects of seasonal variation.[7]

Heatwaves are extended periods of unusually hot weather and their impact on mortality[22,23] and morbidity[18,24] is well established. However, there is some uncertainty about the characteristics that make heatwaves hazardous to human health. Characteristics such as intensity and duration should be considered.[25] In this study, the role of heatwave intensity, duration and timing on daily *Salmonella* notifications and the effect on different serotypes were examined. Intensity was defined as the daily T_{max} of $\geq$35 °C recorded during heatwaves, heatwave duration was the number of days in each episode, and timing was the first, second, or third and so on heatwave of the season.

Statistical analysis

Poisson regression models, assuming an exchangeable correlation structure within each cluster of heatwave days, were fitted using Generalised Estimating Equations (GEE) to examine the effect of heatwaves on daily *Salmonella* cases and number of cases of *Salmonella* specific serotypes during heatwaves compared with non-heatwave periods. Specific serotypes and phage types were selected for further analysis based on the five ranked with the highest frequency over the study period. We used the Quasi-likelihood under the independence model criterion (QIC) to select the best working correlation structure. In the case of over-dispersion a negative binomial model was fitted.

As expected for Adelaide there was little rainfall recorded during heatwave periods and rainfall was not included in the models. Relative humidity was excluded because Adelaide experiences dry, hot weather during the warmer months, therefore humidity is unlikely to confound the relationship with heatwaves. Findings from another study on temperature and salmonellosis conducted in Adelaide found relative humidity was not significant, and so it was not included in their statistical models.[16,21] Day of the week was included in the statistical models as a categorical variable, and public holidays were controlled for with an indicator variable. Variables reflecting linear and quadratic effects of year were included to adjust for long term trends and to allow for a non-linear relationship between counts and time.

Cases with *Salmonella* infection linked to foodborne outbreaks in which a common source had been identified and/or observed numbers of cases exceeded the expected number for the time period were identified from the SA Health Department's notifiable disease surveillance system. These cases were excluded from the analyses, as the relationship of climate conditions may be different for those with a common exposure compared to sporadic cases with no identified source of infection.[10,16,26]

Heatwave characteristics

To estimate the overall effect of heatwaves a binary variable (heatwave and non-heatwave days) was included. We used a categorical variable to identify heatwave days in order to examine the day which produced a greater risk of *Salmonella* infection. Intensity was examined by daily T_{max} within heatwaves and treated as a categorical variable in the Poisson model with four temperature ranges (35–36.9 °C, 37–38.9 °C, 39–40.9 °C, $\geq$41 °C). Duration was defined as three, four and five or more days. An alternative definition was used to examine the effect of short (three days) compared to long duration (four or more days). We considered two aspects of timing. First, timing was defined by the first, second and third heatwave event within each warm season denoting the order of occurrence. Second, timing differed by whether the heatwave event occurred in the early part of the warm season (October–December) or later (January–March).

Separate models were fitted to examine the effects of each heatwave characteristic on daily *Salmonella* counts. The same analyses were repeated for each of the five serotype counts. Incidence rate ratios (IRR) with 95% confidence intervals (CI) are reported with results interpreted as percentage (%) change in the number of daily *Salmonella* counts during heatwave periods compared with non-heatwave periods.

Lag effects

Distributed lag non-linear models (DLNMs) were separately fitted to investigate if there was a delayed effect of T_{max} during heatwaves on daily *Salmonella* and serotype notifications.[27,28] A previous study in SA on salmonellosis and temperature reported a lag of two weeks and based on these results a lag of up to 14 days was selected for this study.[16] We used natural cubic splines with three degrees of freedom (df) for T_{max} during heatwaves and natural cubic splines with three or four df (depending on serotype) for lags of 0–3, 0–7, 0–10, and 0–14 days. In the DLNMs models T_{max} during heatwaves as a continuous variable reflected intensity. We plotted lag-response results at mean T_{max} within heatwaves and three-dimensional plots for the effects of temperature in heatwaves at all lag days. We controlled for the same variables as per the GEE models. The Akaike Information Criterion (AIC) was used to select the best model based on the lowest AIC values. Results are reported from the DLNM analyses as Relative Risks (RR) and 95% CI. Sensitivity analyses were used to evaluate df for heatwave intensity (daily T_{max} as a continuous variable) and df for lags from 0 to 14 days.

