



Rising temperatures and heat waves as drivers of *Salmonella* and *Campylobacter* bacteraemia across diverse climatic zones in Queensland, Australia

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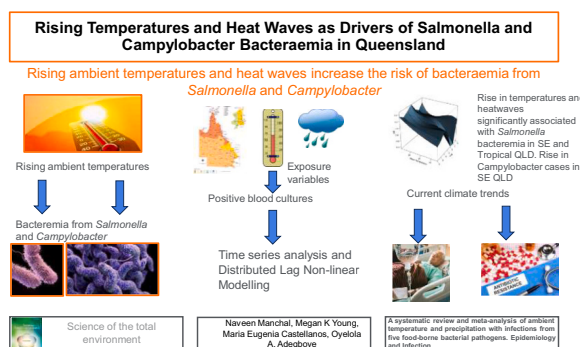
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HIGHLIGHTS

- Rising temperatures significantly increase *Salmonella* bacteraemia risk in Southeast and Tropical North Queensland.
- Temperature-bacteraemia associations vary by climate zone with distinct lag patterns across regions.
- Maximum temperature during heatwaves shows a strong dose-response relationship with *Salmonella* cases (31% increase per 1 °C).
- No significant temperature associations found for *Campylobacter* bacteraemia across any climate zone.
- Findings support targeted, climate-informed public health interventions for invasive bacterial infections.

GRAPHICAL ABSTRACT



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ABSTRACT

Climate change, with rising ambient temperatures and heatwaves, has been linked to an increased risk of food-borne infections. Using a time series quasi-Poisson regression with a Distributed Lag Non-Linear Model (DLNM), we examine the associations between ambient temperature and heatwaves and the risk of *Salmonella* and *Campylobacter* bacteraemia in Queensland (QLD), Australia. A total of 1034 *Salmonella* and 234 *Campylobacter* bacteraemia cases were recorded during 2010–2019. Deseasonalised mean temperatures showed a significant positive relationship with *Salmonella* bacteraemia in Southeast (SE) QLD and Tropical North QLD. In SE QLD, a 1 °C increase in mean temperature increased risk of *Salmonella* bacteraemia by 8–20% across the significant portion of the dose-response curve (RR range: 1.08–1.20, 95% CI: 1.01–1.40), while maximum temperature was linked to 0.3–16% increase (RR range: 1.003–1.16, 95% CI: 0.84–1.57). In Tropical North QLD, a 1 °C rise in

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mean temperature was associated with an 18% increase, peaking at 27.2 °C (RR 1.70, 95% CI: 1.01–2.86). At maximum temperatures above 28 °C, each 1 °C increase corresponded to an 11–105% rise in risk (RR range 1.11–2.05, 95% CI: 1.01–4.73). In Central QLD, *Salmonella* risk exhibited a modest upward trend across temperature ranges of 11 °C to 17.5 °C (mean) and 16 °C to 22 °C (maximum), but estimates were not statistically significant. Cases peaked within one week in SE QLD and after three weeks in Tropical North QLD. Heatwave exposure increased *Salmonella* bacteraemia cases by 31% per degree rise in maximum temperature (IRR: 1.31, 95% CI: 1.16–1.48), reflecting the heatwave-specific intensity–response effects and is not directly comparable to the DLNM temperature RR estimates. No significant associations were identified for campylobacteriosis. These findings confirm the significant impact of rising ambient temperatures and heatwaves on *Salmonella* bacteraemia risk, underscoring the need for climate-adaptive public health strategies.

1. Introduction

Climate change and increased ambient temperatures, along with increasingly frequent heatwaves, pose a significant public health challenge globally (Bell Michelle et al., 2024). Rising temperatures have been linked to increased transmission risks of various zoonotic diseases such as malaria, dengue and food-borne pathogens (Zhang et al., 2024). A meta-analysis found a 7% increase in all-cause diarrhoea for every °C increase (Carlton et al., 2016). These climatic events are expected to alter the epidemiology of various food-borne pathogens, posing new health risks. In Australia, the economic burden of salmonellosis is substantial. A study estimated that salmonellosis and its sequelae cost a median of 146.8 million AUD in 2015 (Ford et al., 2019). The public health impact in QLD is even greater given that climatic factors are even more pronounced (Xu et al., 2023).

Many studies have demonstrated an association between rising ambient temperatures and an increase in gastroenteritis cases caused by food-borne pathogens, particularly *Salmonella* and *Campylobacter* species (Aik et al., 2018; Akil et al., 2014; Djennad et al., 2019; Kuhn et al., 2020b). The potential mechanisms for the association between climatic factors and bacterial gastroenteritis are many, including behavioural changes among the population brought about by extreme temperatures, breaks in cold chain systems during food processing and food contamination. Additionally, higher ambient temperatures may enhance bacterial replication rates and promote expression of virulence factors in *Salmonella* and *Campylobacter* species, potentially increasing their invasive capacity in human hosts (Akil et al., 2014; Jiang et al., 2015; Semenza et al., 2012; Zhang et al., 2010).

The progression in severity from gastroenteritis to invasive infection is a critical escalation. Parisi and colleagues analysed passive surveillance data in QLD, Australia and noted a rise in the incidence of salmonellosis over ten years (Parisi et al., 2019). Not only did the incidence increase over the study period, but the authors also reported an increase in the invasiveness of *Salmonella* infection. Approximately 92% of these invasive infections were diagnosed on blood culture and were more common in the elderly (Parisi et al., 2019). Bacteraemia, compared to gastroenteritis, is a more invasive infection that can lead to prolonged hospitalisation, increased morbidity and mortality and increased use of antimicrobials, contributing to resistance.

Immunocompromising conditions like HIV, organ transplants, hematologic malignancies, hypogammaglobulinemia and co-infection with malaria predispose to *Salmonella* and *Campylobacter* bacteraemia (Lee et al., 2021; Marchello et al., 2022; Otsuka et al., 2023). Our recent systematic review (Manchal et al., 2024) affirmed the impact of climate variables (extreme temperatures and precipitation) on bacteraemia from food-borne pathogens, including *Salmonella* and *Campylobacter*. A meta-analysis of three studies demonstrated a pooled relative risk of 1.05 (95% CI: 1.03–1.07) for extreme ambient temperatures (Jiang et al., 2015; Manchal et al., 2024; Morgado et al., 2021; Nili et al., 2021).

