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Climate variations and *Salmonella* infection in Australian subtropical and tropical regions

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ABSTRACT

This study aims to quantify the relationship between climate variations and cases of *Salmonella* infection in subtropical and tropical areas in Australia. Brisbane in a subtropical area and Townsville in a tropical area of Queensland were selected as the study regions. Local meteorological variables and notified cases of *Salmonella* infection from January 1990 to July 2005 were provided by local authorities. Spearman correlation and time-series adjusted Poisson regression were applied controlling for autoregression, lag effects, seasonal variation and long-term trend. Natural cubic spline and Hockey Stick model were used to estimate a potential threshold temperature. Spearman correlation indicated that maximum and minimum temperatures, relative humidity at 9 am and 3 pm, and rainfall were all positively correlated with the number of cases in both Brisbane and Townsville, with the lag values of the effects up to 2 weeks in Brisbane and 2 months in Townsville. Only temperature and rainfall were significantly included in the regression models in both regions. The models suggested that a potential 1 °C rise in maximum or minimum temperature may cause a very similar increase in the number of cases in the two regions. No threshold for the effect of maximum or minimum temperature on *Salmonella* infection was detected in either region. The association between climate variations and *Salmonella* infection could be very similar in subtropical and tropical regions in Australia. Temperature and rainfall may be used as key meteorological predictors for the number of cases in both regions.

1. Introduction

Salmonella infection is caused by the bacterium *Salmonella* and is often transmitted by contaminated food or water (Grassl and Finlay, 2008). *Salmonella* infections typically affect the gastro-intestinal tract, causing diarrhoea, vomiting, fever, and other symptoms that usually resolve without medical treatment. Reservoirs of the bacteria include humans, poultry, swine, cattle, rodents, and pets (Linam and Gerber, 2007). *Salmonella* infection is one of the most common and widely distributed enteric infections, with millions of cases being reported worldwide every year. In Australia, *Salmonella* is one of the most common agents responsible for food-borne disease outbreaks with a total of 9484 cases notified to OzFoodNet in 2007 (15% increase compared with previous five years), an official Australian government agency established to deal with enteric infections (OzFoodNet Working Group, 2008).

The relationship between enteric infections and climate variation has been documented in limited studies conducted in Europe, the USA, Asia and Australia (Rose et al., 2001; D'Souza et al., 2004; Kovats et al., 2004; Patrick et al., 2004; Tam et al., 2006; Fleury et al., 2006; Lake et al., 2009). There is evidence that the growth and dissemination of the micro-organisms responsible for enteric infections could be influenced by weather (Rose et al., 2001; Simental and Martines-Urtaza, 2008), and there is a positive association between food poisoning and environmental temperature (Bentham and Langford, 2001). However, the relationship between climate variation and *Salmonella* infection is far from clear.

Australia has unique geographic characteristics and diverse climatic regions, which makes it vulnerable to climate change manifested by rising sea levels, floods and droughts. Although some studies have demonstrated the adverse impact of climate variation in Australia (2003), the impact of climate variability on various enteric infections in different climatic regions needs to be studied in order to provide evidence for local policy making. Our study, using historical surveillance data, aims to quantify the relationship between climate variation and cases of *Salmonella* infection in subtropical and tropical areas in Australia in order to provide evidence for policy makers, health professionals, relevant industries and local communities to adapt to climate change with an associated increase in temperature.

2. Methods

2.1. Background information of studied areas

In Australia, Brisbane, the capital city of Queensland in a subtropical area, and Townsville, the biggest city from far north

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Queensland in a tropical area, were selected as the target regions (Fig. 1). Brisbane, located at 27°S and 153°E with a population in excess of 1.6 million, has a hot, humid climate with a summer average maximum temperature of around 30 °C and average temperatures of around 15 °C in winter. Precipitation is less in winter than in summer, with average monthly rainfall of about 60 mm in winter and more than 150 mm in summer (Bureau of Meteorology Australia). Townsville lies at 19°S and 146°E with a population of approximately 186,000 and an area of about 1866 km^2. The town experiences two distinct seasons, the 'wet' and the 'dry', running from January through to April, and May through to December respectively. Winter temperatures average 25 °C and summer temperatures rise to around 31 °C during the day.

2.2. Data collection

Meteorological data, including daily maximum and minimum temperatures, relative humidity (9 am and 3 pm), rainfall and air pressure, were retrieved from the Australian Bureau of Meteorology. The Brisbane and Townsville stations, which were used for this study, are local weather stations with more than 50 years of daily recording. Records from these weather stations are more than 99% complete. The Australian Bureau of Meteorology advised that the records from these weather stations would be adequate to describe the climate in the local regions. Notified daily cases of *Salmonella* infection from January 1990 to July 2005 were obtained from the Queensland Department of Health. The datasets include notification number, date of notification, date of onset, age, sex, address and source of infection. Those patients who visited a doctor and had a stool sample test with a positive result from the laboratory were reported to the surveillance system. Notified cases are more likely to be severe patients than non-notified cases (Hall et al., 2008). Due to the widespread use of computerized reporting practices in Australian laboratories for a few years, the mandatory reporting to health departments has been believed to be reliable and virtually complete (Hall et al., 2008).

