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Temporal and geographical distributions of reported cases of *Escherichia coli* O157:H7 infection in Ontario

P. MICHEL[1]*, J. B. WILSON[1,3], S. W. MARTIN[1], R. C. CLARKE[2], S. A. McEWEN[1]
AND C. L. GYLES[4]

[1] *Department of Population Medicine, Ontario Veterinary College (OVC), University of Guelph, Guelph, Ontario, Canada*
[2] *Guelph Laboratory, Health Canada, Guelph, Ontario, Canada*
[3] *Laboratory Centre for Disease Control (LCDC), Health Canada, Ottawa, Ontario, Canada*
[4] *Department of Pathobiology, Ontario Veterinary College (OVC), University of Guelph, Ontario, Canada*

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SUMMARY

The distribution of 3001 cases of verocytotoxigenic *Escherichia coli* (VTEC) reported in the Province of Ontario, Canada, were examined to describe the magnitude of this condition geographically and to evaluate the spatial relationship between livestock density and human VTEC incidence using a Geographical Information System. Incidence of VTEC cases had a marked seasonal pattern with peaks in July. Areas with a relatively high incidence of VTEC cases were situated predominantly in areas of mixed agriculture. Spatial models indicated that cattle density had a positive and significant association with VTEC incidence of reported cases ($P = 0{\cdot}000$). An elevated risk of VTEC infection in a rural population could be associated with living in areas with high cattle density. Results of this study suggested that the importance of contact with cattle and the consumption of contaminated well water or locally produced food products may have been previously underestimated as risk factors for this condition.

INTRODUCTION

Between 1990 and 1995, 7482 cases of verocytotoxigenic *E. coli* (VTEC) were reported in Canada, of which more than 40% were recorded in the Province of Ontario [1]. The infection in humans is associated with a spectrum of illnesses including severe diarrhoea, and haemolytic-uraemic syndrome (HUS) [2]. There have been few attempts to study the temporal and geographical distributions of verocytotoxigenic *E. coli* (VTEC) in a defined population [3, 4]. Identification of high risk groups based on the geographic distribution of cases can be of value in targeting resources as part of control strategies for diseases caused by VTEC. Similarly, the relationship between the spatial distribution of disease incidence and exposure variables can be studied in order to identify risk factors. Currently, one area of interest related to VTEC disease is the relative importance of this condition in rural, as opposed to urban, areas, and the role that exposure to cattle might play in the epidemiology of this infection in humans. The present study combined geographic, demographic, agricultural and disease data to describe the spatial distributions of cases of human VTEC in Ontario between 1990 and 1995 and to evaluate the spatial association between livestock density and the incidence of VTEC in human populations using a Geographical Information System (GIS) and spatial regression models.

* Author for correspondence: Dr Pascal Michel, Guelph Laboratory, Health Canada, 110 Stone Road West, Guelph, Ontario, Canada, N1G-3W4.

MATERIALS AND METHODS

Data sources

Data on the 3001 VTEC sporadic cases reported in Ontario from January 1990 to the end of December 1995 were extracted from the Reportable Disease Information System (RDIS) database from the Ontario Ministry of Health. For the purpose of surveillance data entry in this information system, cases of VTEC infection are defined as: 'persons with compatible clinical signs for which verocytotoxin was detected from stool specimens; persons with compatible clinical signs and for which verocytotoxigenic *Escherichia coli* was isolated from stool or blood; or any symptomatic person with an epidemiologic link to two or more laboratory-confirmed VTEC cases'. Information on the Ontario population distribution was derived from the 1991 census of Canada which provided a profile of census divisions and census subdivisions based on data collected from all households [5]. Farm animal distribution and land use data were obtained from the 1991 Census of Agriculture for Ontario (Statistics Canada).

Rate calculations

Rates over 5 years of VTEC cases per 10000 inhabitants in Ontario counties were standardized using the age structure of the 1991 Canadian population [6]. Calculations for age adjustment were performed using a personally-programmed spreadsheet software (Lotus 123; Lotus development corporation, Georgia, USA).

Time-series and trend analyses

The original time-series consisted of the monthly number of VTEC cases reported in Ontario for the 72 months of the study period. Monthly incidences of VTEC cases were calculated using the total number of new VTEC cases recorded in a region for a given month as the numerator, and the respective total population reported in the 1991 Ontario census as the denominator. Simple regression analysis was used to define the straight-line trend in the VTEC series over the study period.

