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Hierarchical Bayesian bivariate disease mapping: analysis of children and adults asthma visits to hospital

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In spatial epidemiology, detecting areas with high ratio of disease is important as it may lead to identifying risk factors associated with disease. This in turn may lead to further epidemiological investigations into the nature of disease. Disease mapping studies have been widely performed with considering only one disease in the estimated models. Simultaneous modeling of different diseases can also be a valuable tool both from the epidemiological and also from the statistical point of view. In particular, when we have several measurements recorded at each spatial location, one can consider multivariate models in order to handle the dependence among the multivariate components and the spatial dependence between locations. In this paper, spatial models that use multivariate conditionally autoregressive smoothing across the spatial dimension are considered. We study the patterns of incidence ratios and identify areas with consistently high ratio estimates as areas for further investigation. A hierarchical Bayesian approach using Markov chain Monte Carlo techniques is employed to simultaneously examine spatial trends of asthma visits by children and adults to hospital in the province of Manitoba, Canada, during 2000–2010.

Keywords: Conditional autoregressive (CAR) model; disease mapping; geographic epidemiology; hierarchical Bayesian model; multivariate data; spatial statistics

1. Introduction

Asthma is the most widespread chronic respiratory disease in Canada. It is a disease that inflames and narrows the airways in response to a trigger. A trigger can be an allergen or an irritant. An allergen may be animal dander, pollen, mould, dust mites and cockroaches and an irritant may be exercise, cold air, tobacco smoke, respiratory viral infections, and certain chemicals. Symptoms of asthma include, but are not limited to coughing, wheezing, shortness of breath and a tight feeling in the chest. The main risk factors for developing asthma are a family history of asthma or an allergy such as eczema or allergic rhinitis; infants being exposed to high levels of a trigger such as dust mites; and high levels of exposure to tobacco smoke or chemical triggers in a workplace environment [13].

According to the World Health Organization, approximately 235 million people worldwide suffer from asthma [25] and approximately 10% of the people living in Canada are diagnosed as having asthma [13]. According to Statistics Canada, Asthma is most common during childhood and at least 13% of Canadian children are affected by asthma [4]. As well, the major reason for hospitalization of children in Canada is asthma [16]. People suffering from asthma often have to make unscheduled visits to a physician or a hospital and in serious cases asthma can even

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lead to death. There are nearly 250,000 deaths around the world annually due to asthma [25]. Therefore, it is crucial to identify any trends in asthma incidence that may help to detect risk factors and lead to further epidemiological studies. Over a region, trends may be evident so the purpose of our paper is to examine geographical variation in the number of asthma visits by children and adults to hospital during 2000–2010 in the province of Manitoba, Canada.

As part of routine monitoring of geographic areas by public health agencies, disease rate (or ratio) maps are often used to examine trends over space. These agencies need to have reliable maps that are methodologically sound. The statistical and epidemiological literature has devoted considerable attention to this important issue, since regional maps of morbidity and mortality rates are useful tools in determining spatial patterns of disease. Identifying regions with substantively different rates may be suggestive of region-level characteristics that could be responsible for the geographic pattern of disease rates. These characteristics could be further examined to determine any causal relationship with the disease.

It is well known that if we have more than one disease, with measurements recorded at each spatial location, we need to consider multivariate areal data models to handle the dependence among the multivariate components and the spatial dependence between locations; assuming that these diseases are believed to be related [9, 11, 12, 14, 26]. In this paper, we study a multivariate conditional autoregressive (MCAR) model [12] to simultaneously examine spatial trends of asthma visits by children and adults to hospital in the province of Manitoba, Canada, during 2000–2010. To make an inference, a hierarchical Bayesian (HB) approach using Markov chain Monte Carlo (MCMC) techniques is employed.

