Random Effects

- If we believe that underlying structure (potentially) *always* exists in small area health data, then we should attempt to make some allowance for these effects.
- Without knowledge of the specific effects, it is only possible to assume an effect of simple form should be included in the model. This is often called a *random effect*.

- Random effects can be incorporated in models for disease incidence by specifying an extra level of variation in the model. For example, for tract count data, the incidence in each tract may be thought to contain an extra random component of variation (i.e. overdispersion).
 - This extra effect can be included in a model for the intensity in each tract where v_i is the random effect in the *i* th tract:

$$\lambda_i = e_i.m\{F_i.\beta + v_i\}$$

• Usually to estimate such effects we need to assume some *prior* structure for the effect e.g. a prior distribution.

A Bayesian approach is often useful:
a Bayesian model consists of a likelihood and prior distributions for parameters. The product of these yields a posterior distribution for the parameters. The posterior values for parameters can be averaged to give *posterior expected values*.



Denote a conditional distribution as [a|b)]

• For the Poisson count model with simple constant tract rate, a hierarchy for random effects of interest might be:

$$[n_i | v_i] \sim Pois(e_i.v_i)$$
$$[v_i] \sim Gamma(\alpha, \delta)$$

• This leads to the posterior distribution: $[v_i|n_i] \sim Gamma(n_i + \alpha, e_i + \delta)$ and the Bayes estimate of v_i is given by $\frac{n_i + \alpha}{e_i + \delta}$

- Full Bayes analysis would lead to sampling of the $[v_i|n_i]$ distribution, whereas empirical Bayes methods would provide plug-in estimates of α and δ . (Clayton and Kaldor(1987))
 - Maps of the resulting relative risks can be produced.

Uncorrelated and Correlated Heterogeneity

- the exchangeable Gamma prior for the relative risk allows for uncorrelated heterogeneity
- to allow for correlated heterogeneity it is required that we include a prior distribution with spatial correlation

Besag, York and Mollie(1991)[BYM91] first proposed a general model for constant rate tract counts, which included both correlated and uncorrelated heterogeneity:

$$\lambda_i = e_i . \exp\{t_i + u_i + v_i\}$$

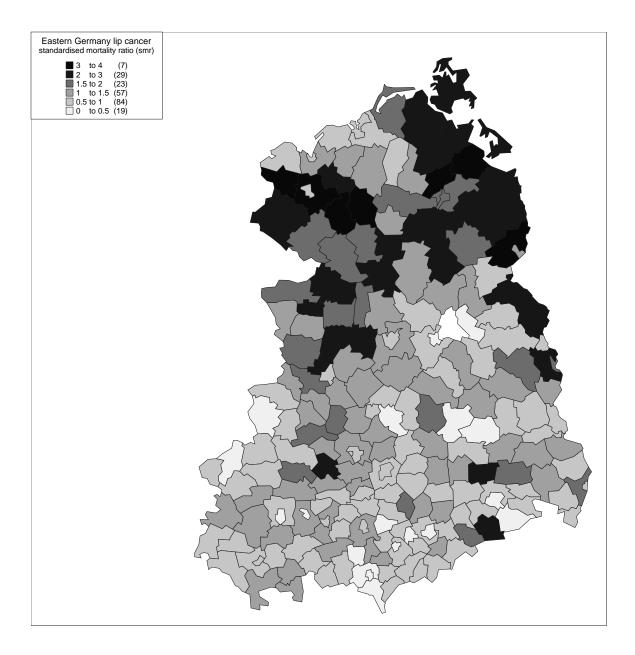
where the uncorrelated heterogeneity is $[v_i|\tau] \sim N(0,\tau)$, and τ has a hyperprior, and $[u_i|\{u_{j\neq i}\}] \propto \frac{1}{\kappa} \exp\{-\frac{1}{\kappa} \sum_{i \in \delta_i} |u_i - u_j|\}$

