Geographic determinants of reported human *Campylobacter* infections in Scotland.

Paul R. Bessell, Louise Matthews, Alison Smith-Palmer, Ovidiu Rotariu, Norval J. C. Strachan, Ken J. Forbes, John Cowden, Stuart W J. Reid, Giles T. Innocent

Abstract

Campylobacteriosis is the leading cause of bacterial gastroenteritis in most developed countries. People are exposed to infection from contaminated food and environmental sources. However, the translation of these exposures into infection in the human population remains incompletely understood. This relationship is further complicated by differences in the presentation of cases, their investigation, identification, and reporting; thus, the actual differences in risk must be separated from the artefactual differences. This paper describes a Generalised Linear Model (GLM) of *Campylobacter* case reporting rates within Scotland. Using data on 34,193 confirmed *Campylobacter* infections in mainland Scotland between 2000 and 2006 (inclusive) a number of risk factors were tested. Social deprivation (measured by the Carstairs index) was protective ($p<0.001$) whilst a greater number of private water supplies per person was associated with increased case rates ($p<0.001$). Once these factors were taken into account significant differences remain in reported infection rates between certain NHS Boards and the expectation; this suggests that either the levels of ascertainment between NHS Boards in Scotland are not uniform or there are determinants of infection which have not been included in the model. Following stratification by age group, population density had a significant protective effect for those under 15 but not for those aged 15 and older, demonstrating that rural children are at greater risk than their urban counterparts. This model demonstrates that the less deprived and children living in rural areas and areas with a large number of private water supplies are at the greatest risk of being reported ac cases of *Campylobacter* infection.

Introduction

Infection with bacteria of *Campylobacter* spp is the leading cause of human bacterial gastroenteritis in most developed countries (Blaser, 1997). In Scotland in 2006 there were 95.3 reported cases per 100,000 (Locking et al., 2007), although this figure is likely to be underreported by a factor of 8 as has been demonstrated in England (Wheeler et al., 1999). Further studies in England and Wales show that approximately 10% of reported cases were admitted to hospital for treatment (Gillespie et al., 2002).

Infection with *Campylobacter* is thought to occur principally via the consumption of contaminated, under-cooked meat (mainly chicken) and cross-contaminated foods (Altekruse et al., 1999; Gormley et al., 2008). However other modes of transmission include direct and indirect contact with animal feces (especially ruminant feces) (Horrocks et al., 2009) and consumption of contaminated water (Savill et al., 2001; Sandberg et al., 2006; Sopwith et al., 2008). Human exposure to these sources is spatially heterogeneous and therefore the spatial pattern of infection is heterogeneous.

Previous studies have identified risk factors; that include eating chicken, eating in restaurants and eating from fast food outlets (Gormley et al., 2008; Danis et al., 2009). Additionally, those who live in rural areas and have regular contact with livestock are at greater risk of infection (Nygard et al., 2004; Devane et al., 2005; Ethelberg et al., 2005; Ellis-Iversen et al., 2009) as are individuals with private water supplies (Sopwith et al., 2008; Danis et al., 2009). Further variations in *Campylobacter* incidences caused by either physiology or differences in exposure relate to the age and gender of the individual. For example, male children are at around 1.5 times greater risk of infection than their female counterparts (Strachan et al., 2008; Unicomb et al., 2008).

In addition to heterogeneity in infection there will be heterogeneity in reporting. Infections may well be under ascertained by a factor of 8 (Wheeler et al., 1999), but this will not necessarily be distributed evenly throughout the population. Reporting may be influenced by the age and gender of the patient (Ethelberg et al., 2005; Strachan et al., 2008), use of primary health care facilities (Olowokure et al., 1999; Simonsen et al., 2008) and the socio-economic status of the patient (Snel et al., 2009a).

This study developed a risk factor model to explain the geographical distribution of *Campylobacter* infections incorporating both sources of heterogeneity. At the level of the community it distinguished between factors that determine risk of *Campylobacter* infections and factors that determine artefactual risk due to differences between NHS Health Board areas. Thus the study provides an overall model of the geographic pattern of *Campylobacter* cases within Scotland. The study has the following aims:

1. To quantify the importance of deprivation in determining *Campylobacter* infections given that deprivation may influence food consumption, environmental contact and propensity to seek medical attention or submit a stool sample.
2. To identify rural-urban differences in *Campylobacter* infections and whether such differences may be explained by proximity to livestock.
3. To identify differences in *Campylobacter* infections between NHS Health Board areas.
4. To identify whether private water supplies influence *Campylobacter* infections.
5. To establish whether these differences are age dependent.

Methods

Data

Data on cases of *Campylobacter* infection were collected from the 12 mainland NHS Boards that existed in Scotland prior to 2006. The island NHS Boards of the Western Isles, Orkney & Shetland were not included because of their small populations and small numbers of cases. Data were collected for the years 2000 to 2006 (inclusive), with the exception of the Ayrshire & Arran NHS Boards for which only the years 2003 to 2006 (inclusive) were available. Data were anonymised and included the age, geneder and postcode sector of the residence of the case, and the date of reporting. Subsequently all analysis was at the level of the postcode sector (median population = 5,426, 25^{th,} and 75th percentile = 2846 and 7,506; median area = 14.5km^2, 25^{th,}and 75th percentile = 2.75 and 83.2km^2).

