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Weather and notified *Campylobacter* infections in temperate and sub-tropical regions of Australia: An ecological study

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Weather and notified *Campylobacter* infections in temperate and sub-tropical regions of Australia

Abstract

Background

The relationship between weather and food-borne diseases has been of great concern recently. However, the impact of weather variations on food-borne disease may vary in different areas with various geographic, weather and demographic characteristics. This study was designed to quantify the relationship between weather variables and *Campylobacter* infections in two Australian cities with different local climatic conditions.

Methods

An ecological-epidemiological study was conducted, using weekly disease surveillance data and meteorological data, over the period 1990-2005, to quantify the relationship between maximum and minimum temperature, rainfall, relative humidity and notifications of *Campylobacter* infections in Adelaide, with a temperate Mediterranean climate, and Brisbane, with a sub-tropical climate. Spearman correlation and time-series adjusted Poisson regression analyses were performed taking into account seasonality, lag effects and long-term trends.

Results

The results indicate that weekly maximum and minimum temperatures were inversely associated with the weekly number of cases in Adelaide, but positively correlated with the number of cases in Brisbane, with relevant lagged effects. The effects of rainfall and relative humidity on *Campylobacter* infection rates varied in the two cities.

Conclusion

Weather might have different effect on *Campylobacter* infections in different cities.

Further studies are needed for a better understanding of these relationships for they may indicate epidemiologic factors important for control of these infections.

Keywords: *Campylobacter* infections, Climate, Sub-tropical, Temperate

BACKGROUND

Campylobacter infection has emerged as a leading bacterial cause of gastroenteritis in developed countries since the 1970s [1]. New Zealand and Australia have the highest incidence rates [2, 3]. In Australia, enteric infection is an important public health issue with approximately 17 million cases annually and 4 to 7 million of these estimated as foodborne [3]. It is estimated that foodborne diseases cause about 76 million illnesses in the US each year [4]. High incidence of enteric infections was also found in the UK [5] and Canada [6]. In Australia, the cost of enteric infections was estimated to be over \$1.2 billion each year approximately [7]. In 2004, the Australian national foodborne diseases surveillance system, recorded 24,313 notifications of eight potentially foodborne diseases, along with 118 outbreaks of foodborne disease. The most common sporadic disease was *Campylobacter* infections (15,640 cases) [8]. All age groups are affected, but infections are prominent in children under five years and in young adults [8].

The relationship between environmental factors and enteric infections has been studied, and the results from the USA and Europe indicate that environmental factors, including weather variables, may influence the reservoir, the growth and dissemination of the micro-organisms responsible for enteric infections including *Campylobacter* infection [5, 6, 9-12]. It is not certain whether the results from these previous studies can be applied to different ecological/meteorological regions, given various population characteristics, eating behaviors, food processing chains, socio-economic status and climate types. Therefore, we have studied the relationship between weather variables and weekly notified *Campylobacter* infections, using time-series analysis, in two Australian cities, Brisbane and Adelaide, with sub-tropical and temperate climatic conditions, respectively. The objective of this study is to determine whether there is an association between weather variables and *Campylobacter* infection in different climatic areas in Australia, because this may lead to discerning of epidemic factors important for control of these infections.

MATERIALS AND METHODS

Background information

Adelaide is the capital city of the State of South Australia. Situated at the intersection of latitude: 34° 56'S and longitude: 138° 35'E with an altitude of 43 meters (Figure 1). The population was approximately 1.1 million over the study period. The city has a typical Mediterranean climate with mild-to-cool, wet winters and hot, dry summers. Brisbane is the capital city of the State of Queensland, lying at the intersection of latitude: 27° 23'S

and longitude: 153° 7' E (Figure 1). Over the study period, the population was approximately 1.6 million. It has a sub-tropical climate with very hot humid summers and mild, dry, sunny winters. Most rain falls during summer, between November and February.

(Figure 1 about there)

Data collection

Disease surveillance data. Laboratory-confirmed *Campylobacter* infections from 1990 to mid-2005 in Adelaide and Brisbane were provided by the Communicable Diseases Control Branches (CDCB) of the South Australian Department of Health and Queensland Department of Health. The data include daily, weekly and monthly counts of the cases over the study period. These notification systems record the onset of disease, which is more useful for epidemiological studies than the date of notification which for the most part has been used in previous research.