2. Methodology

Let y_{iktl} and n_{iktl} denote frequency of incidences and population for the i^{th} region, k^{th} gender, t^{th} time-period and l^{th} disease ($l = 1, 2$). Then the observed cases for each region and disease (y_{il}) are assumed to follow a Poisson distribution with an unknown mean θ_{il}

$$y_{il} \sim Poisson(\theta_{il}), \tag{1}$$

$$\log(\theta_{il}) = \log(e_{il}) + \alpha_l + \eta_{il},$$

where e_{il} is the gender and time-period standardized expected values for the i^{th} region and l^{th} disease, which is given by

$$e_{il} = \sum_k \sum_t n_{iktl} \frac{y_{.kt.}}{n_{.kt.}},$$

where $i = 1, \ldots, I$, $k = 1, 2$, $t = 1, \ldots, T$ and $l = 1, 2$, with $n_{.kt.} = \sum_i \sum_l n_{iktl}$ and similarly $y_{.kt.} = \sum_i \sum_l y_{iktl}$; α_l is an intercept term that corresponds to the log relative risk of disease l across the entire study region and η_{il} is the log relative risk of incidence for the i^{th} region and l^{th} disease. For disease l it is assumed that the log relative risks are spatially correlated across regions. It is also assumed that

all diseases are correlated within the i^{th} region because the diseases depend on the same unmeasured risk factors in that region.

To capture the spatial variation of η_{i1} (for example disease 1) in the univariate case, a variety of conditional autoregressive (CAR) models may be used by taking a collection of mutually compatible conditional distributions $p(\eta_{i1}|\eta_{-i1}), i = 1, ..., I$ where $\eta_{-i1} = \{\eta_{j1} : j \neq i, j \sim i\}$ and $j \sim i$ refers a set of neighbours for the *i-th* region [2]. We consider the following general model which *uniquely* determines the distribution of η_{i1},

$$(\eta_{11}, ..., \eta_{I1})' \sim N(0, \Sigma_\eta), \tag{2}$$

$$\Sigma_\eta = \sigma^2(D - \gamma C)^{-1},$$

where $D = diag(m_i)$ with m_i as the number of neighbours of region $i; C$ is the adjacency matrix of the map (i.e., $C_{ii} = 0$, and $C_{ij} = 1$ if $i \sim j$, and 0 otherwise); σ^2 is the spatial dispersion; γ is a smoothing parameter which measures spatial association ($\gamma_{min} \leq \gamma \leq \gamma_{max}$) where $\gamma_{\min}^{-1}$ and γ_{max}^{-1} are the smallest and largest eigenvalues of $D^{-1/2}CD^{-1/2}$. As a result, this CAR model is proper since $(D-\gamma C)$ is a non-singular matrix.

When we have information available about two sets of incidences (or mortality) data from the same population groups or regions, we can relate the incidences cases of two different diseases with spatial effects. To that end, we use a multivariate CAR (MCAR) model to capture the spatial random effects (η_{i1}, η_{i2}). A variety of MCAR models may be used [9, 11, 12, 14, 26]. We consider the following model which *uniquely* determines the joint distribution of $\eta_1 = (\eta_{11}, ..., \eta_{I1})'$ and $\eta_2 = (\eta_{12}, ..., \eta_{I2})'$ as

$$\begin{pmatrix} \eta_1 \\ \eta_2 \end{pmatrix} \sim N\left(\begin{pmatrix} 0 \\ 0 \end{pmatrix}, \begin{pmatrix} \Sigma_{11} \; \Sigma_{12} \\ \Sigma_{12}' \; \Sigma_{22} \end{pmatrix} \right), \tag{3}$$

where $\Sigma_{kl}, k, l = 1, 2$, are $I \times I$ covariance matrices [12]. From standard multivariate normal theory, we have $E(\eta_2|\eta_1) = \Sigma_{12}\Sigma_{11}^{-1}\eta_1$ and $var(\eta_2|\eta_1) = \Sigma_{22} - \Sigma_{12}\Sigma_{11}^{-1}\Sigma_{12}' := \Sigma_{11.2}$. Let $A = \Sigma_{12}\Sigma_{11}^{-1}$, we can then write $\Sigma_{22} = \Sigma_{11.2} + A\Sigma_{11}A'$ and $\Sigma_{12} = A\Sigma_{11}$. To uniquely define joint distribution of (η_1, η_2), we need to know that Σ_{11} and $\Sigma_{11.2}$ are positive definite. To write the joint distribution of (η_1, η_2), we need to specify the matrices $\Sigma_{11.2}, \Sigma_{11}$, and A.