2.3. Data analyses

Spearman correlation analyses were performed between each meteorological variable and the number of cases over the study period. Time-series adjusted Poisson regression was applied to quantify the relationship between meteorological variables and number of notified cases. Several regression models have been tried previously to study the association between climatic variables and *Salmonella* infection (Zhang et al., 2008). Although seasonal autoregressive integrated moving average (SARIMA) models showed its effectiveness in examining the relationship between climatic variations and *Salmonella* infection in Adelaide, a temperate city with a population size of 1.1 million (Zhang et al., 2008), it is difficult to apply SARIMA models in this study because the number of cases in one of the study regions was so small and sparse that the time-series data were non-stationary and the use of differencing and transformations could not stabilize the data. In order to compare the results from the two study regions, time-series adjusted Poisson regression was applied to data from both Brisbane and Townsville, with consideration of autocorrelation, seasonality, long-term trend and lagged effects.

Data were analysed on a weekly basis in Brisbane and monthly basis in Townsville to ensure sufficient cases for the time-series analysis. To control for the autocorrelation in the outcome variables (Y_t), autoregressive variables (at order d), (Y_{t-1}, Y_{t-2},..., Y_{t-d}) were included in the regression models. Lag values were included in the regression models in order to control for the autocorrelation of the explanatory variables. Seasonality and long-term trends, which vary

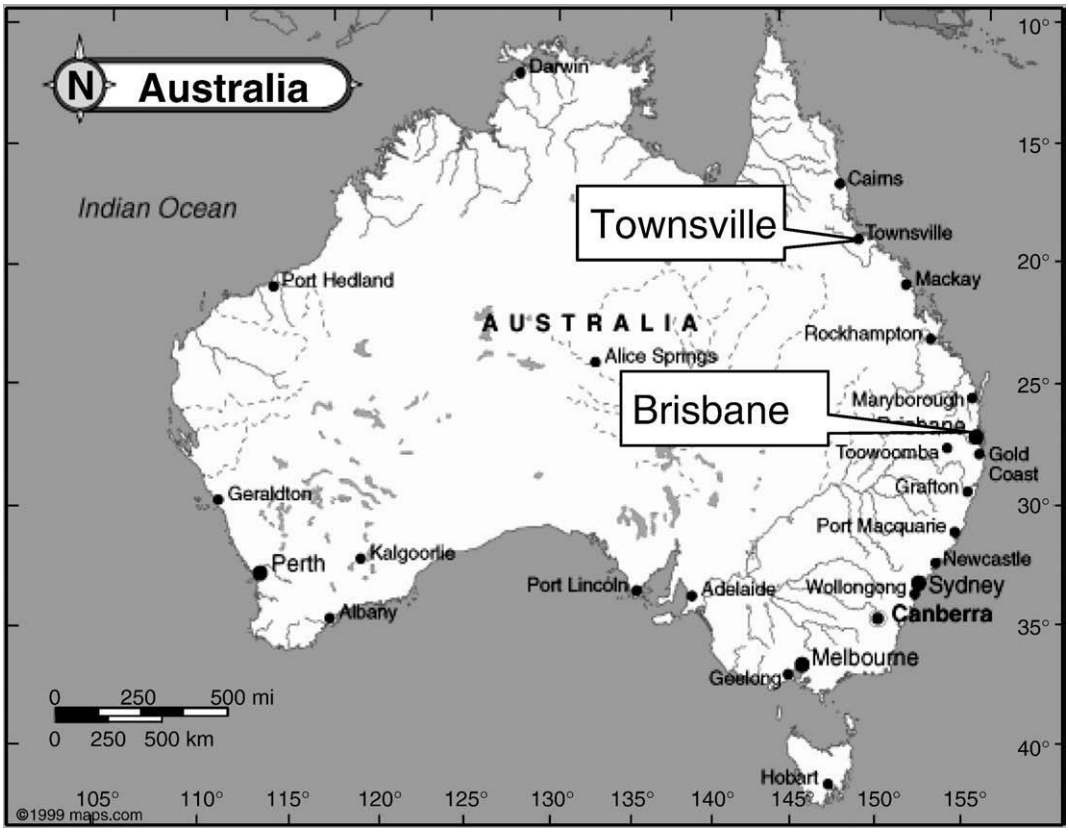
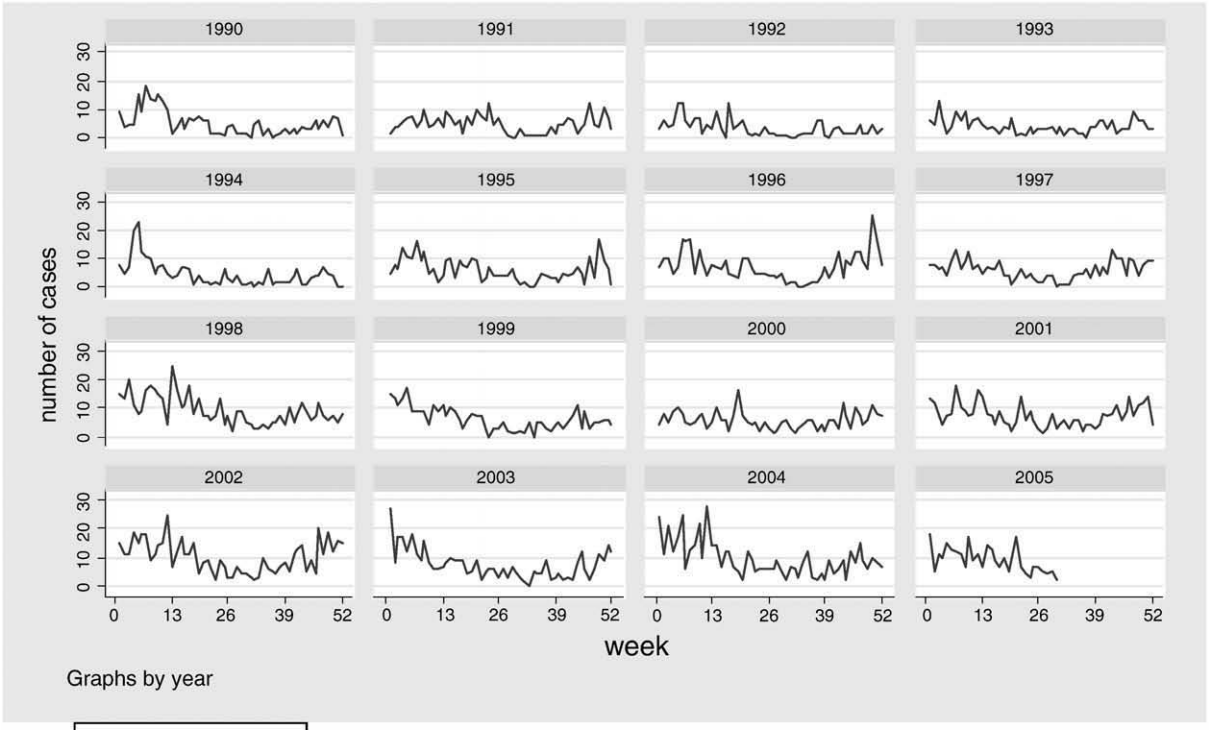