Spatial manipulation and mapping

All cartographic outputs were produced by a Geographical Information Systems (GIS) (ArcView 2.1, ESRI, Redlands, CA, USA). In addition to relational linkage between databases and the production of maps, the GIS environment was used to perform several spatial manipulations including the calculation of the county's centroid coordinates (latitude and longitude), the Euclidian distances between each pair of counties, and the production of contiguity and inverse distance matrices used for spatial autocorrelation and regression.

Spatial distribution

The method of nested mean rates was used to delimit counties with extreme or unusual risks of VTEC cases [7]. For mapping purposes, the measures of animal densities were categorized into four classes according to the natural breaks in their cumulative frequencies. Two indices of spatial autocorrelation, the Moran's I statistic [8] and the G statistic [9, 10], were calculated to explore the spatial distribution of VTEC cases across the 49 counties of Ontario. An observation-specific measure of spatial autocorrelation derived from the G statistic, the G_i statistic, was also calculated.

Regression models

Counties located in the northern regions of the Province (n = 11) were omitted from the spatial regression analysis because of the great differences in their geographic and demographic characteristics as compared to the regions included in the southern counterpart [11]. Metropolitan areas were created in 6 of the 39 counties located in the southern section of the Province. These new areas were formed to improve the homogeneity in the measure of exposure (animal density) within each area. Metropolitan areas were constructed by extracting the demographic and geographic information of a census sub-division (a geographic sub-element of the county) or merging and then extracting this information from contiguous census sub-divisions. The metropolitan areas thus defined were required to comply with all of the following criteria: (1) population greater than or equal to 100000; (2) population density greater than or equal to 1500 pop/km^2; (3) livestock enumeration information matching exactly the boundary of the new metropolitan area available from the agriculture census profile of 1991; (4) no livestock numerated in the new metropolitan areas; (5) frequency of VTEC cases for both the metropolitan and the rest of the county can be calculated based on the patient's forward sortation area (FSA) information recorded in the disease database. Following these criteria, six counties which included major metropolitan areas were divided into two sub-areas. Since Toronto County consists of a single metropolitan area, this county was not further sub-divided.

Variables used in the modelling process included: proportion of the total land which is cultivated (total cultivated area/total area); cattle density (total cattle/total cultivated area); dairy cattle density (total dairy cattle/total cultivated area); density of 'livestock other than cattle' (total pig+total sheep+total horse+total poultry/total cultivated area) and livestock density (animal units/total cultivated area). The 'animal unit' variable is an index calculated by Agriculture and Agri-Food Canada and includes a standardized measure of the number of cattle, horses, sheep, swine and poultry based on the production of nitrogen in their manure [12]. Other predictors considered in the regression analyses were human population density (total population/total county), latitude and longitude of the county centroids. Variables were selected using a backward stepwise manner.

Regression models were constructed using four methods: ordinary least square (OLS) procedure with adjustment of the variance matrix, OLS with a jackknife variance estimate, maximum likelihood estimation (MLE) including a spatial lag coefficient and MLE including a spatial error coefficient. The first OLS method adjusts the variance matrix (White adjustment) [13] for the possible departure from linear regression assumptions (homoscedastic and uncorrelated error terms) due to the spatial structure of the data. An alternative method used to estimate a robust covariance matrix for the OLS estimates in the presence of heteroscedasticity was based on the jackknife resampling method [14]. Inference on the coefficients of these models was based on the most conservative of the jackknife or adjusted variance estimates.

Two classes of spatial models were evaluated, namely a model with spatial error dependence and a model with a spatially lagged dependent variable. These two classes of spatial models, respectively, take into account the geographical inter-dependence among the errors (spatial error model), and among the observed values (spatial lag model) in a specific model. For more technical details relating to the spatial error and the spatial lag models, we refer the reader to Ord [15], Anselin [16], Anselin [17] and Anselin and Hudak [18].