Following the univariate CAR structure, it is assumed that the conditional distribution for $\eta_2|\eta_1$ is

$$\eta_2|\eta_1 \sim N(A\eta_1, ((D - \gamma_2 C)\sigma_2^{-2})^{-1}), \tag{4}$$

and the marginal distribution of η_1 is

$$\eta_1 \sim N(0, ((D - \gamma_1 C)\sigma_1^{-2})^{-1}), \tag{5}$$

where γ_2 is the smoothing parameter associated with the conditional distribution of $\eta_2|\eta_1$, γ_1 is similar for the marginal distribution of η_1, and σ_1^2 and σ_2^2 are spatial dispersions of η_1 and $\eta_2|\eta_1$, respectively. Hence, the joint distribution will be always

proper if these two CAR distributions (4)-(5) are valid. It is also well known that these two CAR models are proper since $(D-\gamma_1 C)$ and $(D-\gamma_2 C)$ are non-singular matrices. The values of parameters γ_1 and γ_2 are restricted between 0 and 1. Also, to determine the relationship between η_1 and η_2, it is assumed that the matrix A as $A = \{a_{ij}\}_{i,j=1}^{I}$ where $a_{ij} = \phi_0$ if $j = i$, $a_{ij} = \phi_1$ if $j \sim i$, and 0 otherwise. Hence, we have $A = \phi_0 N_I + \phi_1 C$ where ϕ_0 and ϕ_1 are the bridging parameters associating η_{i2} with η_{i1} and $\eta_{j1}(j \sim i)$, respectively, and N_I is an identity matrix with dimension I. The resulted MCAR model, which has six parameters $(\gamma_1, \gamma_2, \sigma_1^2, \sigma_2^2, \phi_0, \phi_1)$, is proper and uniquely determined by the joint distribution of (η_1, η_2).

3. Bayesian inference

With advances in computational power, much progress in HB modeling has been made that enables stable estimators for mortality rates in small areas by using information from all areas to derive estimates for individual areas. A comprehensive account of HB models for spatial data is given by Banerjee, Carlin, and Gelfand [1].

The Bayesian approach is employed to estimate the model parameters as well as the relative risks. The Gibbs sampler (e.g., [5, 7]) is used to obtain the posterior mean and variance of model parameters. To generate samples from the posterior distribution using MCMC method via the Gibbs sampler, we need to sample from the full conditional distributions. Note that in our model, all of these full conditional distributions are standard distributions that can be easily sampled. To implement our application in the HB setup, we use the OpenBUGS software [15].

The hyperparameters σ_1^2 and σ_2^2, which determine the variation of the spatial trends, have to be estimated from the data. We assign the gamma distributed priors to the precision of spatial effects, $(\sigma_1^{-2}, \sigma_2^{-2})$, where the gamma prior is given by $\sigma^{-2} \sim G(a, b)$(say), with mean a/b and variance a/b^2, to avoid problems with improper hyperpriors. Gamma distributed priors are computationally convenient because the full conditional of σ^{-2} is again gamma distribution. We also assign $N(0, 10^6)$ for $\alpha_1, \alpha_2, \phi_0, \phi_1$ and $U(0, 1)$ for γ_1 and γ_2.

For each model considered in the "Application" section, we independently simulate two parallel runs ($c = 2$), each of length $D = 2d$ with $d = 25,000$. To reduce the effects of the starting values on the final results, the first 10,000 iterations of each run are deleted (a burn-in sequence). We take every fifth iteration of the remaining 40,000 iterations to reduce the autocorrelation in the run (i.e., thinning), leading to 8,000 iterations for each run for analysis purposes. Hence, we have two runs with sample size $8,000$ for each run. To monitor the convergence of the model parameters, we use several diagnostic methods implemented in the Bayesian output analysis (BOA) program [20], a freely available package created for R [18]. In particular, we evaluate descriptive diagnostic tests such as the autocorrelation of generated samples of model parameters from the posterior distribution and convergence diagnostic tests such as Brooks, Gelman, and Rubin tests [3, 7] and Heidelberger and Welch test [10]. None of these tests indicated non-convergence of the model parameters.

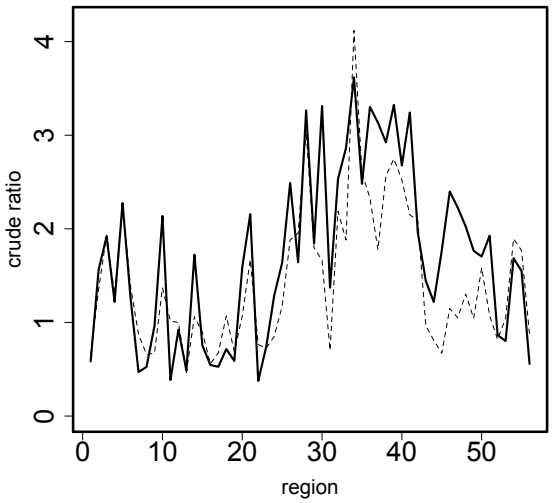

Figure 1. Plots of SDR for children (solid line) and adults (dashed line) asthma visits to hospital in the province of Manitoba, Canada.