A significance level of 0.05 was accepted for all statistical tests. Analyses were conducted using StataSE 13 (StataCorp LP, College Station, Texas) or R version 3.1.1 (R Foundation for Statistical Computing, http://cran.r-project.org/). The latter was used to fit the DLNM models with the 'dlnm' package.

Ethics statement

All data analysed were non-identifiable with ethics approval 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

A total of 7845 *Salmonella* cases (excluding outbreak cases) were reported in the study period from 1990 to 2012, 4412 of which had an illness onset date in the warm season of October–March. Of the 4412 cases, 51% were female and 49% were male. The 1–9 (26%) and 20–39 (25%) year age groups had the highest percentage of cases, followed by 40–59 (16%), 60+ (12%), 10–19 (10%) and <1 year of age (9%). Of the 4412 *Salmonella* cases reported in the warm season, 1217 (27.5%) were included in the top five serotype and phage types in our analyses. There were 178 unique *Salmonella* serotypes reported across the study period, with the five highest in frequency during the study period warm season being: *Salmonella enterica* serovar Typhimurium phage type 9 (*Salmonella typhimurium* PT9) (n = 422), *Salmonella infantis* (*S. infantis*) (n = 229), *S. typhimurium* PT108 (n = 209) is also typed as *S. typhimurium* PT170 in other jurisdictions, *S. typhimurium* PT44 (n = 179) and *S. typhimurium* PT135 (n = 178). Approximately 60% of the total numbers of serotype cases were notified in the warm season.

Table 1 presents summary statistics for temperature. The mean daily T_{max} in Adelaide was 38.4 °C (Standard deviation (*SD*) = 2.22) during heatwaves. A total of 50 heatwave events (comprising 213 days of three or more consecutive days met the heatwave definition) and was recorded in 50 separate events across the study period. In 2009, a nine day heatwave (with temperature above 40 °C on six consecutive days) was reported. The highest T_{max} recorded during any heatwave was 45.7 °C. Duration of heatwaves ranged from three to 15 days with a mean of 3.17 (*SD* = 2.40) days. No heatwaves were recorded outside of the warm season and none were recorded in 1990, 1996 and 2005.

Effect of heatwaves and heatwave characteristics on *Salmonella*

Fig. 1 illustrates the overall effect of heatwaves for all notified *Salmonella* cases and serotypes. The effect on daily *Salmonella* counts compared with those on non-heatwave days was not significant (IRR 1.02, 95% CI 0.84–1.25). Table 2 summarises the heatwave effect estimates. Heatwave intensity rather than duration had a greater impact on daily *Salmonella* infections. A 34% increase (IRR 1.34, 95% CI 1.01–1.78) in *Salmonella* cases was estimated if

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

Time period	Maximum temperature (T_{max})							
	Mean	5th percentile	25th percentile	50th percentile	75th percentile	90th percentile	95th percentile	99th percentile
1990–2012[a]	22.3 °C	14.1 °C	17 °C	21.1 °C	26.6 °C	32.3 °C	35.3 °C	39.8 °C
Cool[b]	18.2 °C	13.4 °C	15.3 °C	17.2 °C	20.2 °C	24.1 °C	26.4 °C	30.6 °C
Warm[c]	26.5 °C	17.8 °C	21.9 °C	25.7 °C	31 °C	35.3 °C	37.7 °C	41.2 °C
Heatwaves[d]	38.4 °C	35.4 °C	36.6 °C	38.3 °C	39.8 °C	41.5 °C	42.9 °C	43.9 °C

[a] *Salmonella* cases (n = 7845) notified in the study period.
[b] *Salmonella* cases (n = 3433) notified in the cool season (April–September).
[c] *Salmonella* cases (n = 4412) notified in the warm season (October–March).
[d] *Salmonella* cases (n = 238) notified during heatwaves.

T_{max} was ≥41 °C (compared to temperature ranges between 35–36.9 °C, 37–38.9 °C and 39–40.9 °C). *Salmonella* cases were less frequent in the early months of the warm season compared to the later months.

Effect of heatwaves and heatwave characteristics on *Salmonella* serotypes

The effect of heatwaves on daily counts was only significant for *S. typhimurium* PT135 as depicted in Fig. 1, such that the risk doubled during heatwaves (IRR 2.08, 95% CI 1.14–3.79). As shown in Table 2, there was a twofold increase in risk on day three (IRR 2.09, 95% CI 1.04–4.20) and a greater increase on day five (IRR 2.72, 95% CI 1.20–6.12) for *S. typhimurium* PT135 relative to non-heatwave days.