The inverse of the Euclidian distances between the county's centroids (inverse distance matrix) was used for all regression calculations which required the specification of a spatial weight matrix. The Akaike Information Criteria (AIC, measure of fit) [19], the parsimony and interpretability of the model, as well as the results from the residual diagnostic tests were considered in the selection and evaluation of the competing spatial regression models. Spatial regression analysis was conducted using the software SpaceStat (SpaceStat ver 1·8; Regional Research Institute, West Virginia University, VI, USA).

RESULTS

The temporal distribution of VTEC cases showed a marked seasonal pattern with peaks centred on the month of July (Fig. 1). During the summers of 1990, 1991 and 1995, over 100 cases per month were reported at the highest peak of reporting. Approximately 75 cases per month were reported at a similar time for 1992, 1993 and 1994. Except for two increases in case numbers in the spring of 1992 and 1995, the overall pattern of VTEC was very regular over the study period. For Ontario and all regional sub-series (northern, southern, eastern, central, western), a non-significant slope coefficient indicated no evidence of a systematic long term increase or decrease in the average monthly number of reported cases.

The geographic distribution of standardized rates of VTEC cases is presented in Figure 2. According to the nested means categorization technique, 8 counties were classified as having a very low (0–1·76), 11 low (1·77–2·44), 13 average (2·45–3·34), 8 high (3·35–4·00) and 9 very high (4·01 and higher) incidence of VTEC cases (rate are per 10000 population). Counties with a very high incidence of VTEC cases as compared to the Ontario average were situated in the western and central part of the western region, the north-west section of the central region, and the far western part of the eastern region, all of which areas of predominantly mixed agriculture. Areas belonging to the northern region generally had a low incidence of VTEC cases. Counties which represent or include large urban areas were associated with either average or low levels of VTEC rates.

The Moran's *I* index indicated an overall significant spatial autocorrelation of VTEC incidence in Ontario

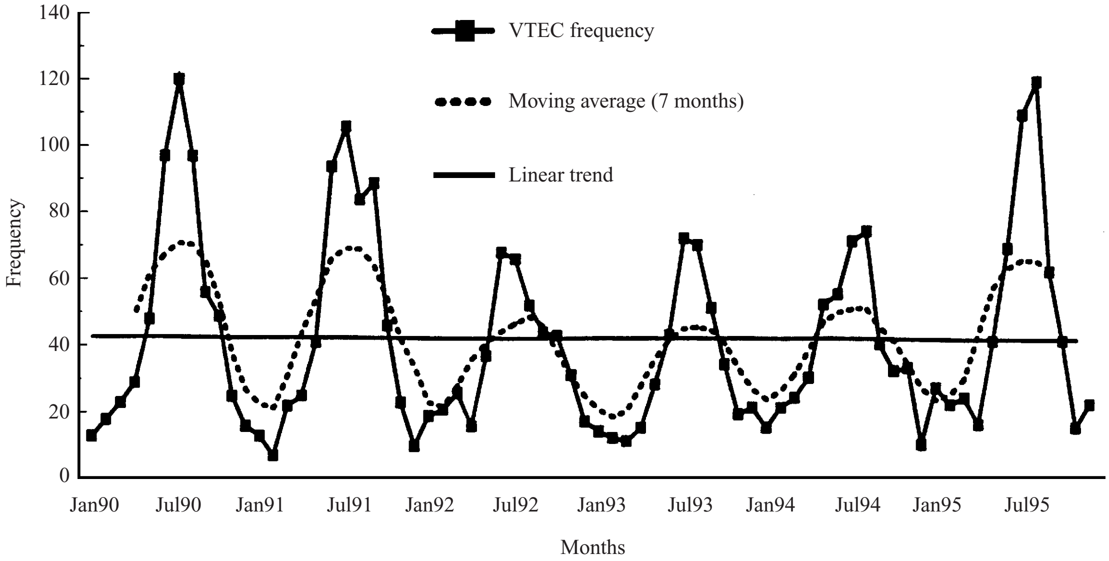

Fig. 1. Linear trend and moving average for VTEC time-series, Ontario (1990–5).

