Bayesian Spatial Modeling of Malnutrition and Mortality among Under-five Children in sub-Saharan Africa

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Dedication

'Acquire knowledge, it enables its possessor to distinguish right from wrong; it lights the way to Heaven; it is our friend in the desert; our society in solitude, our companion when friendless; it guides us to happiness; it sustains us in misery" .... *The Prophet Mohammad(pbuh)*.

To:
Nike
my Lovely wife and friend
&
Halimat Adebusola(F) and Abdulbasit Adedeji(M)
my blessed children.

Declaration of Authorship

I, RASHEED ALANI ADEYEMI, declare that this thesis titled 'Bayesian spatial Modeling of Malnutrition and Mortality among under-five children in sub-Saharan Africa' and the work presented in it are my own. I confirm that:

- This work was done wholly or mainly while in candidature for a research degree at this University.

- No part of this thesis has previously been submitted for a degree or any other qualification at this University or any other institution.

- Where I have consulted the published work of others, this is always clearly attributed.

- Where I have quoted from the work of others, the source is always given. With the exception of such quotations, this thesis is entirely my own work.

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List of Publications

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1. **Rasheed A. Adeyemi**, Temesgen Zewotir & Shaun Ramroop. "Bayesian Multinomial Ordinal Models to analyze the risk factors and spatial patterns of childhood anaemia in Tanzania" **published** Annual Proceedings of the South African Statistical Association (SASA), Congress 1, Nov **2016**, p. 9 - 16 Available at: http://hdl.handle.net/10520/EJC198858.

2. **Rasheed A. Adeyemi**, Temesgen Zewotir & Shaun Ramroop. Spatial-Temporal Modeling of Under-five mortality rates in the context of a developing country *Paper Presented at UKZN College Research Day inQubate, Westville Durban South African.* **2018***.*

3. **Rasheed A. Adeyemi**, Temesgen Zewotir & Shaun Ramroop. Spatial Patterns Of Childhood Mortality And Morbidity In Sub-Saharan Africa: A Bayesian

Geo-Additive Multinomial Models Approach *Paper Presented at UKZN College Research Day Oct.* **2017**.

4. **Rasheed A. Adeyemi**, Temesgen Zewotir & Shaun Ramroop. "Bayesian Multinomial Ordinal Models to analyze the spatial patterns of Childhood anaemia in Tanzania" **presented** 58th *Annual Conference of the South African Statistical Association (SASA) held at University of Cape Town* November **2016**.

5. **Rasheed A. Adeyemi**, , Temesgen Zewotir & Shaun Ramroop. Bayesian Joint modeling of Disease Co-morbidity among under-five children in Nigeria and Tanzania; *Accepted for Presentation 2016.: UKZN College Research Day: Postgraduate*

Abstract

The aim of this thesis is to develop and extend Bayesian statistical models in the area of spatial modeling and apply them to child health outcomes, with particular focus on childhood malnutrition and mortality among under-five children. The easy availability of a geo-referenced database has stimulated a paradigm shift in methodological approaches to spatial analysis. This study reviewed the spatial methods and disease mapping models developed for areal (lattice) data analysis. Observational data collected from complex design surveys and geographical locations often violates the independent assumption of classical regression models. By relaxing the restrictive linearity and normality assumptions of classical regression models, this study first developed a flexible semi-parametric spatial model that accommodates the usual fixed effect, nonlinear and geographical component in a unified model. The approach was explored in the analysis of spatial patterns of child birth outcomes in Nigeria. The study also addressed the issue of disease clustering, which is of interest to epidemiologists and public health officials. The study then proposed a Bayesian hierarchical analysis approach for Poisson count data and formulated a Poisson version of generalized linear mixed models (GLMMs) for analyzing childhood mortality. The model simultaneously addressed the problem of over-dispersion and spatial dependence by the inclusion of the risk factors and random effects in a single model. The proposed approach identified regions with elevated relative risk or clustering of high mortality and evaluated the small scale geographical disparities in sub-populations across the regions. The study identified another challenge in spatial data analysis, which are spatial autocorrelation and model misspecification. The study then fitted geoadditive mixed (GAM) models to analyze childhood anaemia data belonging to a family of exponential distributions (Gaussian, binary and multinomial). The GAM models are extension of generalized linear mixed models by allowing the inclusion of splines for continuous covariate (or time) trends with the parametric function. Lastly, the shared component model originally developed for multiple disease mapping was reviewed and modified to suit the binary data at hand. A multivariate conditional autoregressive (MCAR) model was

developed and applied to jointly analyze three child malnutrition indicators. The approach facilitated the estimation of conditional correlation between the diseases; assess the spatial association with the regions and geographical variation of individual disease prevalence. The spatial analysis presented in this thesis is useful to inform health-care policy and resource allocation. This thesis contributes to methodological applications in life sciences, environmental sciences, public health and agriculture. The present study expands the existing methods and tools for health impact assessment in public health studies.

Acknowledgements

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List of Abbreviations

AIC	Akaike Information Criterion
AOR	Adjusted Odds Ratio
BIC	Bayesian Information Criterion
BYM	Besag, York and Mollie
CAR	Conditional Autoregressive
DHS	Demographic and Health Survey
DIC	Deviance Information Criterion
EB	Empirical Bayes
GAMM	Generalized Additive Mixed Model
GCV	Generalized Cross Validation
GLM	Generalized Linear Model
GLMM	Generalized Linear Mixed Model
INLA	Integrated Nested Laplace Approximation
LBW	Low Birth weight
LL	log-likelihood
mage	mother's age at birth
mbmi	mother's body mass index
MCAR	Multivariate Conditional autoregressive
MCMC	Markov chain Monte Carlo
MRF	Markov Random Field
MSE	Mean Squared Error
NB	Negative Binomial
NDHS	Nigeria Demographic and Health Survey
PCAR	Proper Conditional Autoregressive
P	Poisson
pD	effective number of parameters
PG	Poisson-Gamma
PLN	Poisson-lognormal
RR	Relative Risk
SIR	Standardized Incidence Rate
SMR	Standardized Mortality Ratio
SSA	sub-Saharan Africa
STAR	Structured additive regression
UH	Uncorrelated Heterogeneity
WHO	World Health Organization
UNICEF	United Nations Children's Fund
ZINB	Zero Inflated Negative Binomial
TDHS	Tanzania Demographic and Health Survey

List of Notations and Symbols

σ^2	variance
τ^2	precision
Σ	variance-covariance matrix of multivariate normal variate
Ω	Precision matrix of multivariate response variate
$v_i \sim \mathrm{N}(0, \sigma^2)$	uncorrelated heterogeneity normal
	distribution of a discretize region i
$u_i	u_j$	correlated random spatial distribution
	of a region i given region j
$\boldsymbol{\Phi_i} | \boldsymbol{\Phi}_j$	Multivariate Conditional Autoregressive distribution
	of a region i given region j , where the j are neighbouring regions to i

Chapter 1

Introduction

Nutritional status is defined by Pridmore & Carr-Hill (2009) as the physiological condition of an individual that reflects the balance between nutrient requirements and intake together with the ability of the body to use these nutrients. Ge & Chang (2001) describe malnutrition as a pathological state resulting from in- adequate nutrition, Both overnutrition, that is being overweight or obese due to consumption of excess energy foods and other nutrients, and undernutrition are forms of malnutrition. Undernutrition due to insufficient intake of energy or protein foods or of other nutrients. Therefore even with enough food intake for energy requirements, a child may be considered undernourished if he or she has inadequate micro-nutrients. Deficiency disease arises from insufficient intake of one or more specific nutrients, such as vitamins or minerals, as reported in Ge & Chang (2001).