Heatwave intensity increased the risk of *S. typhimurium* PT135 by almost threefold (IRR 2.96, 95% CI 1.21–7.24) within T_{max} range of 39–40.9 °C. Short duration increased the risk twofold for *S. typhimurium* PT135 (IRR 2.10, 95% CI 1.05–4.20) and duration by length of days had effects at four (IRR 3.30, 95% CI 1.34–8.13) and five days (IRR 2.47, 95% CI 1.04–5.88). Four day duration increased *S. infantis* notifications close to threefold (IRR 2.84, 95% CI 1.26–6.41). An increase in *S. typhimurium* PT135 infections was estimated with the second heatwave event in the season (IRR 2.53, 95% CI 1.07–6.01), and a much higher risk in the third (IRR 4.24, 95% CI 2.19–8.22). The number of cases was lower in the early months of the warm season compared to the later months for all serotypes and phage types, with the exception of a non-significant effect estimate for *S. typhimurium* PT44.

Lag effects

Fig. 2 presents the lag effects up to 14 days of T_{max} during heatwaves on *Salmonella* and serotype cases and Fig. 3 shows the three-dimensional plots for significant RR for *Salmonella* and two of the serotypes. As shown in Fig. 2A, the

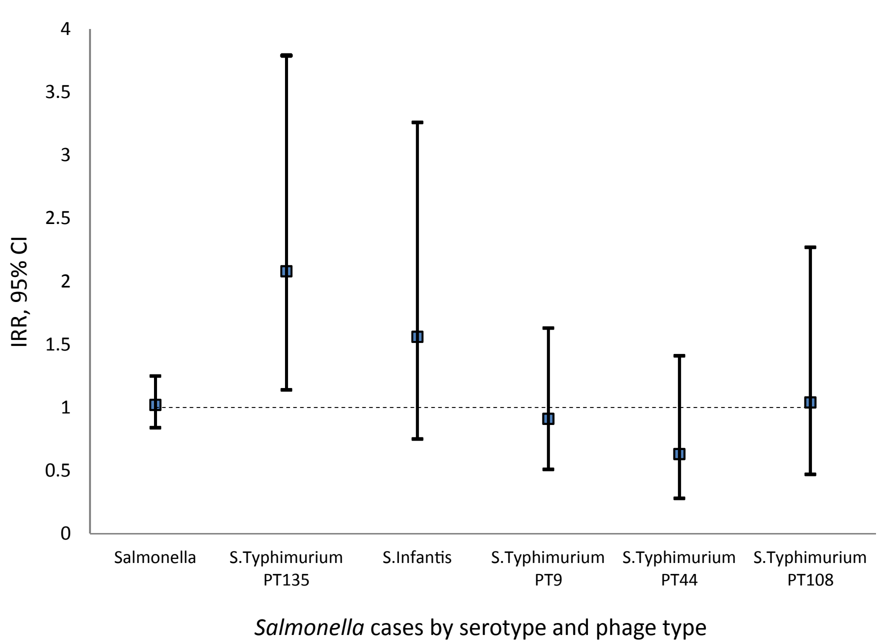

Figure 1 Effect estimates of heatwaves for all notified *Salmonella* cases and specific serotype and phage types, 1990–2012, Adelaide, South Australia.

Table 2 Effect estimates of heatwave characteristics on daily *Salmonella cases* and specific serotypes and phage types.