Data on the human population were collected from the 2001 Scottish census (from the UK Borders at EDiNA® http://edina.ac.uk/ukborders/) along with data on the Carstairs index of deprivation; cattle, sheep and poultry numbers were obtained from the Scottish agricultural census (from EDiNA®, http://edina.ac.uk/agcensus; 2004 estimates) and data on private water supplies from Environmental Health Departments of Local Authorities. Data on recent travel was available for the Lothian and Grampian NHS Health Board areas from the Health Protection Scotland (HPS) enteric forms.

Risk factors

The following risk factors were included for initial screening:

- NHS Board area of residence - factor with 13 levels - the Argyll & Clyde NHS Board was broken into Argyll and Clyde due to the ten fold difference in case rates between the two but treated as an independent NHS Health Board area (Table 1).
- The Carstairs deprivation score (Carstairs and Morris, 1990).
- Easting and northing of the postcode sector centroid.
- Population density (people / km^2) of the postcode sector.
- Density of cattle, sheep and poultry (head / km^2) in the postcode sector.
- Density of private water supplies (supplies / person) in the postcode sector ($\log_{10}$ transformed to normalise its distribution).
- The case rate (cases per 1,000) in the five neighbouring postcode sectors - a spatial lag term.

The model

The risk factors listed above were screened in univariate generalised linear models (GLM) with a poisson error term and those with $p<0.25$ selected for insertion into the multivariable model. For modelling in a GLM, factor variables require a reference level – a level which acts as the base of comparison for the other levels. In the case of NHS Board there is no natural reference level to act as the base of comparison, so the model was fitted with no reference level for the NHS Board and instead an individual intercept for each level of the NHS Board. The significant of the intercept is evaluated by testing whether it is significantly different to zero. In the case of the NHS Board predictor it is necessary to test whether the case rate within that board is significantly different from the national mean, therefore, the expected number of cases within that postcode sector offset the model. The expected number of cases was based upon the population of the postcode sector and the national case rate; the expectation in postcode sector *i* ($E(Y_i)$) was calculated as:

$$E(Y_i) = \frac{\sum_{j=1}^{j=n} Y_j}{\sum_{j=1}^{j=n} P_j} P_i$$

where *Y* is the number of *Campylobacter* cases and *P* is the population in postcode sector *i* or *j* respectively.

In order to compare between the NHS Health Board areas the intercept (NHS board area) is centered by adding the mean of the coefficients for the NHS Board of each data point ($\bar{H}$) to the offset. Thus the model is run without ($\bar{H}$) in order to calculate this term for addition to subsequent models.

The model took the form:

$$\log(y_i) \sim b_1 H_i + b_2 x_1 + ... + b_n x_{n-1} + (\log(E(Y_i)) + \bar{H})$$

where β represents the fitted coefficients, *x* the risk factors and H_i the NHS Board of postcode sector *i*. Non-significant ($p>0.05$) risk factors were removed in turn from the multivariable model starting with the greatest value of p until only significant risk factors remained. When removed, the effect of the removal on remaining risk factors was monitored, as a substantial change in the estimate would indicate correlation. Overdispersion in the model was handled using a quasipoisson error term and the significance of risk factors further checked by performing an anova (F-test).

To allow for the differences in the ages of cases (Strachan et al., 2008) and to test for the age dependent differences in the effect of rurality noted in Denmark (Ethelberg et al., 2005), separate models were constructed for those aged under 15 years and those 15 and over. Further sensitivity analysis will be conducted by building models for just the Lothian and Grampian NHS Board areas and running the model with and without the cases that had travelled overseas in the previous 14 days. Evaluation of the relative change in the model coefficients will indicate whether the model results were a result of foreign travel. The coefficients for the risk factors in these models were compared. Spatial autocorrelation in the model was examined by plotting a spatial corellogram of Moran's *I* with ten lags, in the spdep package for R (Bivand, 2009). Significant spatial autcorrelation in the initial model was handled by including the neighbouring case rate term as a spatial lag component. All analyses were conducted in the R statistical package (R Development Core Team, 2008).

Results

NHS Board differences

A total of 34,193 cases were reported over the course of the study period. There was a ten-fold difference in the rates reported in the Argyll relative to the Clyde areas of the Argyll & Clyde NHS Board (Table 1). Case rates in the Ayrshire & Arran NHS Board were around 50% lower than those seen in other NHS Board areas (Table 1).

Once the other risk factors have been taken into account (Table 3), the Argyll sector of the Argyll & Clyde NHS Board and the Ayrshire & Arran NHS Board remain significantly lower than the expected number of cases. Seven of the 13 NHS Board areas had case rates which were significantly higher than expectation, with the greatest difference being in the Lothian NHS Board area.

Risk factor analysis

All risk factors with the exception of sheep and poultry densities were significant at $p<0.25$ in univariate screening and were therefore entered into the multivariable model (Table 2). In the multivariable modelling NHS Board, Carstairs deprivation index, density of private water supplies and the neighbouring rate were significant and retained (Table 3). Greater deprivation was associated with lower case rates, whilst private water supplies and the neighbour rate were risk factors. The fitted values from the model closely match the data (Figures 2 and 3) and the residuals show no spatial pattern (Moran's I $p > 0.5$). Sensitivity analysis for foreign travel in the Lothian and

Grampian NHS Health Board areas indicate that there is no sensitivity to foreign travel as there was no significant difference in the model coefficients with and without the foreign travel cases.