4. Application

Our study was based on a yearly dataset of asthma visits to a hospital by Children (age $\leq$ 20) and adults (age $>$ 20) in the province of Manitoba, Canada, during the 2000–2010 fiscal years. Eleven Regional Health Authorities, which were subdivided into 56 Regional Health Authority Districts (RHAD), were responsible for the distribution of health care services in the province of Manitoba and these RHADs are the geographic unit used in our model and all data were linked to these geographic boundaries. A population-based centroid was provided for each RHAD although the centroid was not necessarily the geographic centre of that region. The regions were labelled 1,2,...,56 and the data were aggregated over the study period 2000 to 2010. The number of asthma visits by children totaled 14,691 over the study period with mean and median number of yearly cases per region of 26 and 17 (range 3 to 422), respectively, while the number of asthma visits to hospital by adults totaled 77,095 over the study period with mean and median number of yearly cases per region of 138 and 52 (range 11 to 3885), respectively. The population of Manitoba was stable during the study period from 1.15 million in 2000 to 1.20 million in 2010. The region population sizes varied from 920 to 663,000, with mean and median numbers of 20,900 and 7922, respectively. The largest population was in region 56 while region 38 had the least population.

For each region, asthma visits to a hospital by children was considered disease 1 and asthma visits to a hospital by adults was considered disease 2. The expected number of diseases is adjusted by gender and year. The standardized disease ratios (SDRs) (i.e., $SDR_{il} = y_{il}/e_{il}$) shown in Figure 1 exhibit the evidence of spatial correlation between two diseases, motivating use of the MCAR model.

We fit the model (1) to our datasets. Table 1 reports the model parameters estimate and corresponding 95% credible intervals. It seems that the model is able to capture some variations presented in the data as the model parameters are mostly significant. Regarding estimation of the spatial association parameters, (λ_1, λ_2), we observe that we have strong degree of spatial association in the random effects η_1 as the point estimate and corresponding 95% equal-tail interval estimates

Table 1. Parameters estimate and corresponding 95% credible intervals of bivariate spatial mixed model using the HB method; children and adults asthma visits to hospital in the province of Manitoba, Canada, 2000–2010.

Parameter	Estimate	95% CI
α_1	0.344	(-1.049,1.205)
α_2	0.232	(-0.337,0.856)
λ_1	0.969	(0.893,0.998)
λ_2	0.723	(0.240,0.981)
σ_1^2	0.666	(0.446,0.997)
σ_2^2	0.323	(0.210,0.486)
ϕ_0	0.639	(0.448,0.832)
ϕ_1	0.003	(-0.046,0.052)

are 0.969 and $(0.893, 0.998)$, while the moderate point estimate and wide confidence interval suggest a relatively modest degree of spatial association in the random effects η_2. It is also important to note that in our set-up, λ_1 measures spatial association in the children random effects η_1, while λ_2 measures spatial association in the adults random effects η_2 given the children random effects η_1.

Turn now to spatial dispersion for each disease, i.e., σ_1^2 and σ_2^2. It is resulted from Table 1 that we have relatively small variability in both random effects although again comparison is difficult here since σ_1^2 is a marginal dispersion for η_1 while σ_2^2 is a conditional dispersion for η_2 given η_1.

Note that from linking parameters ϕ_0 and ϕ_1, it is also resulted from Table 1 that the two diseases have positive spatial correlation. This is also evident from the posterior means and corresponding 95% credible intervals of the SDRs for two diseases (Table 2).

In particular, Figure 2 shows that the two diseases are strongly correlated with higher fitted ratios in the north-central part of the province. More investigation may be warranted to explore the reasons for seemingly higher ratios of asthma cases in the north-central part of Manitoba compared to other parts of the province.

5. Sensitivity analysis

We now investigate the choice of priors through a sensitivity study for our data analysis. Full details of the prior sensitivity and choice of models appear in [17]. The hyperprior distributions of the variance components are generally set to be vague to get the most information from the data. In general, the prior for the precision of the random effects (σ^{-2}) is often specified as a gamma distribution with scale and shape parameters both equal to 0.001. One may also use a uniform prior for the standard errors σ [8].