Heatwave characteristic	*Salmonella* IRR (95% CI)	*p*	*S.* typhimurium PT135 IRR (95% CI)	*p*	*S* infantis IRR (95% CI)	*p*	*S.* yyphimurium PT9 IRR (95% CI)	*p*	*S.* typhimurium PT44 IRR (95% CI)	*p*	*S.* typhimurium PT108 IRR (95% CI)	*p*
Heatwave	1.02 (0.84, 1.25)	0.77	2.08 (1.14, 3.79)	0.01	1.56 (0.75, 3.26)	0.23	0.91 (0.51, 1.63)	0.76	0.63 (0.28, 1.41)	0.26	1.04 (0.47, 2.27)	0.91
Day of heatwave												
Day 3	1.05 (0.85, 1.29)	0.63	2.09 (1.04, 4.20)	0.03	2.02 (0.95, 4.29)	<0.01[a]	0.87 (0.49, 1.54)	0.65	0.63 (0.25, 1.61)	0.34	0.70 (0.30, 1.64)	0.42
Day 4	0.94 (0.63, 1.41)	0.79	1.74 (0.57, 5.24)	0.32	0.57 (0.04, 6.55)	0.65[a]	0.95 (0.35, 2.57)	0.92	0.54 (0.08, 3.64)	0.52	2.02 (0.69, 5.90)	0.19
Day 5	0.96 (0.65, 1.42)	0.86	2.72 (1.20, 6.12)	0.01	0.28 (0.00, 30.62)	0.60[a]	1.18 (0.47, 3.00)	0.71	0.81 (0.23, 2.80)	0.74	1.75 (0.54, 5.64)	0.34
Duration												
Short	1.05 (0.85, 1.29)	0.63	2.10 (1.05, 4.20)	0.03	2.01 (0.95, 4.27)	0.06	0.87 (0.49, 1.54)	0.65	0.63 (0.25, 1.61)	0.34	0.70 (0.30, 1.64)	0.42
Long	0.95 (0.66, 1.36)	0.79	2.05 (0.77, 5.40)	0.14	0.40 (0.02, 6.21)	0.51	1.04 (0.43, 2.49)	0.92	0.63 (0.19, 2.06)	0.45	1.91 (0.72, 5.04)	0.18
Duration by length												
3 days	0.93 (0.68, 1.29)	0.70	0.76 (0.20, 2.90)	0.69	1.03 (0.15, 7.04)	0.97	1.09 (0.57, 2.07)	0.77	0.37 (0.50, 2.50)	0.31	0.66 (0.18, 2.33)	0.52
4 days	1.20 (0.86, 1.67)	0.27	3.30 (1.34, 8.13)	<0.01	2.84 (1.26, 6.41)	0.01	0.71 (0.29, 1.69)	0.44	1.08 (0.44, 2.64)	0.85	0.62 (0.18, 2.17)	0.46
5 days	0.96 (0.66, 1.38)	0.83	2.47 (1.04, 5.88)	0.04	0.99 (0.37, 2.65)	0.99	0.97 (0.28, 3.30)	0.96	0.46 (0.08, 2.49)	0.36	1.82 (0.61, 5.42)	0.27
Timing by order of occurrence in a season												
1st heatwave	1.00 (0.74, 1.34)	0.99	0.31 (0.0, 2.2)	0.24	2.01 (0.68, 5.97)	0.20	1.13 (0.44, 2.86)	0.79	0.54 (0.14, 2.01)	0.35	2.07 (0.87, 4.92)	0.09
2nd heatwave	1.00 (0.77, 1.29)	0.97	2.53 (1.07, 6.01)	0.03	0.64 (0.09, 4.41)	0.65	1.11 (0.50, 2.44)	0.79	0.38 (0.06, 2.16)	0.28	0.45 (0.15, 1.40)	0.17
3rd heatwave	1.08 (0.70, 1.67)	0.70	4.24 (2.19, 8.22)	<0.01	1.86 (0.66, 5.21)	0.23	0.49 (0.17, 1.39)	0.18	1.00 (0.35, 2.87)	0.99	c	c
Timing by months in season												
Early	0.68 (0.64, 0.73)	<0.01	0.57 (0.4, 0.8)	<0.01	0.44 (0.33, 0.58)	<0.01	0.37 (0.29, 0.47)	<0.01	0.87 (0.60, 1.25)	0.47	0.55 (0.40, 0.77)	<0.01
Intensity by temperature range												
35–36.9 °C	0.93 (0.64, 1.35)	0.71	1.30 (0.41, 4.06)	0.64	1.93 (0.54, 6.87)	0.30	1.06 (0.40, 2.81)	0.89	0.93 (0.63, 1.35)	0.71	0.33 (0.04, 2.77)	0.31
37–38.9 °C	0.97 (0.68, 1.38)	0.87	2.34 (0.85, 6.44)	0.09	1.96 (0.80, 4.75)	0.13	0.83 (0.31, 2.22)	0.71	0.98 (0.69, 1.39)	0.92	0.57 (0.14, 2.33)	0.44
39–40.9 °C	0.99 (0.74, 1.33)	0.98	2.96 (1.21, 7.24)	0.01	1.11 (0.28, 4.29)	0.87	0.66 (0.22, 1.92)	0.45	1.14 (0.91, 1.42)	0.24	1.88 (0.62, 5.63)	0.25
≥41 °C	1.34 (1.01, 1.78)	0.04	1.74 (0.60–5.01)	0.30	0.70 (0.09, 5.05)	0.72	1.15 (0.60, 2.19)	0.65	b	b	2.21 (0.83, 5.90)	0.11

Incidence Rate Ratio (IRR), 95% Confidence Interval (CI), *p*-value (0.05 significance level).