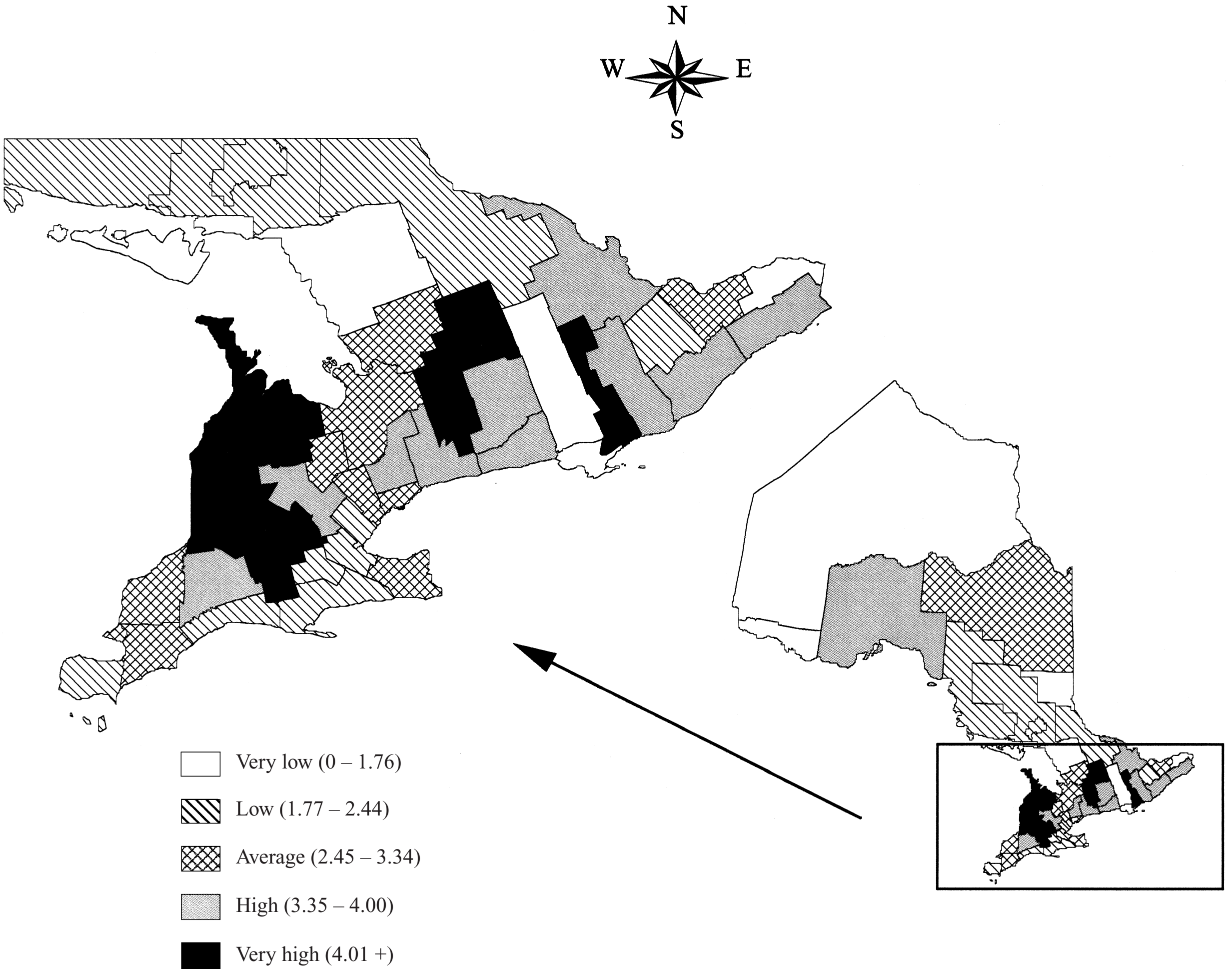

Fig. 2. Incidence of cases of VTEC infection in Ontario, 1990–5 (cases per 10000 population).

regardless of the underlying null distribution or the weight matrix chosen for the calculation ($P < 0{\cdot}005$). An overall spatial inter-dependence of the VTEC incidence across Ontario was also found by calculating the G statistic ($P = 0{\cdot}002$).