Meteorological data. The weather stations used in this study are the Kent Town station in Adelaide and Brisbane Airport station. Both stations have a long history of weather records which represent the local weather variability very well, according to the Australian Bureau of Meteorology. Daily, weekly and monthly weather variables, including mean maximum and minimum temperatures, relative humidity and rainfall over the study period were provided.

Data analysis

After a descriptive summarization of weather variables and disease incidence, Spearman's correlation coefficients were calculated between weather variables and the notified number of the cases on a weekly basis. To examine lagged effects, lags of 1-15 weeks were included. The lagged effects of climatic variables were explored by cross-correlation analysis and the climatic variables with the maximum correlation coefficient are presented.

In contrast with standard regression models, it is desirable to allow the dependent variable to be influenced by both the past values of independent variables and possibly by its own past values [13]. Time-series adjusted Poisson regression analysis was performed, using Stata [14], allowing consideration of autocorrelation, seasonality and lag effects. Potential seasonal variation was controlled for by including a categorical seasonal variable. Test of the possibility of over-dispersion of Poisson distribution was performed, and the result did not demonstrate extra-Poisson variation in this study. Seasons in

Australia were defined according to local weather: 0-summer (weeks49-8), 1-spring (weeks37-48), 2-autumn (weeks9-22), 3-winter (weeks23-36). Moreover, in order to control for the interannual trend in the number of cases over the 15-year study period, a variable was included in the regression model specifying the year of onset. To avoid the problem of multicollinearity due to the high correlation among the climatic variables in both cities, particularly between weekly mean minimum and maximum temperatures ($r>0.8$), and between relative humidity at 9am and 3pm ($r>0.7$), two models were set up. Model I had maximum temperature and humidity at 9am as explanatory variables, and Model II had minimum temperature and humidity at 3pm.

The primary Poisson regression model adjusted for autocorrelation for this study was:

$$\ln(Y_t) = \alpha + \beta_1 Y_{t-1} + \beta_2 Y_{t-2} + \dots + \beta_p Y_{t-p} + \beta_{p+1} \text{temperature}_t + \beta_{p+2} \text{temperature}_{t-1} + \dots + \beta_{p+q} \text{temperature}_{t-q} + \beta_{p+q+1} \text{rainfall}_t + \beta_{p+q+2} \text{rainfall}_{t-1} + \dots + \beta_{p+q+r} \text{rainfall}_{t-r} + \beta_{p+q+r+1} \text{humidity}_t + \beta_{p+q+r+2} \text{humidity}_{t-1} + \dots + \beta_{p+q+r+s} \text{humidity}_{t-s} + \beta_{p+q+r+s+1} \text{Season} + \beta_{p+q+r+s+2} \text{Year}$$

where p, q, r are the lags determined by correlation analysis. The stepwise method was used in the analysis to include variables as long as there was a significant improvement determined by calculation of the maximum likelihood [15]. Only final parameter estimates of regression Model I are presented. The diagnosis of the models was performed by goodness-of-fit and residual plotting. Data from 1990-2003 were used to develop the model and data from January, 2004 to July, 2005 were used to test the forecasting ability of the model. The significance level in the analysis was $\alpha=0.05$.

RESULTS

Description of *Campylobacter* infections in Brisbane and Adelaide, 1990-2005

In Adelaide, there were 20,211 notified cases of *Campylobacter* infection over the study period, with 20.12% of notified cases occurring in children under 5 years. There is no consistent obviously seasonal distribution (Figure 2), however averaging across years reveals that major peak weeks over the study period occurred between weeks 45 and 48 (late spring), and between weeks 28 and 33 in winter.

In Brisbane, there were 14,697 notified *Campylobacter* infections over the study period. As in Adelaide, there is no clear seasonal distribution (Figure 3). However, on the average, major peak weeks over the study period occurred between the weeks 40 and 47 (late spring), and between the weeks 52 and 2 in summer. This seasonal difference suggests that different causal mechanisms may be occurring in the two cities.

(Figures 2 and 3 about here)

Weather and *Campylobacter* infections in Adelaide

(Table 1 about here.)

As shown in Table 1, there was an inverse correlation in Adelaide between weekly mean minimum and maximum temperatures and weekly notifications of *Campylobacter* infections, with the highest correlation coefficients having a lag of 9 weeks, while rainfall

was correlated at a marginal level. Relative humidity was positively associated with the numbers of cases with a lag of 14 weeks.

The time-series adjusted Poisson regression Model II, using minimum temperature as an explanatory variable is not presented in this paper because of its great similarity to Model I with maximum temperature.