Adjusted for seasonality (warm season), long term trends, day of the week (reference day is Sunday) and public holidays. The reference group are non-heatwave days.

[a] *S. infantis* – re-categorised day of increased risk and fitted to the model.

[b] *S. typhimurium* PT44-intensity by temperature range – a variable with three levels was fitted to the model.

[c] *S. typhimurium* PT108 – timing by order of occurrence – a variable with two levels was fitted to the model.

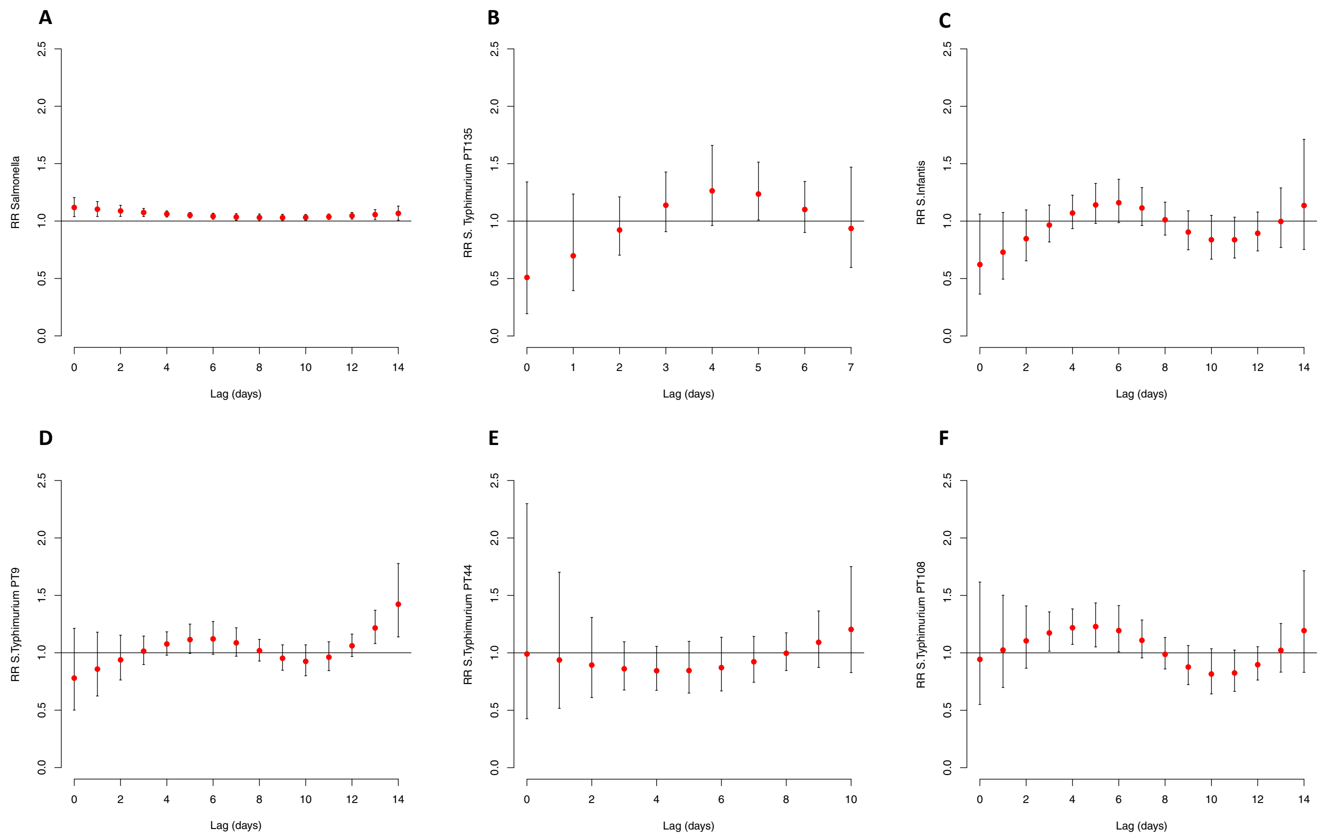

Figure 2 Relative risks of daily *Salmonella* and serotype and phage type counts at 38.4 °C (mean T_{max} during heatwaves), using a natural cubic spline—natural cubic spline DLNM with 3 df natural cubic spline for T_{max} and 3 df for *Salmonella* (A), *S. typhimurium* PT135 (B), *S. typhimurium* PT44 (E) or 4 df for *S. infantis* (C), *S. typhimurium* PT9 (D), *S. typhimurium* PT108 (F) for lag days, 1990—2012, Adelaide, South Australia.