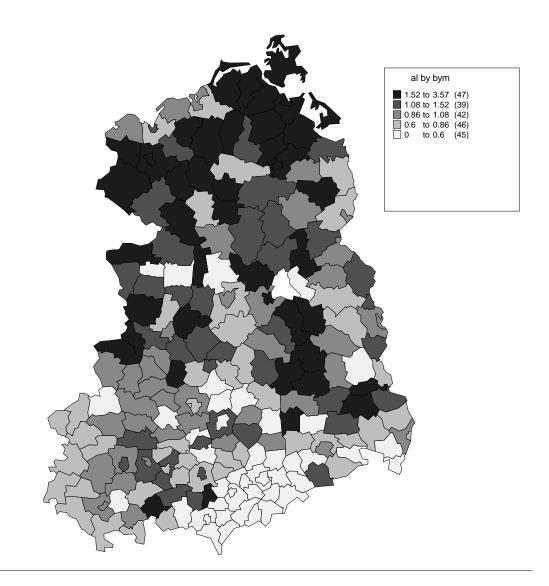
where δ_i is the neighbourhood of the *i* th tract/region. A hyperprior for κ is also assumed.

- The data likelihood is still Poisson but the relative risk is allowed to have components for trend/covariates (*t_i*) and heterogeneity (*u_i*, *v_i*)
- Note that there is a log-normal type of link to the counts and not a direct Gamma prior



• This model is very general as it includes known and unknown confounders and expected rates.





Lip Cancer Eastern Germany: BYM model

Lip Cancer Tutorial

Comparative Analysis of Mapping Methods: East German lip cancer

Aims:

- To acquaint participants with analysis of the East German lip cancer mortality dataset
 - To allow participants to view relative risk surfaces based on the set of 219 landkeise with incidence of lip cancer for 1980-1989

Questions:

1) What is the overall pattern in the SMR map?

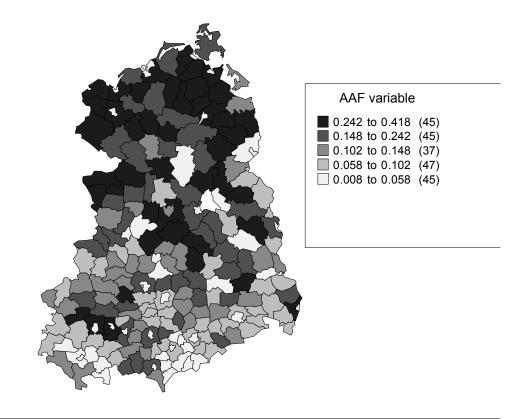
2) What happens to the map when you choose different percentile scales?



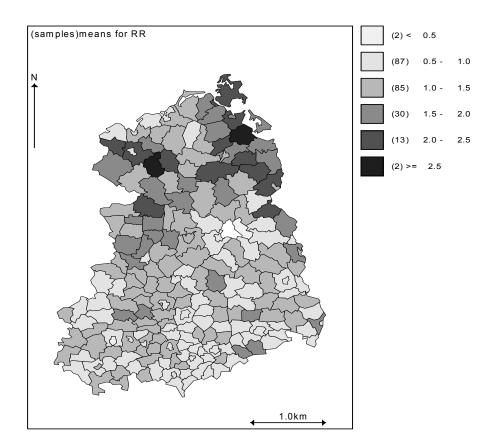
create the crude SMR map with 10 classes create the crude SMR map with 5 classes

A full Bayesian analysis is provided: the maps are AFF, lognormal model; lognormal +AFF; BYM model.; BYM model+AFF; BYM model +AFF: h; BYM model +AFF: b

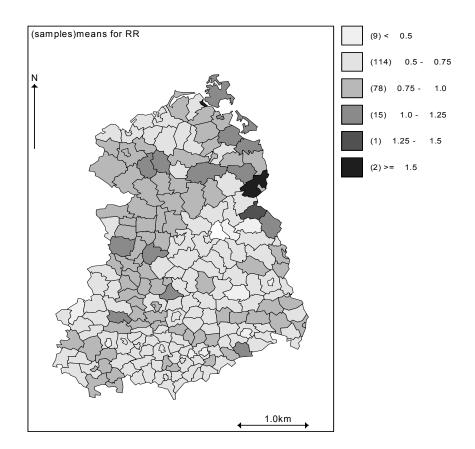
Comment on the differences between the maps.



