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Mapping gender variation in the spatial pattern of alcohol-related mortality: A Bayesian analysis using data from South Yorkshire, United Kingdom.

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Abstract

Gender variation in the spatial pattern of alcohol-related deaths in South Yorkshire, UK for the period 1999 and 2003 was explored using two Bayesian modeling approaches. Firstly, separate models were fitted to male and female deaths, each with a fixed effect deprivation covariate and a random effect with unstructured and spatially structured terms. In a modification to the initial models, covariates were assumed estimated with error rather than known with certainty. In the second modeling approach male and female deaths were modeled jointly with a shared component for random effects. A range of different unstructured and spatially structured specifications for the shared and gender-specific random effects were fitted. In the best fitting shared component model a spatially structured prior was assumed for the shared component, while gender-specific components were assumed unstructured. Deprivation coefficients and random effect standard deviations were very similar between the gender-specific and shared component models. In each case the effect of deprivation was observed to be greater in males than in females, and slightly larger in the measurement error models than in the fixed covariate models. Greater variation was observed in the spatially smoothed estimates of risk for males versus females in both gender-specific and shared component models. The shared component explained a greater proportion of the male risk than it did the female risk. The analysis approach reveals the residual (unexplained by deprivation) gender-specific and shared risk surfaces, information which may be useful for guiding public health action.

Keywords: Alcohol mortality; Bayesian disease mapping; shared-component models; ecological models; error-in-covariate.

1 Introduction

Alcohol consumption contributes directly to a range of adverse health outcomes through its toxic effects. alcohol-related causes of death mediated through these toxic effects include acute alcohol poisoning, alcoholic liver cirrhosis, alcoholic pancreatitis, alcoholic gastritis and alcoholic cardiomyopathy. Across Europe, spatio-temporal patterns in alcohol-related mortality are complex with increases in some countries, including the United Kingdom (UK), and decreases in others (Office for National Statistics, 2010; Leon and McCambridge, 2006). The harmful use of alcohol is widespread within the UK (Jones et al., 2008) despite efforts to address the problem (Prime Minister's Strategy Unit, 2004; Department of Health, 2007; BMA Board of Science, 2008). In 2004 it was estimated that 38% of men and 16% of women aged 16 to 64 years had an alcohol use disorder, implying a total burden of approximately 8.2 million people in England alone (Department of Health, 2005). The rising prevalence of binge drinking, defined as drinking more than eight units on at least one day in the past week for men and more than six units on at least one day for women (Goddard, 2006), is of particular concern since this pattern of alcohol consumption increases the risk of death both from alcohol-related causes, as well as from causes not directly related to alcohol (Laatikainen et al., 2003).

Several national reports have highlighted the above issues and suggested possible ways to reduce alcohol-related harm (Prime Minister's Strategy Unit, 2004; Department of Health, 2007; BMA Board of Science, 2008). Options to reduce alcohol-related harm range from population wide interventions to approaches targeting individuals at high risk. Targeted approaches need to first identify individuals at high risk and one method is to identify geographical areas with high rates of alcohol-related disease. Further investigation and additional resources could then be focussed on these areas in order to understand reasons for high rates and attempt to reduce alcohol-related harm.

Certain sections of the population may experience greater than average risk of alcohol-related mortality, including younger age groups and those who experience greater levels of socioeconomic disadvantage (Harrison and Gardiner, 1999; Mar-

tikainen et al., 2003; Erskine et al., 2010). The association with socioeconomic deprivation is seen for both men and women (Erskine et al., 2010). However, men drink more than women and alcohol-related mortality is higher amongst men (Goddard, 2006; Erskine et al., 2010). The evidence suggests that there ought to be similarities as well as differences in the geographical pattern of alcohol-related mortality for men and women.

In this paper, we illustrate a number of options for comparing the spatial patterns of alcohol-related mortality in men and women. We examine a range of Bayesian models based on differing prior specifications for the underlying spatial structure in mortality, and different assumptions regarding the shared nature of risk factors between men and women. We examine the effect of explicitly modeling area-level socioeconomic deprivation as a covariate, as well as presenting the residual spatial pattern in mortality once deprivation is adjusted for.

2 Methods

2.1 The data

We obtained counts of alcohol-related deaths for the period 1999-2003 for the 94 'Standard Table Wards' of the county of South Yorkshire in England, by five year age band and by sex. Standard Table Wards (hereafter called 'wards') are a standard geographical unit for the purposes of aggregating 2001 Census information. Deaths were allocated to wards based on the deceased's usual place of residence. We calculated ward-level expected numbers of deaths for males and for females by applying the overall South Yorkshire age sex specific rates to the ward-level denominator populations. We used as our denominator the estimated 2001 ward-level population counts that were obtained from the 2001 Census, and assumed that these were valid estimates for the whole period 1999-2003. For each ward we calculated the standardised mortality ratio (SMR) as the ratio of observed to expected mortality counts.

Alcohol-related mortality was defined according to the Office for National Statistics agreed set of ICD (International Classification of Diseases) codes (Of-

fice for National Statistics, 2006). This categorisation includes only those causes of death regarded as being most directly attributable to alcohol consumption. Deaths in our data set were coded using the ICD9 system for the years 1999 and 2000, and the ICD10 system for the years 2001 to 2003. See appendix 1 for the ICD codes used to define alcohol-related mortality.