Fig. 1. The location of study regions in Australia.

over time and may be associated with weather conditions, are potential confounders of the relationship between climate variation and the disease. Given the seasonal distribution of *Salmonella* infection, a triangular function, $\sin(2\pi t/12)$ (for monthly data) and $\sin(2\pi t/52)$ (for weekly data), was included in the model to control for seasonality (Singh et al., 2001). To control for potential long-term trends over the study period, a 'year' variable was included in the models. The diagnosis of the time-series adjusted Poisson regression was performed by the goodness-of-fit, plotting of model residuals and testing of forecasting ability. Data from 1990 to 2003 were used to



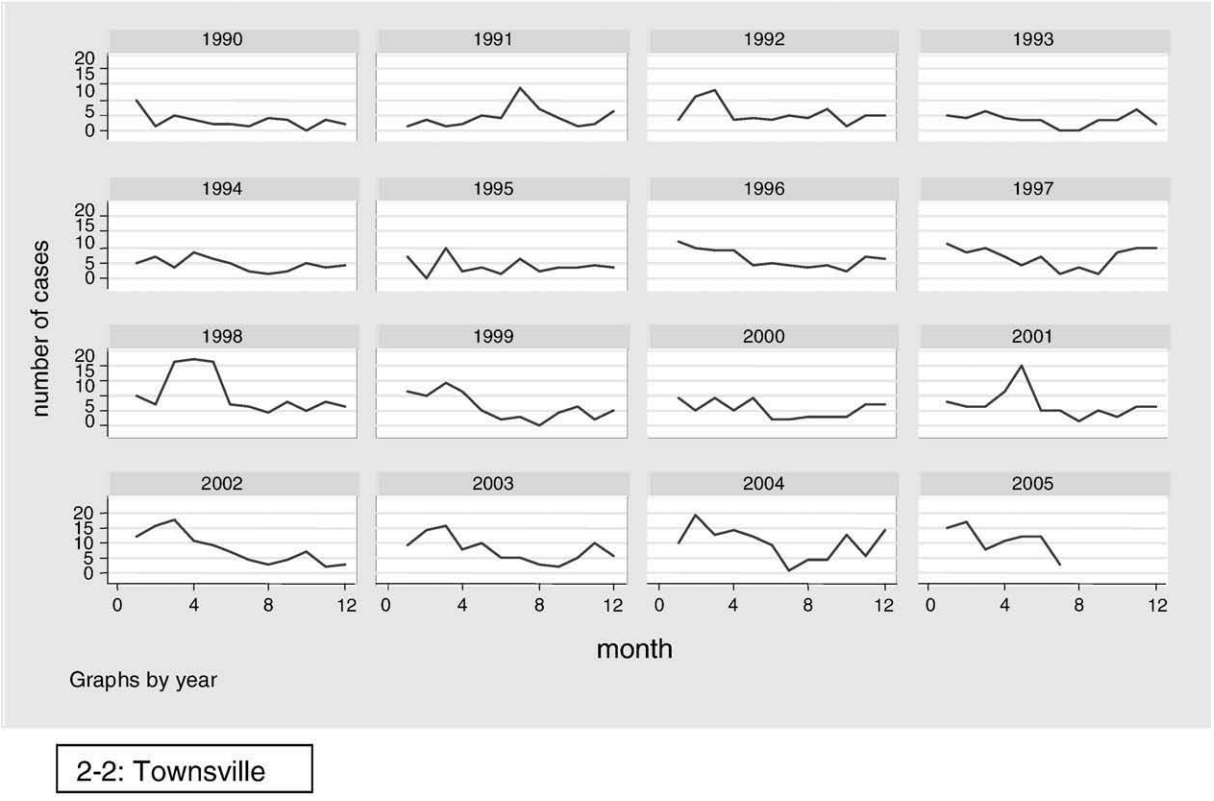


Fig. 2. Annual distribution of weekly cases of *Salmonella* infection in Brisbane and Townsville, January 1990–July 2005.

build up the models and data between 2004 and 2005 were used to test the forecasting ability of the models.