greatest effect of T_{max} during heatwaves on overall *Salmonella* cases was at day-0 lag (at 38.4 °C, RR 1.12, 95% CI 1.04—1.20) and the effect remained elevated up to day-14 lag (at 38.4 °C, RR 1.07, 95% CI 1.01—1.13). Lagged effects of T_{max} during heatwaves decreased over time and increased with higher temperature (Fig. 3A). The effects of T_{max} during heatwaves on *S. typhimurium* PT44 (Fig. 2E) and *S. infantis* (Fig. 2C) cases over lags of 0—14 days were not significant and marginally significant at day-5 lag for *S. typhimurium* PT135 cases (Fig. 2B). The effect of temperature during heatwaves on *S. typhimurium* PT108 notifications was elevated between day-3 (at 38.4 °C, RR 1.17, 95% CI 1.01—1.36) and day-6 lag (at 38.4 °C, RR 1.19, 95% CI 1.01—1.41) (Fig. 2F). As demonstrated in Fig. 3C, lagged effects on *S. typhimurium* PT108 decreased over time and increased with higher temperature. The effect of T_{max} during heatwaves on *S. typhimurium* PT9 cases was elevated between four and six days, and greatest at day-14 lag (at 38.4 °C, RR 1.42, 95% CI 1.14—1.78 (Figs. 2D and 3B).

Discussion

To our best knowledge, this study, for the first time, reports the effect of heatwaves and the role of heatwave intensity, duration and timing on daily *Salmonella* counts and on different strains. Our findings indicate that higher intensity increases the risk of infection, and that of specific serotypes and phage types. This association is biologically plausible because *Salmonella* growth occurs between 5.2 and 46.2 °C, and within an ideal temperature range of 35—43 °C.[3]

Heat wave duration of four days had a pronounced effect on *S. typhimurium* PT135 and *S. infantis* infection. Our findings are congruent with the results from studies examining the effects and characteristics of heatwaves on enteric infections, but not salmonellosis. Intensity (above 99th percentile) and duration (three days) were associated with hospital emergency department visits for childhood diarrhoea,[29] and hospital admissions for infectious gastroenteritis increased by 4.7% with every additional day in a heatwave.[30] In our study the effect of heatwaves on overall salmonellosis increased by 34% if T_{max} was $\geq$41 °C. As well, day three and five of a heatwave, and subsequent heatwave events after the first in a warm season, increased the number of *S. typhimurium* PT135 notifications. This could in part be explained by the accumulated effects of T_{max} during heatwaves on different stages of food processing, as we found an increase in salmonellosis cases persisted up to 14 days at high temperatures. A better understanding of the likely mechanisms on the effects of heatwaves on