We constructed a measure of socioeconomic deprivation at Census Output Area level. Output Areas are the smallest areas of Census enumeration and consist of approximately 125 houses. We based our deprivation measure on the UV67 Census Table, which reports for each Output Area a cross tabulation of the number of households by the number of four selected deprivation related characteristics present per household. The four household level characteristics were: (1) a member of the household aged 16-74 (who is not a full-time student) is either unemployed or permanently sick; (2) no member of the household aged 16 to pensionable age has at least 5 GCSEs (grade A-C) or equivalent AND no member of the household aged 16-18 is in full-time education; (3) a member of the household has general health 'not good' in the year before the Census or has a limiting long term illness; (4) the household's accommodation is either overcrowded OR is in a shared dwelling OR does not have sole use of bath/shower and toilet OR has no central heating. For each Output Area we took the mean number of characteristics per household as the measure of deprivation for that area.

Each ward comprises a unique set of contiguous Output Areas, and all ward and Output Area boundaries are non-overlapping. Within South Yorkshire the number of Output Areas per ward ranges from 22 to 115 with a mean of 46.

2.2 Analysis stage 1 - modeling male and female mortality separately

In the first stage of the analysis we modeled deaths in males and females separately. For each gender in turn we assumed the following mixed effects model for the observed counts,

$$O_i|\boldsymbol{b} \sim \text{Pois}(\mu_i) \quad (1)$$

$$\log(\mu_i|\boldsymbol{b}) = \log(E_i) + \alpha + \beta x_i + b_i, \quad (2)$$

where i indexes the 94 wards, O_i and E_i are the observed and expected counts for ward i, x_i is the ward-level UV67 measure of socioeconomic deprivation entered as a fixed effect with corresponding regressor β, α is an intercept term and $\boldsymbol{b} = (b_1, \ldots, b_{94})'$ are ward-level random effects.

Because we had deprivation scores at a finer spatial resolution than ward level we were able to enter the deprivation covariate x_i into the model in two ways. In the first instance we calculated for each ward the sample mean of the UV67 scores for the Output Areas comprising the ward, and entered these into the model as quantities assumed to be known with certainty. In the second instance we assumed a measurement error model for the deprivation covariate. Given Output Area level deprivation scores y_{ij} where i indexes the ward, and $j = 1, \ldots, n_i$ indexes the n_i Output Areas within ward i, we assumed the following hierarchical model (based on Spiegelhalter et al., 1996)

$$y_{ij} \sim N(x_i, \tau_i^2), \tag{3}$$

$$x_i \sim N(\theta, \phi^2), \tag{4}$$

with a diffuse $N(0, 10^4)$ prior on the hyperparameter θ, and weakly informative Gamma$(0.001, 0.001)$ priors on the inverse variances, $1/\tau_i^2$ and $1/\phi^2$.

For the random effects we assumed a 'convolution' prior (Besag et al., 1991), i.e. the random effect b_i is the sum of two independent components, a spatially structured term s_i, and an unstructured term u_i. We placed a Gaussian conditional autoregressive (CAR) prior on s_i and a zero mean Gaussian *iid* prior on u_i, i.e.

$$u_i \sim N(0, \sigma_u^2), \tag{5}$$

$$s_i | s_{\delta_i} \sim N(\bar{s}_{\delta_i}, \sigma_s^2 / n_{\delta_i}). \tag{6}$$

The term s_{δ_i} denotes the spatially structured random effects for those wards that are 'neighbours' of ward i. We define the neighbours of ward i as those wards that have a contiguous boundary with ward i. The term $\bar{s}_{\delta_i}$ denotes the mean of the spatially structured random effects for the neighbours of ward i, and n_{δ_i} as the number of those neighbours.

We assumed weakly informative $N(0, 10^4)$ priors for the intercept α and regressor β, and weakly informative Gamma$(0.001, 0.001)$ priors for the precisions

$1/\sigma_u^2$ and $1/\sigma_s^2$. To determine robustness to the specification of the priors on the precision terms we re-ran both models with Gamma$(0.5, 0.0005)$ priors.

2.3 Analysis stage 2 - joint modeling of male and female mortality

In stage 2 of the analysis we jointly modeled deaths in males and in females assuming a shared component model (Knorr-Held and Best, 2001). We assumed Poisson distributions for the data,

$$O_{ik}|\boldsymbol{b}_k, \boldsymbol{\phi} \sim \text{Pois}(\mu_{ik}), \tag{7}$$

with linear predictors

$$\log(\mu_{i1}|\boldsymbol{b}_1, \boldsymbol{\phi}) = \log(E_{i1}) + \beta_1 x_i + b_{i1} + \phi_i \delta, \tag{8}$$

$$\log(\mu_{i2}|\boldsymbol{b}_2, \boldsymbol{\phi}) = \log(E_{i2}) + \beta_2 x_i + b_{i2} + \phi_i / \delta, \tag{9}$$

where gender is indexed $k = 1$ for males and $k = 2$ for females, $\boldsymbol{b}_k = (b_{1k}, \ldots, b_{94k})'$ are random effects representing gender-specific risks for gender k, and $\boldsymbol{\phi} = (\phi_1, \ldots, \phi_{94})'$ is a random effect representing a shared component of risk. The parameter δ ensures identifiability with the squared term δ^2 being interpreted as the ratio of the strength of association between mortality and the unmeasured shared risk in males, to the strength of association between mortality and the unmeasured shared risk in females (Dabney and Wakefield, 2005).

In our first shared component model we assumed convolution priors (i.e. spatially structured and unstructured terms) for both b_{ik} and ϕ_i. We denote this the *full model*. Because the full model is heavily overparameterized we explored the range of more parsimonious alternatives that result from dropping either the structured or unstructured prior term from either the shared or gender-specific components in the full model (see table 2). For each model we computed the DIC and pD values in order to guide model selection.