For most regions, the geographic distribution of

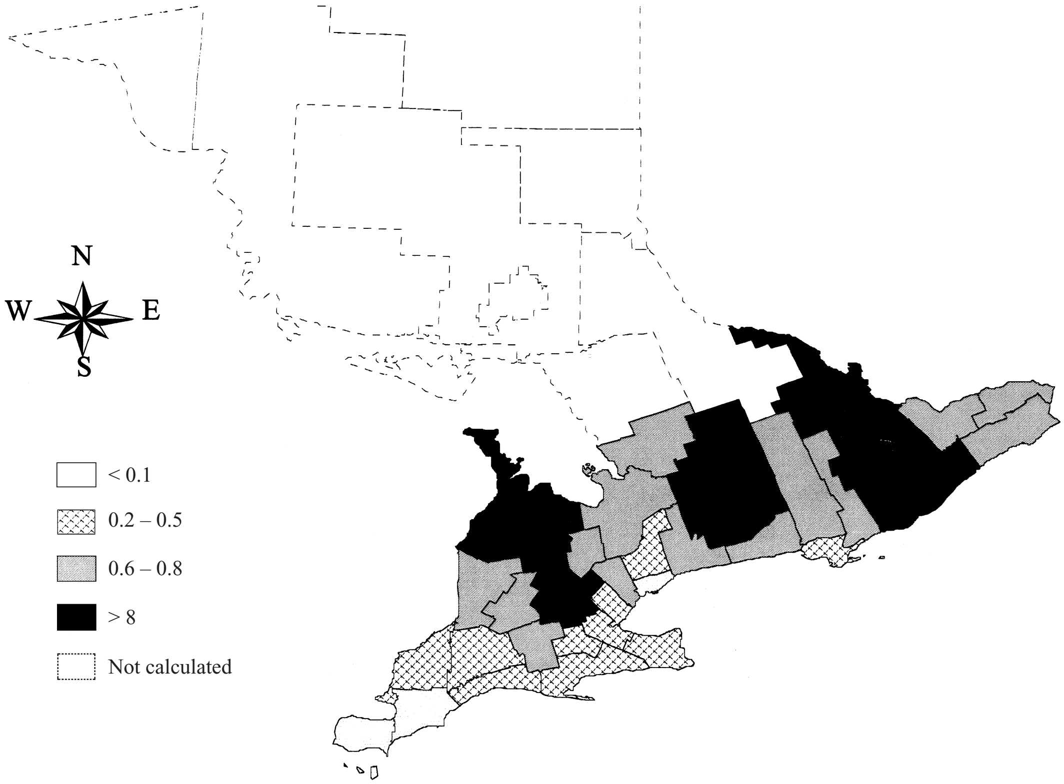

Fig. 3. Cattle density in southern Ontario, 1991. Total number of cattle per cultivated area (hectare).

Table 1. *Competing models for spatial regression of VTEC incidence in Ontario*

Models	Variables	Coefficients	S.E.*	*P* values	AIC†
A	Constant	2·42	0·16	< 0·001	111·25
	Cattle density	1·47	0·33	< 0·001	
B	Constant	2·59	0·2	< 0·001	114·85
	Livestock density	0·61	0·2	0·002	

* Based on the jackknife resampling method [14].

† AIC, Akaike Information Criteria [19].

cattle density by county presented a similar geographic pattern as the one described for human VTEC cases. Counties with very high cattle density were distributed in three areas of Ontario (Fig. 3).

The results of two competing models (A and B) which included 44 geographical units of southern Ontario are presented in Table 1. The first model (A) indicated a positive and significant spatial association between cattle density and human VTEC incidence ($P < 0{\cdot}001$). Model B also indicated a positive and significant spatial association between a measure of livestock density (animal unit) and human VTEC incidence ($P = 0{\cdot}002$). For both models, the estimation of a 'robust' covariance matrix for the ordinary least square estimates, when the assumptions of homoscedastic and uncorrelated error terms were not verified, were based on the jackknife method.

DISCUSSION

Although the surveillance definition for VTEC infection in Ontario is inclusive of all serotypes, the majority (> 95 %) of the VTEC cases recorded in the database were typed as *E. coli* O157 VT+ [11] and epidemiological interpretation based on the following results should be made accordingly. The monthly distribution of VTEC cases in this study concurs with previous reports of the seasonal distribution of this disease. In a 2-year study performed in Alberta, 77·4% of the VTEC cases occurred between June and September with a marked peak in July [20]. In a 10-year summary of *E. coli* O157:H7 surveillance in Scotland, Coia and colleagues also reported that over 60% of the reported cases occurred between May and September [21]. The cause of the increased incidence of disease during the summer months is unknown, although it is presumably related in some manner to increased ambient temperature. It is possible, for example, that elevated environmental temperature favours multiplication of VTEC in the farm environment and on food products during processing, distribution and preparation for consumption.