Undernutrition is pervasive in sub-Saharan Africa, where its prevalence is among the highest in the world. A recent report by Black et al. (2008) indicated that among children under-five years of age in developing countries undernutrition is highly prevalent. They estimate that 178 million children show stunting due to chronic malnutrition. Stunting is indicated when a child's height-for-age Z score is lower than - 2 standard deviations (SD). Similarly, a child is considered underweight if the weight-for-age Z score is lower than - 2 SD. It is estimated that 112 million children are underweight. Wasting, or acute malnutrition afflicts an estimated 55 million children, is indicted by their having a weight-for-height Z score of between -2 SD and -3 SD. In 2011, it was reported that 165 million children were stunted, and 52 million were wasted (UNICEF et al., 2012).

Protein-energy malnutrition and micro-nutrient deficiencies are two major forms of malnutrition that contribute the most to the health burden in developing countries (Soja & Kiran, 2016; Müller & Krawinkel, 2005). Globally, malnutrition in any form,

is a recognized health issue, being the most important causes of illness and deaths in pregnant women, and young children (Müller & Krawinkel, 2005). Marasmus and kwashiorkor are forms of malnutrition due to, respectively, insufficient protein or energy. Micronutrient deficiencies most common in developing countries include insufficiencies of iron, iodine, vitamin A and zinc (Müller & Krawinkel, 2005).

There is evidence that undernutrition erodes the physical and intellectual development in children, and retards their educational attainment (Alderman et al., 2006), which then also affects the health and labour market productivity at adulthood (Haddad et al., 2004; Victora et al., 2008). In other words, long-term consequences of stunting affect adult health and human capital (Victora et al., 2008). Studies have established that severe malnutrition in children arising from fetal growth restriction, stunting, wasting, micro-nutrient deficiencies, and sub-optimal breastfeed leads to high population mortality. Recent studies have established that childhood malnutrition resulting from inadequate food intake of nutrients or from chronic diseases that may lead to mild to moderate anaemia, is a major contributor to under-five mortality (Ehrhardt et al., 2006; Stevens et al., 2013; Alkema et al., 2014).

Furthermore, childhood malnutrition has a direct relationship with disease morbidity and child mortality because it compromises the immune function, increases susceptibility to infectious diseases, hasten the progression and severity and duration of disease, as shown by the findings of Bhutta et al. (2013). Undernutrition in under -five children is also a consequence of poor health, as infectious diseases (e.g. diarrhea, acute respiratory infections, TB and HIV) increase energy requirements but also often reduce appetite and nutrient absorption, as reported in Bhutta et al. (2013) and WHO (2013). Empirical studies reported in De Onis (2017) have shown that poor nutritional status in a child is highly correlated with his or her vulnerability to disease infections, to delayed physical and mental development, and to an increased risk of dying. Although the proportion of under-five children who were underweight is reported to have declined by 36 percent between 1990 and 2011, undernutrition among children under five is nevertheless estimated to be associated with 45% of child deaths worldwide.

According to [1] United Nation Convention on the Rights of the Child to Food and Nutrition reported in Weingärtner (2009). The report emphasized the importance of reducing child malnutrition and mortality as enshrined in the then United Nations Millennium Development Goals (MDGs), which sets target for 50% reduction in the prevalence of being underweight among under-five children between 1990

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[1]United Nation Convention on the Rights of the Child to Food and Nutrition. Article 24(c) requires States parties "to combat disease and malnutrition". The Convention further emphasizes that States parties shall ensure the provision of adequate nutritious food. This was reaffirmed in Article 11 of the ICESCR, which requires States parties to ensure access to nutritionally adequate, culturally appropriate and safe food and to combat malnutrition.

and 2015, while MDG4 targets a two-third reduction in the mortality rate among children under five years of age. The disparities in achieving these goals, which vary from region to region, may hinder the universal progress across the world, particularly in the sub-Saharan Africa. According to (Easterly, 2009) and the poverty index in the Petrou & Kupek (2010) report, high mortality and malnutrition still persists among young children in eastern Asia, whilst the majority of countries in sub-Saharan Africa had made little or non-substantial progress in this area and failed to achieve the target goals by 2015. In 2012, Global Nutrition Targets (WHO GNT) were introduced by the World Health Organization (WHO) member states endorsement of a broader agenda to improve nutrition by 2025, which includes stunting, wasting, low birth weight and overweight in under-five children, as reported in McGuire (2015). The Sustainable Development Goals (2.2), which call for an end to all forms of malnutrition by 2030, mean that attaining such universal progress cannot be separated from many other goals concerning child health ambitions (Forouzanfar et al., 2016; Nilsson et al., 2016).

This study focused on modeling the prevalence of malnutrition among children and child bearing mothers using childbirth outcomes and mortality among under-five children in selected sub-Sahara African countries. It makes use of Bayesian disease mapping in hierarchical modeling with applications to non-Gaussian data. This thesis was motivated by the availability of an enormous amount of geo-referenced database from demographic and health surveys with inherent spatial dependence properties. The output of the findings would be useful to policy makers allocating public health resources, in disease management, designing intervention programmes and to identify the potential risk factors of key nutrition indicators. The study explored spatially aggregated data and sought to review the developments in spatial statistical theory and its applications to understand one of the critical health problems in Africa.

1.1 Statement of Research Problem and Objective

A variety of methods have been developed to deal with spatial data modeling and disease mapping, for both single and multiple diseases response outcomes. The spatial models are based on the use of random effects, which are split into spatial and non-spatial components. The normality assumption in a non-spatial regression model is barely met when data comes from spatially correlated units. Nowadays, huge geo-referenced data resources and complex survey designs exhibit autocorrelation and spatial heterogeneity across the locations or data points, which may

vary in space or time. The statistical challenges are to develop robust and flexible methods that extend the existing models, while at the same time accommodating the complex data structures. The proposed methods relax the restrictive assumptions of the classical regression models concerning linearity and normality. This thesis explored several semi-parametric models for analyzing (lattice) areal data, which vary over space. The aim of this thesis was to develop statistical models for spatially aggregated (areal) data and apply the methods to investigate the child health outcomes extracted from demographic and health survey (DHS) data. The specific objectives for this thesis are:

- To review disease mapping models for single and multiple diseases.

- To develop a flexible model that captures all unobserved factors of regional effects and individual effects and applied to estimate the underlying risk factors of child birth outcomes and anaemia among under-five children.

- To investigate the associations between various socio-demographic, bio-social, and environmental factors and the child mortality rates and geographic distribution.

- Recognizing extra components of variability associated with a small area health survey, the study developed models that incorporate over-dispersion and spatial heterogeneity for under-five mortality in a developing context.

- To construct models which jointly estimate the conditional correlation among multiple malnutrition indicators among under- five children and their spatial association within the region.

1.2 Outlines of the Thesis

This thesis developed techniques for analyzing spatial or spatial-temporal data arising from demographic and health surveys for public health, epidemiology, environmental and ecological studies relating to child health problem. Chapter 1 provided the background about childhood malnutrition and developing techniques in spatial epidemiology. In Chapter 2, relevant literature relating to statistical methods, disease mapping models and spatial epidemiology are reviewed and systematically evaluated. The remaining chapters in this thesis constitute the spatial method, its applications and include material published in high impact journals or conference proceedings.