As in stage 1 we assumed diffuse $N(0, 10^4)$ priors for the regressors, and weakly informative Gamma$(0.001, 0.001)$ priors for all precision terms. We placed a $N(0, 5.9^{-1})$ distribution on $\log(\delta)$, which represents a prior belief that there is

a 95% probability that δ^2 is between 1/5 and 5 (Dabney and Wakefield, 2005). Again, we tested robustness to the specification of the priors on the precision terms by re-running the model with Gamma$(0.5, 0.0005)$ priors.

2.4 Model implementation

We implemented all of the Bayesian models in OpenBUGS 3.2.1 (Lunn et al., 2009). For each model we ran three MCMC chains and determined convergence by examining Gelman Rubin plots. After discarding an adequate burn-in of 100,000 samples from each chain we based our posterior inference on 100,000 subsequent samples. We computed the DIC and pD values for both the observed death counts and the output level deprivation scores for each model (Spiegelhalter et al., 2002).

3 Results

There were 391 alcohol-related deaths in males and 216 in females in South Yorkshire between 1999 and 2003. Numbers of deaths per ward ranged from 0 to 12 in males, and 0 to 8 in females. The denominator person years at risk was 3,088,300 for males and 3,244,075 for females giving an overall annual alcohol-related mortality of 12.66 per 100,000 population for males and 6.66 per 100,000 population for females. This compares with an annual alcohol-related mortality rate across the whole of England and Wales (population 52.4 million) of 14.6 per 100,000 population for males and 7.6 per 100,000 population for females.

Figure 1 (upper panel) shows a thematic map by quintile of ward-level deprivation. The major urban areas of South Yorkshire are centred on the city of Sheffield and the three towns of Barnsley, Rotherham and Doncaster. These urban areas lie in a band running North-South down the centre of the county, with the exception of Doncaster, which is located in the central Eastern part of the county. Socioeconommic deprivation is highest in the urban centres, and lowest in the more rural areas lying in the West of the county, and in the rural areas lying between the central North-South urban belt and Doncaster.

Figure 1 (lower panel) shows a thematic map by quintile of ward-level SMRs across South Yorkshire, for males and for females. Quintiles are defined with

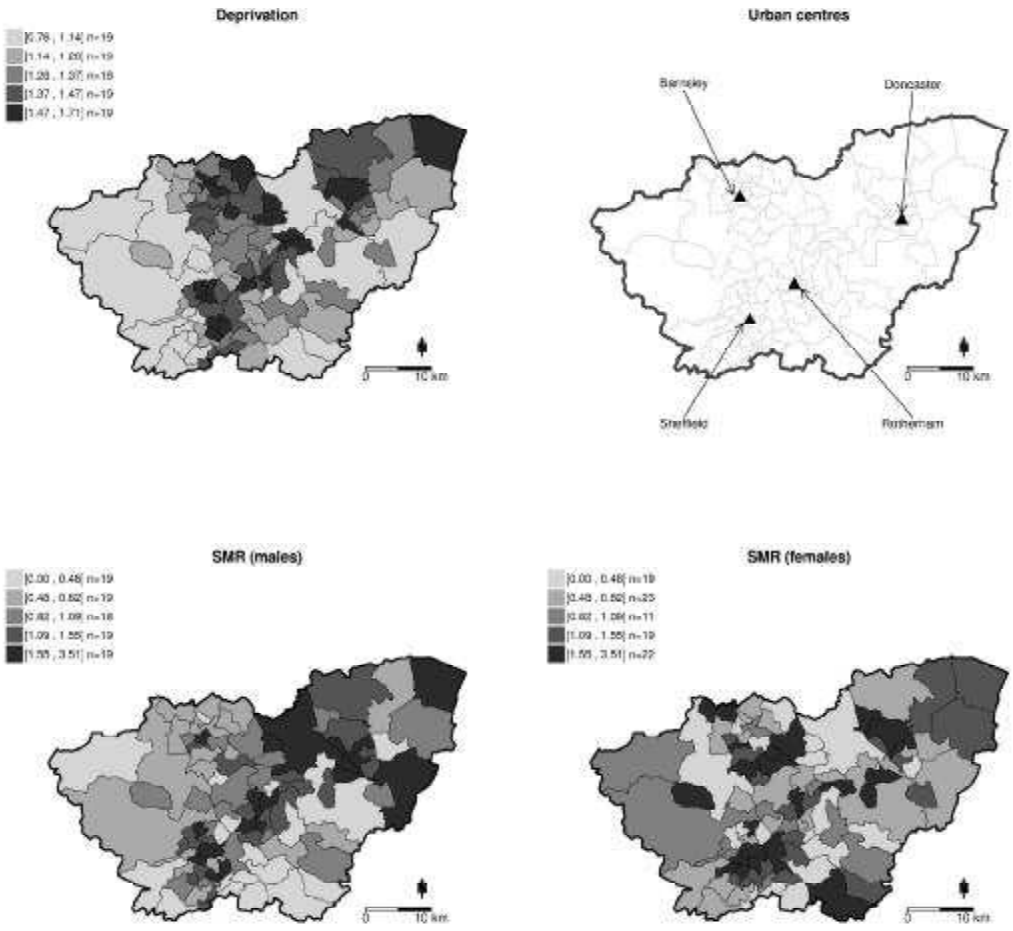

Figure 1: Ward level UV67 deprivation measure by quintile (upper panel). Male and female SMRs, mapped by male quintile cut-points (lower panel)

respect to the male SMRs. Note the contrasting patterns for males and females.