Previous studies on ambient temperature and *Salmonella* (Aik et al., 2018; Bi et al., 2006; Bi et al., 2009; Britton et al., 2010; Chua et al., 2022; Fleury et al., 2006; Grjibovski et al., 2013; Jiang et al., 2015; Kendrovski et al., 2011; Milazzo et al., 2016; Robinson et al., 2022; Wang et al., 2018; Zhang et al., 2008; Zhang et al., 2010) and

Campylobacter (Bi et al., 2008; Carev et al., 2018; Cousins et al., 2020; Djennad et al., 2019; Kovats et al., 2005; Kuhn et al., 2020a; Kuhn et al., 2020b; Lake et al., 2019; Mun, 2020; Patrick et al., 2004; Rosenberg et al., 2018; Semenza et al., 2012) infections have primarily focused on gastroenteritis, the most common manifestation of these infections. This is the first study to address the critical gap in understanding how climate variables can drive invasive food-borne bacterial infections. Specifically, this study aimed to investigate the association between rising ambient temperatures, heatwaves across QLD, Australia, and the incidence of bacteraemia caused by two genera of bacterial food-borne pathogens, *Salmonella* and *Campylobacter*, over a 10-year period (2010–2019). We hypothesised that warmer climatic conditions, reflected by increasing ambient temperatures and heatwaves, increase the risk of bacteraemia from *Salmonella* and *Campylobacter* species in QLD.

2. Methods

2.1. Study location

QLD is the second largest state in Australia, with a population of over 5.3 million concentrated mainly in the Southeast (Australian bureau of statistics, 2025). Due to its vast size, the state experiences significant climatic variation, encompassing three predominant climatic zones (Fig. 1):

1. The tropical north (Zone 1): Characterised by a hot, humid summer season between December and February and a warm winter with average maximum temperatures of 32 °C.
2. Subtropical southeast coastline in SE QLD (zone 2): Exhibiting a warm, humid summer, mild winter and average maximum temperatures of 29 °C,
3. Arid western region in Central QLD (zone 3 and 5): Characterised by hot, dry summer and warm winter with average maximum temperatures ranging from approximately 37 °C in summer to 24 °C in winter.

Fig. 1 illustrates the diverse climatic conditions. The projected climate scenarios for the state indicate an annual rise of 0.5–1.5 °C by 2030, with hotter days, less rainfall in the Southeast and a warmer ocean with rising sea levels (Intergovernment Panel On Climate Change, 2024).

2.2. Climate data

Climate data were obtained from the Australian Bureau of Meteorology (BOM). The primary exposure variables included weekly maximum and mean temperatures. Additionally, precipitation data were collected to account for its potential impact. The BOM defines a heatwave as a positive Excess Heat Factor (EHF), which incorporates two indices: the average temperatures over a 3-day period compared to both the historical data (95th percentile) for the region and the preceding 30 days. Any week in our time series that fell within a BOM-declared heatwave period for SE QLD was flagged as a heatwave week. Using this definition, we then extracted the daily maximum

temperature values for these identified periods to create our heatwave-day exposure variable. The variability in climate parameters can be significant even within different climate zones. Therefore, climate variables were extracted from weather stations in the closest proximity to the postcode of each residence for cases of bacteraemia. QLD has a well-distributed network of weather stations that provide spatially relevant exposure estimates. The BOM website provides a selection tool that identifies the closest operational weather station to a given postcode. For each postcode, the nearest station with complete meteorological data was selected for extracting the climate variables. In rare instances where the closest station had incomplete data, the next nearest station with complete records was used. All bacteraemia cases had matched climate exposure data.

2.3. Case data

Salmonella and *Campylobacter* infections are notifiable conditions in QLD, and the confirmed results are registered in the Notifiable Conditions System (NOCS). A case was defined as any person with a laboratory-confirmed blood culture for *Salmonella* or *Campylobacter* species. The corresponding ICD-10-AM codes for bacteraemia outcomes were A02.1 (*Salmonella* sepsis) and R78.81 (bacteraemia). While *Campylobacter* enteritis cases are coded as A04.5, our study specifically included only those with documented bacteraemia (R78.81) (Authority IHaACP, 2025). The blood culture results from public health care settings were obtained from Pathology QLD, and the private lab results were available on NOCS. Patient age, sex, postcode of residence, and dates of blood culture collection were collected. If the same patient had multiple positive blood cultures, the date of the first culture-positive sample was considered. Data linkage between the two databases with case information was performed by the Statistical Services Branch, QLD. To account for the baseline variability in climate in the state, the cases were analysed in clusters for the three main climate zones: subtropical SE QLD, humid Tropical North QLD and dry Central QLD.

2.4. Statistical analysis

The climatic variables were the independent variables, while the dependent variable was the weekly count of notifications for positive blood cultures. Weekly counts were used to account for the variable incubation periods of the pathogens (1–5 days) and the scarcity of daily cases, which would lead to many zero-count days. It also allowed for reporting delays in the laboratory confirmation of bacteraemia. Time series analysis was used to investigate the seasonal trends in bacteraemia cases.

Annual incidence rates (cases per 100,000 population) were calculated for each region. Temporal trends were assessed using linear regression of incidence against year, from which slope coefficients (representing the annual change in incidence), 95% confidence intervals, and *p*-values were derived. The total percentage change over the study period and the annual percentage change rates were also computed. Trends were considered statistically significant at $p < 0.05$.