(Table 2 and Figure 4 about here)

Table 2 and Figure 4 show that the number of cases was 3rd-order autoregressive, indicating that the number of notified cases in the current week was related to the numbers of cases occurring in the previous one, two and three weeks. More importantly, weekly maximum temperature had an inverse effect on the *Campylobacter* infections but rainfall had a positive effect in Adelaide. Season also made a contribution to the number of disease notifications. Relative humidity was not significant and excluded from the model.

As shown in Figure 4, the estimated/expected number of cases from Model I fits very well the observed number of cases in Adelaide for the period 1990-2005, including the peak values. In the diagnosis of the residuals of the model, a random distribution was observed with no autocorrelation among them.

Weather and *Campylobacter* infection in Brisbane

(Table 3 about here)

Table 3 indicates that there was a positive correlation in Brisbane between mean weekly minimum temperature, maximum temperature and rainfall and weekly new notifications of *Campylobacter* infections, with a 6-week and 1-week lagged effect, respectively; while relative humidity had an inverse correlation with the weekly notifications with a lag time of 3 weeks.

The results from the time-series adjusted Poisson regression (Table 4) show that the number of cases is 4th-order autoregressive, indicating that the number of notified *Campylobacter* infections in the current week is related to the numbers of cases occurring in the previous one, two, three and four weeks. The year of onset was included in the model as an independent variable indicating that there was an increase of disease notification over the study period (this is not the case in Adelaide). More importantly, after controlling for the long-term trend and seasonality, weekly maximum temperature had a positive association with the weekly notifications of *Campylobacter* infections, but relative humidity had a negative relationship. Rainfall was not significant and excluded from the model. As shown in Figure 5, the estimated/expected number of cases from the regression model fits very well to the observed number of *Campylobacter* cases in Brisbane, 1990-2005, including the peak values.

(Table 4 and Figure 5 about there)

DISCUSSION

We systematically studied the impact of weather on the number of cases of *Campylobacter* infections in two Australian cities with different climatic conditions, using weekly disease surveillance data. Our results indicate that the seasonal distribution of the number of cases varied in these different climatic areas. After controlling for the seasonal variation, the lag effects and long-term trends, the results indicate that both weekly mean maximum and minimum temperatures have an association with the number of *Campylobacter* infections. However, the direction of the association is different—negative in Adelaide, the temperate city, but positive in Brisbane, the subtropical region.

The seasonal distribution of *Campylobacter* infections may vary in different geographic, latitude and climatic regions, therefore it may be too simplistic to study *Campylobacter* epidemiology at the national level. This is one reason for selecting different climatic regions in Australia for this study. A recent international study incorporating 13 countries including Canada, Europe, Australia and New Zealand, indicated many, but not all, populations have a peak in spring [11]. A study in Portugal, another country with a Mediterranean climate, found that most cases occurred in winter [16]. Moreover, a very recent Canadian study, focusing on two provinces, suggested that the peaks of *Campylobacter* infection were evident in early summer in Alberta and Newfoundland-Labrador [6]. This was also observed in Denmark [12]. In New Zealand, there was a different seasonal distribution between north and south islands [11]. We consider that

these different seasonal distributions in different areas may indicate that there are different roles for the various reservoirs, host and environmental factors.

Temperature may impact on the growth of foodborne pathogens [10], animal reservoirs and host behaviors. It may affect eating habits, the type of food prepared in households and the meat industry. A 1°C rise in temperature has been found to correspond to a 5% increase of reported *Campylobacter* infections in England [5] and a 4.5% increase in Newfoundland-Labrador, Canada, with a threshold temperature of 0°C [6]. The associations between temperatures and *Campylobacter* infection in the two cities of our study indicate that weather may be one of the contributing factors for *Campylobacter* infection, although the direction of the association is different. The possible underlying

causes of the different directions of the association between the two cities might include the different local weather conditions and their impacts on local animal reservoirs or food processing processes. Unlike Brisbane with a humid subtropical climate, Adelaide has a typical Mediterranean climate with wet, mild winters and dry, hot summers. The different seasonal distribution of the cases indicates the possible different impacts of the local climate on the transmission of the infection.

The length of the lag before the effects of temperature were observed were similar in the two cities (up to six or nine weeks) and could be due to the role that temperatures play in the transmission of this disease by affecting processes in the food chain, including the farm, the processing plant, or the home, thus having an impact on the risk of infection.