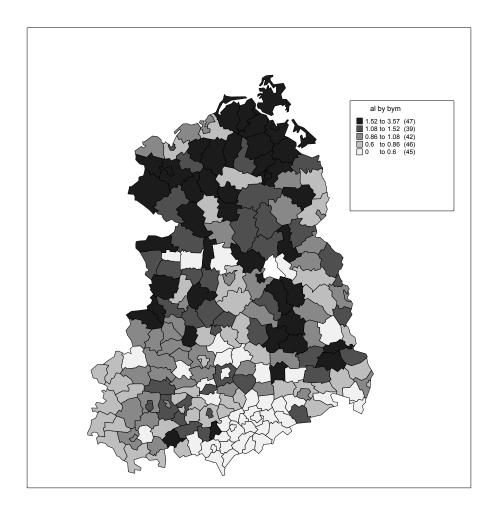
AFF variable



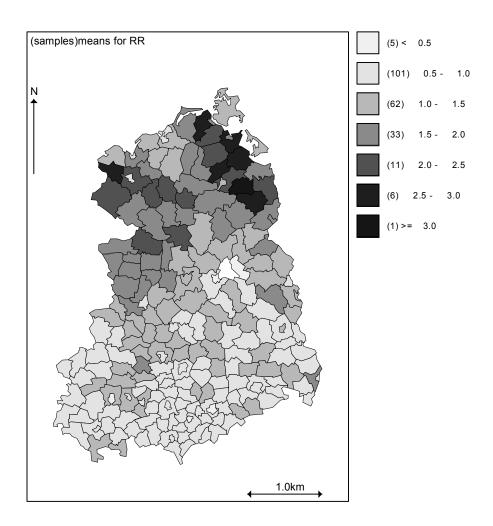
LNmodel : Relative risk



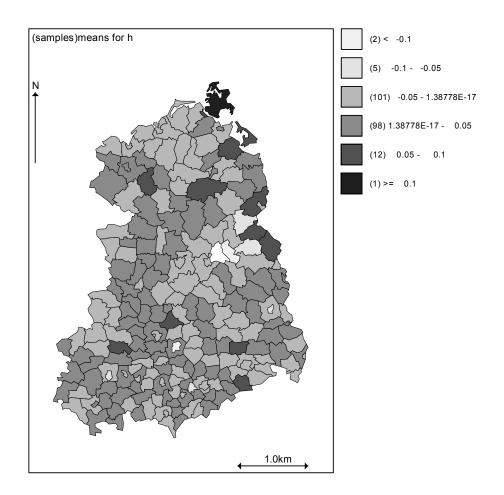
LN model: RR with AFF



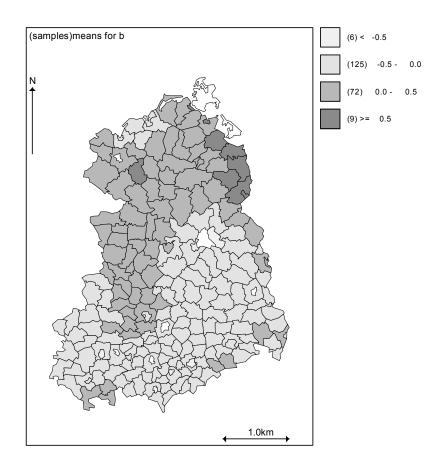
posterior BYM relative risks



BYM model: RR with AFF



BYM model: h



BYM model: b