Age-stratified analysis

Comparison of the coefficients for the Carstairs deprivation score and population density amongst individuals aged under 15 years compared to 15 and over shows that there is little change in the estimate for Carstairs deprivation index (Figure 4). However, population density changes from being a non-significant predictor in those aged 15 and over to a highly significant risk factor in under 15s (Figure 4) with high population densities being protective in the under 15 groups.

Discussion

Reported *Campylobacter* infections are more common among the least deprived and amongst children living in rural areas. At least part of the difference is likely to be a result of real differences in rates of infection, although some may be due to differences in ascertainment. These results are in line with findings from other countries for both *Campylobacter* and other gastrointestinal diseases (Simonsen et al., 2008; Snel et al., 2009a). A number of potential explanations have been offered for the relationship with deprivation:

1. Acquired immunity through exposure to household sources of infection at a young age amongst the more deprived. The level of exposure among younger age groups to bacterial sources of infection within the household may increase with deprivation. However, Figure 4 demonstrates that there is no significant difference in the Carstairs deprivation score in the age-stratified analysis. If acquired immunity were the explanation then the younger groups would be more commonly infected in more deprived areas whilst older age groups would be more commonly infected in less deprived areas, however Figure 4 does not support this. These findings are supported by other studies that suggest that there is no difference between age and deprivation (Simonsen et al., 2008; Snel et al., 2009a).
2. Deprivation may be associated with differences in dietary habits (Simonsen et al., 2008); differences in the quality of the available fresh food have been observed elsewhere (Cummins et al., 2009). If there is greater consumption of processed rather than fresh meat among the more deprived there will be less *Campylobacter* because the process of freezing reduces the number of *Campylobacter* organisms (Ritz et al., 2007). Furthermore, the less deprived may also eat at restaurants more frequently, demonstrated as a risk factor in other studies (Danis et al., 2009).
3. Differences in environmental exposure associated with different leisure activities, differences in access to rural areas or people working in rural areas.

4. Differences in reporting. Lower reporting rates for gastrointestinal disease among the more deprived have been noted in the UK (Olowokure et al., 1999; Snel et al., 2009c, b), Denmark (Simonsen et al., 2008) and New Zealand (Snel et al., 2009c, b).
5. Differences in foreign travel. However, the sensitivity analysis in the two NHS Health Board areas for which travel data were available indicated that this is not the case.

Further research is necessary to fully understand the processes operating, for example comparing hospitalisation rates, however, it is likely that some combination of these factors is responsible for the relationship with deprivation.

The significance of the protective effect of population density among children confirms findings from Denmark (Ethelberg et al., 2005) where significantly higher case rates were found among children in rural areas. This may be the result of differences in the tolerance level that determines whether a patient reports to a doctor, which is likely to be age dependant. Furthermore, this evidence does not suggest that consumption of chicken products or eating in restaurants would be greater in rural areas. That the effect of rurality is greatest among children suggests that playing outdoors and becoming exposed to environmental reservoirs of infection is important, and may additionally be compounded by poor hygiene among younger groups.

Whilst one of the greatest sources of *Campylobacter* in rural areas is likely to be livestock (Brown et al., 2004; French et al., 2005; Horrocks et al., 2009), our analysis did not show density of livestock to be associated with *Campylobacter* infections. Rather, this model suggests that a major reservoir from which individuals are becoming infected is private water supplies. These findings suggest that environmental exposures, whilst these may ultimately be the result of contamination from livestock sources, are best characterised by low population densities. This is exacerbated in the Grampian NHS Board area in which a large number of people obtain their water from private supplies.

Environmental exposures may arise following travel outside of the postcode sector of residence and this was accounted for in the model by the inclusion of the case rate in neighbouring postcode sectors. Whilst most exposure to infection is likely to occur in the postcode sector of residence this model suggests that there remains some infection challenge outside the postcode sector. The model assumes that all error is associated with the measured outcome, in this case the postcode sector case rate. Use of neighbouring postcode case rates as an explanatory variable violates this assumption. However, since we amalgamate the outcomes from several postcode sectors into this explanatory variable the error is reduced considerably. This, whilst not ideal, allows us to use this method without producing excessive bias, although p-vaues can no longer be considered a slight over-estimate. Alternative solutions including aggregating the data by postcode district and including postcode district or NHS Health Board areas as a random effect were tried but neither of these removed the spatial autocorrelation.