To detect a potential threshold for the effect of temperature on enteric infections, a Hockey Stick model was used to estimate a threshold temperature (Kovats et al., 2005). A natural cubic spline curve was fitted to explore the shape of the relationship before performing the Hockey Stick model. The assumption of the Hockey Stick model is that temperature has no effect on disease transmission until a threshold value is reached. Autocorrelation variables were included in the models to control for the autocorrelation of the number of cases. This approach uses the Stata–nl hockey (non-linear hockey) estimation program, which estimates complex linear and non-linear models by least squares (StataCorp, 2003). "nl" is used to estimate whether, and if so where, a change in slope occurs in the relationship between two variables by iterative numerical methods (Bi et al., 2007).

Stata 8.2 (StataCorp, 2003) was used in all the analyses with a significant level of 0.05.

3. Results

3.1. Salmonella infection and weather variables in Brisbane, southern Queensland, Australia (a subtropical area)

There were 5294 cases of *Salmonella* infection notified in Brisbane from January 1990 to July 2005, with a trend of increasing cases over the study period. A seasonal distribution of cases was observed with most occurring in summer (Fig. 2-1). Spearman correlation analyses suggested that maximum temperature ($r=0.57$, $p<0.001$), minimum temperature($r=0.56$, $p<0.001$), relative humidity at 9 am ($r=0.09$, $p<0.05$) and 3 pm ($r=0.12$, $p<0.05$) and rainfall ($r=0.23$, $p<0.001$) were all positively correlated with the weekly number of cases in Brisbane with relevant lag times from zero to 2 weeks.

Due to the high correlation between maximum and minimum temperature ($r=0.9$) and relative humidity at 9 am and at 3 pm ($r=0.8$), in order to reduce the multicollinearity in the regression modelling, two separate models were examined with Model 1 including maximum temperature and humidity at 9 am and Model 2 including minimum temperature and humidity at 3 pm. The adjusted Poisson regression for data from 1990 to 2003 suggested that the number of cases was 2-order autoregressive with a significant seasonal distribution. The 'year' was included in the models, indicating an increase in the cases over the study period. After controlling for the autocorrelation, seasonality and the increase over time, maximum or minimum temperature and rainfall, with 2 week lags, had a positive association with the number of cases in Brisbane (Table 1).

The models suggest that a potential 1 °C rise in mean weekly maximum temperature may be related to an 8.8% (95%CI: 7.6%–10.0%) increase in the weekly number of cases, and a 1 °C rise in mean weekly minimum temperature may lead to a 5.8% (95% CI: 5.0%–6.7%) increase in the weekly number of cases. The residuals of the models are randomly distributed. The observed cases, model fit cases, and predicted cases for data from 2004 to 2005 by Model 1 are demonstrated in Fig. 3-1. Diagnostic plotting of Model 2 is not presented here due to its similarity to the plots for Model 1. No threshold for the effects of maximum temperature or minimum temperature on *Salmonella* infection was detected in Brisbane. The spline curves of the relationship between cases of *Salmonella* infection in Brisbane and temperature are presented in Fig. 4-1.

3.2. Salmonella infection and weather variables in Townsville, far north Queensland, Australia (a tropical area)

In total, 1170 cases of *Salmonella* infection were notified in Townsville over the study period with a trend to increasing numbers. Most cases occurred in the summer months but the seasonal pattern was not obvious in every year over the study period (Fig. 2-2). Spearman correlation analyses demonstrated that maximum temperature ($r=0.54$, $p<0.001$), minimum temperature ($r=0.50$, $p<0.001$),

Table 1
Parameters from time-series adjusted Poisson regression for *Salmonella* infection in Brisbane.

	Coefficient	Std. err.	z	p	[95% conf. interval]
Model 1					
Lag1 case count	0.0279	0.0037	7.48	<0.001	[0.0206, 0.0352]
Lag2 case count	0.0163	0.0037	4.35	<0.001	[0.0090, 0.0236]
Lag2 max temp	0.0881	0.0062	14.31	<0.001	[0.0760, 0.1001]
Lag2 rainfall	0.0020	0.0003	6.28	<0.001	[0.0014, 0.0026]
sin(2πt/52)	27.3695	16.2749	1.68	0.033	[4.5288, 50.2102]
Year	0.0367	0.0041	8.96	<0.001	[0.0287, 0.0447]
Constant	−74.0947	8.1962	−9.04	<0.001	[−90.1590, −58.0304]
Model 2					
Lag1 case count	0.0311	0.0037	8.41	<0.001	[0.0239, 0.0384]
Lag2 case count	0.0162	0.0038	4.31	<0.001	[0.0088, 0.0235]
Lag2 min temp	0.0583	0.0044	13.36	<0.001	[0.0498, 0.0669]
Lag2 rainfall	0.0013	0.0003	3.85	<0.001	[0.0006, 0.0019]
sin(2πt/52)	34.7144	16.2028	2.14	0.032	[2.9574, 66.4713]
Year	0.0380	0.0041	9.24	<0.001	[0.0299, 0.0460]
Constant	−75.2489	8.2116	−9.16	<0.001	[−91.3434, −59.1545]

Lag1/Lag2: records occurred one-/two-week prior.