In Chapter 3, by relaxing the linearity assumptions of the classical statistical models, a semi-parametric model is proposed to investigate socio-demographic factors and

geographic location on the child birth defects in a developing country. A cumulative multinomial regression model was proposed to analyze ordinal data of child birth size. It recognizes child birth weight as an important health parameter for obstetricians and gynecologists and a good health indicator of a child-bearing mother. In survey reporting, the mothers or nurses frequently estimate their infant's birth weight and make a classification in ordinal category (low, normal, large) instead of recording an actual birth weight. The study fitted a Geoadditive logistic regression model to analyze the binary outcome and the polytomous response with different kind of covariates in a unified manner. The fixed effect components of the model are estimated in a parametric manner and the non-linear effect was modeled using penalized P-spline. The spatial component was modeled using conditional autoregressive error. A penalized maximum likelihood estimation was performed to estimate the model parameters. The proposed method was applied to child birth weight data of children born between 2003–2008 obtained from the 2008 Nigeria Demographic and Health Survey (NDHS).

Chapter 4 deals with Poisson counts that are spatially aggregated over regions A Poisson log-linear model with random effects was proposed to investigate the relative risk and the underlying risk factors of mortality among children less than five years. The study applied Bayesian hierarchical spatial models to assess the geographical variation in child death counts over areal units defined by administrative areas (states) or sub-region. A crude standardized mortality ratio (SMR) was first estimated for each state (district) and mapped to assess an elevated relative risk or unusual clusters of low (high) child mortality across the regions in the country. Spatial generalized linear mixed models (GLMMs) formulated from Poisson distributions were proposed to analyze the mortality counts that accommodate the overdispersion and spatial dependence in the data. The commonly used Poisson-gamma model was first constructed to investigate the extra variability in the death counts. We later introduced Poisson log-normal, conditional autoregressive (CAR) and the convolution model known as Besag, York and Mollie (BYM) model. A full Bayesian inference was performed via the Markov chain Monte Carlo computation technique to estimate the model parameters and potential risk factors. The goodness of fit measure by means of the deviance information criterion (DIC) was used to compare the models performance.

In Chapter 5, we discuss three probability distributions for different data struc- ture. The distributions were applied to analyze of anaemia in children less than five years of age. Spatial generalized additive mixed models (GAMM) were formulated and applied on the response variables belonging to a family of exponential distribu tions (Gaussian, binomial and multinomial). GAMMs are extension of generalized linear mixed models (GLMMs) utilized in the previous chapters (3 and 4). The advantage

of GAMMs lies in their use of splines, which is a general term for describing the class of smooth and piecewise polynomials. Given data over a defined over a time period or a metrical covariate, the splines are, in essence, used to capture complicated temporal trends (or continuous variables), which are often missed by a single parametric function.

In the previous chapters, the discussion was focused on one single univariate outcome at a time, so the spatial correlation induced in the case of multiple diseases was neglected. However, two or more diseases may co-vary and share common risk factors or a geographical pattern. Therefore in Chapter 6, the shared component model, originally developed for Poisson count in multiple diseases modeling, is reviewed and modified to suit the binary data at hand for the study. A multivariate conditional auto-regressive (MCAR) model is proposed to analyze, jointly, the three malnutrition indicators among under-five children and the small area geographical variation. The MCAR analysis permits the joint estimation of conditional correlation between the diseases, the spatial association within the region, and the marginal geographical variation in prevalence of each disease across regions or districts. The multivariate approach provides a versatile and robust method to model simultaneously two correlation structures; firstly, between the multiple diseases and, secondly, within the geographical areas through the variance co-variance structure. The MCAR model was constructed via a conditionally induced model that allows estimation of conditional correlation between the diseases, which would have been neglected in a separate univariate analysis.

Chapter 7 presents a summary of the study, highlights the conclusions, makes recommendations and indicates the potential for future research.

Chapter 2

Literature Review

This chapter contains an introductory section in which the relevant literature providing the theoretical basis of this thesis is reviewed. The purpose of this chapter is to provide an overview of the key methodological concepts employed in the research. The chapter is organized as follows. Section 2.1 explains the spatial data and survey data that feature throughout this research. Section 2.2 explains the theory of Markov random fields and describes some related models for areal or lattice data that are used throughout the thesis. Section 2.3 describes the spatial modeling and disease mapping. Statistical models are outlined and the concept of smoothing is also discussed. Section 2.4 describes the Bayesian estimation approach, sampling procedure and shows its relevance in the research.

2.1 Demographic and Health Survey database

The data which motivated this work was acquired from the Demographic and Health Surveys (DHS) programme. The DHS programme (Corsi et al., 2012) is designed to collect and analyze reliable demographic and health data for regional and national family and health planning. The U.S. Agency for International Development (US-AID) provided the funds for the survey data collection and it is implemented by Macro International Inc. The standard procedure of DHS methodology involves collecting complete birth histories from women of child bearing age concerning family planning, reproductive health, maternal health, child survival, and the control and prevention of sexually transmitted diseases such as HIV and AIDS. The data are made available to researcher, who should register and submit a research proposal through the website by sending a request to the MEASURES Program. The

researcher then get permission to download and use the data strictly for intended research purpose (www.measureDHS.com). The strategic objective of MEASURE is to improve and institutionalize its health development activities by supporting data collection, analysis and evaluation. The procedures are designed to improve programme performance and to better understand programme impact in the health sectors of many developing countries. To date, the DHS and related programmes have provided technical assistance for more than 100 surveys in Africa, Asia, the middle-east, Latin America, and the Caribbean.

For the purpose of the present study, the DHS database was assessed and downloaded the needed data relating to children health in some sub-Saharan African countries. The information provided is self-reported by child bearing women of reproductive age between 15-49 years and men aged 15-59 years. The data include information on family planning, household members, home environment, demographic characteristics, all children born, place of delivery, child birth weight, nutritional status, child deaths, distance to health facilities, antenatal care, and skilled health professionals. In addition, the interviewers also recorded geographic information system (GIS) data for the locations (latitude-longitude) of the respondent's place of residence.

2.2 Spatial Epidemiology

Spatial epidemiology, sometimes called geographical epidemiology, is the study of the geographical distribution of mortality or disease incidence and its relationship to potential risk factors. The analysis of such incidence plays a prominent role in the understanding of the aetiology of diseases in public health and epidemiological studies. To efficiently carry out analysis on spatial data, the analyst must account for spatial dependence and extra variability in the population size (Aregay et al., 2017). Spatial epidemiological study can be classified into three broad areas of study, which Elliot et al. (2000) listed as disease mapping, geographical correlation studies and disease clustering in population surveillance.

Spatial data are data which have a certain location in space in relation to their place of collection. Let $\mathbf{s} \in \mathbb{R}^d$ be a data location in a $\mathbf{d}-$dimensional Euclidean space and assume the potential datum $\mathbb{Z}(\sim)$ at spatial location $\mathbf{s}$ is a random quantity. Suppose a random variable s varies over a set of locations, index $\mathbf{D} \subset \mathbb{R}^d$, then a multivariate random field (or random process) is defined by $\left\{ Z(s) : s \in \mathbb{R}^d \right\}$ could be formed as suggested by Cressie (1993). In spatial data analysis, two types of spatial data are encountered namely: point referenced and lattice(areal) data.