There are few differences between NHS Health Boardarea with the exception of the Argyll area of the Argyll & Clyde NHS Health Board area, which, whilst it has a low population density (13.9 people/km^2), it is not the lowest. However this pattern of reporting may be the result of differences in access to health services in this area. The Ayrshire & Arran NHS Health Board area also reported significantly lower case numbers than expected. The Ayrshire & Arran NHS Health Board area also reported less GI disease per head of population than any other NHS Health Board area in Scotland for both *Salmonella* and *Cryptosporidium* infections (N. Strachan pers. comm.). Otherwise, whilst there is evidence for differences in the pattern of reporting between health boards, the fitted model is able to account for these differences. This may be because the differences in case rate arise from differences in the risks identified by the analysis. An alternative interpretation is that regional differences are being accounted for by the inclusion of the spatial lag effect. This is equivalent to identifying that the underlying risk varies across Scotland in a smooth manner. This in turn may be due to one or more unmeasured factors, possibly environmental.

This study has demonstrated that there are real differences in the geographic distribution of *Campylobacter* infections within Scotland caused by differences in exposure to infection as well as bias due to reporting between NHS Health Boards. Those at greatest risk are children in rural environments and those supplied by private water sources. The results demonstrate that the relationship with deprivation is unlikely to result from differences in acquired immunity. Furthermore, those less deprived may be more exposed to infection or may be more willing to seek medical attention. However large differences remain in reported disease incidences between the deprived and the less deprived as well as differences in ascertainment between the boards administering the health care.

References

Altekruse, S.F., Stern, N.J., Fields, P.I., Swerdlow, D.L., 1999. Campylobacter jejuni--an emerging foodborne pathogen. Emerg Infect Dis 5, 28-35.

Bivand, R., 2009. spdep: Spatial dependence: weighting schemes, statistics and models.

Blaser, M.J., 1997. Epidemiologic and clinical features of *Campylobacter jejuni* infections. Journal of Infectious Diseases, S103-105.

Brown, P.E., Christensen, O.F., Clough, H.E., Diggle, P.J., Hart, C.A., Hazel, S., Kemp, R., Leatherbarrow, A.J., Moore, A., Sutherst, J., Turner, J., Williams, N.J., Wright, E.J., French, N.P., 2004. Frequency and spatial distribution of environmental Campylobacter spp. Appl Environ Microbiol 70, 6501-6511.

Carstairs, V., Morris, R., 1990. Deprivation and health in Scotland. Health Bull (Edinb) 48, 162-175.

Cummins, S., Smith, D.M., Taylor, M., Dawson, J., Marshall, D., Sparks, L., Anderson, A.S., 2009. Variations in fresh fruit and vegetable quality by store type, urban-rural setting and neighbourhood deprivation in Scotland. Public Health Nutr, 1-7.

Danis, K., Di Renzi, M., O'Neill, W., Smyth, B., McKeown, P., Foley, B., Tohani, V., Devine, M., 2009. Risk factors for sporadic Campylobacter infection: an all-Ireland case-control study. Euro Surveill 14.

Devane, M.L., Nicol, C., Ball, A., Klena, J.D., Scholes, P., Hudson, J.A., Baker, M.G., Gilpin, B.J., Garrett, N., Savill, M.G., 2005. The occurrence of Campylobacter subtypes in environmental reservoirs and potential transmission routes. J Appl Microbiol 98, 980-990.

Ellis-Iversen, J., Pritchard, G.C., Wooldridge, M., Nielen, M., 2009. Risk factors for Campylobacter jejuni and Campylobacter coli in young cattle on English and Welsh farms. Prev Vet Med 88, 42-48.

Ethelberg, S., Simonsen, J., Gerner-Smidt, P., Olsen, K.E., Molbak, K., 2005. Spatial distribution and registry-based case-control analysis of Campylobacter infections in Denmark, 1991-2001. Am J Epidemiol 162, 1008-1015.

French, N., Barrigas, M., Brown, P., Ribiero, P., Williams, N., Leatherbarrow, H., Birtles, R., Bolton, E., Fearnhead, P., Fox, A., 2005. Spatial epidemiology and natural population structure of Campylobacter jejuni colonizing a farmland ecosystem. Environ Microbiol 7, 1116-1126.

Gillespie, I.A., O'Brien, S.J., Frost, J.A., Adak, G.K., Horby, P., Swan, A.V., Painter, M.J., Neal, K.R., 2002. A case-case comparison of Campylobacter coli and Campylobacter jejuni infection: a tool for generating hypotheses. Emerg Infect Dis 8, 937-942.

Gormley, F.J., Macrae, M., Forbes, K.J., Ogden, I.D., Dallas, J.F., Strachan, N.J., 2008. Has retail chicken played a role in the decline of human campylobacteriosis? Appl Environ Microbiol 74, 383-390.

Horrocks, S.M., Anderson, R.C., Nisbet, D.J., Ricke, S.C., 2009. Incidence and ecology of Campylobacter jejuni and coli in animals. Anaerobe 15, 18-25.

Locking, M., Browning, L., Smith-Palmer, A., Brownlie, S., 2007. Gastro-intestinal and foodborne infections. HPS Weekly Report 41, 3-4.