To investigate the influence of hyperprior specifications in our set-up, we conduct a sensitivity analysis with respect to the prior distributions for the precision of random effects parameter σ_1^{-2} and σ_2^{-2}, assuming a variety of different gamma priors $G(a, b)$. Following [19, 21, 23, 24], we use the following combinations in our experimental design: $(a, b) = (0.001, 0.001)$, $(0.5, 0.0005)$, $(0.01, 0.01)$, $(0.1, 0.1)$, $(1, 0.1)$, $(2, 0.001)$, $(0.2, 0.0004)$, and $(10, 0.25)$, which are denoted by A, B, C, D,

Table 2. Posterior mean of SDRs and corresponding 95% credible interval of bivariate spatial mixed model using the HB method; children and adults asthma visits to hospital in the province of Manitoba, Canada.

	Children		Adults	
Region	Estimate	95% CI	Estimate	95% CI
1	0.600	(0.465 , 0.764)	0.653	(0.578 , 0.735)
2	1.548	(1.427 , 1.680)	1.355	(1.300 , 1.413)
3	1.910	(1.668 , 2.175)	1.924	(1.775 , 2.083)
4	1.203	(1.036 , 1.394)	1.296	(1.215 , 1.380)
5	2.212	(1.831 , 2.649)	2.233	(2.053 , 2.429)
6	1.221	(0.966 , 1.525)	1.383	(1.267 , 1.506)
7	0.482	(0.415 , 0.555)	0.870	(0.821 , 0.920)
8	0.533	(0.430 , 0.653)	0.658	(0.591 , 0.732)
9	0.916	(0.789 , 1.058)	0.678	(0.624 , 0.738)
10	2.065	(1.832 , 2.319)	1.373	(1.271 , 1.482)
11	0.453	(0.335 , 0.600)	1.003	(0.922 , 1.089)
12	0.867	(0.638 , 1.157)	0.994	(0.877 , 1.123)
13	0.461	(0.368 , 0.573)	0.471	(0.426 , 0.521)
14	1.713	(1.467 , 1.988)	1.084	(0.915 , 1.272)
15	0.731	(0.536 , 0.972)	0.897	(0.816 , 0.984)
16	0.540	(0.473 , 0.613)	0.559	(0.519 , 0.600)
17	0.531	(0.445 , 0.626)	0.674	(0.624 , 0.728)
18	0.737	(0.577 , 0.932)	1.057	(0.963 , 1.157)
19	0.585	(0.484 , 0.704)	0.688	(0.628 , 0.754)
20	1.568	(1.416 , 1.729)	1.073	(1.018 , 1.130)
21	2.141	(1.909 , 2.393)	1.669	(1.569 , 1.775)
22	0.410	(0.348 , 0.481)	0.759	(0.723 , 0.797)
23	0.736	(0.634 , 0.850)	0.728	(0.683 , 0.774)
24	1.280	(1.070 , 1.517)	0.851	(0.777 , 0.928)
25	1.632	(1.457 , 1.823)	1.171	(1.084 , 1.261)
26	2.468	(2.229 , 2.726)	1.879	(1.697 , 2.070)
27	1.668	(1.473 , 1.882)	1.950	(1.870 , 2.030)
28	3.227	(2.909 , 3.567)	3.027	(2.886 , 3.176)
29	1.854	(1.682 , 2.040)	1.795	(1.713 , 1.880)
30	3.194	(2.780 , 3.645)	1.668	(1.557 , 1.784)
31	1.366	(1.225 , 1.517)	0.712	(0.649 , 0.779)
32	2.539	(2.153 , 2.978)	2.182	(1.925 , 2.455)
33	2.837	(2.248 , 3.532)	1.862	(1.545 , 2.230)
34	3.636	(3.195 , 4.130)	3.985	(3.554 , 4.450)
35	2.505	(2.222 , 2.810)	2.555	(2.327 , 2.805)
36	3.269	(2.929 , 3.632)	2.327	(2.088 , 2.589)
37	3.113	(2.876 , 3.367)	1.782	(1.604 , 1.969)
38	2.900	(2.256 , 3.677)	2.463	(2.059 , 2.920)
39	3.275	(2.735 , 3.890)	2.704	(2.306 , 3.162)
40	2.695	(2.382 , 3.039)	2.482	(2.208 , 2.777)
41	3.192	(2.839 , 3.576)	2.156	(1.894 , 2.436)
42	1.938	(1.381 , 2.666)	2.066	(1.714 , 2.483)
43	1.411	(1.180 , 1.671)	0.974	(0.871 , 1.088)
44	1.233	(1.019 , 1.479)	0.807	(0.726 , 0.894)
45	1.722	(1.514 , 1.951)	0.680	(0.627 , 0.737)
46	2.361	(2.102 , 2.646)	1.151	(1.070 , 1.236)
47	2.150	(1.804 , 2.545)	1.056	(0.934 , 1.189)
48	2.014	(1.733 , 2.334)	1.299	(1.191 , 1.418)
49	1.748	(1.474 , 2.058)	1.042	(0.941 , 1.152)
50	1.712	(1.515 , 1.925)	1.581	(1.505 , 1.659)
51	1.862	(1.624 , 2.132)	1.082	(1.011 , 1.156)
52	0.890	(0.743 , 1.056)	0.821	(0.760 , 0.884)
53	0.843	(0.712 , 0.993)	1.041	(0.980 , 1.105)
54	1.675	(1.447 , 1.929)	1.890	(1.792 , 1.993)
55	1.564	(1.385 , 1.762)	1.763	(1.686 , 1.843)
56	0.558	(0.541 , 0.575)	0.869	(0.861 , 0.878)