To account for the delayed effects and the potential non-linear associations between climate variables and health outcomes, distributed lag non-linear modelling (DLNM) through quasi-Poisson regression was used. Prior studies have shown non-linear and lagged effects for infections caused by these pathogens (Aik et al., 2018; Bi et al., 2006; Chua et al., 2022; Zhang et al., 2010). This method is particularly well-suited for capturing complex bi-dimensional associations by modelling both exposure levels (e.g., temperature) and lag periods (Chipman et al., 2010; Gasparrini, 2011). Climatic variables were deseasonalised before model inclusion, removing seasonal components (Aik et al., 2018; Grjibovski et al., 2013). Seasonal variation was controlled using paired sine and cosine functions (Bhaskaran et al., 2013; Gasparrini et al., 2011; Peng et al., 2006). Long-term temporal trends were controlled using linear and quadratic functions of time. Annual population estimates in

all postcodes were not available, and thus, we could not obtain the interpolated weekly population estimates needed to produce a population offset term for *Salmonella* and *Campylobacter* bacteraemia cases. Weekly case counts were analysed without population offsets in the DLNM framework. This approach is standard for DLNM analyses of sparse count data, as temporal controls (seasonal and trend terms) adequately account for population-related secular changes. Additionally, the weekly temporal granularity (520 observations) with sparse case counts would make rate-based models unstable. However, as the number of heat wave periods were 13, for each heatwave period, we aggregated case counts and used the corresponding annual postcode-level population estimate (obtained from the Australian Bureau of Statistics) as an offset term: $\log(\text{population})$. This allowed calculation of incidence rate ratios (IRR), representing the change in bacteraemia rate per 100,000 population for each 1 °C increase in maximum temperature during heatwave periods.

Model specifications were determined a priori based on regional climate characteristics, sample sizes, and established DLNM methodology, not through post-hoc data exploration. Model selection was guided by Quasi-Akaike Information Criterion (QAIC). For *Salmonella* in SE and Central QLD, natural splines with three degrees of freedom (df) were used. These specifications were chosen to accommodate moderate temperature variability while maintaining parsimony, given the larger sample sizes in these regions. The lag-response function was specified for up to six weeks using polynomial functions with two dfs. The 6-week lag period was chosen as previous studies (Chua et al., 2022; Manchal et al., 2024) have reported effects up to this period, accounting for factors such as the incubation period and delays in health-seeking behaviour. Precipitation was included in all models to adjust for potential confounding effects. The functional form was determined through model comparison: linear specifications provided optimal fit for *Salmonella* (QAIC = 376.29 vs. 400.80 for nonlinear, $\Delta\text{QAIC} = 24.82$), while natural splines were tested for *Campylobacter* given this pathogen's moisture sensitivity. For Tropical North QLD, greater model flexibility was required due to more extreme temperature ranges and smaller sample sizes. The Tropical North QLD model used basic splines with three dfs for temperature and natural splines with five dfs for lag weeks to balance model flexibility with overfitting risk. Seasonal adjustment had no significant effects in this region. To validate these specifications, sensitivity analyses systematically tested alternative parameterisations: dfs (2–5), lag periods (4–8 weeks), and polynomial degrees (1–3). Temperature-*Salmonella* associations remained consistent across specifications, confirming robustness. The results are presented as relative risk (RR) and 95% confidence intervals (CI) for bacteraemia cases, calculated at specified temperature quintiles. Statistical significance was evaluated at the 5% level. These outcomes were tested with separate DLNM models for mean and maximum temperatures for each pathogen and climate region. This approach allowed for a region-specific evaluation of the impact of temperature variability, accounting for differences in baseline climate conditions across the state's diverse climatic zones. Predictions were centred at the regional average weekly mean temperature (SE QLD: 21.0 °C, Central QLD: 22.3 °C, Tropical North QLD: 24.0 °C) and average maximum temperature (SE QLD: 28.1 °C, Central QLD: 27.4 °C, Tropical North QLD: 27.6 °C). Risk estimates were calculated at temperature quintiles (5th, 15th, 25th, 75th, 85th, 95th percentiles) specific to each region's temperature distribution. We also tested the models with time as a covariate, accounting for long-term trends that could potentially confound the effect of temperature.

To assess the relationship between heatwaves and weekly bacteraemia rates (cases per 100,000 population), a separate analysis was conducted using Poisson regression models. For each identified heatwave period, we extracted the daily maximum temperature values and aggregated case counts. Maximum temperature was chosen as the exposure metric because peak daily temperatures are most relevant to food safety risks through effects on bacterial replication rates and cold chain integrity. This approach is consistent with established methods for

analysing discrete extreme weather events in climate-health research (Morgado et al., 2021; Scalley et al., 2015). Our heatwave-day exposure was modelled as a continuous variable, as it only included maximum temperature values from heatwave periods. The analysis specifically examined the dose-response relationship between temperature intensity during heatwave periods and bacteraemia risk, rather than comparing heatwave versus non-heatwave days.

All analyses were implemented in R version 4.0.3 (R Core Team, 2020). The main R package was “dlnm” (Gasparrini, 2011).

3. Results

3.1. Study cohort

A total of 1034 cases of *Salmonella* and 234 cases of *Campylobacter* were reported during the study period (Table 1). The age distribution of the cohort revealed that children and adolescents under 20 years accounted for 36.3% of the *Salmonella* sample and 1.3% of the *Campylobacter* sample (Fig. 2A). The most common species identified were *S. Virchow* (264/1034, 25.5%), and *C. jejuni* (153/234, 65.4%) (Fig. 2B). Males comprised the majority of the cohort, accounting for 58% (Table 1).

The mean weekly cases by demographics are presented in Table 1. Considering that 3.8 million of the state’s 5 million population live in SE QLD, this translates to an annual crude incidence rate of 2.07 cases per 100,000 in SE QLD, 4.12 cases per 100,000 in Central QLD, and 5.5 cases per 100,000 in Tropical North QLD. The number of *Salmonella* bacteraemia cases per year increased in the study period in SE and Tropical North QLD (Fig. 2C). In SE QLD, the incidence of *Salmonella* bacteraemia more than doubled from 1.42 cases per 100,000 in 2009 (estimated population 3,055,685) to 4 cases per 100,000 in 2019 (estimated population 3,710,455). This corresponds to a significant upward trend in SE QLD (slope = 0.25, $p < 0.001$) and Tropical North QLD (slope = 0.21, $p = 0.020$), whereas no statistically significant trend was detected in Central QLD ($p = 0.067$) (Fig. 3).

In contrast, *Campylobacter* bacteraemia was less common, with a total of 234 cases reported. The highest number of cases was in SE QLD (199), followed by Tropical North QLD (22) and Central QLD (13). This translates to incidence rates of 0.48 (SE QLD), 1.05 (Tropical North), and 1.0 (Central QLD) cases per 100,000.