Nygard, K., Andersson, Y., Rottingen, J.A., Svensson, A., Lindback, J., Kistemann, T., Giesecke, J., 2004. Association between environmental risk factors and campylobacter infections in Sweden. Epidemiol Infect 132, 317-325.
Olowokure, B., Hawker, J., Weinberg, J., Gill, N., Sufi, F., 1999. Deprivation and hospital admission for infectious intestinal diseases. Lancet 353, 807-808.
R Development Core Team, 2008. R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria.
Ritz, M., Nauta, M.J., Teunis, P.F., van Leusden, F., Federighi, M., Havelaar, A.H., 2007. Modelling of Campylobacter survival in frozen chicken meat. J Appl Microbiol 103, 594-600.
Sandberg, M., Nygard, K., Meldal, H., Valle, P.S., Kruse, H., Skjerve, E., 2006. Incidence trend and risk factors for campylobacter infections in humans in Norway. BMC Public Health 6, 179.
Savill, M.G., Hudson, J.A., Ball, A., Klena, J.D., Scholes, P., Whyte, R.J., McCormick, R.E., Jankovic, D., 2001. Enumeration of Campylobacter in New Zealand recreational and drinking waters. J Appl Microbiol 91, 38-46.
Simonsen, J., Frisch, M., Ethelberg, S., 2008. Socioeconomic risk factors for bacterial gastrointestinal infections. Epidemiology 19, 282-290.
Snel, S.J., Baker, M.G., Kamalesh, V., French, N., Learmonth, J., 2009a. A tale of two parasites: the comparative epidemiology of cryptosporidiosis and giardiasis. Epidemiol Infect, 1-10.
Snel, S.J., Baker, M.G., Venugopal, K., 2009b. The epidemiology of cryptosporidiosis in New Zealand, 1997-2006. N Z Med J 122, 47-61.
Snel, S.J., Baker, M.G., Venugopal, K., 2009c. The epidemiology of giardiasis in New Zealand, 1997-2006. N Z Med J 122, 62-75.
Sopwith, W., Birtles, A., Matthews, M., Fox, A., Gee, S., Painter, M., Regan, M., Syed, Q., Bolton, E., 2008. Identification of potential environmentally adapted Campylobacter jejuni strain, United Kingdom. Emerg Infect Dis 14, 1769-1773.
Strachan, N.J., Watson, R.O., Novik, V., Hofreuter, D., Ogden, I.D., Galan, J.E., 2008. Sexual dimorphism in campylobacteriosis. Epidemiol Infect 136, 1492-1495.
Unicomb, L.E., Dalton, C.B., Gilbert, G.L., Becker, N.G., Patel, M.S., 2008. Age-specific risk factors for sporadic Campylobacter infection in regional Australia. Foodborne Pathog Dis 5, 79-85.
Wheeler, J.G., Sethi, D., Cowden, J.M., Wall, P.G., Rodrigues, L.C., Tompkins, D.S., Hudson, M.J., Roderick, P.J., 1999. Study of infectious intestinal disease in England: rates in the community, presenting to general practice, and reported to national surveillance. The Infectious Intestinal Disease Study Executive. BMJ 318, 1046-1050.