- Point referenced data are observations collected at geographical locations, say

s over a fixed continuous space. For example, point level data arises when the exact geographical locations of the individual cases of disease is known, which is expressed in terms of longitude and latitude of the location. Sometimes the point referenced data are described as in the geostatistical data analysis or geocoded data, as detailed by Banerjee et al. (2014). With such data, the research interest is to make inferences on the distribution of health outcome over the entire region under study and the analyses are based on the information collected at location, s. Such analyses do account for spatial dependence as a function of distance between pairs of locations in $\mathbb{R}^d$, and such measurements are facilitated by the availability of Geographic Information Systems (GIS) tools. The information that captures the precise individual location (place) through the use of point referenced data are rarely used for disease mapping. In particular, the confidentiality issues, which surrounds the regulations of individual health outcomes (Jin et al., 2005).

- The second level of spatial data is the areal or lattice data. The areal type observation are data measured on a collection of subsets of points in a region $\mathbb{R}^d$, which is spatially aggregated over the regions formed. The areal data come from either irregular or regular areas. A location which consists of a set of regularly spaced points are called regular lattice, and such data are used in remote sensing from satellite observation of earth images, known as pixels. The second category of areal data are called irregular lattice and they do neither follow a predictable pattern nor precise geometrically connections. The irregular lattice data are in the forms of administrative units, local government area, district, counties, regions, states etc and they are commonly utilized in disease mapping. The geographical distribution of disease summary can be modelled at aggregated district level, administrative units or any sub-national level are relatively easy to obtain than the individual health outcome for the sake of confidentiality issues. Disease mapping is an important subtopic in spatial epidemiology usually conducted at aggregated areal unit, especially with sensitive health outcome like HIV/AIDS. Recent studies have adopted the setting as proposed in the work of Elliot et al. (2000), which is popularly used for disease mapping.

Disease mapping provides a linkage between two other areas of spatial epidemiology: small area estimation and ecological-spatial regression. Disease maps as described by Banerjee (2016) are often useful to identify spatial clusters of unusual high and low incidence, generate hypotheses regarding common underlying environmental, demographic, or cultural factors shared by the neighboring regions. In

spatial data analysis of small area-specific level, it is important to take into account unobserved hetereogeniety and spatial dependence in the statistical model. Ignoring autocorrelation in spatial analysis can lead to underestimation of the model parameters and as such leading to erroneous conclusions about the hypothesis (Wakefield & Elliott, 1999; Waldhoer et al., 2008). In addition, another prominent challenges frequently encountered in small area estimation include data sparsity, which often arises from a large population size, over-dispersion, and measurement errors (Beale et al., 2008; Hampton et al., 2011; Caprarelli & Fletcher, 2014).

Over the years, random effect models are commonly formulated as a means of modeling the spatial autocorrelation and heterogeneity in disease mapping models. In a simple representation, a random effect, ϕ_i, represents a random effect specific to area i, which is used to account for unexplained spatial autocorrelation. For a set of random effects, $\phi = (\phi_1, \phi_2, \ldots, \phi_n)$ are commonly represnted by conditional autogregresive (CAR) prior distribution first proposed by Besag (1974). The CAR prior introduced by Besag (1974) and later extended by Besag et al. (1991), called the intrinsic CAR (ICAR) prior, is defined by the set of conditional distributions as

$$\phi_i | \phi_j, j \neq i, {\sigma_\phi}^2 \backsim N \left(\sum_{j \neq i} \frac{w_{ij}\phi_j}{w_{ij}}, \frac{{\sigma_\phi}^2}{w_{ij}} \right) \tag{2.1}$$

The CAR priors can also be modeled via a multivariate normal distribution defined as

$$\phi \sim \mathrm{N}(0, \Sigma), \text{with } \Sigma = \sigma^2 (D - \mathbf{W})^{-1} \tag{2.2}$$

with a covariance function, Σ that reflects the spatial correlation between the random effects. Besag et al. (1991) later proposed intrinsic CAR (ICAR) prior with covariance matrix, $\Sigma = \sigma^2 (D - \mathbf{W})^{-1}$, where σ is a conditional variance parameter and D and W are matrices determined by the neighbourhood structure of the data. In equation (2.1) above, $i \sim j$ denotes region i and j sharing a common boundary and ϕ_{-i} represents the random effects of all regions excluding region i, and w_{ij} is the element of a spatial weights matrix $\mathbf{W}$ corresponding to row i and column j in matrix 2.1b and ${\sigma_\phi}^2$ is the variance parameter that controls the amount of spatial smoothness as reported in Rue & Held (2005) and Banerjee et al. (2014).

In the case of areal data analysis, the producure to evaluate the spatial dependence is by the introduction of conditional autoregressive (CAR) prior error, such methods and applications can be found in Li & Lin (2006) and Waller & Gotway (2004). One popular strategy to capture the spatial dependence is by utilizing the variance-covariace matrix structure to model each element of the correlation matrix as a fuc-

tion of distance. For example, in Best et al. (2005), the variance-covariace matrix is expressed $\Sigma_{u(i,j)} = \exp(-d_{i,j}\phi)$, where $d_{i,j}$ measures the distance between the centroids in region i and j and ϕ represents a common parameter describing the overall extent of spatial dependence involved. Other procedure for estimating spatial correlation in analysing geostatistical data can be found in Cressie (1993) and Leroux et al. (2000).

Furthermore, the extent of spatial correlation may be induced in the the response may be determined by the smoothensss arising from the ommited factors, which may vary from acrosss the regions. Clayton & Kaldor (1987) extended the use of mixed models for geographical data to account for the extra-Poisson variability through the introduction of random effects, where the random effects are often spatially correlated in a disease mapping context.

The autocorrelation and overdispersion arising from the population sizes and spatial dependence are re-occurring challenges in public health and survey data, especially when rare diseases are involved. The sparsity of population usual result in large variability in the estimated mortality rates or disease incidence, thus masking the true variability from the potential underlying factors. To handle the true variability (spatial risk ensemble), several approaches have been proposed in the form of mixed effect models. One of the popular method is spatially varying random effects, which explores the borrowing strength from the neighbouring regions to mitigate the effect of sparsely populated regions and produce a better inference, for more readings, see Banerjee et al. (2014); Lawson (2013) and Leroux et al. (2000).

Disease mapping of mortality rates is very useful to epidemiologist, and public health. The use of crude mortality rates or disease incidence to estimate rare disease risk in small areas such as administrative units, health centers and ward levels, is problematic as the approach does not account for the high variability in population sizes over the regions nor account for spatial variation of the regions understudy. More so, the interpretation of spatial distribution of the disease or mortality rates are also misleading. Bayesian inference provides an alternative way to produce a standardized smooth maps by borrowing information from the neighboring areas across the regions. The early developments of disease mapping models include the use of empirical Bayes (EB) techniques discussed by several authors among them are well documented in Clayton & Kaldor (1987); Clayton (1992); Mollié (1999) and Leyland & Davies (2005) to estimate model parameters and plug-in approximations for the statistical inference. These eventually yielded unbiased estimates of the relative risk, but underestimate the variance. However the EB approach does not account for uncertainty resulting from the hyper-parameters.