This was a **bullets**-longer than the study results in Denmark, where a positive association

was identified between temperature and *Campylobacter* infections, with a four-week lagged effect [12]. While, in the international study by Kovats et al international study on the relationship between temperature and peak-time of *Campylobacter* cases, a lag time of more than two-months lag-time was reported [11]. The lag time could vary in different areas depending on local weather conditions and food processing. The biological-plausibility-for-relatively longer lags could indicate the significance of the conditions earlier in the food production process, eg animal husbandry and slaughtering or processing and distribution, rather than the importance of food storage and hygiene close to the point of consumption or it could be that the food processing chain takes longer in Australia.

As *Campylobacter* does not survive very well in dry conditions, high relative humidity and/or rainfall may contribute to the survival of these organisms in the environment [12]. In this study, we did not find a consistent relationship between rainfall and *Campylobacter* infections in the two cities. Rainfall was significant in the regression model in Adelaide, which is the first time this has been reported in such studies. However, the effect was not significant in Brisbane. Relative humidity was significantly associated with *Campylobacter* infections in Brisbane but not in Adelaide. This could be because ~~that~~ Adelaide had higher levels of disease recorded in winter (its wetter season) and Brisbane had higher levels in summer (its season of higher relative humidity).

It is of significance to find different relationships between weather variables and *Campylobacter* infections in the two Australian cities with different climates. Though

weather, in particular temperature, may affect the growth of food-borne pathogens [10], this is probably not a factor of importance in *Campylobacter* epidemiology, as far as the organisms in the food processing chain are concerned. ~~However, Weather~~ weather may impact on reservoirs, e.g. animals as well as human eating behaviour. ~~Kovats's study~~ The study by Kovats et al. did not find a strong effect of temperature variability on the infections at either the short-time scale or at longer-time scales [~~11~~]. ~~This do suggests~~ that more studies are necessary to identify the relationship between temperatures and Campylobacter infections, especially in different climatic regions, ~~and found no seasonal distribution either in the prevalence of positive flocks, or in the percentage of birds infected [11].~~

In both Adelaide and Brisbane, the regression models indicate that the number of new cases in a given week could be related to the number of cases in previous weeks. This may provide an indicator to local community and health authorities. As soon as an increase in cases is detected by a surveillance system, preventive public health action could be initiated by an early warning system and educating the meat industry and the local community to conduct relevant health promotion campaigns so as to change people's behaviors.

Weekly disease surveillance data rather than monthly data were used in this study and it was an appropriate choice. Monthly data might not be able to provide up-to-date information for foodborne disease, and would be less sensitive in detecting any climate impacts. Daily data were too scarce to run appropriate analyses. Therefore, we strongly

recommend the use of weekly data in similar studies, especially as weekly data should have a better alignment with weather data. In addition, date of onset, rather than date of notification was used in the study, which would be expected to provide a more accurate estimation of the relationship between weather variables and disease outcome.

The transmission of *Campylobacter* is a complicated ecological process with multiple hosts and routes involved [17]. A wide range of animals and birds comprise the reservoirs of *Campylobacter* and many external environmental factors including weather factors may affect this cycle. There are also many risk factors for *Campylobacter* infections among human beings. For instance, poor handling or consumption of raw or undercooked chicken meat may account for approximately 30% of *Campylobacter* cases each year in Australia [18]. Temperature might impact on *Campylobacter* carriage and/or the sources of contamination within the farm environment, such as migratory birds, beetles and rodents, which are temperature dependent [12]. Weather variables might also influence the whole *Campylobacter* infection transmission process, including any animal reservoirs, animal/human interaction and human-to-human contact.

Limitation of this study should be acknowledged. Under-reporting of food-borne diseases is inevitable in any infectious disease surveillance system, but there is no reason to suspect that this introduces any systematic biases because it is believed that under-reporting was consistent over the study period. In addition, further studies including more local influencing factors, such as socio-economic status and public health services, would

be important to fully understand the causes of the various effects of weather variables on such diseases.

In conclusion, the seasonal distribution of *Campylobacter* infections in the Australian cities studied is different. After controlling for seasonality, this study indicates that temperature could be an environmental factor that affects the transmission of *Campylobacter* infections, although the direction of the association could be different depending on climatic region. Further studies are necessary to fully understand the relationship.

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There has been no ethical approval necessary for the project.

Authors' contributions

PB and SC designed the study, YZ conduct the data analysis, PB draft manuscript, and KP, and SC revised it.