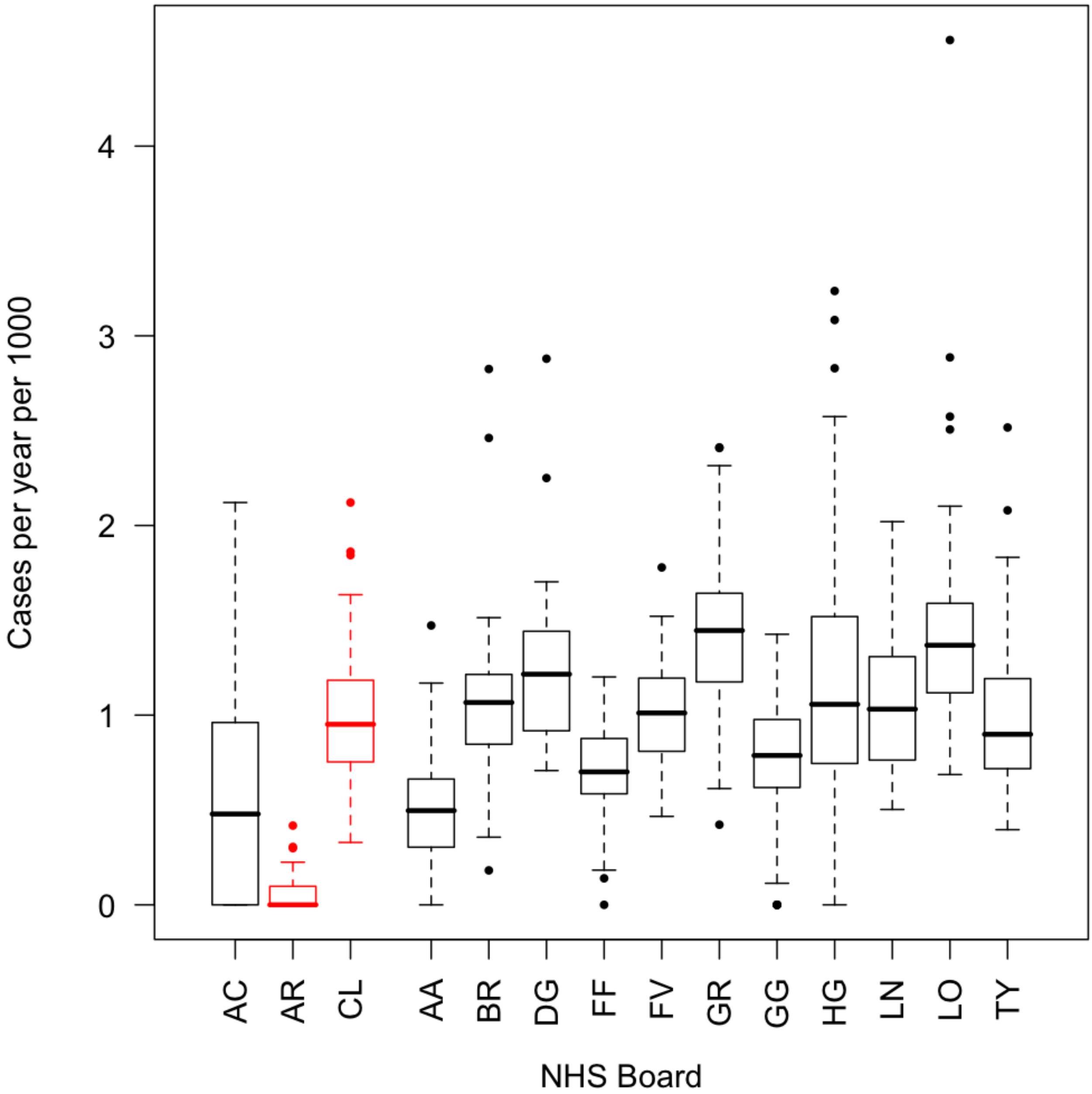

Figure 1. Boxplot of case rates per year for the 12 NHS Boards (in black) and the Argyll and Clyde NHS Board divided into in the separate units (in red). NHS Board abbreviations are expanded in Table 1.

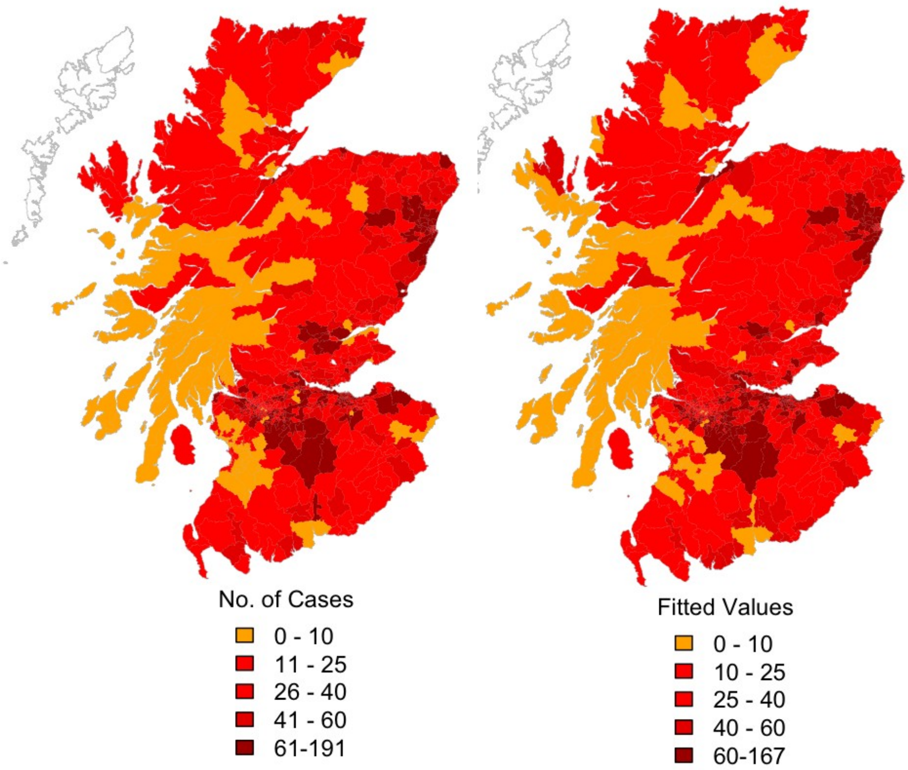

Figure 2. Maps of the number of cases and fitted values from the model in Table 3 by postcode sector.