Clustering in the geographical distribution of VTEC incidence in Ontario was supported by significant indices of spatial autocorrelation regardless of the statistical method used. Detailed analysis of the autocorrelation pattern also revealed local non-stationnarities particularly in the northern regions of the Province. In these regions, the VTEC incidence in a given county was not strongly correlated with the corresponding magnitude in proximate counties suggesting local non-stationarity in the spatial process. This finding underlines the numerous analytical problems associated with the evaluation of geo-political areas with low population densities and characterized by less precise exposure and outcome measures [22].

Adjustment of the regression model to accommodate the spatial nature of the data, and more specifically the estimation of a 'robust' covariance matrix can be accomplished in various ways. Two approaches for such estimation were evaluated during the regression analysis: the first based on the adjusted White variance and the second based on a jackknife method. This latter method gave slightly more conservative estimates than the alternative robust approach based on the adjusted White variance and was adopted. Similarly, other possible spatial adjustments were explored during the first stage of the analysis. In an attempt to adjust the regression models for spatial effects, a spatial lag coefficient and a spatial error term were offered to the regression models. However these spatial terms were not statistically significant.

Important observations made in this study were the relatively high incidence of the condition in rural regions as opposed to urban areas, and the consistent spatial association between VTEC incidence in human populations and measures of cattle or livestock density in the southern section of the Province. The presence of livestock density in a competing model, and the constant failure to include the variable representing 'livestock other than cattle' alone or conditional on the presence of cattle density, suggest that the explanatory power of livestock density was possibly due to the strong contribution of cattle in the calculation of that index. Although the present results consistently revealed the spatial association between measures of cattle density and human VTEC incidence, it is not clear how this association should be interpreted. First, the observed effect could suffer from a bias due to the presence of significant metropolitan centres in some counties also having a high cattle population density. Assuming a true association between cattle density and VTEC disease in humans, and considering that a relatively high proportion of the county population will be located in urban centres, the presence of cities with a large population could dilute or reverse the observed cattle-VTEC association when measured at the county level. One way of minimizing such bias is to make the groups, within each geographic units, as homogeneous as possible in regard to the exposure measure [23]. Therefore in the present study, seven of the largest metropolitan areas of southern Ontario, representing approx. 44% of the total population, were extracted from their counties of origin. This spatial manipulation created seven urban geographical units with a measure of cattle density equal to zero. However, the use of a similar 'extraction' procedure for smaller metropolitan areas was limited by the joint precision of the disease, census and geographical databases. The magnitude and direction of this bias on the cattle-VTEC association was not evaluated in the context of the present study. Secondly, it is also possible that the observed association is not causal. Regardless, the potential importance of this association substantiates the need for further research on the topic.

The overall spatial association of cattle density and human VTEC incidence suggests that living in an agricultural region where cattle are raised could be an important risk factor for the acquisition of VTEC disease. In the past, VTEC organisms have been isolated from the faeces of healthy cattle and other farm animals, thereby identifying them as reservoirs for human infection [24–27]. People living or working on dairy farms have an increased exposure to VTEC strains than urban controls [28] and are known to be at higher risk of carrying VTEC organisms [29]. This increased carriage rate might be explained by a closer contact of farm families with cattle and cattle manure and by the consumption of unpasteurized milk by some individuals [30]. Whether or not this evidence of increased exposure to VTEC organisms actually translates into higher reported cases as compared to the rest of the population is unknown. However, in regions with higher cattle density, other transmission patterns involving the contamination of various elements of the environment are likely to exist. Since cattle manure is used as fertilizer for crops and pastures, contamination of vegetables, shallow water wells and recreational water is possible. An example of the contamination of water wells in intense agricultural areas was illustrated in a 1992 Ontario survey of ground water quality in which 32% of wells exceeded the maximum acceptable concentration for coliform bacteria and 25% had faecal coliform bacteria isolated [31]. Possible contamination of drinking water and exposure to animals were also underlined as influential factors for VTEC cases following the preliminary findings of a Scottish surveillance study which documented that 20% of cases lived on or had visited a farm, 17% had been in contact with animal manure, 12% had a private water supply and 2% had recent water failures in their homes [21]. The impact of living in an agricultural area on the risk of VTEC cases was further suggested by a recent study comparing VTEC isolation rates of diarrhoeic patients living in urban and rural regions of Mexico. In this study, Parra and co-workers reported a higher VTEC isolation rate in patients who lived in rural regions compared to those in urban areas [4]. Unfortunately, no information on the definition of rural regions, no denominators and no details concerning the density of farm animals in these regions were given by the authors, making the study results difficult to compare with those of the present investigation. It is also possible that geographic variations in human VTEC incidence could reflect regional differences in meat production, handling and distribution practices, although investigation of these factors was beyond the scope of the present investigation.