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Table 1. Spearman correlations between weekly weather and notifications of *Campylobacter* infections in Adelaide

	Maximum Coef.	Lagged weeks	<i>p</i>
Mean Maximum temperature	-0.25	9 weeks	<0.001
Mean Minimum temperature	-0.26	9 weeks	<0.001
Total Rainfall	-0.07	0	0.079
Mean Relative humidity 9am	0.21	14weeks	<0.001
Mean Relative humidity 3pm	0.18	14weeks	<0.001

Table 2. Parameters estimated by time-series adjusted Poisson Model I for *Campylobacter* infections in Adelaide

	Coefficient	Std. Err.	<i>z</i>	P> <i>z</i>	[95% Conf. Interval]
Lag1_case	0.016	0.001	19.25	<0.001	(0.015, 0.018)
Lag2_case	0.005	0.001	4.66	<0.001	(0.003, 0.007)
Lag3_case	0.006	0.001	6.50	<0.001	(0.004, 0.007)
Lag9_max_temp	-0.007	0.002	-3.40	0.001	(-0.011, -0.003)
Rainfall	0.010	0.004	2.64	0.008	(0.003, 0.018)
Season	-0.021	0.009	-2.23	0.025	(-0.039, -0.003)
Constant	2.671	0.060	44.68	<0.001	(2.55, 2.79)

* Lag1~3_case: number of cases 1~3 week prior; Lag9_max_temp: mean maximum temperature 9-week prior.

Table 3. Spearman Correlations between weekly weather and notifications of *Campylobacter* infection in Brisbane

	Maximum Coef.	Lagged weeks	CI (95%)
Maximum temp	0.10	6 weeks	0.010
Minimum temp	0.07	3 weeks	0.010
Rainfall	0.05	1 week	0.086
Relative humidity 9am	-0.10	3weeks	0.021
Relative humidity 3pm	-0.06	3weeks	0.072

Table 4. Parameters estimated by time-series adjusted Poisson Model I for *Campylobacter* infections in Brisbane

	Coef.	Std. Err.	z	P> z	95% Conf. Interval
Lag1_case	0.014	0.002	9.77	<0.001	(0.011, 0.017)
Lag2_case	0.013	0.002	8.71	0.010	(0.010, 0.016)
Lag3_case	0.004	0.002	2.58	0.010	(0.0009, 0.006)
Lag4_case	0.003	0.001	2.55	0.011	(0.0008, 0.007)
Lag6_max_temp	0.009	0.003	3.18	0.001	(0.004, 0.015)
Lag3_rh	-0.005	0.001	-5.14	<0.001	(-0.007, -0.003)
Year	0.009	0.002	3.94	<0.001	(0.005, 0.014)
Constant	-16.71	4.829	-3.46	0.001	(-26.18, -7.25)

*Lag1~4_case: number of cases occurred 1~4 weeks prior; Lag6_max_temp: maximum temperature 6 weeks prior; Lag3_rh9: relative humidity 9am 3 weeks prior.