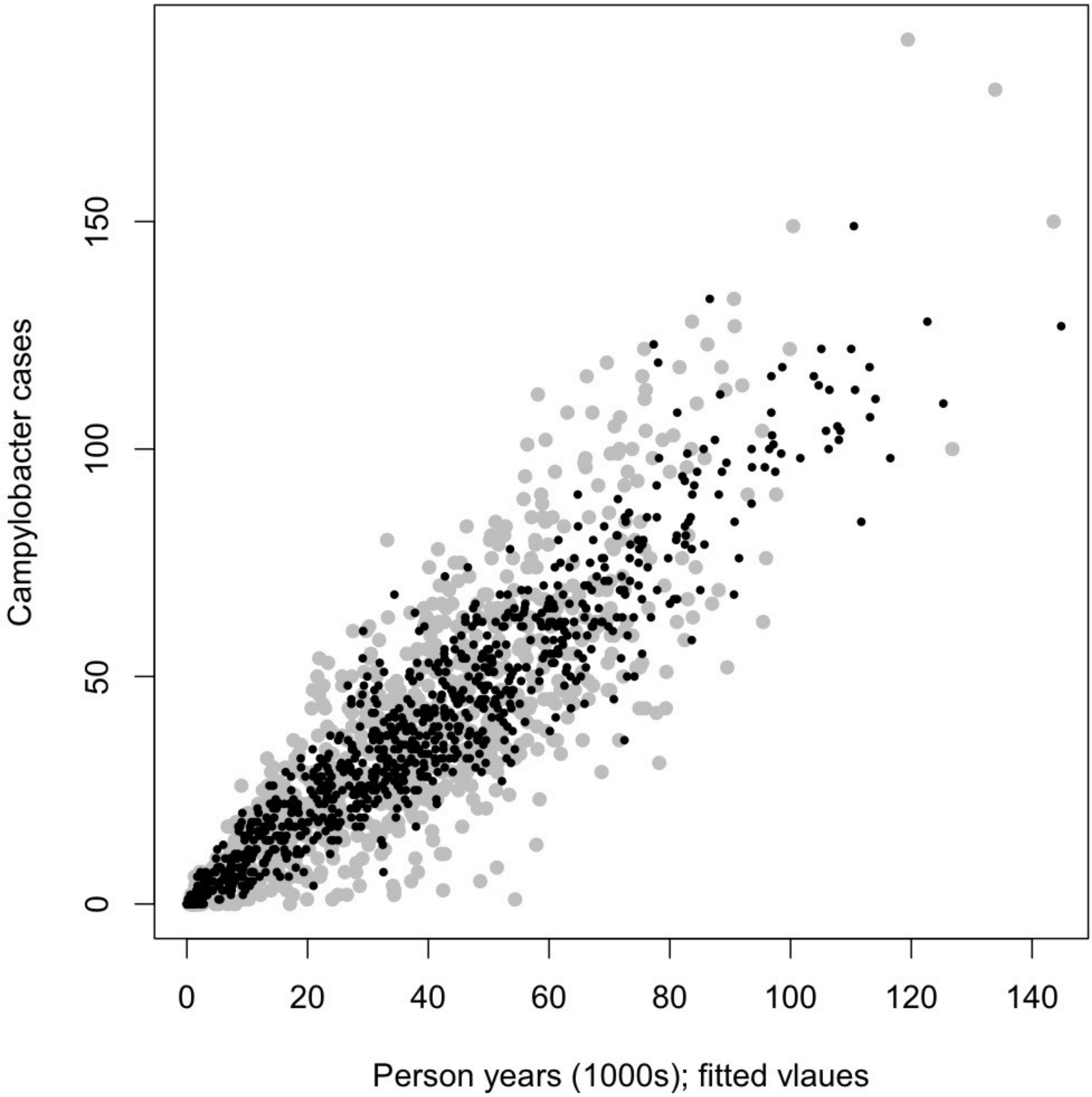

Figure 3. Plot of population against the number of cases (grey points) and the fitted values from the full multivariable model (Table 3) against the observed cases (black points).