Recently, the full Bayesian (FB) approach has gained prominence in the field of dis-

ease mapping and modeling. Inference is based on Markov Chain Monte Carlo(MCMC) simulation techniques due to their pioneering work by Besag et al. (1991) commonly referred to as the BYM, and subsequent applications by Bernardinelli & Montomoli (1992); Ugarte et al. (2009); Catelan et al. (2010) and Congdon (2014). Their proposal was to model the relative risks of each region by combination of fixed and random effects. Under CAR, the conditional distribution could be modeled via neighborhood structure where the random effects in a region given all the other regions is simply taken as the weighted average of all the other random effects. Besag et al. (1991) considered the weights based on neighboring structure on adjacent areas, where regions sharing a common boundary were assigned a weight matrix of 1 and 0 otherwise. Gosoniu et al. (2010) extended their model by including both spatially unstructured random effects and spatially structured random effects through the convolution model. Research has shown that the fully Bayesian model produced better estimates than the EB as reported in Moura & Migon (2002) and Gosoniu et al. (2010). Bernardinelli & Montomoli (1992) compared the EB methods with the fully Bayesian methods and concluded that the fully Bayesian approach offers greater flexibility and better estimates of the credible regions of geographical distribtion for disease rates. There is a vast amount of literature on the development and application of disease mapping techniques among them Besag et al. (1991); Torabi & Rosychuk (2012) and Best et al. (2005).

Other previous studies on modeling count data have seen considerable development on space-time models. Spatial and spatio-temporal disease mapping models are common tools for the estimation of the risk of disease, identifying regions and periods with high risk and time trends for disease incidence or prevalence. Bernardinelli & Montomoli (1992) approach is one of the first for spatio-temporal models for count data. They assumed a Poisson GLM with a linear predictor containing separate terms for space and time as well as a space-time interaction effect which allows for different temporal trend in different in different areas or regions. Waller et al. (1997) proposed a spatio-temporal model which is an extension of the BYM or the convolution model due to Besag et al. (1991). In their study, Waller et al. (1997) assumed that the covariate effects are constant over time and the disease counts followed a Poisson distribution. They treated each year as a separate time period and estimated the overall trend of increasing lung cancer deaths by incorporating both spatial clustering and uncorrelated heterogeneity. Knorr-Held & Best (2001) proposed spatio-temporal model consisting of a pair of area-specific random effects, that included both unstructured and structured random effects via convolution prior on space and also a similar prior for temporal trends to describe the spatial and spatio-temporal variation.

Traditionally, Markov random fields (MRF) have been successful used in a non-

hierarchical setting as a direct approach for the observed data, but not as a model for unobserved parameter such has been used in spatial context of Besag (1974) and Künsch (1987) nor the approach which corresponds to what is known as state-space models in the temporal domain in the work by Harvey & Stock (1989). The Markovian property facilitates the estimation of high dimensional models than it is usually done using general multivariate Gaussian models.

Markov random fields belongs to the class of parametric models which utilizes the Markov property theory. MRFs play a pivotal role in the development of statistical methodology for analyzing spatial data. For detail, see Clifford (1990) and Haran (2011), and its importance for disease mapping models. Sometimes, a situation may occur in which a location includes all other locations as neighbours, in this case, it is not a MRF, the situation is known as 'saturated' neighbourhood structure. Such neighbourhood structure that assumes a random field approach instead a MRF for spatial modeling are discussed in details, in Besag & Kooperberg (1995); Cressie & Verzelen (2008) and Wolpert & Ickstadt (1998). The neighbourhood structure can
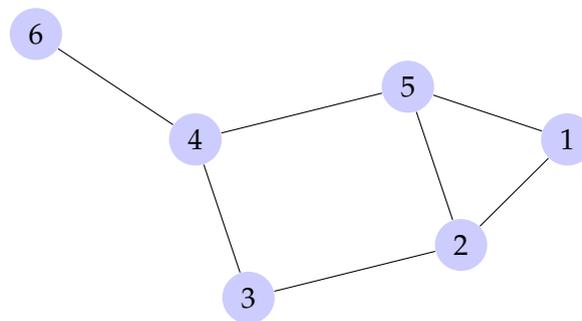
(a) Relationship between nodes in an undirected graph

$$\mathbf{W}=$$

$$\begin{bmatrix} 0 & 1 & 0 & 0 & 1 & 0 \\ 1 & 0 & 1 & 0 & 1 & 0 \\ 0 & 1 & 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 & 1 & 1 \\ 1 & 1 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \end{bmatrix}$$

(b) Adjacency matrix consisting of of six (6) nodes

take two popular forms of representation: as a finite graph displayed in Figure 2.1a with six nodes or as an adjacency matrix, $\mathbb{W}$. Figure 2.1a shows an example of a simple undirected graph and a corresponding adjacency matrix, displayed in Figure 2.1b. According to (Assunção & Krainski, 2009), the neighbourhood matrix, as displed in Figure(2.1b) is defined as a $n \times n$ matrix, which contains diagonal elements of $\mathbf{W}$ of zeros and off-diagonal element $w_{ij} = 1$ if regions i and j share a common

boundary and zero otherwise.

Alternatively, a *first-order* neighbourhood can also be contructed for each region and the adjacency matrix is defined by a symmetric representation of the neighbourhood arrangement (Harary, 1962). For more complex models, higher-order neighbourhood configuration are defined in the literature. For example, a good number of different neighborhood strutures and guidelines have been investigated and techniques are documented in Earnest et al. (2007) and Stakhovych & Bijmolt (2009). From the Hammersley–Clifford Theorem and Brook's Lemma, for details see Besag & Kooperberg (1995); White & Ghosh (2009),and Banerjee et al. (2014), which states that the conditional prior distribution in equation (2.1) can be written jointly as a prior distribution of ϕ as Gaussian Markov Random Field (GRMF) defined by

$$\phi \backsim N_m \left(\mathbf{0}, \frac{1}{\sigma_\phi^2}(\mathbf{B} - \mathbf{W})^{-1} \right) \tag{2.3}$$

where $\mathbf{B}$ is defined as a diagonal matrix with $B_{ii} = m_i$. Re-parameterizing in term of covariance matrix, Leroux et al. (2000) replaced $(\mathbf{B} - \mathbf{W})$ with an equivalent matrix, $\mathbf{R}$ with element satisfying

$$R_{ij} = \begin{cases} 1 & \text{if } i = j \\ -w_{ij} & i \neq j \end{cases} \tag{2.4}$$

Compared to the joint modeling approach in equation (2.3), it is obvious that the CAR model in (2.1) would enhance a more efficient sampling of each ϕ_i, as it becomes amenable to Bayesian estimation via Markov Chain Monte Carlo (MCMC) simulation technique.

A close examination of equations (2.1) and (2.3), it indicates that covariance matrix, $(\mathbf{B}-\mathbf{W})$ is singular, meaning it cannot be inverted. Though a full conditional of each ϕ_i can result into proper prior, but the joint distribution of the covariance matrix in equation (2.3) does not exit, leading to impropriety of the joint prior. In order to salvage the problem of improper prior, one can add a constant, (say, c) to each element in ϕ, and then the joint probability density, $p(\phi)$ becomes unaffected. The equation (2.3) can therefore be re-written as a pairwise difference representation defined by

$$p(\phi) \propto \exp \left(-\frac{1}{2\sigma_\phi^2} \sum_{i \neq j} (\phi_i - \phi_j)^2 \right) \tag{2.5}$$

Doing so, the elements in ϕ tends to be determined uniquely to a constant. Invariably, the remedies provided are either imposition of a sum-to-zero constraint, i.e.

$\sum_i \phi_{\mathbf{i}}^{\mathbf{m}} = 0$ over the ϕ_i's or the exclusion of intercept from the full model. Alternatively, Cressie (1993) provided a proper CAR model in terms of the neighborhood matrix, $\mathbf{W}$ for equation (2.3) by multiplying by a constant, α, making the matrix, $(\mathbf{B} - \alpha \mathbf{W})$ non-singular.