3.1 gender-specific model results

Table 1 shows the posterior parameter estimate for the effect of deprivation (expressed on the relative risk scale) along with the standard deviations of the structured and unstructured random effect terms, for both the model assuming fixed covariates and the measurement error model. In both cases the effect of deprivation is stronger in males than in females, and in females the credible interval includes 1, the null value. The coefficients for the deprivation effect are slightly larger in the measurement error model, and credible intervals are slightly wider reflecting the additional uncertainty.

The standard deviation of the unstructured component of the random effect (σ_h) is similar for males and females in both the model assuming fixed covariates and the measurement error model, whereas the empirical marginal standard deviation of the spatially structured random effect (σ_{es}) is much larger for males than for females in both cases. This is also reflected in the proportion of the variance of the random effect that is due to the spatially structured component, which is higher for males (78.6% in the fixed covariate model; 80.1% in the measurement error model) than for females (32.9% in the fixed covariate model; 33.8% in the measurement error model).

3.2 Shared component model selection

Table 2 shows DIC and pD values for the competing shared component models in which the deprivation covariate is assumed known with certainty. The DIC values suggest that Models H best fits the data. Model H includes an unstructured prior on each of the gender-specific random effects and a spatial CAR prior on the shared component.

Table 3 shows the posterior parameter estimates obtained from model H. Results for the measurement error model are very similar to those of the fixed covariate model. As with the gender-specific models, the deprivation regression coefficient is slightly larger in the measurement error, and is estimated with slightly

Table 1: **Results for gender-specific models**

Deprivation covariate treated as known with certainty		
	Males	Females
Parameter	Mean (95% CrI)	Mean (95% CrI)
$\exp(\beta)$	3.972 (1.828, 8.741)	1.385 (0.648, 2.956)
σ_h (95% CrI)	0.183 (0.028, 0.473)	0.178 (0.028, 0.443)
σ_{es} (95% CrI)	0.418 (0.099, 0.604)	0.100 (0.015, 0.318)
Proportion variance spatial	0.786 (0.050, 0.997)	0.329 (0.004, 0.972)
	DIC (pD)	DIC (pD)
Model fit (deaths)	423.1 (38.8)	352.2 (13.2)

Measurement error model for deprivation covariate		
	Males	Females
Parameter	Mean (95% CrI)	Mean (95% CrI)
$\exp(\beta)$	4.172 (1.862, 9.450)	1.399 (0.640, 3.034)
σ_h	0.173 (0.027, 0.450)	0.174 (0.028, 0.439)
σ_{es}	0.423 (0.155, 0.603)	0.099 (0.015, 0.310)
Proportion variance spatial	0.808 (0.128, 0.997)	0.338 (0.004, 0.970)
	DIC (pD)	DIC (pD)
Model fit (deaths)	423.0 (38.7)	352.2 (13.0)
Model fit (deprivation)	1083.0 (186.1)	1084.0 (186.7)

greater imprecision. The deprivation effect estimates are very similar to those obtained in the gender-specific models for both males and females. For males the proportion of the variance of the random effect that is due to the shared component is 77% in both models, whereas for females it is 62%. These ratios should be treated with caution given the imprecision that is inherent in estimating such ratio parameters. The value of 1.497 (95% CrI 0.879, 2.410) for δ^2 suggests that the shared component is more important for males than for females, though posterior credible intervals are wide.

Figure 2 shows the smoothed relative risk map for males and females following

Table 2: **Model comparison**

Model	Prior specification			DIC (pD)
	Male random effect	Female random effect	Shared component	
Full	CAR+iid	CAR+iid	CAR+iid	773.9 (54.6)
A	CAR+iid	CAR+iid	CAR	774.0 (52.6)
B	CAR+iid	CAR+iid	iid	775.0 (55.6)
C	CAR	CAR	CAR+iid	773.7 (48.5)
D	iid	iid	CAR+iid	772.8 (51.6)
E	CAR	CAR	CAR	773.4 (44.3)
F	iid	iid	iid	779.7 (56.0)
G	CAR	CAR	iid	774.2 (48.9)
H	iid	iid	CAR	772.3 (49.5)
I	CAR	iid	CAR	772.9 (48.9)
J	CAR	iid	iid	772.6 (50.3)
K	iid	CAR	CAR	780.5 (52.8)
L	iid	CAR	iid	773.0 (46.7)

CAR: Spatially structured Gaussian conditional autoregressive prior; iid: zero mean Gaussian unstructured prior.

fitting of gender-specific models (upper panel) and the shared component model (middle panel). In all cases results for the measurement error model are shown. Cut points for the thematic shading for all four maps are those that define the quintiles for the SMRs for males. The choice of modeling strategy (gender-specific versus shared component) makes very little difference to the smoothed estimates of the relative risk of alcohol-related mortality. The DIC values for shared and separate models are also comparable. The variation in risk for males is greater than that for females, but maps are otherwise similar, and clearly reflect an association between deprivation and alcohol-related mortality.