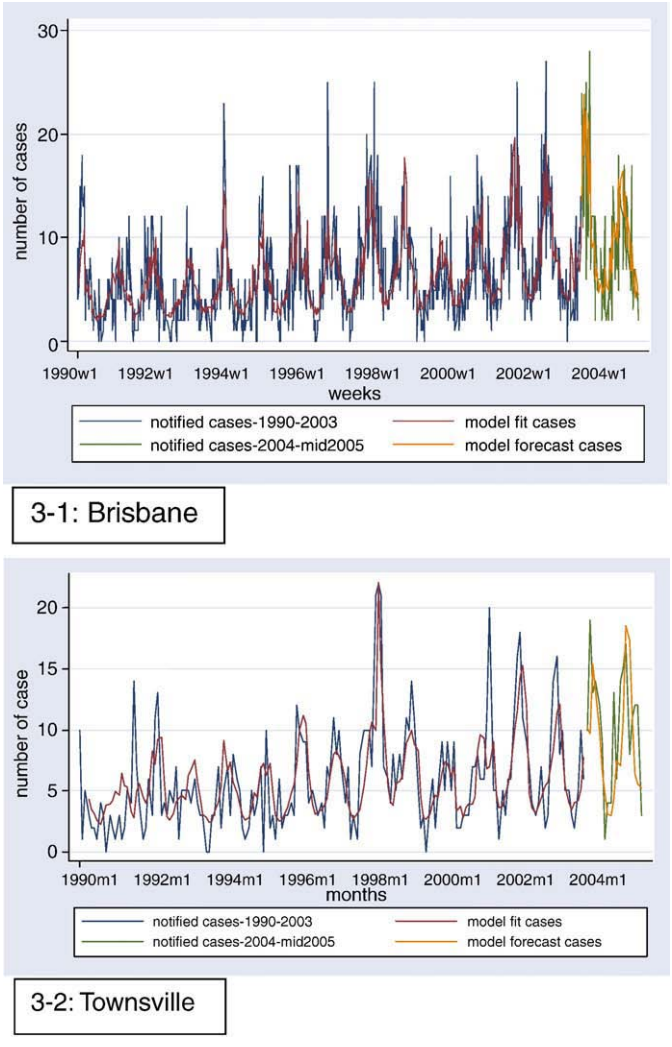


Fig. 3. Notified cases vs. model fit cases (1990–2003) and predicted cases of *Salmonella* infection (2004–2005) in Brisbane and Townsville according to Model 1.