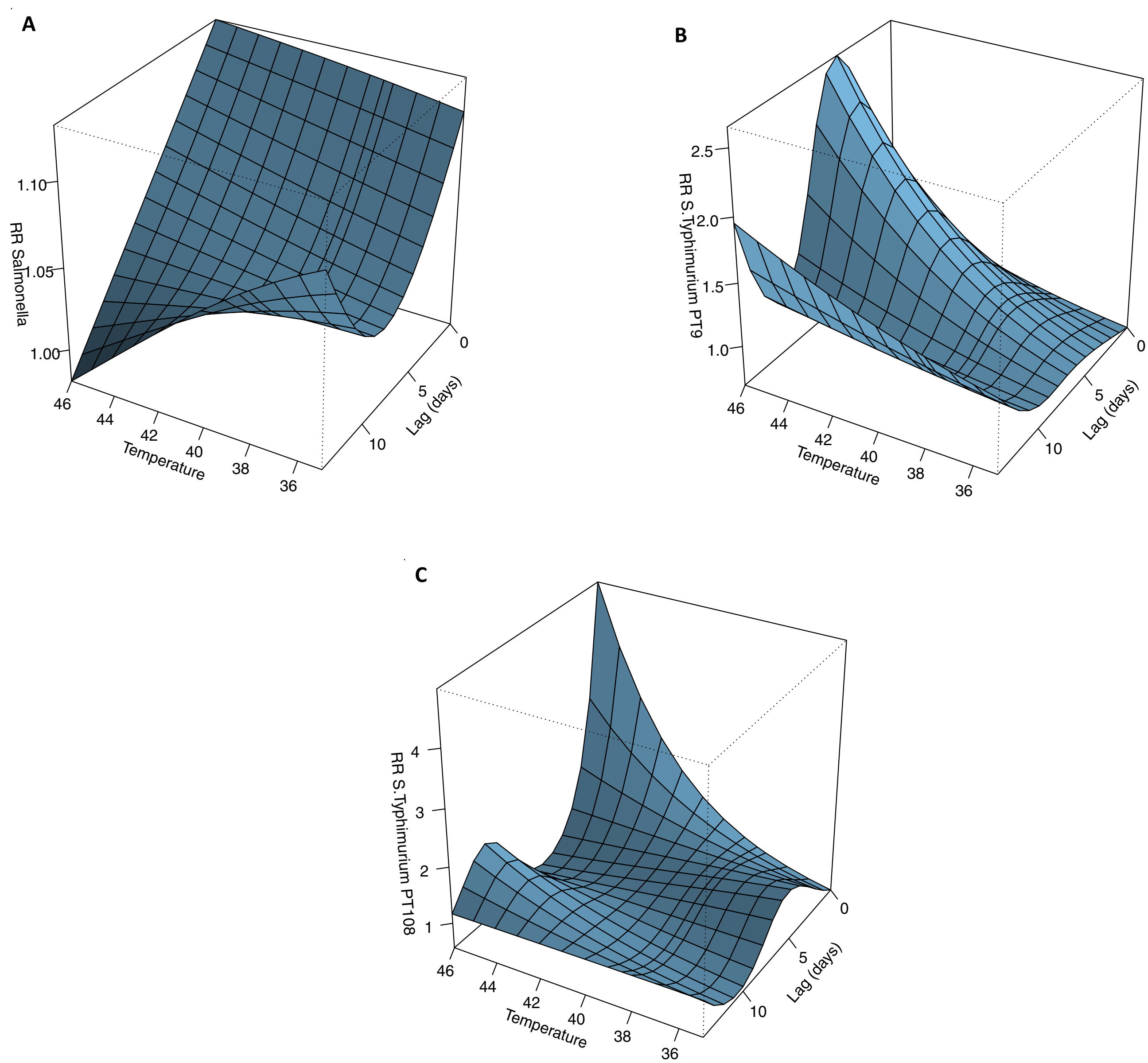

Figure 3 Three-dimensional plots for risk of *Salmonella* (A), *S. typhimurium* PT9 (B) and *S. typhimurium* PT108 (C) infection by T_{max} during heatwaves and lag days 0–14, 1990–2012, Adelaide, South Australia (Temperature = T_{max} during heatwaves).

salmonellosis infection is needed so that public health messages can be targeted most effectively to those at greatest risk.

Our study has provided evidence that *Salmonella* serotypes are sensitive to the effects of heatwaves. This is important because not all *Salmonella* serotypes are pathogenic and cause human infection; there are different pathways for transmission[15] and the relationship with temperature is not well established. Previous studies found that the effect of temperature on incidence of *Salmonella* serotypes was statistically significant for *S. typhimurium* and *Salmonella enteritidis* infections.[10,11] However, the associations were examined at the serotype and not phage type level, and as there are over 2500 *Salmonella* serotypes,[3] identification of a possible relationship with temperature at the level of phage type may inform refined intervention strategies. As well as considering specific phage types, we focussed on the effect of heatwaves while these previous studies estimated the effect of temperature. Hence, public health implications for incidence of foodborne disease and prevention may vary depending on the mechanisms of these relationships on transmission of *Salmonella* infection.