There are five areas for which the posterior 95% credible interval for the relative risk of mortality lies above 1, and 10 areas for which the interval lies below 1. For females there is one area in which the posterior credible interval lies below 1. This

Table 3: **Results for shared component model H**

Deprivation covariate treated as known with certainty	
Parameter	Mean (95% CrI)
$\exp(\beta)$ males	4.054 (1.882, 8.794)
$\exp(\beta)$ females	1.411 (0.663, 3.022)
σ_b males	0.185 (0.028, 0.458)
σ_b females	0.153 (0.028, 0.399)
σ_ϕ shared	0.269 (0.101, 0.404)
δ^2	1.483 (0.890, 2.379)
Proportion variance shared, males	0.773 (0.082, 0.996)
Proportion variance shared, females	0.619 (0.058, 0.985)

Measurement error model for deprivation covariate	
Parameter	Mean (95% CrI)
$\exp(\beta)$ males	4.251 (1.928, 9.450)
$\exp(\beta)$ females	1.428 (0.648, 3.139)
σ_b males	0.186 (0.028, 0.458)
σ_b females	0.157 (0.027, 0.408)
σ_ϕ shared	0.272 (0.118, 0.405)
δ^2	1.497 (0.879, 2.410)
Proportion variance shared, males	0.774 (0.118, 0.996)
Proportion variance shared, females	0.621 (0.071, 0.986)

is shown in the lower panel in figure 2.

The portion of the variation in alcohol-related mortality that is unexplained by the fixed effect, and therefore modeled by the random effects terms is shown in figure 3 (again, results for the measurement error model are shown). For the gender-specific models the term $\exp(b_i)$ is mapped, and for the shared component models $\exp(b_i+\phi_i\delta)$ is mapped for males, and $\exp(b_i+\phi_i/\delta)$ is mapped for females. Again, note the close similarity between the maps resulting from the two modeling strategies. For males, there are three areas in which the posterior 95% credible interval for the unexplained component of risk lies above 1, and four areas for which the interval lies below 1. For females, the posterior credible interval includes

1 in all areas. This is shown in the lower panel in figure 3.

The shared component and gender-specific components of risk are shown in figure 4. The spatial structure that was *a priori* assumed for the shared component is seen for both males and females (upper panel). The female map shows less variation than the male map, reflecting the lesser importance of the shared component in explaining female variation in mortality. The maps for the gender-specific risks (lower panel) show little variation in either gender, reflecting the smaller contributions of the gender-specific components relative to the shared component.

4 Discussion

Across South Yorkshire we found significant spatial variation in ward-level alcohol-related mortality for males. A portion of this variation is explained by ward-level socioeconomic deprivation, but even after adjustment for deprivation, significant variation exists with several areas appearing to be outliers. For females, the spatial variation in mortality is much less marked with no significant association between deprivation and mortality. From a public health perspective, we might be interested in identifying those areas that have an overall elevated risk, but also those that have an elevated *unexplained* risk, since in this latter group factors other than those related to deprivation may be particularly important. Potential causes of the unexplained variation in mortality include differences in patterns of consumption that are not modeled by the deprivation covariate (Jefferis et al., 2007; Bellentani et al., 1997), variation in the prevalence of obesity (an independent risk factor for alcoholic liver disease, Naveau et al., 1997), and differences in the availability and/or quality of services for those with alcohol-related problems (Department of Health, 2005).

Modeling male and female mortality jointly by allowing a portion of the risk to be shared led to estimates of association with deprivation that were essentially the same as those produced by the separate gender-specific models. The model we selected on the basis of the lowest DIC value was that in which the spatially structured variation in risk was modeled by the shared component. We observed