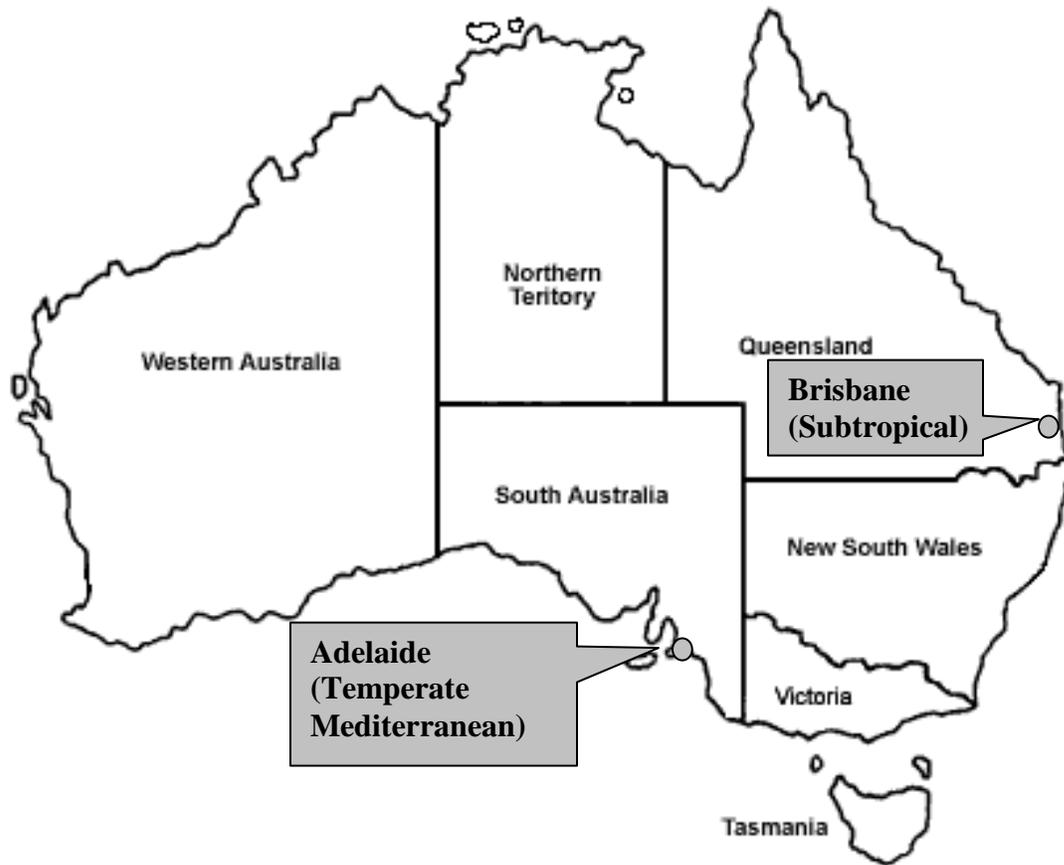


Figure 1 Geographic location and climatic zones of Brisbane and Adelaide, Australia

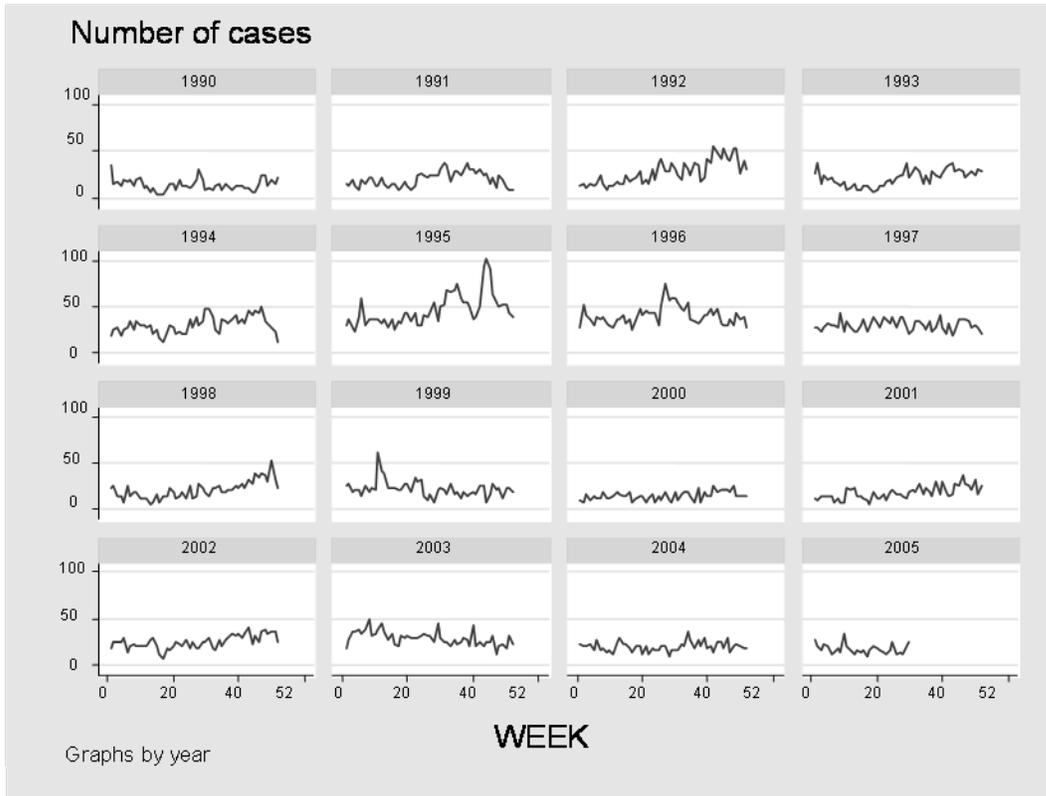


Figure 2 Annual trend of the number of cases of Campylobacter infections in Adelaide, 1990-2005