2.3   Bayesian Inference

From a Bayesian framework, there is no fundamental distinction between observations and parameters of a statistical model: all are considered random quantities. Let y denote the observed data, and θ denote the model parameters and missing data. Unlike the classical statistical inference which summarizes θ with a point estimate, Bayesian inference provides a distribution of plausible values for the parameter given the observed data (Carlin et al., 2010). The distribution which describes the the parameters after the data is observed is called posterior distribution and it is obtained as the product of the likelihood and the prior distribution through Bayes theorem (Banerjee et al., 2014). The posterior distribution can be expressed as:

$$p(\theta|y) = \frac{p(y|\theta)p(\theta)}{p(y)} = \frac{P(y|\theta)p(\theta)}{\int p(y|\theta)p(\theta)\,d\theta} \tag{2.6}$$

where $p(\theta)$ is the prior distribution of the parameter, and $p(y)$ is the marginal distribution of y, given as

$$p(y) = \int p(y|\theta)p(\theta)\,d(\theta) \tag{2.7}$$

$p(y)$ does not depend on observed model parameters. The denominator in equation (2.6) i.e $\int p(y|\theta)p(\theta)\,d(\theta)$ is called the normalizing constant equal to the marginal pdf of y . The likelihood can be expressed as the product of individual contributions, as follows:

$$p(\boldsymbol{y}|\boldsymbol{\theta}) = \prod_{i=1}^{n} p(y_i|\boldsymbol{\theta}) \tag{2.8}$$

The posterior distribution in equation (2.6) can be specified in proportional terms and mathematically expressed as:

$$p(\theta|y) \propto p(y|\theta)p(\theta|\lambda) \tag{2.9}$$

Thus, the expression in quation (2.9) is often treated as the un-normalized posterior distribution, see Bernardo et al. (2008) and Polson et al. (2013) for more details.

The integration in equation (2.7) to determine $P(y)$ does not have to be a closed form, since the posterior distribution is estimated using Markov Chain Monte Carlos (MCMC) simulation.

It is common practice to treat λ as unknown and the posterior distribution as intractable. The method of dealing with these unknown quantities determines the type of Bayesian approach. Empirical Bayes can be used to replace λ in the right hand side of equation (2.9). In other hands, a full Bayesian approach can be applied to estimate the unknown information prior and conditional distributions via the marginal distributions of the observations, detialed techique can found in the literature, see Ghosh et al. (2006) and Carlin et al. (2010) for more readings.

However, the variance of empirical Bayes estimators are known to be too small such that they do not account for additional variability to the parameter estimate in hierarchical modeling as detailed in Carlin et al. (2010) and Berger et al. (1994). More so, for the Bayesian approach adjust to variance estimates, the full Bayesian methods are recommended to guarantee convergence and adequate representation of the distribution of underlying rates and details are documented in Ghosh et al. (1999) and Lawson (2013). In the disease mapping nomenclature, this model occurs when the data likelihood is Poisson and there is a common relative risk parameter with a single gamma prior distribution as explained in Lesaffre & Lawson (2012).

The theory of the MCMC algorithm is commonly used to perform posterior inference in the case where the product of the likelihood and the prior are analytically intractable. By considering conditional independence between the parameters, the posterior distribution in equation (2.6) can be rewritten as

$$p(\boldsymbol{\theta}|\boldsymbol{y}) \propto p(\boldsymbol{y}|\boldsymbol{\theta})p(\theta_1|\theta_{\searrow 1})\ldots\ldots p(\theta_T|\theta_{\searrow T})$$

where T is the total number of parameters in the model including the hyper-parameters. The marginal posterior distribution is useful in order to make inference about each parameter. Thus the marginal posterior distribution for the tth parameter is obtained by integrating out the other parameters..

$$p(\theta_t|y) = \int \ldots \int p(\theta_1, \ldots, \theta_g, \ldots \theta_T|\boldsymbol{y}) \, d(\boldsymbol{\theta}_{\searrow t}) \tag{2.10}$$

$$= \int \ldots \int p(\theta_t|\boldsymbol{\theta}_{\searrow t}p(\boldsymbol{\theta}_{\searrow t}) \tag{2.11}$$

The integration needed to compute $p(\theta_t|y)$ is often impracticable according to Banerjee et al. (2014) and Gilks et al. (1995). This method involves a set of procedures which use iterative simulation of parameter values from the posterior distribution

via a Markov Chain (MC) approach.

The MCMC techniques enable the analyst to estimate the parameter of highly complicated models corresponding to the posterior distribution with high accuracy. This method has greatly contributed to the development, advancement and propagation of Bayesian theory. Gómez-Rubio & Rue (2018) stated that the MCMC is a generic algorithm that ensures the Markov Chain converges eventually to the target (stationary) distribution and under mild assumptions, see also Besag (1974) and Cressie (1993) for more discussion. A Markov chain is a stochastic process in which future states are independent of the past given the present states and the chain converges to a stationary distribution which is the unnormalized posterior distribution as desired was discussed in Geman & Geman (1984) and Christensen & Meyer (2001). This process is defined by $\theta^{(1)}, \theta^{(2)}, \ldots, \theta^{(T)}$ such that $P(\theta^{(t+1)}|\theta^{(t)}, \ldots, \theta^{(1)}) = P(\theta^{(t+1)}|\theta^{(t)})$. That is, the distribution of θ at time $t + 1$ given all the preceding θ values (for $t, t - 1, \ldots, 1$) depends only on the value $\theta^{(t)}$ of the previous sequence t.

As $t \to \infty$, the distribution of $\theta^{(t)}$ converges to its equilibrium, which is independent of the initial of the chain $\theta^{(0)}$. This condition occurs, when the MC is irreducible, aperiodic and positive- recurrent as stated in Roberts et al. (2004) and Gelfand (2000). The MCMC sampling method for constructing the Markov chain is summarized in the following steps:

1. Select an initial value $\theta^{(0)}$

2. Sequentially generate $\theta^{(t+1)}|\theta^{(t)}$ values from $P(\theta|y)$ until the equilibrium distribution is reached.

3. Monitor the converge of the algorithm using the convergence diagnostics. If convergence diagnostics fails, we generate more samples.

4. Cut off the first B observations. B is called the burn-in period meaning the first B iteration values are eliminated from the sample to avoid the influence of initial values.

5. Consider $\left\{\theta^{(B+1)}, \theta^{(B+2)}, \ldots, \theta^{(T)}\right\}$ as the sample for the posterior analysis.

6. Plot the posterior distribution.

7. Finally, obtain summary results of the posterior distribution.

There are other alternative MCMC techniques such as the Gibbs sampler algorithms developed by Geman & Geman (1984) and later generalized by Gelfand (2000), slice sampling algorithm due to Brooks et al. (2011) and Walker (2007) can also be used. MCMC sampling simulations are routinely performed with software such as R programming software (Team, 2014) and WinBUGS (Lunn et al., 2000).

Chapter 3

Semiparametric Multinomial Ordinal Model to Analyze Spatial Patterns of Child Birth Weight in Nigeria

A child's birth weight is the result of fetal growth and a good predictor of infant morbidity and mortality. Birth weight, according to Bassler et al. (2009), is a strong indicator of not only the mother's health but also of a newborn's chances of survival, growth, long term health and psychological development. In the last three decades, there has been increasing evidence that low birth weight and pregnancy complications are independently associated with the increased risk of mortality and early morbidity in babies, and poor maternal health outcomes (Goldstein, 1981; Rees et al., 1996; Madsen et al., 2010). Black et al. (2016) give an estimate of 303,000 maternal deaths occurred in 2015 worldwide due to complications in pregnancy or childbirth, and 5.9 million deaths among children of under five years age; of which 45% occurred within the first 28 days of life.