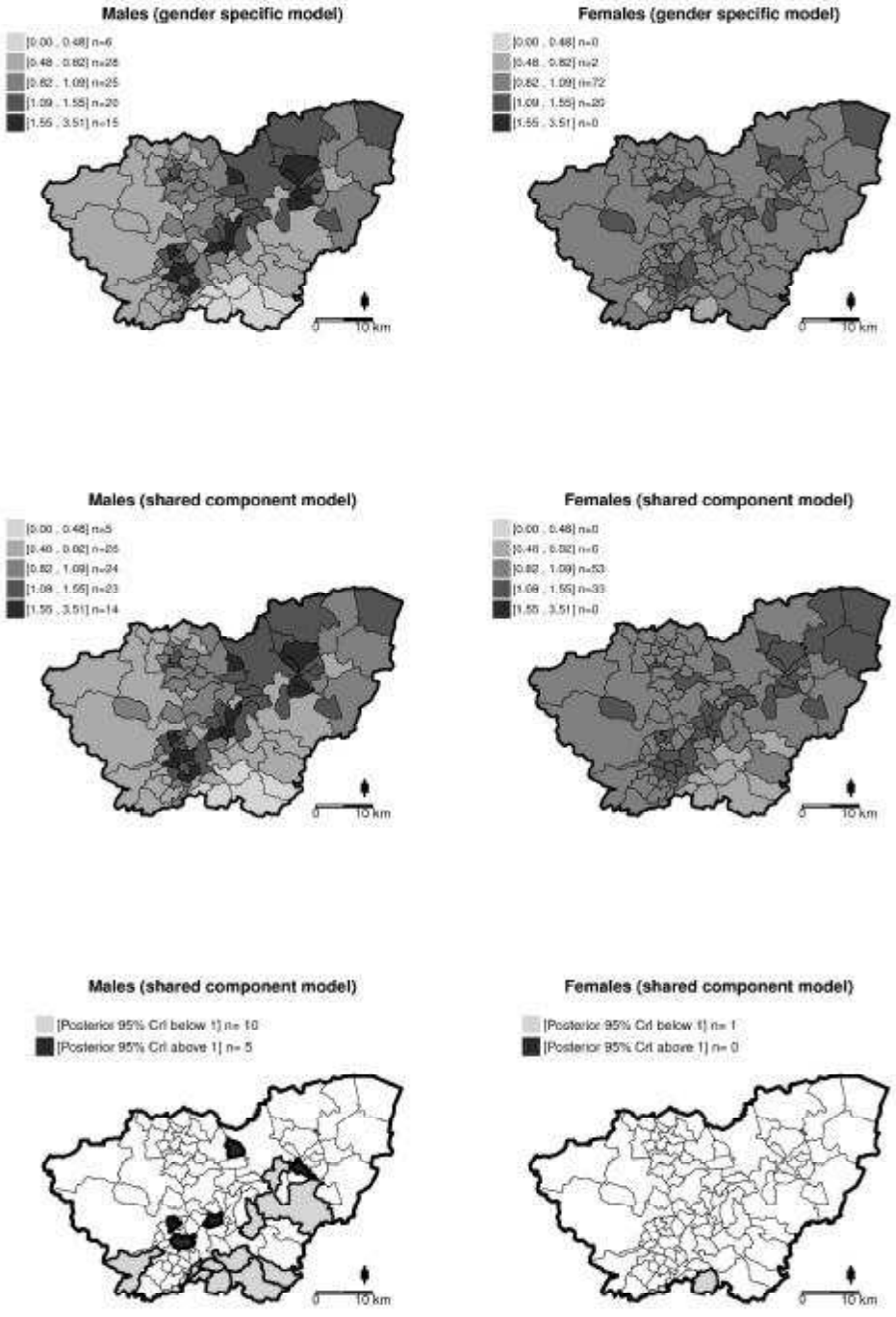

Figure 2: Smoothed relative risk for males and females

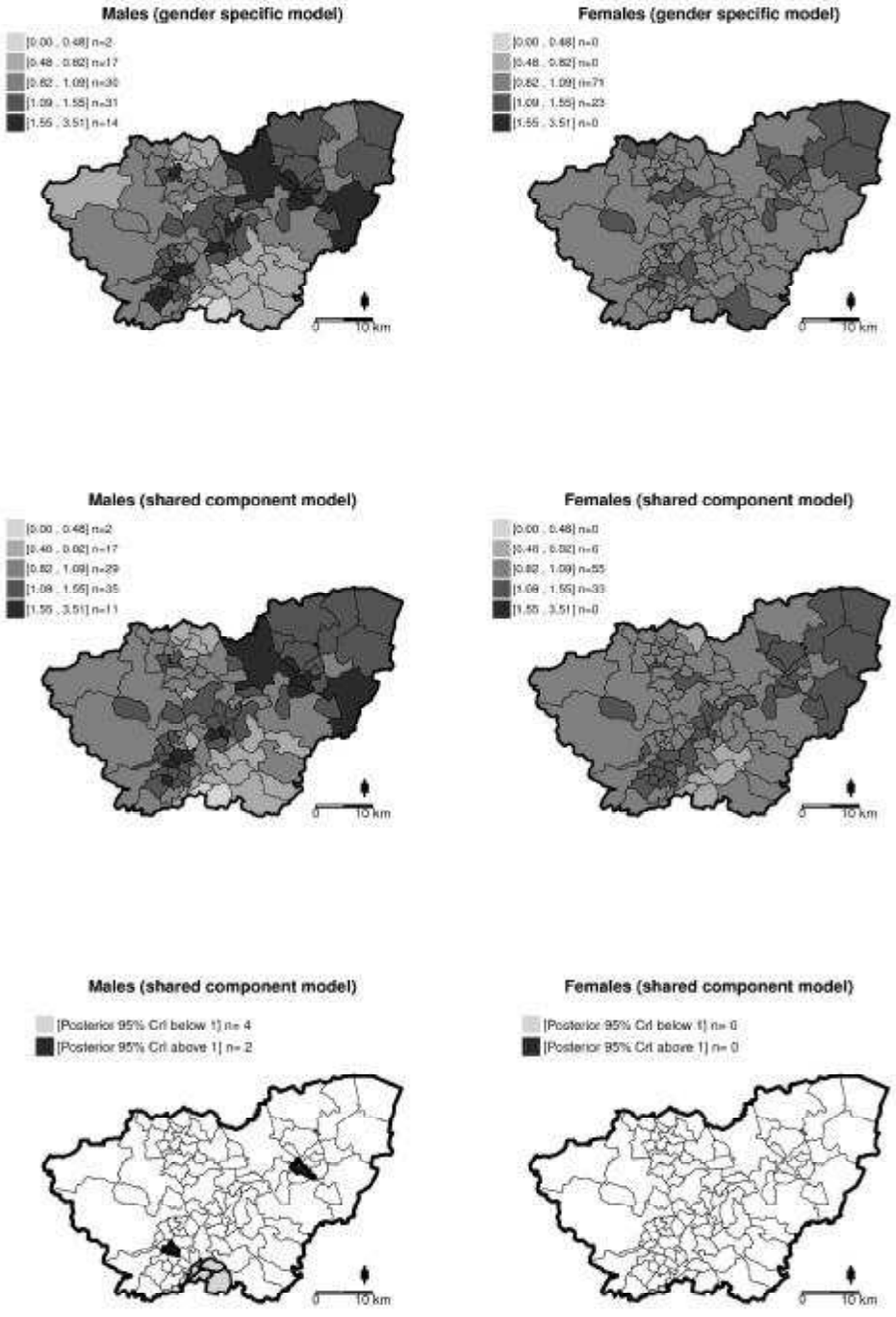

Figure 3: Unexplained variation for males and females

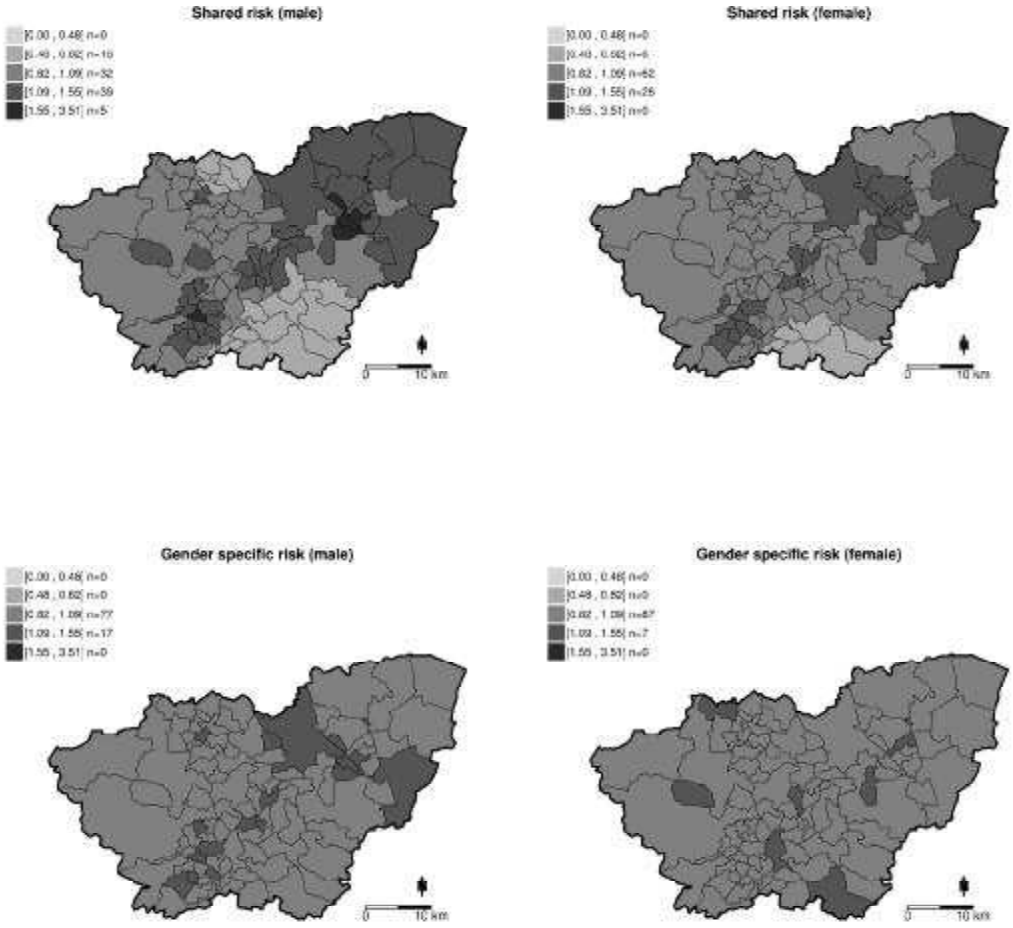

Figure 4: Shared and gender-specific random effects components estimated by the shared component model

a slightly stronger effect of deprivation when we used the Output Area level deprivation information in a measurement error model, rather than assuming that the ward level deprivation scores were estimated with certainty.

As other authors have cautioned (Held et al., 2005), we must be careful with the interpretation of results. In our shared component model we assumed *a priori* spatial structure for the shared component and non-spatial structure in the individual gender-specific components. In our initial exploratory work we found that different assumptions regarding the structure of the priors result in quite different spatial patterns in the shared component, as we would expect, but that the smoothed risk surface was relatively robust to the hyperprior specification.

Our results suggest that there are similarities as well as differences in the spatial pattern of alcohol-related mortality in men and women, although we have to be cautious about this interpretation as numbers of deaths amongst women were small, leading to area risk estimates with high uncertainty. Results also suggest that there is significant spatial variation in alcohol-related mortality that is unexplained by deprivation. Identifying the factors that are associated with elevated risk may help to inform the targeting of harm reduction interventions.

We have presented results for both gender specific and shared component modeling strategies in our case study. In many applications one of the two approaches will clearly be the more appropriate, with the choice of modeling strategy being guided by the purpose of the investigation. If the task is to identify areas in which the risk of mortality is particularly high (or low) for each gender, then posterior risk inference based on separate gender-specific models is likely to be more plausible than that based on a single shared component model. One of the advantages of a shared component model, that it facilitates 'borrowing strength' across genders, is also a potential limitation in that the spatial variation that is specific to each gender will to some extent be 'pulled' towards a common pattern of spatial variation. In a shared component model gender specific patterns in mortality may be (at least partially) lost in the smoothing process. Alternatively, if the object of the analysis is to estimate a risk surface that relates to causes that are common to both male and female mortality, with less concern as to whether males or females will benefit from any subsequent risk reduction, then a shared component may be

useful.

In conclusion, we found that compared with a gender specific modeling approach, the shared component model offered useful additional information when modeling alcohol-related mortality in men and women. The analysis revealed the residual (unexplained by deprivation) shared risk surfaces, information which may be useful for guiding public health action that is targeted at overall risk reduction.