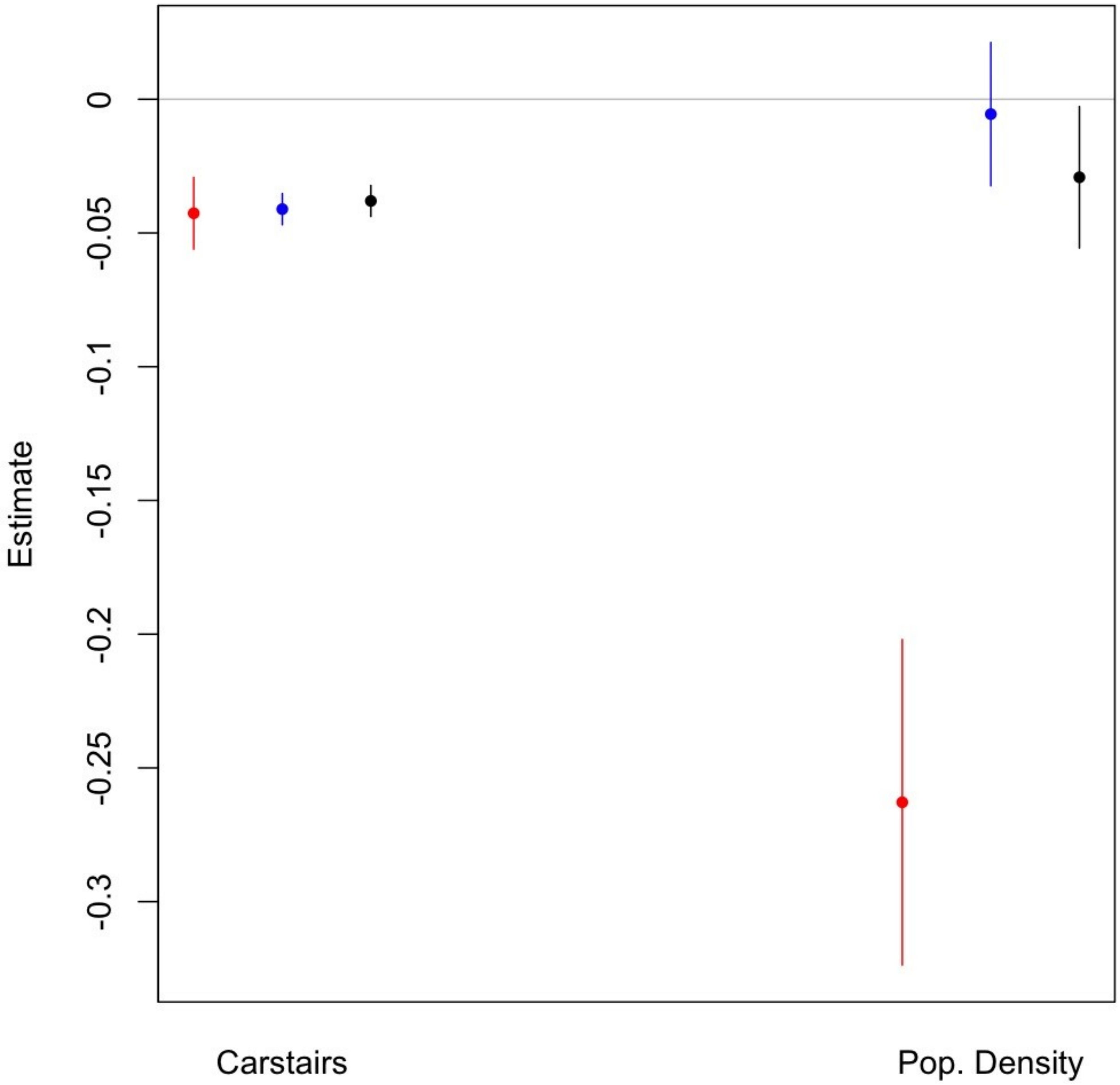

Figure 4. Point estimates and 95% confidence intervals (CIs) for multivariable models parameterised with NHS Board, Carstairs and population density for cases under 15 (red), 15 and over (blue) and all data (black).

Table 1. Summary statistics for NHS Boards.

NHS Board	Population	Number of postcode sectors	Population density	Total number of cases	Cases/100000/yr
Argyll & Clyde (AC)	420,010	97	59.3	2,317	78.8
Argyll (AR)	*88,384*	*46*	*13.9*	*58*	*9.4*
Clyde (CL)	*331,626*	*51*	*464.1*	*2,259*	*97.3*
Ayrshire & Arran (AA)	368,149	71	111.4	750	50.9
Borders (BR)	105,580	29	22.4	791	106.7
Dumfries & Galloway (DG)	147,625	34	22.2	1,214	117.4
Fife (FF)	346,580	50	255.8	1,762	72.6
Forth Valley (FV)	282,900	47	97.4	2,049	103.5
Grampian (GR)	525,595	101	59.9	5,112	138.9
Greater Glasgow (GG)	869,520	140	1527.6	4,992	82.0
Highland (HG)	208,223	90	8.4	1,396	95.8
Lanarkshire (LN)	552,397	62	228.5	4,086	105.7
Lothian (LO)	779,223	124	463.3	7,285	133.6
Tayside (TY)	387,272	71	51.3	2,439	90.0

Table 2. Univariate poisson GLM analysis of risk factors.

Predictor	Unit	Estimate	Std. error	t-value	p-value
Easting	m	3.533×10^{-6}	2.457×10^{-7}	14.38	<0.001
Northing	m	9.412×10^{-7}	1.835×10^{-7}	5.13	<0.001
Carstairs score		-0.050	0.004	-14.23	<0.001
Private water density[1]	$\log_{10}$(supplies/person)	4.754	0.709	6.709	<0.001
Human density[1]	people/ km^2	-0.033	0.015	-2.156	0.031
Cattle density	cattle/ km^2	-6.401×10^{-4}	3.854×10^{-4}	-1.661	0.097
Sheep density	sheep/ km^2	-4.655×10^{-6}	1.631×10^{-4}	-0.029	0.977
Poultry density	poultry/ km^2	-2.460×10^{-6}	3.654×10^{-6}	-0.673	0.501
Neighbouring case rate	cases/1000	0.890	0.029	30.89	<0.001

Table 3. Reduced multivariable poisson GLM of risk factors for *Campylobacter* cases.

Predictor	Unit	Estimate	Std. error	t-value	p-value
NHS Board	Argyll	-1.941	0.192	-10.09	<0.001
	Ayrshire & Arran	-0.290	0.058	-4.993	<0.001
	Borders	0.109	0.068	1.595	0.111
	Clyde	0.193	0.048	3.986	<0.001
	Dumfries & Galloway	0.189	0.064	2.950	0.003
	Fife	-0.060	0.046	-1.294	0.196
	Forth Valley	0.140	0.052	2.678	0.007
	Grampian	0.196	0.059	3.300	0.001
	Greater Glasgow	0.116	0.041	2.819	0.005
	Highland	0.059	0.056	1.054	0.292
	Lanarkshire	0.187	0.050	3.729	<0.001
	Lothian	0.237	0.058	4.069	<0.001
	Tayside	0.065	0.047	1.383	0.167
Neighbouring case rate	cases / 1000	0.049	0.041	12.04	<0.001
Carstairs		-0.033	0.003	-12.60	<0.001
Private water supplies	$\log_{10}$(supplies/person)	1.885	0.622	3.031	0.002

←

←

←