The results of the present study suggest that human VTEC incidence is high in rural regions as compared to urban areas and that farm animal density, more particularly cattle density, is a significant predictor of this incidence in many regions of Ontario. These findings support the possible influence of direct and indirect contact with reservoir animals as an important mode of transmission of VTEC organisms. In areas with higher cattle density, factors that could be responsible for VTEC transmission include the contamination of surface water and shallow wells by cattle manure used as fertilizer; working with, or being in close contact with cattle; and consumption of food produced and processed locally. It is understood that, under the limitations of the present study design, the observed association between human VTEC incidence and cattle density may not be causal. However, the importance of such information for the public health and agricultural sectors is such that further studies, including specific evaluations of the comparative risk of disease acquisition between rural and urban human populations, as well as investigations of environmental risk factors associated with human exposure to cattle, are warranted. Finally, this study also illustrates the value of existing population-based surveillance data to describe the geographical distribution of reportable enteric diseases as a means of both orienting resources and efforts to areas of higher risk and guiding policy-making.

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REFERENCES

1. Notifiable Diseases Annual Summary, 1995. Canada Comm Dis Rep 1997; **23S9**: 92–3.
2. Griffin PM, Tauxe RV. The epidemiology of infection caused by *E. coli* O157:H7, other enterohemorrhagic *E. coli*, and the associated hemolytic uremic syndrome. Epidemiol Rev. 1991; **13**: 60–97.
3. Waters JR, Sharp JCM, Dev VJ. Infection caused by *Escherichia coli* O157:H7 in Alberta, Canada, and in Scotland: a five-year review, 1987–1991. Clin Infect Dis. 1994; **19**: 34–43.
4. Parra MR, Torres LJ, Camorlinga PM, Giono CS, Gonzalez AS, Munoz HO. Frequency of identification of cytotoxigenic strains of *Escherichia coli* in cases of diarrhea from rural and urban communities. Arch Invest Med (Mex) 1991; **22**: 217–22.