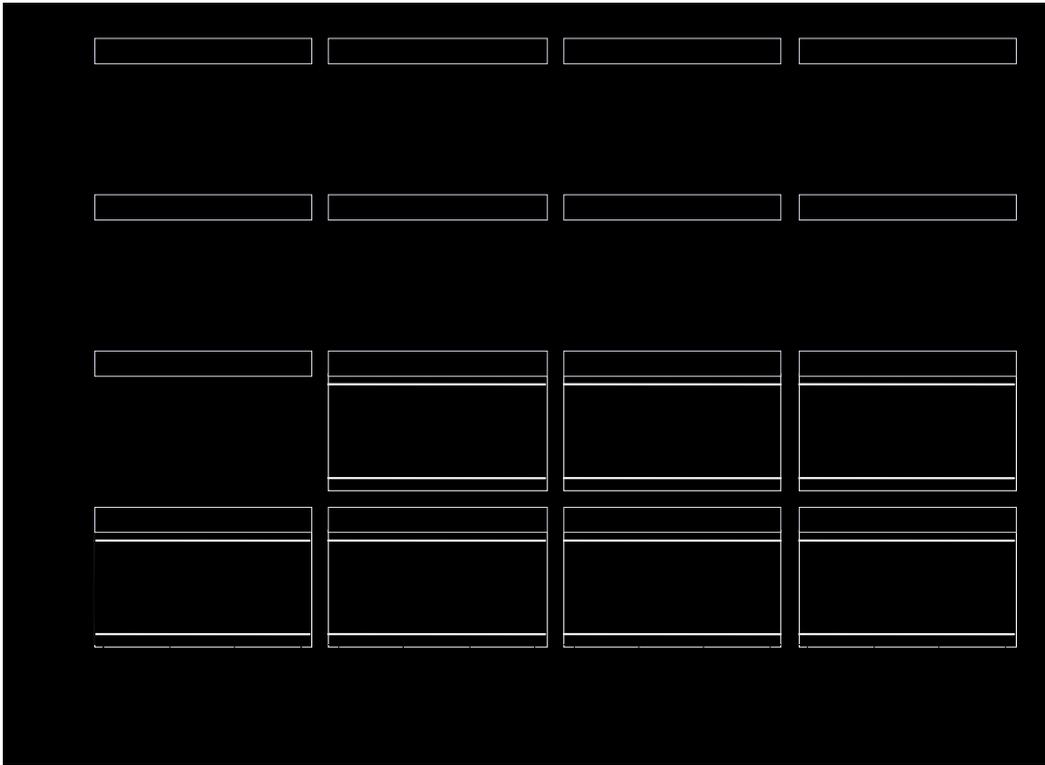


Figure 3 Annual trend of the number of notified cases of *Campylobacter* infection in Brisbane, 1990-2005