The weekly mean temperatures in SE, Central and Tropical North QLD were 21 °C, 21.5°C and 23.3°C, respectively. The weekly mean maximum temperatures in the regions were 28.1 (SE QLD), 27.4 (Central QLD) and 27.3 (Tropical North QLD). The mean precipitation was 20.7 mm (SE QLD), 12.3 (Central QLD) and 24 (Tropical North QLD) (Table 2).

Table 1
Demographic and geographic characteristics of study participants by pathogen type and mean weekly cases.

Category	Salmonella				Campylobacter			
	Total	Weekly mean	95% CI	SD	Total	Weekly mean	95% CI	SD
Overall	1034	2.51	(2.36, 2.66)	1.58	234	1.34	(1.25,1.43)	0.61
Sex								
Female	465	1.72	(1.6,1.84)	0.99	66	1.08	(1,1.17)	0.33
Male	569	1.73	(1.63,1.83)	0.95	168	1.30	(1.2,1.4)	0.58
Age group								
<18 years	444	1.63	(1.52,1.74)	0.92	12	1.00	(1,1)	0.00
18–39 years	217	1.28	(1.18,1.37)	0.62	43	1.07	(0.99,1.16)	0.27
40–64 years	162	1.19	(1.12,1.26)	0.43	76	1.06	(1,1.11)	0.23
≥65 years	211	1.13	(1.08,1.19)	0.36	103	1.14	(1.05,1.24)	0.44
Climate zone								
Central QLD	91	1.20	(1.10,1.30)	0.25	22	1.00	(1,1)	0.00
SE QLD	785	2.15	(2.00,2.30)	1.30	182	1.20	(1.12,1.28)	0.55
Tropical North QLD	158	1.20	(1.10,1.30)	0.50	30	1.00	(1,1)	0.00

3.2. Temperature and bacteraemia association

The time series analysis, showing the DLNM fitted values for weekly mean and maximum temperature trends, is presented in the Supplementary Material (Figs. S1 and S2). DLNM models were used separately for mean and maximum temperatures in the three regions of QLD. There was a significant association between a rise in temperature and *Salmonella* bacteraemia cases in SE QLD (mean temperature) and Tropical North QLD (mean and maximum temperatures). However, no significant association was found for Central QLD.

Table 3 presents the RR and percentage changes associated with temperature quantiles of weekly mean and maximum temperatures across the three regions of QLD. The significant cumulative effects (RR) over the 6-week period for *Salmonella* was as follows. In SE QLD, a non-linear significant increase in RR was noted above the threshold mean weekly temperature of 21.1 °C. A 1 °C rise in deseasonalised mean temperature was associated with an 8–20% increase in risk (RR range: 1.08–1.20, 95% CI varying from 1.01 to 1.40, all $p < 0.05$) with the strongest effects occurring at 2–4 week lags. Conversely, cooler temperatures below 21 °C showed protective effects, with each 1 °C decrease associated with a 6–25% reduced risk (RR range: 0.75–0.94, 95% CI: 0.64–0.99, all $p < 0.05$). Maximum deseasonalised temperatures above 28.1 °C were associated with higher risk, with a 1 °C rise corresponding to 0.3–16% increase in risk (RR range: 1.00–1.16, 95% CI: 0.84–1.57, all $p > 0.05$). Cooler maximum temperatures between 25.0 and 25.9 °C were associated with protective effects, with a 1 °C decrease in this range corresponding to a 21–24% reduced risk (RR range: 0.74–0.79, 95% CI: 0.55–0.99, all $p < 0.05$).

In Central QLD, a rise in deseasonalised mean temperature from about 11 °C to 17.5 °C showed an upward trend in risk, with RR values rising from 0.56 [95% CI: 0.31–1.02] at 11 °C to 0.80 [95% CI: 0.54–1.19] at 17.7 °C. A similar gradual increase was observed for deseasonalised maximum temperature between 16 °C and 22 °C, where RR values ranged from 0.64 [95% CI: 0.36–1.15] at 17 °C to 0.89 [95% CI: 0.60–1.31] at 23.6 °C. In Tropical North QLD, above the threshold deseasonalised mean weekly temperature of 24 °C, an 18% increase in RR was seen per additional degree, with peak significant effect at 27.2 °C (RR 1.70, 95% CI: 1.01–2.86). For deseasonalised maximum temperatures above a threshold of 28 °C, a 11–106% increase in RR was seen per additional degree rise in temperature (RR range 1.11–2.05, 95% CI: 1.01–4.73). For both mean and maximum temperatures, effects were pronounced at 3-week lags (Supplementary Material, Figs. S3a to S3f, S4a to S4f, Table 3).

Campylobacter bacteraemia was only modelled for SE QLD as the number of cases was low in the other areas. For both deseasonalised mean and maximum temperatures, no significant association was found for the rise in temperatures (Supplementary Material Figs. S5 and S6).