Appendix 1

Table 4: ICD codes defining alcohol-related deaths (Office for National Statistics, 2006)

ICD9 Code	Description
291	Alcoholic psychoses
303	Alcohol dependence syndrome
305.0	Non-dependent abuse of alcohol
425.5	Alcoholic cardiomyopathy
571	Chronic liver disease and cirrhosis
571.0	Alcoholic fatty liver
571.1	Acute alcoholic hepatitis
571.2	Alcoholic cirrhosis of liver
571.3	Alcoholic liver damage, unspecified
571.4	Chronic hepatitis
571.5	Cirrhosis of liver without mention of alcohol
571.6	Biliary cirrhosis
571.7	Other chronic nonalcoholic liver disease
571.8	Unspecified chronic liver disease without mention of alcohol
E860	Accidental poisoning by alcohol

ICD10 Code	Description
F10	Mental and behavioural disorders due to use of alcohol
I42.6	Alcoholic cardiomyopathy
K70	Alcoholic liver disease
K73	Chronic hepatitis, not elsewhere classified
K74	Fibrosis and cirrhosis of liver
X45	Accidental poisoning by and exposure to alcohol

References

Bellentani, S., Saccoccio, G., Costa, G., Tiribelli, C., Manenti, F., Sodde, M., Croce', L. S., Sasso, F., Pozzato, G., Cristianini, G., Brandi and the Dionysos Study Group, G. (1997). Drinking habits as cofactors of risk for alcohol induced liver damage, *Gut*, **41 (6)**: 845–850.

Besag, J., York, J. and Mollié, A. (1991). Bayesian image restoration, with two applications in spatial statistics, *Ann I Stat Math*, **43 (1)**: 1–20.

BMA Board of Science (2008). Alcohol misuse: tackling the UK epidemic.

Dabney, A. R. and Wakefield, J. C. (2005). Issues in the mapping of two diseases, *Stat Methods Med Res*, **14**: 83–112.

Department of Health (2005). Alcohol Needs Assessment Research Project (ANARP): The 2004 national alcohol needs assessment for England, `http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_4122341`.

Department of Health (2007). Safe. Sensible. Social. the next steps in the National Alcohol Strategy, `http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyandGuidance/DH_075218`.

Erskine, S., Maheswaran, R., Pearson, T. and Gleeson, D. (2010). Socioeconomic deprivation, urban-rural location and alcohol-related mortality in England and Wales, *BMC Public Health*, **10 (1)**: 99.

Goddard, E. (2006). General Household Survey 2006. Smoking and drinking among adults, 2006, `http://www.parliament.uk/deposits/depositedpapers/2008/DEP2008-2395.pdf`.

Harrison, L. and Gardiner, E. (1999). Do the rich really die young? Alcohol-related mortality and social class in Great Britain, 1988-94, *Addiction*, **94**: 1871–1880.

Held, L., Natario, I., Fenton, S. E., Rue, H. and Becker, N. (2005). Towards joint disease mapping, *Stat Methods Med Res*, **14 (1)**: 61–82.

Jefferis, B. J. M. H., Manor, O. and Power, C. (2007). Social gradients in binge drinking and abstaining: trends in a cohort of British adults, *Journal of Epidemiology and Community Health*, **61 (2)**: 150–153.

Jones, L., Bellis, M., Dedman, D., Sumnall, H. and Tocque, K. (2008). Alcohol-attributable fractions for England: alcohol-attributable mortality and hospital admissions, `http://www.lape.org.uk/`.

Knorr-Held, L. and Best, N. G. (2001). A shared component model for detecting joint and selective clustering of two diseases, *J R Stat Soc Series A Stat Soc*, **164 (1)**: 73–85.

Laatikainen, T., Manninen, L., Poikolainen, K. and Vartiainen, E. (2003). Increased mortality related to heavy alcohol intake pattern, *J Epidemiol Community Health*, **57**: 379–384.

Leon, D. A. and McCambridge, J. (2006). Liver cirrhosis mortality rates in Britain from 1950 to 2002: an analysis of routine data, *Lancet*, **367**: 52–56.

Lunn, D., Spiegelhalter, D., Thomas, A. and Best, N. (2009). The BUGS project: Evolution, critique, and future directions, *Stat Med*, **28**: 3049–3067.

Mäkelä, P., Keskimäki, I. and Koskinen, S. (2003). What underlies the high alcohol related mortality of the disadvantaged: high morbidity or poor survival?, *Journal of Epidemiology and Community Health*, **57 (12)**: 981–986.

Martikainen, P., Kauppinen, T. M. and Valkonen, T. (2003). Effects of the characteristics of neighbourhoods and the characteristics of people on cause specific mortality: a register based follow up study of 252,000 men, *J Epidemiol Community Health*, **57**: 210–217.

Naveau, S., Giraud, V., Borotto, E., Aubert, A., Capron, F. and Chaput, J. (1997). Excess weight risk factor for alcoholic liver disease, *Hepatology*, **25 (1)**: 108–111.

Office for National Statistics (2006). Defining alcohol-related deaths, `http://www.ons.gov.uk/ons/rel/subnational-health4/`

alcohol-related-deaths-in-the-united-kingdom/
defining-alcohol-related-deaths/index.html.

Office for National Statistics (2010). Alcohol-related deaths in the United Kingdom, 1991-2008, http://www.ons.gov.uk/ons/rel/subnational-health4/alcohol-related-deaths-in-the-united-kingdom/alcohol-related-deaths/statistical-bulletin.pdf.

Prime Minister's Strategy Unit (2004). Alcohol harm reduction strategy for England, Tech. rep., Prime Minister's Strategy Unit.

Spiegelhalter, D. J., Best, N. G., Carlin, B. P. and Van Der Linde, A. (2002). Bayesian measures of model complexity and fit, *J R Stat Soc Series B Stat Methodol*, **64 (4)**: 583–639.

Spiegelhalter, D. J., Best, N. G., Gilks, W. and Inskip, H. (1996). Hepatitis B: a case study in MCMC methods, in *Markov Chain Monte Carlo in Practice*, edited by Gilks, W., Richardson, S. and Spiegelhalter, D. J., London: Chapman and Hall.