5. Statistics Canada. Profile of census divisions and census subdivisions – part A. Ottawa: Supply and Services Canada, 1992. 1991 Census of Canada. Service number 846–031.
6. Pickle LW, White AA. Effects of the choice of age-adjustment method on maps of death rates. Stat Med 1995; **14**: 615–27.
7. Brown P, Hirschfield A, Marsden J. Analysing spatial patterns of disease: some issues in the mapping of incidence data for relatively rare conditions. In: de Lepper MJC, Scholten HJ, Stern RM, eds. The added value of geographical information systems in public and environmental health. The Netherlands: Kluwer Academic Publishers, 1995: 145–63.
8. Moran P. The interpretation on statistical maps. J Royal Stat Soc 1948; **B10**: 243–51.
9. Getis A, Ord JK. The analysis of spatial association by use of distance statistics. Geogr Anal 1992; **24**: 189–206.
10. Ord JK, Getis A. Local spatial autocorrelation statistics: distributional issues and an application. Geogr Anal 1995; **27**: 286–306.
11. Michel P. An epidemiologic study of human cases of verocytotoxigenic *Escherichia coli* infection reported in Ontario [dissertation]. Ontario, Canada; University of Guelph, 1997.
12. Ministry of Agriculture and Food, Ministry of the Environment, Ministry of Housing of Ontario. Agricultural Code of Practice, 1996: 14–5.
13. White H. A heteroskedastic-consistent covariance matrix estimator and a direct test for heteroskedasticity. Econometrica 1980; **48**: 817–38.
14. Efron B. The jackknife, the bootstrap and other resampling plans. Philadelphia: SIAM J Appl Math 1982.
15. Ord K. Estimation methods for models of spatial interaction. J Am Stat Assoc 1975; **70**: 120–6.
16. Anselin L. Estimation methods for spatial autoregressive structures. New York, NY: [Regional Science Dissertation and Monograph Series], 1980.
17. Anselin L. Spatial econometrics, methods and models. Dordrecht. Kluwer Academic, 1988.
18. Anselin L, Hudak S. Spatial econometrics in practice: a review of software options. Reg Science Urb Econ 1992; **22**: 509–36.
19. Akaike H. Likelihood of a model and information criteria. J Econometrics 1981; **16**: 3–14.
20. Pai CH, Ahmed N, Lior H, Johnson WM, Sims HV, Woods DE. Epidemiology of sporadic diarrhea due to verocytotoxin-producing *Escherichia coli*: a two-year prospective study. J Infect Dis 1988; **157**: 1054–7.
21. Coia JE, Sharp JCM, Curnow J, Reilly WJ. Ten years experience of *Escherichia coli* O157 in Scotland (1984–1993). In: Karmali MA, Goglio AG, eds. Recent advances in verocytotoxin-producing *Escherichia coli* infections. 2nd international conference on VTEC, Bergamo, Italy. [Amsterdam: Elsevier Science B. V, 1994]: 41.
22. Smans M, Estève J. Practical approaches to disease mapping. In: Elliot P, Cuzick J, English D, Stern R, eds. Geographical and environmental epidemiology; methods for small-area studies [Oxford: Oxford Medical Publications, 1996.]
23. Morgenstern H. Uses of ecologic analysis in epidemiologic research. Am J Pub Health 1982; **72**: 1336–44.
24. Chapman PA, Wright DJ, Norman P, Fox J, Crick E. Cattle as a possible source of verocytotoxin-producing *Escherichia coli*-O157 infections in man. Epidemiol Infect 1993; **111**: 439–47.
25. Clarke RC, McEwen SA, Harnett N, Lior H, Gyles CL. The prevalence of verotoxin-producing *Escherichia coli* (VTEC) in bovines at slaughter. Abstr Annu Meet Am Soc Microbiol, 1988.
26. Wray C, Randall LP, McLaren IM, Wooddward MJ. Verocytotoxic *Escherichia coli* from animals, their incidence and detection. In: Karmali MA, Goglio AG, eds. Recent advances in verocytotoxin-producing *Escherichia coli* infections, 2nd international conference on VTEC, Bergamo, Italy. [Amsterdam: Elsevier Science B. V, 1994]: 69.
27. Conedera G, Zuin A, Marangon S. Seroprevalence of neutralizing antibodies to *Escherichia coli* verocytotoxins in domestic and wild animals in Italy. In: Karmali MA, Goglio AG, eds. Recent advances in verocytotoxin-producing *Escherichia coli* infections, 2nd international conference on VTEC, Bergamo, Italy. [Amsterdam: Elsevier Science B. V; 1994]: 81.
28. Reymond D, Johnson RP, Karmali MA, et al. Neutralizing antibodies to *Escherichia coli* vero cytotoxin 1 and antibodies to O157 lipopolysaccharide in healthy farm family members and urban residents. J Clin Microbiol 1996; **34**: 2053–7.
29. Wilson JB, Clarke RC, Renwick R, et al. Verocytotoxigenic *Escherichia coli* infection on dairy farms in southern Ontario. In: Karmali MA, Gogli AG, eds. Recent advances in verocytotoxin-producing *Escherichia coli* infections, 2nd international conference on VTEC, Bergamo, Italy. [Amsterdam: Elsevier Science B. V, 1994]: 61.
30. West AM, Martin SW, McEwen SA, Clarke RC, Tamblyn SE. Factors associated with the presence of *Salmonella* sp. in dairy farm families in southwestern Ontario. Can J Pub Health 1988; **79**: 119–23.
31. Agriculture and Agrifood Canada. The Ontario farm groundwater quality survey; Winter 1991/1992.