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Campylobacteriosis in New Zealand

To the Editor:
The recent paper by Rind & Pearce ‘The spatial distribution of campylobacteriosis in New Zealand, 1997–2005’ [1] is a further attempt to understand this highly prevalent disease, but is unfortunately marred by errors which are likely to have affected their interpretation of their results.

Their statement that ‘the pathogen occurs more frequently during the winter months because the organism grows well in water below 10 °C’ is incorrect, as Campylobacter species are fastidious, thermophilic organisms. Campylobacter species are well known to require low oxygen tension and high temperatures (mammal and bird body temperatures) for growth [2]. Studies in New Zealand river systems show a higher summer incidence concordant with contamination of water during the typical summer seasonal peaks in bird and animal/human sources [3–5].

A further error is their misinterpretation of low seasonal variation in the north of the North Island as being ‘low summer incidence’, given that the cited paper is clear that the summer incidence is higher than in winter, although relatively small compared to the large seasonal differences further south [6].

Have these errors affected their choice of variables to include in this study?

One of these variables is ethnicity, with Europeans found to have a significant association with the disease, although it proved unstable in the multivariate model. Rind & Pearce considered it to be an expected outcome, based on an earlier study quoted at some length [7]. Unfortunately, that study was based on a faulty assumption that the data used contained a breakdown into ethnic groups. EpiSurv data (www.nzpho.org.nz/NotifiableDisease.aspx), the source for reported campylobacteriosis cases in both studies, do not reliably report ethnicity. This is easily demonstrated by checking ethnic reporting of campylobacteriosis. For example in the Auckland region, EpiSurv reports no Māori cases, while census data shows this region is about 24% Māori. Further, EpiSurv does not report an ethnicity called ‘European’ at all, but rather reports Māori, Pacific Peoples, and ‘Other’.

Has this error affected interpretation of other social factors?

The age-related risk is also incorrect. The EpiSurv data shows the 5–14 years age group very consistently exhibiting a low rate of campylobacteriosis, and the <5 years age group a consistently high rate. Rind & Pearce have combined these two groups, which does not make sense. The 25–44 years age group is also an unusual combination for analysis, as EpiSurv data readily demonstrate a marked peak in rates for the 20–29 years age group, dropping noticeably for higher age groupings.

The authors suggest that socioeconomic conditions might be a key factor in explaining the spatial differences in campylobacteriosis rates in New Zealand, but provide no convincing evidence for this statement. It is unfortunate that the highly variable spatial pattern of population in New Zealand was not included in their study, especially considering the very high proportion of population in the North Island and the known geographic gradation of campylobacteriosis rates increasing from north to south [6]. A thorough study to tease out population/geographic factors and campylobacteriosis/geographic trends would be useful to attempt to elucidate the role of socioeconomic factors on risk for this common and debilitating disease.

Declaration of Interest

None.
Our paper was an ecological study investigating large-scale trends based on area-level data and we clearly introduced and discussed limitations of this approach. We interpreted our data accordingly and provided evidence for a relatively unexplored and plausible relationship between the spatial variation in Campylobacter notifications, social deprivation, and the distribution of fresh food outlets. These findings demonstrate relatively low summer incidence and low inter-seasonal variation for the rural North Island as follows: ‘Rural North Island: dominant seasonality pattern is characterized by relatively low summer incidence and low inter-seasonal variation’ [7, p. 344].

Third, Nelson comments on the incorporation of the ‘ethnicity’ variable in our modelling procedure. We are well aware of the problematic reporting of ethnicity in EpiSurv – hence our use of census variables to capture ethnic differences across the Territorial Local Authorities (TLAs). While our univariate model showed a significant association between Campylobacter notifications and the percentage of Europeans per TLA, we excluded this variable from our multivariate models because of the multicollinear effects we observed. Capturing comprehensive ethnicity data for notifiable disease cases in New Zealand is an important priority.

Fourth, Nelson comments on the use of our selection of age groups. Our age-related variables were derived from census data available in six age groups (<5, 5–14, 15–24, 25–44, 45–64, >65 years). We combined those age groups that showed similar associations with the Campylobacter rate to obtain a greater population. Due to multicollinear effects we incorporated only the variable representing younger adults in our multivariate models.

Finally, Nelson states that we do not provide convincing evidence for the potential role of socio-economic conditions in explaining spatial variations in campylobacteriosis. This assertion is surprising because our results show a clear inverse and stable association with the variable representing socio-economic deprivation. Nelson also implies that we did not consider the highly variable spatial pattern of population in New Zealand. However, all of our variables representing exposure or surveillance characteristics were related to the appropriate TLA population, either as index, percentage or rate (per capita). Therefore, Nelson is not correct to imply that we did not account for the distribution of population across New Zealand.

The authors reply:

We appreciate Nelson’s letter on our paper on the spatial distribution of campylobacteriosis in New Zealand. However, we disagree with a number of the concerns raised. Many of the points result from Nelson’s misinterpretation of our results. We clarify these issues below.

First, Nelson misrepresents our statement about the appearance of Campylobacter during winter months. As we noted, our assertion concerned the occurrence of the pathogen in the environment. Numerous previous studies have observed that the survival of Campylobacter in water is highest at temperatures of around 5 °C, and significantly lower at temperatures in excess of about 15 °C [1–6].

Second, Nelson suggests that we misinterpreted the results presented by Hearnden et al. [7] in reporting low summer incidence and low inter-seasonal variation in rural areas across the North Island. However, Nelson is mistaken as Hearnden et al. clearly
deserve further investigation in New Zealand and elsewhere.

References


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