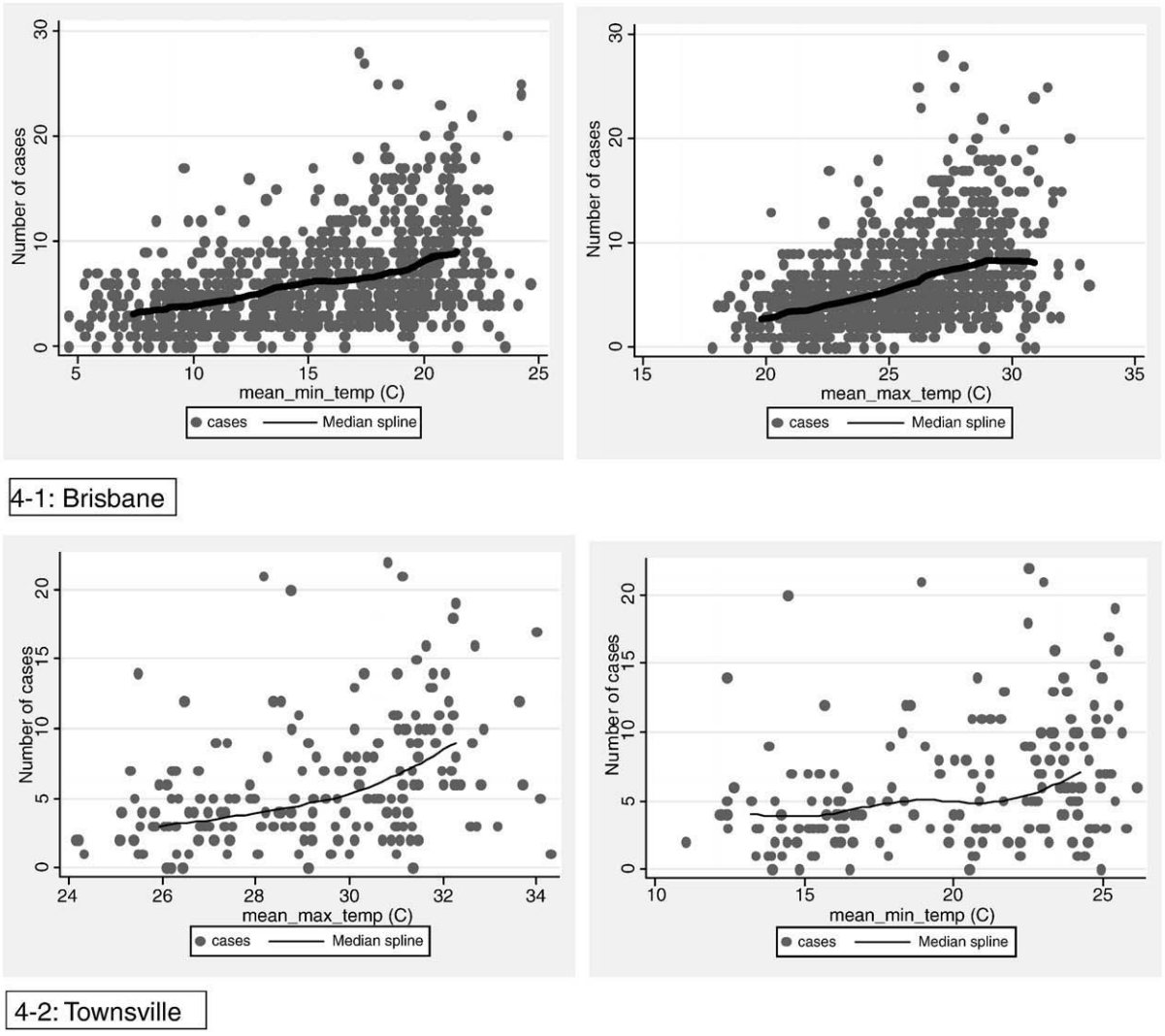


Fig. 4. Relationship between temperature and *Salmonella* infection in Brisbane and Townsville.

relative humidity at 9 am ($r=0.21$, $p<0.001$) and 3 pm ($r=0.35$, $p<0.001$), and rainfall ($r=0.40$, $p<0.005$) were all positively correlated with monthly cases of *Salmonella* infection in Townsville with 2-month lags for each of the climatic variables.

Two separate models were examined for *Salmonella* infection in Townsville with Model 1 including maximum temperature and humidity at 9 am and Model 2 including minimum temperature and humidity at 3 pm. The adjusted Poisson regression models indicate that the number of cases of *Salmonella* infection was 2-order autoregressive and had a significant seasonal distribution. Similar to Brisbane data, the models show that maximum and minimum temperatures and rainfall were significantly associated with the number of cases in Townsville (Table 2), indicating that a 1 °C rise in mean monthly maximum temperature is related to an 11.9% (95% CI: 8.7%–15.1%) increase in the monthly number of cases of *Salmonella* infection and a 1 °C rise in mean monthly minimum temperature is related to a 5.9% (95% CI: 4.0%–7.8%) increase in the number of monthly cases in Townsville.

A comparison between the models for Brisbane and Townsville, reveals that although a 1 °C rise in temperature may cause a similar relative increase in the number of cases, the lag times for the effects of temperatures were shorter and the lag times for the effects of rainfall were longer in Townsville. The model diagnosis plots are demonstrated in Fig. 3-2. As with Brisbane, in Townsville there was no threshold for the effect of maximum and minimum temperatures on *Salmonella* infection detected. The spline curves of the relationship between cases of *Salmonella* infection and temperatures in Townsville are presented in Fig. 4-2.

Table 2
Parameters from adjusted Poisson regression for *Salmonella* infection in Townsville.

	Coefficient	Std. err.	z	$P>\|z\|$	[95% conf. interval]
Model 1					
Lag1 case count	0.0439	0.0082	5.36	0.000	[0.0278, 0.0599]
Lag2 case count	0.0266	0.0088	3.02	0.002	[0.0094, 0.0439]
Mean max temp	0.1188	0.0162	7.33	0.000	[0.0870, 0.1505]
Lag3 rainfall	0.0006	0.0002	3.19	0.001	[0.0002, 0.0009]
Year	0.1501	0.0231	8.70	0.000	[0.1050, 0.1952]
sin(2πt/12)	99.5097	38.381	2.59	0.010	[24.2833, 174.7360]
Constant	0.2059	0.2500	0.85	0.382	[−0.2841, 0.6959]
Model 2					
Lag1 case count	0.0470	0.0082	5.73	0.000	[0.0309, 0.0631]
Lag2 case count	0.0249	0.0089	2.80	0.005	[0.0075, 0.0423]
Mean min temp	0.0587	0.0096	6.09	0.000	[0.0398, 0.0776]
Lag3 rainfall	0.0005	0.0002	2.79	0.005	[0.0001, 0.0008]
Year	0.1567	0.0198	7.82	0.000	[0.1250, 0.1884]
sin(2πt/12)	110.4626	38.8100	2.85	0.004	[34.3964, 186.5289]
Constant	0.2186	0.2500	0.87	0.382	[−0.2712, 0.7085]

Lag1/Lag2/Lag3: Records occurred one-/two-/three-month prior.

4. Discussion

This study of the effect of climate variation on *Salmonella* infection in subtropical and tropical regions in Australia indicates that increases in both maximum and minimum temperatures are associated with an increase in *Salmonella* infection in both the study regions. The positive relationship between temperatures and *Salmonella* infection is similar to recent findings from Europe, North America and Asia, which reported associations between temperatures and *Salmonella* infection (D'Souza et al., 2004; Kovats et al., 2004; Fleury et al., 2006), Hepatitis A (Hu et al., 2004), campylobacteriosis (Patrick et al., 2004; Kovats et al., 2005; Tam et al., 2006; Bi et al., 2008) and other enteric infections (Pinfold et al., 1991; Nath et al., 1992; Bentham and Langford, 2001; Singh et al., 2001; Charron et al., 2004).