E, F, G, and H, respectively. In fact, the Gamma priors $(0.1, 0.1)$ and $(2, 0.001)$ have lowest and highest means and corresponding variances while other Gamma priors are between these two priors. We also study the uniform distribution $U(0, 100)$ for standard errors σ_1 and σ_2 denoted by U. We consider the same set-up as in our data analysis, noting that the scenario A is the same set-up employed in the data analysis Section.

Table 3 provides the model parameters estimates (and corresponding standard

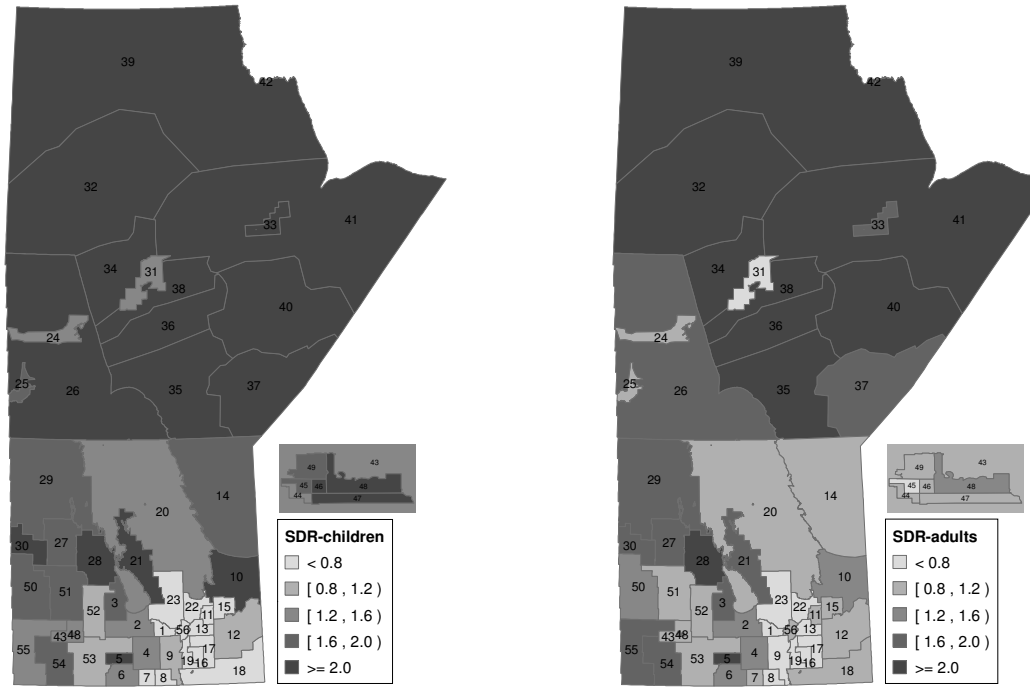

Figure 2. Maps of posterior mean of SDRs for children and adults asthma visits to hospital in the province of Manitoba, Canada. Major urban centre (Winnipeg region) is provided as inset.