The sensitivity plots for model fit are shown in Supplementary

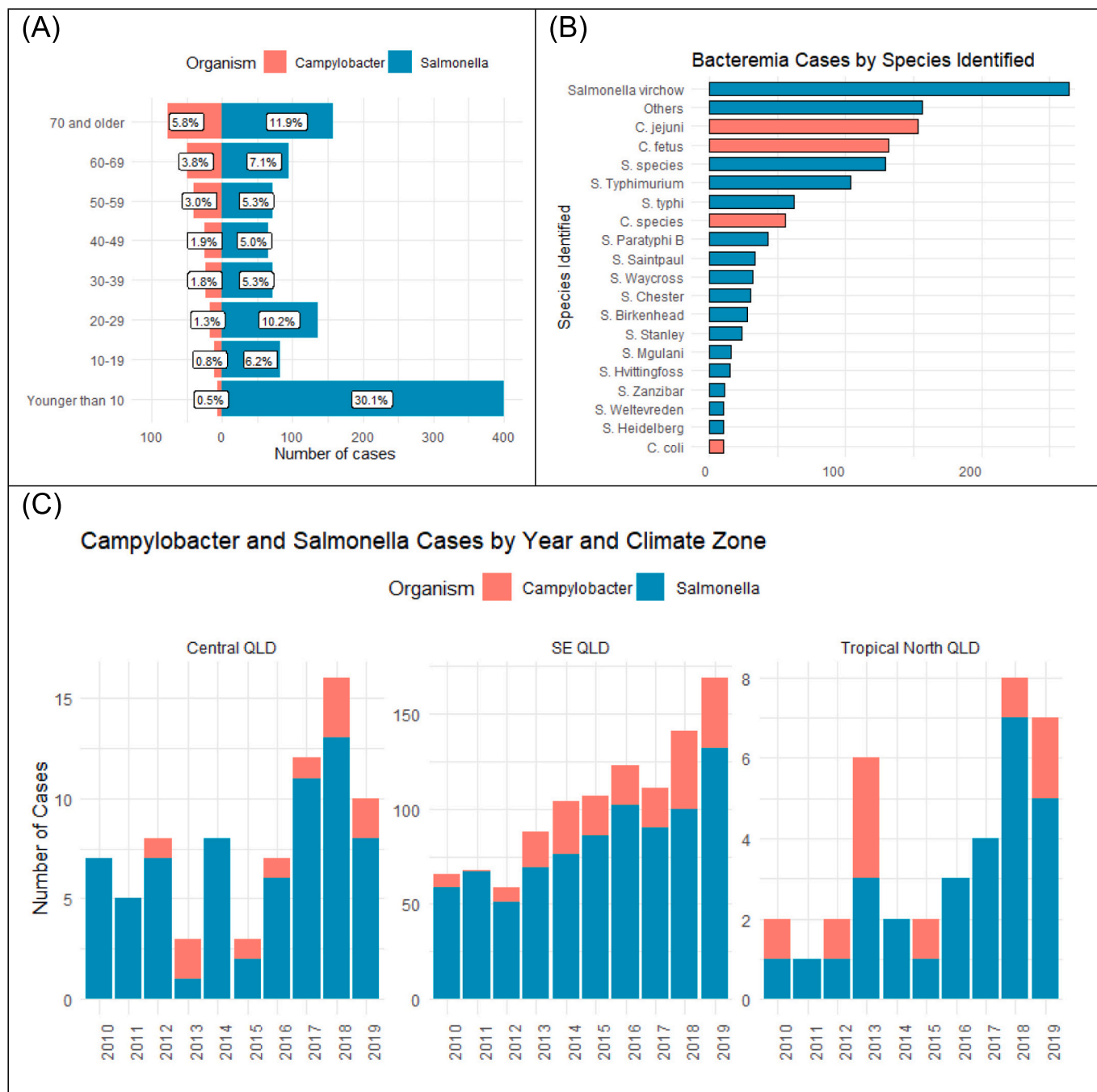


Fig. 2. Distribution and trends of *Salmonella* and *Campylobacter* bacteraemia cases by (A) age, (B) species, and (C) climate zone in Queensland, Australia, between 2010 and 2019.

Material Figs. S7-S10. Increasing precipitation had a marginal protective effect on *Salmonella* but no effect on *Campylobacter* cases in SE QLD. The effects in Central QLD and Tropical North QLD were variable, and all were statistically non-significant. The cumulative risk plots for precipitation are shown in the Supplementary Material (Fig. S11).

Heatwave exposure was investigated with a separate model for the dose-response relationship of maximum temperature and *Salmonella* bacteraemia. The incident rate ratio (IRR) was 1.31 (95% CI: 1.16–1.48, $p < 0.001$) which shows that each 1 °C increase in maximum temperature during the heatwave is associated with a 30.6% increase in *Salmonella* cases (Fig. 4). Low case numbers precluded assessment of whether there was an association between heatwaves and *Campylobacter* cases.

4. Discussion

Our study tested the hypothesis that increasing ambient temperatures and heatwaves increase the risk of *Salmonella* and *Campylobacter* bacteraemia in QLD, Australia. We found partial support for this hypothesis, as a significant association was found for *Salmonella* but not for *Campylobacter*. Specifically, we found that increases in both mean and maximum temperatures were significantly associated with a higher number of *Salmonella* bacteraemia cases in SE and Tropical North QLD. However, no statistically significant association was found in Central QLD.

The observed temperature-*Salmonella* associations are likely a reflection of multiple pathogen and human behavioural factors acting