The control of *Salmonella* infection is an ongoing challenge and it is worthwhile understanding the underlying causes, including climate variation particularly global warming (O'Brien and Valk, 2003; Hall et al., 2002). Temperature may affect the transmission of *Salmonella* infections via several causal pathways, such as direct effects on bacterial proliferation and indirect effects on eating habits during hot days. The optimum temperature for the growth of *Salmonellae* is between 35 °C and 37 °C. The growth is greatly reduced at less than 15 °C (Doyle and Mazzotta, 2000). Although not all cases of *Salmonella* infections in Australia are food-borne, more than 70% of cases are believed to be transmitted by food (The OzFoodNet Working Group, 2008). Ambient temperature influences the development of *Salmonellae* at various stages in the food chain, including bacterial loads on raw food production, transport and inappropriate storage (Jiménez et al., 2009; Komitopoulou and Peñaloza, 2009). The potential thresholds for the effect of temperature on *Salmonella* infection have been investigated in this study. Contrary to our expectation and reports from other studies (D'Souza et al., 2004; Kovats et al., 2004), no threshold temperature was detected in the subtropical and tropical regions in Queensland, Australia. This may reflect the local climate conditions with a narrow range of temperatures in both regions.

In the subtropical and tropical regions of Queensland, rainfall is positively associated with the number of cases *of Salmonella* infection. Rainfall, especially heavy rainfall events, may affect the frequency and level of contamination of drinking water, and hence enteric infection. A strong association between drinking water quality, precipitation and gastroenteritis in New Zealand has been described (Weinstein and Woodward, 2005). Relative humidity was not significantly included in the regression models, although it was significant in the correlation analysis. Further studies are needed to examine the effects of interactions among the weather variables in predicting the number of cases.

Climatic variables may not affect *Salmonella* infection directly but via a variety of pathways as mentioned above. Therefore, lagged effects of these variables on the number of cases of *Salmonella* infection are observed by this study and by another recent study conducted in the UK, reporting 2–5 weeks lagged effect of temperature on *Salmonella* infection (Lake et al., 2009). By comparing results from these two study areas from our study, it can be seen that the lagged effects of the meteorological variables on *Salmonella* infection varied. Lag times for the impact of maximum and minimum temperatures are shorter in Townsville (0 months), the tropical region, than that in Brisbane (2 weeks), the subtropical region. This minor difference may be due to the narrower range of temperatures in the tropical region. It may also be because of the different frequency of the data used in the models, which requires more precise modellings to verify the different time lag of the effects of temperature on *Salmonella* infection in these two regions. However, the lag effects of rainfall on *Salmonella* infection are longer (3 months) in Townsville than that in Brisbane (2 weeks), which may be due to less rainfall in the tropical region or more contamination happened closer to food consumption in the subtropical region. No matter what the reason might be, it has implications for local public health practitioners. Given the variety of the lagged effect of climatic variables on *Salmonella* infection, strategies for the prevention and control of this disease should take into account local climatic conditions. The high quality of disease surveillance data in Australia enhances the research strength of this study. Date of disease onset were used in the analysis rather than notification date because the onset date is more closely correlated with the date of infection. Therefore, as discussed by Kovats et al. (2004), the use of the onset date overcomes the shortcoming of recording only the date of notification in most disease surveillance systems, which generally entails a delay of approximately 1 week for diarrhoeal diseases. This is very important for the epidemiology of enteric infection when considering prevention and control.

Under-reporting is an important issue in disease surveillance systems, especially for enteric infections. Generally only those patients with severe symptoms go to see the doctor and are notified to health authorities. It is reported that only approximately one in fifteen cases of enteric infection is notified in Australia (Hall et al., 2005) and the number of annual community infection of *Salmonella* infection would be 49,843 (95% CI: 28,466–118,518) (Hall et al., 2008). Under-reporting obviously has an impact on the study results. However, it is to be believed that the trend in under-reporting remained consistent over the study period. Therefore, the association between climatic variables and *Salmonella* infection could be detected by the time-series analyses in controlling for the confounders. The other limitation of our study is the change of communicable disease notification system in Australia after 1992. In order to have enough time-series points to guarantee the statistical power for the time-series analysis, it was assumed there are no important differences between GP and laboratory reports of *Salmonella* infection caused by the change of the notification system. In addition, there are more than 2000 serotypes of *Salmonellae* in the natural environment and not all of them are food-borne, which may have different sensitivity to climate variation (O'Brien and Valk, 2003; Hall et al., 2005). This study did not distinguish different serotypes of *Salmonella* infection.

The transmission of *Salmonellae* to humans is a complex ecological process. Meteorological variables are a component of a causal network—neither necessary nor sufficient for the transmission of the infection. Warmer temperatures, in combination with differences in eating behaviour, may contribute to enteric infections including *Salmonella* infection. Furthermore, many factors not as yet examined, such as socioeconomic factors may play a significant role in enteric infection. Studies on the independent impact of climate variation on host/reservoirs, patients and bacterial survival, are necessary in the future.

References

Bentham G, Langford IH. Environmental temperatures and the incidence of food poisoning in England and Wales. Int J Biometeorol 2001;45:22–6.

Bi P, Zhang Y, Parton K. Weather variables and Japanese Encephalitis in the metropolitan area of Jinan city, China. J Infect 2007;55:551–6.