Table 3. Parameters estimates (and corresponding standard errors) and means of SDRs over regions for sensitivity analysis of prior distributions for different scenarios of gamma and uniform distributions for variance components.

Prior	A	B	C	D	E	F	G	H	U
α_1	0.335(0.413)	0.342(0.425)	0.336(0.424)	0.352(0.406)	0.344(0.413)	0.346(0.410)	0.339(0.394)	0.332(0.409)	0.354(0.401)
α_2	0.227(0.294)	0.230(0.300)	0.226(0.300)	0.239(0.291)	0.232(0.292)	0.233(0.292)	0.229(0.283)	0.222(0.301)	0.239(0.284)
λ_1	0.968(0.030)	0.969(0.028)	0.968(0.030)	0.968(0.030)	0.969(0.029)	0.972(0.026)	0.968(0.029)	0.980(0.019)	0.967(0.030)
λ_2	0.716(0.197)	0.722(0.197)	0.717(0.196)	0.715(0.197)	0.723(0.194)	0.741(0.186)	0.721(0.196)	0.812(0.150)	0.713(0.197)
σ_1^2	0.693(0.150)	0.676(0.144)	0.693(0.150)	0.694(0.151)	0.666(0.142)	0.636(0.132)	0.686(0.147)	0.480(0.088)	0.710(0.155)
σ_2^2	0.333(0.076)	0.324(0.072)	0.333(0.075)	0.337(0.076)	0.323(0.071)	0.303(0.067)	0.330(0.075)	0.226(0.044)	0.342(0.077)
ϕ_0	0.636(0.100)	0.636(0.097)	0.636(0.098)	0.635(0.101)	0.639(0.098)	0.640(0.096)	0.635(0.099)	0.665(0.087)	0.633(0.101)
ϕ_1	0.003(0.025)	0.004(0.024)	0.003(0.024)	0.004(0.025)	0.003(0.024)	0.003(0.024)	0.003(0.025)	0.0001(0.022)	0.004(0.025)
$\overline{SDR}_1$	1.687	1.687	1.687	1.688	1.687	1.687	1.687	1.687	1.687
$\overline{SDR}_2$	1.424	1.424	1.424	1.424	1.424	1.424	1.424	1.423	1.424

errors) and also the means of SDRs over regions ($\overline{SDR}$) for different sceneries. As shown in Table 3, the estimates of model parameters and $\overline{SDR}$s are stable for different scenarios of gamma and uniform distributions for variance components.

6. Conclusion

We illustrate a model for bivariate spatial analysis that pays specific attention to the mapping of areal data of two (or more) diseases ratios which are believed to be correlated. The model accommodates a multivariate conditional autoregressive (MCAR) model to capture the dependence among the multivariate (bivariate) components and the spatial dependence between regions. The fully Bayesian approach is employed for the analysis using Markov chain Monte Carlo techniques. We study the convergence of the samples obtained through diagnostic methods and conclude that convergence was achieved. Our sensitivity analysis using different priors for

the variance components points out that this hierarchical Bayesian bivariate spatial analysis for Poisson data yields similar results regardless of the vague priors considered in the "Application" section.

We adjust our expected number of cases by gender and time (year). However, we may have lost some information about the time trends of incidences for children and adults over years. Also, in our dataset of asthma visits to hospital, some individuals may have been counted multiple times although the number of re-admissions to hospital was relatively small. To capture these variations, we have planned to study spatio-temporal modeling of bivariate disease mapping in a separate manuscript.

We have shown that these two diseases (asthma visits by children and adults) are spatially correlated, so it is evident that we should use this information for disease mapping. Overall, it is suggested by the model estimates that the high asthma incidence ratios for children and adults were mainly located in the north-central part of the province. In fact, in a separate manuscript [22], we have studied these two diseases (children and adults asthma visits to hospital) separately using a frequentist approach and observed that the high asthma incidence ratios for children are also mainly located in the north-central part of the province while for adults only two regions in the south-central and two regions in the north-central part of the province had high asthma incidence ratios. The findings of our analysis may represent real increases or may be indicative of different distributions of important covariates that are unmeasured and unadjusted for in our modeling. Further investigation is needed to look for possible risk factors for the regions with high asthma ratios in the north-central part of Manitoba.

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