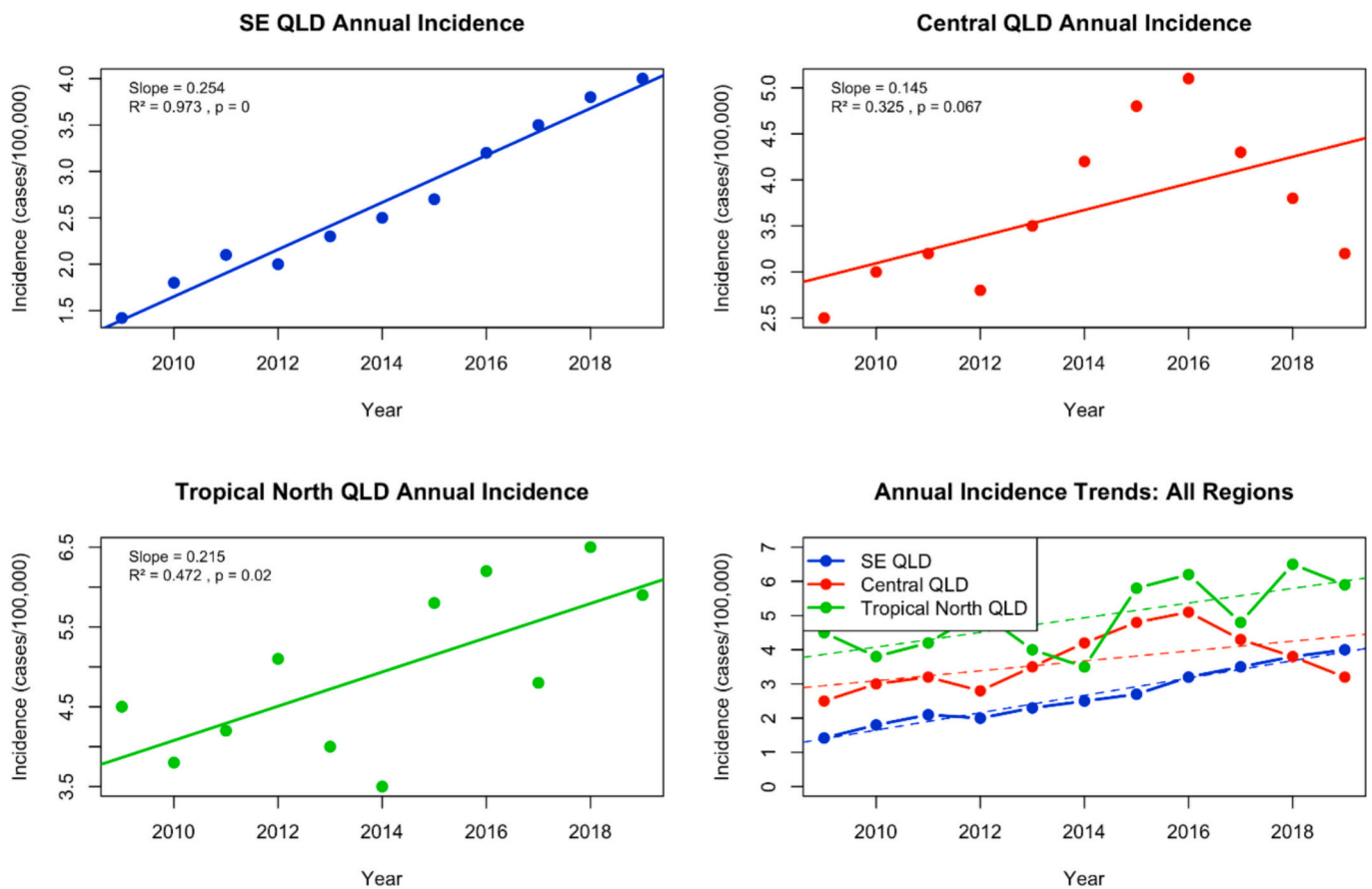


Fig. 3. Annual incidence trends of *Salmonella* bacteraemia across Queensland regions, 2009–2019.

Table 2
Summary statistics of measured weekly climate variables in the three regions of QLD.

Temperature (°C)/precipitation (mm)	Mean	Minimum	Maximum
SE QLD			
Weekly mean temperature	21.0	12.2	29.7
Weekly mean maximum temperature	28.1	19.7	41.2
Weekly precipitation	20.7	0	286.6
Central QLD			
Weekly mean temperature	21.5	9.2	34.2
Weekly mean maximum temperature	27.4	14.5	40.2
Weekly precipitation	12.3	0	280.0
Tropical North QLD			
Weekly mean temperature	23.3	12.8	33.5
Weekly mean maximum temperature	27.3	17.8	39.1
Weekly precipitation	24.0	0	659.3
Number of heatwave periods per week was 0.029 for QLD			

together at different magnitudes. More than 70% of infections in Australia from these pathogens are food-borne (Ford et al., 2016). *Salmonella* replication is enhanced at temperatures between 35 and 37 °C (Bedale, 2024). This species poses a major threat to the food industry as it can adapt to a wide range of temperatures (8–45 °C) outside its optimum growth range (Chlebicz and Śliżewska, 2018). *Campylobacter*, on the other hand, is more temperature sensitive and grows optimally between 40 and 42 °C (Chlebicz and Śliżewska, 2018).

The lag periods for developing infection reflect the potential sources of contamination at different points in the food chain. We noted a rise in cases as early as 1 week, suggesting food contamination closer to the point of consumption. This corresponds to the consumption of raw or undercooked eggs, poultry and nuts as the most common source of

Salmonellosis in SE QLD (Parisi et al., 2019). However, in SE QLD and Tropical North QLD, after an initial peak in cases within 1 week of the temperature rise (Chlebicz and Śliżewska, 2018), another peak was noted after a lag of 3 weeks and up to 6 weeks. This delayed effect suggests that contamination is likely earlier in the food supply chain.

The incidence of *Salmonella* bacteraemia increased during the study period, particularly in SE and Tropical North QLD. This finding is consistent with previously published studies (Parisi et al., 2019). *S. Virchow* was the most common serovar. The significance of serovar and environmental transmission was studied by Fearnley et al. (2018). They noted that, compared to the temperate states in Australia, QLD has a higher proportion of *Non-Typhimurium Salmonella* cases, suggesting that nuts may serve as a potential food-borne source of transmission, possibly due to environmental contamination during production or storage. Children between 0 and 9 years were the most affected. The epidemiology of paediatric salmonellosis in QLD has been described previously, noting the predominance of non-typhoidal gastroenteritis in this age group (Berger et al., 2019).

The lag periods for the rise in cases is similar to previous studies on *Salmonella* gastroenteritis that have found a positive association at lags 2–4 weeks and at temperature ranges of 15 °C–35 °C (Aik et al., 2018; Dewan et al., 2013; Fleury et al., 2006; Robinson et al., 2022). Extreme temperatures, during heatwave periods, were significantly associated with *Salmonella* bacteraemia in QLD. This is consistent with other studies (Milazzo et al., 2016; Morgado et al., 2021), which found that heatwaves have varying effects on *Salmonella* serotypes and the impact lasted up to a lag of 2 weeks. A recent systematic review (Manchal et al., 2024) reported a pooled IRR of 1.05 (95% CI:1.03,1.07) for extreme temperature (95th percentile). Included studies focused mainly on gastroenteritis infections, although they did include infections caused by *Salmonella* and *Campylobacter*. The present study strengthens the

Table 3
Relative Risk (RR) and percentage change for the temperature quantiles of weekly mean and maximum temperature for the three regions of QLD.