Bi P, Cameron AS, Zhang Y, Parton K. Weather and notified *Campylobacter* infections in temperate and sub-tropical regions of Australia: an ecological study. J Infect 2008;57:317–23.

Bureau of Meteorology Australia. Retrieved May 2008, from http://www.bom.gov.au/cliamte/how.

Charron D, Thomas M, Waltner-Toews D, Aramini J, Edge T, Kent R, et al. Vulnerability of waterborne diseases to climate change in Canada: a review. J Toxicol Environ Health A 2004;67:1667–77.

Doyle ME, Mazzotta AS. Review of studies on the thermal resistances of salmonellae. J Food Prot 2000;63:779–95.

D'Souza R, Becker N, Hall G, Moodie KB. Does ambient temperature affect foodborne disease? Epidemiol Infect 2004;15:86–92.

Fleury M, Charron DF, Holt JD, Allen OB, Maarouf AR. A time series analysis of the relationship of ambient temperature and common bacterial enteric infections in two Canadian provinces. Int J Biometeorol 2006;50:385–91.

Grassl GA, Finlay BB. Pathogenesis of enteric *Salmonella* infections. Curr Opin Gastroenterol 2008;24:22–6.

Hall G, D'Souza RM, Kirk M. Foodborne disease in the new millennium: out of the frying pan and into the fire? Med J Aust 2002;177:614–8.

Hall G, Kirk M, Becker N, Gregory JE, Univomb L, Millard G, et al. Estimating foodborne gastroenteritis, Australia. Emerg Infect Dis 2005;11:1257–64.
Hall G, Yohannes K, Raupach J, Becker N, Kirk M. Estimating community incidence of *Salmonella*, *Campylobacter*, and Shiga toxin-producing *Escherichia coli* infections, Australia. Emerg Infect Dis 2008;14:1601–9.
Hu W, McMichael AJ, Tong S. El Nino Southern Oscillation and the transmission of hepatitis A virus in Australia. MJA 2004;180:487–8.
Jiménez SM, Tiburzi MC, Salsi MS, Moguilevsky MA, Pirovani ME. Survival of *Salmonella* on refrigerated chicken carcasses and subsequent transfer to cutting board. Lett Appl Microbiol 2009;48:687–91.
Komitopoulou E, Peñaloza W. Fate of *Salmonella* in dry confectionery raw materials. J Appl Microbiol 2009;106:1892–900.
Kovats R, Edwards S, Hajat S, Armstrong BG, Ebi KL, Menne B. The effect of temperature on food poisoning: a time-series analysis of salmonellosis in ten European countries. Epidemiol Infect 2004;132:443–53.
Kovats RS, Edwards S, Charron D, Cowden J, D'Souza RM, Ebi KL, et al. Climate variability and campylobacter infection: an international study. Int J Biometeorol 2005;49: 207–14.
Lake IR, Gillespie IA, Bentham G, Nichols GL, Lane C, Adak GK, et al. A re-evaluation of the impact of temperature and climate change on foodborne illness. Epidemiol Infect 2009;137:1538–47.
Linam WM, Gerber MA. Changing epidemiology and prevention of *Salmonella* infections. Pediatr Infect Dis J 2007;26:747–8.
Nath G, Singh S, Sanyal SC. Childhood diarrhoea due to rotavirus in a community. Indian J Med Res 1992;95:259–62.
O'Brien S, Valk H. Salmonella-'old organism', continued challenges. Eur Surv 2003;8:29–31.
OzFoodNet Working Group. Monitoring the incidence and causes of diseases potentially transmitted by food in Australia Annual Report of the OzFoodNet network, 2007. Communicable Diseases Intelligence 2008;32:400–24.
Patrick M, Christiansen L, Wainø M, Ethelberg S, Madsen H, Wegener HC. Effects of climate on incidence of *Campylobacter* spp. in humans and prevalence in broiler flocks in Denmark. Appl Environ Microbiol 2004;70:7474–80.
Pinfold J, Horan N, Nara DD. Seasonal effects on the reported incidence of acute diarrhoeal disease in northeast Thailand. Int J Epidemiol 1991;20:777–86.
Rose JB, Epstein PR, Lipp EK, Sherman BH, Bernard SM, Patz JA. Climate variability and change in the United States: potential impacts on water- and foodborne diseases caused by microbiologic agents. Environ Health Perspect 2001;109:211.
Simental L, Martines-Urtaza J. Climate patterns governing the presence and permanence of salmonellae in coastal areas of Bahia de Todos Santos, Mexico. Appl Environ Microbiol 2008;74:5918–24.
Singh RB, Hales S, de Wet N, Raj R, Hearnden M, Weinstein P. The influence of climate variation and change on diarrheal disease in the Pacific Islands. Environ Health Perspect 2001;109:155–9.
StataCorp. Stata statistical software: release 8. College Station, TX: StataCorp LP; 2003.
Tam C, Rodrigues L, O'Brien SJ, Hajat S. Temperature dependence of reported *Campylobacter* infection in England, 1989–99. Epidemiol Infect 2006;34:119–25.
Weinstein P, Woodward A. Ecology, climate, and campylobacteriosis in New Zealand. In: Ebi K, Smith J, Burton, editors. Integration of public health with adaptation to climate change. London: Taylor & Francis; 2005. p. 61–72.
Zhang Y, Bi P, Hiller JE. Climate variations and salmonellosis transmission: a comparison of regression models. Int J Biometeorol 2008;52:179–87.