S. typhimurium PT135 is a commonly reported phage type isolated from human infection, with *S. typhimurium* PT108 becoming more frequent.[31] The serotypes examined in this study are consistent with reports from other states in Australia[31] and have been isolated from chickens and eggs with consumption of both implicated in several outbreaks.[32–36] Despite the exclusion of foodborne outbreaks in our study, the five serotypes considered in our analyses are linked to animal and food sources, suggesting heatwaves may play an important role in transmission of *Salmonella* infection through food contamination. Sustained elevation in temperature may affect transmission of *Salmonella* infection directly by multiplication of bacteria in the environment and in food. Indirect transmission may occur by a change in people's eating behaviours and food preferences (eating out, BBQ cooking) as well as food processing practices (poor food storage) during hotter days.[6,10,15]

The immediate effect of heatwave intensity was associated with an increased risk of *Salmonella* cases. We detected delayed effects, between four and six days for *S. typhimurium* PT9. For *S. typhimurium* PT108, there was evidence of delayed effects from days three through six. We also found the effects of heatwave intensity on *Salmonella* and *S. typhimurium* PT9 cases at day-14 lag indicating persisting effects at higher temperatures. Lag effects importantly indicate where and when food contamination could have occurred. Short lag times points to food contamination closer to the time of consumption, whereas longer time lags could indicate effects at the production processing stages.[11,37] Our results of lags with the greatest effect at zero, six and 14 days suggest that heatwaves may have an effect at the time of food preparation and consumption and at the time of food production and processing. A lag of up to 14 days is consistent with other studies in South Australia that examined the effects of warmer temperature on *Salmonella* cases.[13,16] In keeping with our findings, studies in other countries have detected lags of 1 week,[10] 2 weeks,[14] 1 month[6,8,9,15] and up to 2 months.[7,12,17] Notwithstanding this, consideration should be given to the short incubation period (between 8 and 72 h) for *Salmonella* as lag effects of temperature could be associated with varying incubation periods.[3]

Our study has several limitations. *Salmonella* infection is underreported given its reliance on a passive disease surveillance notification system.[38] However, it is unlikely that underreporting will affect estimates of the association between heatwaves and increased risk of salmonellosis. In addition, underreporting has remained relatively stable over the study period.[16] We were unable to exclude cases that travelled prior to onset of illness because the data were not fully captured 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.[10] As no studies have investigated the effects of heatwaves on salmonellosis notifications we applied the methods and analyses used by other studies that examined the relationship between heatwaves and mortality or morbidity.[39–41] As in these studies, we fitted a Poisson regression model using GEE based on the three studies cited above since this approach is commonly used to model clustered data and allows for autocorrelation to be taken into account. We did not adjust our reporting of statistical analyses for multiple comparisons[42] because of the exploratory nature of our study.

Given that the serotypes and phage types in our analyses were sensitive to the effects of heatwaves and are linked to animal and food sources, predominately chicken and eggs,[32,34,43–45] we postulate that heatwaves may play a role in transmission of infection through food contamination. Our results show that overall salmonellosis and *S. typhimurium* PT135 cases increase over 39 °C in heatwaves — the optimum temperature for bacterial growth and multiplication. Ideal temperatures could enable the different *S. typhimurium* phage types to proliferate and develop quickly increasing risk of food contamination and subsequent risk of human infection.[3] Bacterial contamination of poultry meat, and livestock carcasses at the time of leaving abattoirs has been found to be higher in summer.[46] Additionally, lack of hand washing and improper storage during food handling and preparation in both domestic and retail settings can increase risk of food contamination.[26]

In this instance increased risk of *Salmonella* infection is generally preventable with correct food handling, preparation, storage and effective hygiene practices. Food and personal hygiene practices in domestic, retail and food catering settings promoted prior to the start of a heatwave will help to reduce the burden of *Salmonella* infection. It is essential for regulatory measures within retail and food catering industries to be in place when temperature increases to avoid food spoilage and contamination. Likewise, monitoring and enforcement of industry standards is also important.[47]

These public health interventions are particularly relevant given that populations worldwide are exposed to variability in weather patterns with increasingly common extreme events, such as heatwaves. Heatwaves are predicted to increase in frequency, intensity and duration, with consequential increases in mortality and morbidity arising from the effects of heatwaves. Furthermore, consideration and research should be given to exploring biological plausibility and epidemiological characteristics of *Salmonella* serotypes to better understand the association between heatwaves and salmonellosis, including replication in other areas of Australia and internationally. These findings support the need for targeted public health interventions and will inform development of policy recommendations for food-safety regulations and health-behaviour interventions that will mitigate the consequences of foodborne illness on human health.

Competing interests

The authors declare that they have no competing interests.

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