Temperature quantiles (%)	Mean temperature (°C)	RR (95% CI)	%change in RR	Maximum temperature (°C)	RR (95% CI)	%change in RR
Salmonella- SE QLD						
5%	18.1	0.65(0.50,0.84)	-35%	24.5	0.74(0.55,0.99)	-26%
15%	19.3	0.68(0.54,0.85)	-32%	25.7	0.76(0.59,0.98)	-24%
25%	19.8	0.76(0.64,0.90)	-24%	26.4	0.82(0.67,1.01)	-18%
75%	22.2	1.17(1.02,1.35)	+17%	29.6	1.13(0.93,1.37)	+13%
85%	23.0	1.18(0.96,1.45)	+18%	30.6	1.16(0.87,1.54)	+16%
95%	24.2	1.14(0.90,1.45)	+14%	32.2	1.15(0.84,1.57)	+15%
Average (reference) (reference (reference)	21.0	1		28.1	1	
Salmonella- Central QLD						
5%	11.6	0.56(0.31,1.02)	-44%	17.0	0.64(0.36,1.15)	-36%
15%	14.1	0.56(0.31,1.02)	-44%	19.8	0.64(0.36,1.15)	-36%
25%	17.7	0.80(0.54,1.19)	-20%	23.6	0.89(0.60,1.31)	-11%
75%	25.6	1.05(0.67,1.66)	+5%	32.0	1.01(0.63,1.62)	+1%
85%	27.4	1.05(0.63,1.75)	+5%	33.7	1.05(0.62,1.77)	+5%
95%	30.3	1.05(0.63,1.75)	+5%	36.4	1.05(0.62,1.77)	+5%
Average (reference)	22.3	1	0%	28.0	1	0%
Salmonella-Tropical North QLD						
5%	18.0	0.50(0.23,1.12)	-50%	22.0	0.57(0.29,1.13)	-43%
15%	19.3	0.63(0.38,1.03)	-37%	23.2	0.58(0.35,0.95)	-42%
25%	20.5	0.69(0.46,1.03)	-31%	24.7	0.66(0.49,0.90)	-34%
75%	26.1	1.36(1.00,1.86)	+36%	29.5	1.47(1.06,2.06)	+47%
85%	26.7	1.51(1.01,2.26)	+51%	30.5	1.67(1.08,2.58)	+67%
95%	28.1	1.94(0.99,3.82)	+94%	33.5	2.06(1.03,4.13)	+106%
Average (reference)	24.0	1		27.6	1	
Campylobacter -SE QLD						
5%	15.9	0.97(0.49,1.91)	-3%	22.9	1.06(0.58,1.96)	+6%
15%	17.2	0.95(0.55,1.64)	-5%	24.5	1.04(0.62,1.75)	+4%
25%	18.5	0.93(0.59,1.47)	-7%	25.9	1.00(0.65,1.56)	0%
75%	23.7	1.02(0.63,1.63)	+2%	30.3	1.01(0.63,1.63)	+1%
85%	24.5	0.98(0.58,1.65)	-2%	31.2	1.00(0.57,1.75)	0%
95%	25.6	0.93(0.55,1.74)	-7%	33.5	0.99(0.55,1.78)	-1%
Average (reference)	21.4	1		28.2	1	

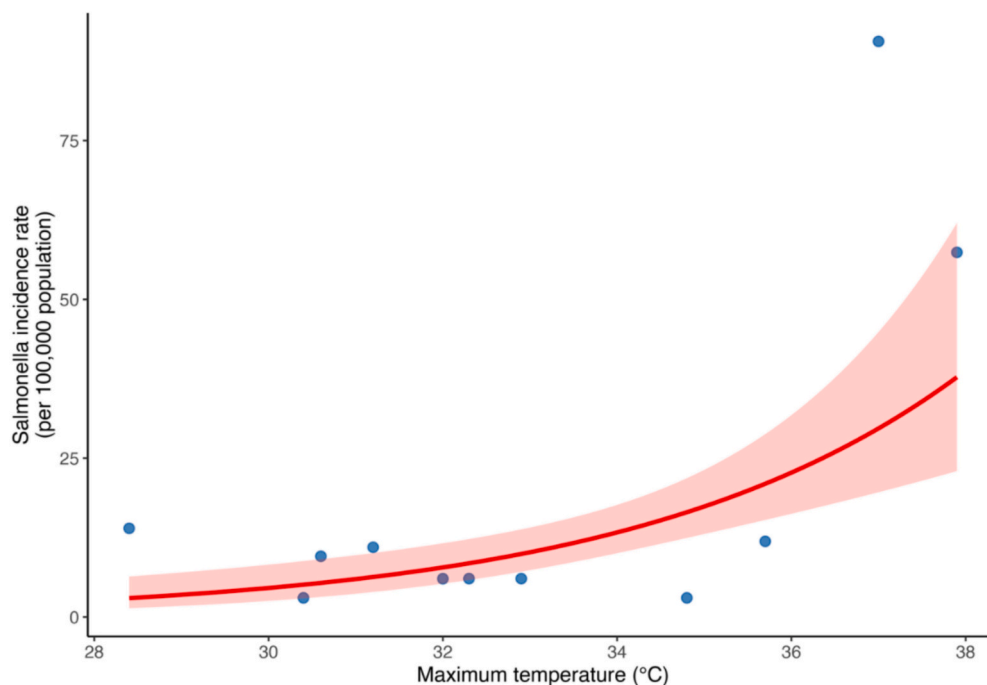


Fig. 4. The effect of heatwave temperatures on *Salmonella* bacteraemia in QLD. The solid line represents the incidence rate ratio (IRR) per 1 °C increase in maximum temperature during heatwaves, Shaded area represents 95% confidence intervals. Each 1 °C increase corresponds to an IRR of 1.31 (95% CI: 1.16–1.48, p < 0.001).

evidence by providing a more targeted and region-specific estimate of this risk in a subtropical Australian setting.

The differential impact of climate variables even within a single state is an important finding. SE QLD, with the highest population density, had a significant increase in cases with a rise in ambient temperature.

Tropical North QLD, despite having a lower population density, also showed significant associations. This could be due to exposure to more extreme temperature ranges and different food handling behaviours. However, Central QLD, with the highest crude incidence rate (4.12/100,000), did not have any significant association with temperature.

This could reflect different transmission pathways or climatic factors not captured in our models.