For a simple epidemiological interpretation, low birth weight (LBW) is defined as a birth weight of less than 2500 g. As UNICEF (2008) reports, LBW is a significant health problem in many parts of the world due to its immediate and long-term consequences. Reports by Villar & Belizan (1982) and Villar et al. (2006) have shown that

about one-half of the LBW outcomes in industrialized countries are due to infants being born preterm (< 37 weeks gestation), while, in developing countries, most LWB infants have been affected by restricted intrauterine growth that may have begun early in pregnancy. A number of studies have investigated the impact of maternal socio-economic characteristics on their newborns' birth weight. However, there is scanty research work that investigates, jointly, the influence of the geographical variations in mothers location and other underlying determinants of birth weight. In epidemiology and social sciences research, generalized linear models (GLM) (McCulloch, 1997) are commonly applied to investigate the influence of underlying factors on the response variable of interest. But, in these cases, the values of dependent variables are qualitative categories (e.g. small, normal, large) and some covariates are not necessarily linear functions of the response variables. Therefore the classical regression model is technically inappropriate, and even ordinary logistic regression may yield biased estimates. A few of these studies on determinants of child birth weight have employed linear and logistic regression models in studying the association between air pollution and birth weight (Currie et al., 2009; Proietti et al., 2013; Charnigo et al., 2010). For example, Uthman (2008b) recently used nonlinear functions to directly describe the relationship between birth weight and infant mortality in Nigeria. Of recent, generalized additive models (GAMs) and their extension have been successfully applied in many fields of study, see Khatab & Fahrmeir (2009) and Sapra (2013) for more discussion.

This study therefore proposes a structured additive regression model to examine the influence of different kinds of covariates on a categorical response variable. The motivation behind this study is the utility of the approach in analyzing a multivariate natural ordered response, while simultaneously handling covariates of different types in a unified framework. We also explore a small area estimation of the spatial residual effects in child birth weight that are not captured by the underlying factors, and which would have been neglected in the classical regression approach.

This chapter is structured as five sections. In Section 3.1, we discuss the data in terms of sampling procedure and statistical methods. The models to be used are outlined in Sections 3.2 and 3.3. Section 3.4 presents the results of the data analysis, while Section 3.5 is a discussion of the results. In Section 3.6, the concluding remarks are given.

3.1 The Data

For data at national household-level on fertility, family planning and children's nutritional status the demographic and Health Surveys (DHS) are reliable sources. The

2008 Nigeria DHS (NDHS) was designed to provide, among other things, information on maternal health, prenatal and postnatal care, attitudes and micronutrient supplementation during pregnancy. The survey covers a representative national sample of women of reproductive age (15-49 years) and it includes data on children's weights at birth and up 59 months . Other variables relate to levels and trends in fertility; sexual partners; fertility preferences; awareness and use of family planning methods; infant's and young children's feeding practices; nutritional status of mothers and childhood mortality. As noted earlier, maternal health outcomes such as child birth weight play a vital role in understanding the epidemiology of public health problems, children's growth restriction, infant mortality trend and childhood survival. The detailed information about the sampling techniques of the NDHS survey have been published in the final report of National Population Commission (2009).

Nigeria is made up of six geopolitical zones, which when subdivided at the secondary administrative level give overall 36 states and the Federal Capital Territory (FCT) . The states and FCT are further subdivided into local government areas. Figure 3.1 displays the map of Nigeria showing the states grouped into geopolitical zones. NDHS-2008 utilizes a two-stage probability sampling method. At the first stage, 888 clusters, consisting of 286 urban and 602 rural areas, were selected from the sampling frame as used during the 2006 National Population Census (NPC). The primary sampling unit, is defined by an enumeration area (EA) for the 2006 NPC, and known as a cluster in the 2008 NDHS. The sample frame of households in each selected cluster was obtained and the households were randomly drawn for interviewing. At the second stage, a sample representative consisting of 36,800 households was randomly drawn for an interview with a minimum target of 950 interviews per state, in at least 41 clusters. In each state, the number of households selected for interview was proportionally distributed among urban and rural areas. Interviews were conducted for 33 385 women aged 15-49 and 15 486 men aged 15-9, resulting in a total response rate of 97% interviewed. This means over two-thirds of the respondents were women.
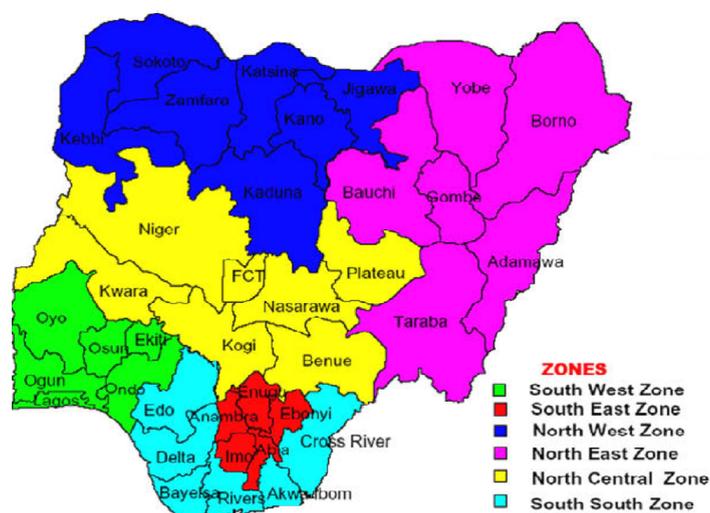
Figure 3.1: Map of Nigeria showing 36 states (districts) and Federal Capital Territory (FCT) by geopolitical zones.

The selection of explanatory variables was done on the basis of previous empirical studies such as by Uthman (2008b) and Frederick et al. (2008) among others. In the original 2008 NDHS, the mother's estimation of the size of her baby at birth was asked for children born in five years before the survey. The birth size was given in five scale categories: very small, small, average, larger than average, and very large. In this report, very small and small are combined into one category to represent the detrimental birth outcome. The other three categories are considered non-detrimental. The response variable is constructed by categorizing the child birth size into four-ways by (low, average (normal), large, very large) and the percentage distribution presented in the Table 3.1. This type of categorization is consistent with previous reports by Rutstein (2000) and Wardlaw (2004). The percentages computed for the four-way birth category were low size, 4239 (14.82%), larger than average 7852 (27.4%), very much larger than average, 5160 (18%) and normal size 10,732 (37.5%). The total number of births in 2008 NDHS classified by sizes was 27,983 (97%) children out of total live births of 28,647 births.

The categorical covariates are grouped into two broad categories according to birth sizes. The bivariate analysis that used χ^2-statistic test is presented in Table 3.2. The variables are derived from child and mother characteristics, as well as environmental factors, e.g., place of residence and childhood diseases (diarrhea, fever, cough) and nutrition status of the child, for further analysis. The analysis showed that all the covariate groups differ significantly in their proportional distribution in the two-way classification of birth size.

Table 3.1: Frequency distribution of child birth weights by size category in the 2008 Nigeria Demographic and Health Survey (NDHS).