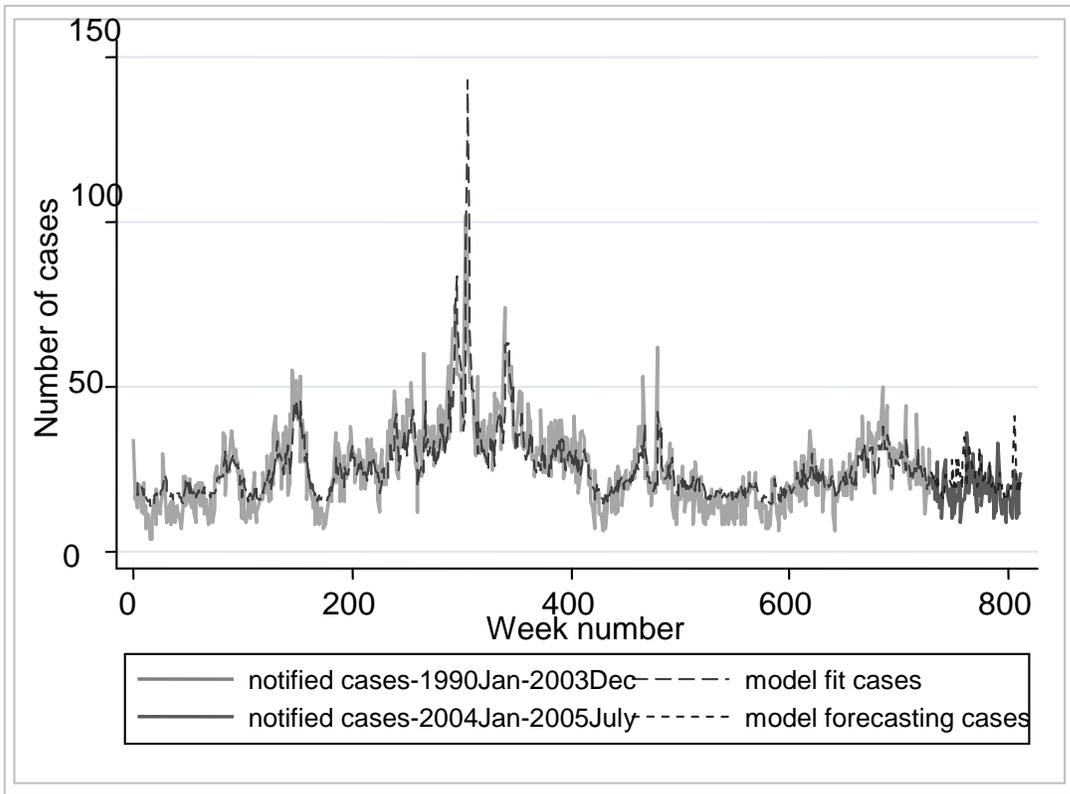


Figure 4 Regression model I having maximum temperature as one of the predictors for *Campylobacter* infections in Adelaide

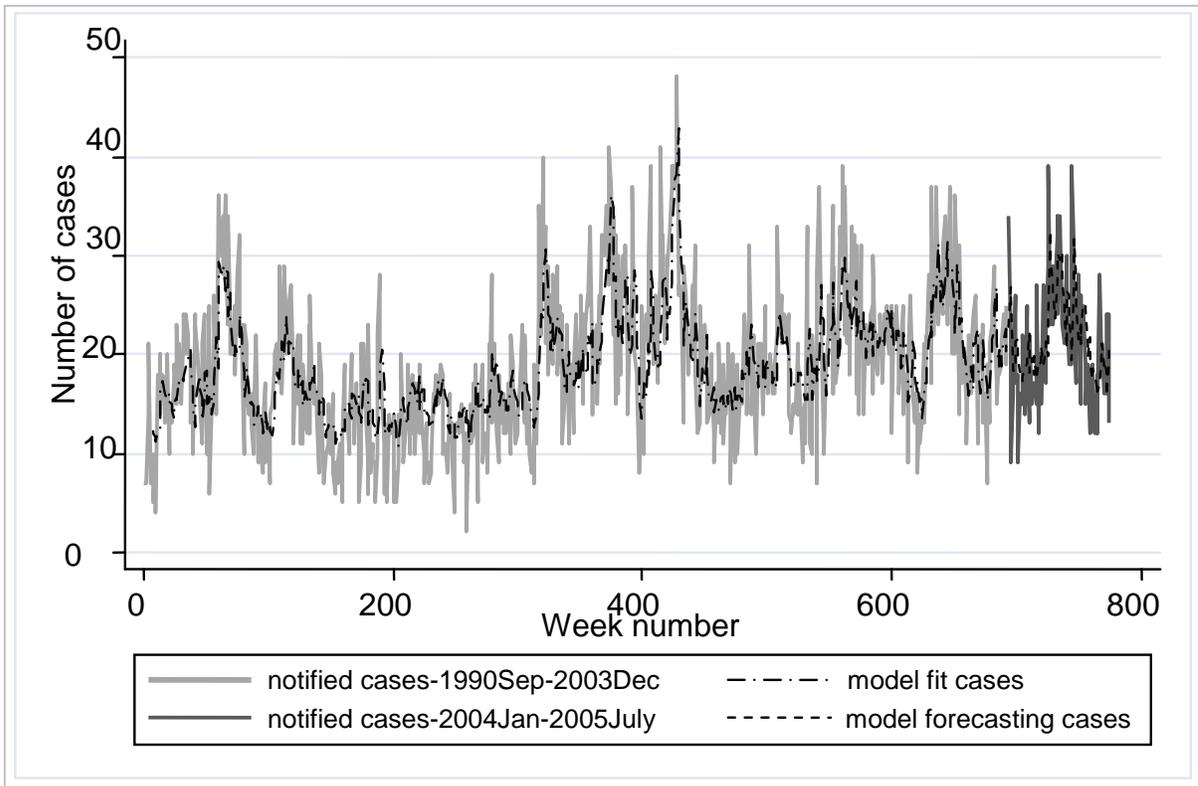


Figure 5 Regression model I having maximum temperature as one of the predictors for *Campylobacter* infection in Brisbane