We found a marginal protective effect of precipitation independent of temperature and seasonal factors in SE QLD and variable effects in other regions. Only one previous Australian study noted the negative impact of precipitation on Salmonellosis (Zhang et al., 2008). However, the effect was statistically non-significant, like in this study. This contrasts with previous studies on precipitation and Salmonellosis that have found positive associations, mainly with extreme precipitation, in the Northern hemisphere (Aik et al., 2018; Jiang et al., 2015; Liu et al., 2018; Manchal et al., 2024; Morgado et al., 2021). The lack of positive effect seen in this study could be due to moderate precipitation levels and the rarer outcome of bacteraemia compared to gastroenteritis. Similarly, studies conducted in regions with moderate precipitation and temperate climates have found no significant association (7, 24, 27, 57). This is similar to the other regions in QLD in this study.

Campylobacter transmission is a complex process that involves several points of contamination in the food processing chain, ranging from consumption of undercooked chicken to breaks in preservation during storage and transport. Human behaviour during hot conditions with increased outdoor activity and rapid cooking techniques could also increase the risk of consuming undercooked food. *Campylobacter* does not replicate well in dry conditions, and studies showing a positive effect with rising temperature are primarily from Europe (Kovats et al., 2005; Kuhn et al., 2020b; Lake et al., 2019; Nichols et al., 2009). However, *C. jejuni* can tolerate arid conditions better than other species, and this was the most common isolate in the blood found in this study. However, despite our model demonstrating good sensitivity for the small number of cases, no significant positive association was found with a rise in ambient temperature. Previous studies have found a positive effect of rising temperatures between lags 0–3 weeks in the Northern hemisphere, with human behavioural changes in summer cited as the most likely explanation (Kuhn et al., 2020b; Lake et al., 2019). However, studies in Australia and New Zealand have not found any temperature-related temporal trends (Milazzo et al., 2016; Spencer et al., 2012). A systematic review found that there are mixed results for the impact of precipitation and humidity on campylobacteriosis (Austhof et al., 2024). The lack of impact of these climate variables in SE QLD, which has a humid subtropical climate, is consistent with previous studies (Bi et al., 2008; Carev et al., 2018).

We acknowledge several limitations in this study. The small sample size for *Campylobacter* did not allow for analysis in Central and Tropical North QLD, or to study the impact of heat waves on bacteraemia from these bacteria. Weather stations cannot always match exact residence postcodes, which could result in misclassification of the temperature to which the cases were exposed. Similarly, we could not account for exposure to climatic conditions outside the case residential area; for example, if cases travelled during their exposure period (exposure misclassification). Due to the ecological study design, it is not possible to make individual-level inferences (ecological fallacy). However, it provides population-wide exposure, balancing statistical power with temporal precision. We did not include relative humidity and socio-demographic factors as confounders. Humidity can promote bacterial growth and survival, particularly for *Campylobacter*, which is sensitive to desiccation (Park, 2002). However, previous studies have shown no relationship between humidity and the two pathogens (Aik et al., 2018; Bi et al., 2008; Carev et al., 2018; Zhang et al., 2010). Furthermore, the mean temperatures in our study were calculated using minimum temperatures that are directly proportional to humidity levels (Nairn and Fawcett, 2015). Socio-demographic factors, such as economic status, population density, and food handling practices, can influence the outcome. Cases were grouped by postcodes, which capture some spatial variation but not the other social confounders. Our DLNM analyses modelled weekly case counts without population denominators, consistent with standard DLNM methodology for sparse count data. We controlled for population-related secular trends through seasonal

adjustment and linear/quadratic time terms. However, we cannot rule out that unmeasured changes in population age structure or spatial distribution may have influenced observed temporal patterns. While quasi-Poisson regression appropriately addresses overdispersion in count data, our models did not explicitly account for potential residual temporal or spatial autocorrelation. If present, such correlation could affect standard error estimation and statistical inference. However, the inclusion of seasonal terms, temporal trends, and region-specific analyses likely mitigated much of this concern. Our study utilised notifiable, laboratory-confirmed surveillance data. However, temporal changes in blood culture ordering practices, laboratory detection methods, healthcare access, and reporting completeness may have affected case ascertainment independently of temperature. Our findings represent associations specifically for bacteraemia in QLD and may not generalise to all *Salmonella* or *Campylobacter* infections or in regions with substantially different climates. Lastly, as clinical data were not collected, the proportion of immunosuppressed cases was unknown, which could have influenced their propensity to develop more invasive infections.

Our study has several strengths. We analysed the impact of ambient temperature in different climate zones across QLD to demonstrate the different levels of risk and lag periods in developing bacteraemia. The rising incidence of invasive non-typhoidal *Salmonella* infection in children has significant implications for public health. This is the first study to quantify the risk of food-borne bacteraemia from these two important pathogens from the rise in ambient temperatures.

The positive temperature-*Salmonella* bacteraemia association found in this study has important implications for public health preparedness and food safety systems. Public health authorities should consider implementing targeted climate-driven strategies such as enhanced food safety messaging and surveillance during heat events. Food safety regulators may need to reassess temperature control requirements for food handling and storage in warming climates. The DLNM modelling approach provides a methodological framework for real-time disease surveillance systems that could integrate meteorological forecasting to provide early warning of increased transmission risk.

5. Conclusion

Our ecological time series analysis in QLD has demonstrated an increased risk of bacteraemia from *Salmonella* with rising ambient temperatures and heatwaves. However, no association was found with climate variables and *Campylobacter* bacteraemia. With current climate trends and rising average temperatures, the burden of bacteraemia can be expected to rise with increasing need for hospitalisation, longer duration of antibiotics and potentially increased mortality. The findings of this study highlight the importance of interdisciplinary collaboration among environmental health, food safety, and climate adaptation communities in developing evidence-based strategies for mitigating climate-sensitive health risks.

CRedit authorship contribution statement

Naveen Manchal: Writing – original draft, Software, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Megan K. Young:** Writing – review & editing, Validation, Supervision, Investigation. **Maria Eugenia Castellanos:** Writing – review & editing, Validation, Supervision, Investigation. **Oyelola A. Adegboye:** Writing – review & editing, Visualization, Validation, Supervision, Methodology, Investigation.

Informed consent/patient consent

This is a retrospective study involving patient records. There was no direct patient contact, and data were deidentified.

Ethics

This project was approved by James Cook University Human Ethics (H9034) under the project HREC/22/QPCH/87676.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.scitotenv.2026.181610>.

Data availability

The data that has been used is confidential.

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