Birth Size	Birth Weight Intervals (in kg)	Response	Frequency	Percent
Low	<2.5	1	4239	14.82
Average	2.5–3.2	2	10,732	37.46
Large	3.3–4.0	3	7852	27.41
Very large	>4.0	4	5160	18.11

A four-way classification of the birth weight distribution was presented in Table 3.3. The proportion of low weight deliveries exhibits an inverse relationship with the birth order 1–4, except order 5 (5 + order). Further, the low birth deliveries also decline linearly with rise in maternal educational levels. Further, the low birth weight also shows an inverse linear relationship with increase in household wealth index. In other words, the household wealth index can be expressed as an inverse association with low birth weight. It is also noteworthy that there are differential variations in patterns across the six geopolitical zones. The highest prevalence of low birth weights were recorded in the North East and North West regions of the country. Table 3.3 presents the summary of the descriptive analysis of social and demographic characteristics of the households interviewed in the survey , which is categorized into four-way child birth size. The percentage distribution in Table 3.3 is similar to Table 3.2 and includes other categories. Analysis in Table 3.3 shows that a higher proportion of the women did not attend antenatal sessions. Also, the proportion of women, who did not attend antenatal checks were higher than those who attended, which cuts across the four birth strata. About half of these women (48%) received iron syrup supplementation during pregnancy. Of these women, 77 percent of the children they delivered (within the last five year) did not receive postnatal vitamin A supplementation. The analysis further showed that a substantial percentage of the children who were born with low birth weight also suffered from childhood undernutrition (such as stunting and wasting). The percentage of malnourished children declines linearly as the birth size increases. The descriptive statistics in Table 3.4 also includes mean and standard deviations of some metrical covariates, considered in the multivariate analysis.

Table 3.2: Frequency distribution of some categorical covariates by Two-way classification and Bivariate analysis based on 2008 Nigeria DHS.

	Low Size	Normal Size	
	$cbw < 2500$ n(%)	$cbw \geq 2500$ (n%)	*p*-Value
North Central	463 (22.5)	4534 (17.3)	<0.001 **
North East	306 (14.8)	6197 (23.7)	
North West	367 (17.8)	7528 (28.7)	
South East	221 (10.7)	2191 (8.3)	
South South	269 (13.1)	2974 (11.3)	
South West	434 (21.1)	2850 (10.8)	
Child's sex			
Female	1977 (47.0)	12,302 (52.0)	<0.001 **
Male	2262 (53.0)	11,442 (48.0)	
Residence			
Rural	3320 (78.0)	17,231 (73.0)	<0.001 **
Urban	919 (22.0)	6513 (27.0)	
Birth Types			
Single	4005 (94.5)	23,068 (97.0)	<0.001**
Twin	234 (5.5)	676 (2.8)	
Birth order			
1st order	803 (19.0)	4412 (19.0)	<0.001 **
2nd order	660 (16.0)	4195 (18.0)	
3rd order	586 (14.0)	3703 (16.0)	
4th order	523 (12.0)	3111 (13.0)	
$\geq$5th order	1667 (39.0)	8323 (35.0)	
Mother education			
Incomplete Primary	2772 (65.4)	11,275 (47.0)	<0.001 **
Primary	765 (18.0)	5646 (23.8)	
Secondary	591 (13.9)	5617 (23.7)	
High	111 (2.6)	1206 (5.1)	
Mother employed			
No	1522 (36.0)	7279 (31.0)	<0.001 **
Yes	1813 (43.0)	11,773 (50.0)	

Table 3.2: *Cont.*

	Low Size	Normal Size	
	$cbw < 2500g$ n(%)	$cbw \geq 2500$ g n(%)	*p*-Value
Maternal wealth Index			
Poorest	1490 (35.1)	5982 (25.2)	<0.001 **
Poor	1103 (26.0)	5584 (23.5)	
Average	756 (17.8)	4684 (19.7)	
Rich	529 (12.5)	4123 (17.4)	
Richest	361 (8.5)	3371 (14.2)	
Diarrhea			
No	3079 (72.0)	18,967 (79.9)	<0.001 **
Yes	512(12.1)	2108 (8.9)	
Fever			
No	2988 (70.0)	17,745 (75.0)	<0.001 **
Yes	604 (14.0)	3317 (14.0)	
Cough			
No	3089 (73.0)	18,615 (78.0)	<0.001 **
Yes	502 (12.0)	2420 (10.0)	
Vitamin A Supp.			
No	2219 (52.0)	11,257 (47.0)	<0.001 **
Yes	433 (10.0)	3547 (15.0)	
Antenatal			
No	1656 (39.0)	6683 (28.0)	<0.001 **
Yes	1003 (24.0)	8224 (35.0)	
Stunted			
No	1448 (34.0)	10,125 (43.0)	<0.001 **
Yes	1217 (29.0)	6026 (25.0)	
Wasted			
No	1639 (39.0)	11,764 (50.0)	<0.001 **
Yes	1026 (24.0)	4387 (18.0)	
Underweight			
No	2230 (52.0)	14,142 (59.0)	<0.001 **
Yes	435 (10.0)	2011 (8.5)	
Iron			
No	1656 (39.0)	6683 (28.0)	<0.001 **
Yes	1003 (24.0)	8224 (35.0)	

The *p*-value marked with ** indicates that the variable was significant at 1% level. All *p*-values correspond to Pearson Chi-square test of contingency.

Table 3.3: Descriptive summary of the socio-demographic characteristics (covariates) used in the model by four-way category of birth size in 2008 NDHS.

| Covariates | Four-Way Category of Child Birth Weight | | | | |
	Low n (%)	Average n (%)	Large n (%)	Very Large n (%)	Total N
Zones					
North Central (NC)	772 (18.2)	1638 (15.3)	1286 (16.4)	1220 (23.6)	5046
North East (NE)	1255 (29.6)	2670 (24.9)	1467 (18.7)	1062 (20.6)	6559
North West (NW)	1270 (30.0)	2744 (25.6)	2343 (29.8)	1372 (26.6)	7947
South East (SE)	293 (6.9)	1147 (10.7)	665 (8.5)	281 (5.4)	2450
South South (SS)	292 (6.9)	1389 (12.9)	934 (11.9)	629 (12.2)	3327
South West (SW)	357 (8.4)	1144 (10.7)	1157 (14.7)	596 (14.6)	3318
Place of residence					
Rural	3320 (78.0)	7825 (73.0)	5645 (72.0)	3761 (73.0)	21,034
Urban	919 (22.0)	2907 (27.0)	2207 (28.0)	1399 (27.0)	7613
Sex of child					
Male	1977 (46.6)	5373 (50.1)	4083 (52.0)	2846 (55.2)	14,604
Female	2262 (53.4)	5359 (49.9)	3769 (48.0)	2314 (44.8)	14,043
Child birth type					
Singleton	5064 (98.1)	4005 (94.5)	10,364 (96.6)	7640 (97.3)	27,685
Twin	96 (1.9)	234 (5.5)	368 (3.4)	212 (2.7)	962
Malaria drug during pregnancy					
No	1656 (39.0)	3115 (29.0)	2180 (28.0)	1388 (27.0)	8420
Yes	1003 (24.0)	3479 (32.0)	2775 (35.0)	1970 (38.0)	9295
Child birth order					
1st order	803 (19.0)	1975 (18.0)	1495 (19.0)	942 (18.0)	5353
2nd order	660 (16.0)	1901 (18.0)	1406 (18.0)	888 (17.0)	4969
3rd order	586 (14.0)	1670 (16.0)	1219 (16.0)	814 (16.0)	4388
4th order	523 (12.0)	1426 (13.0)	1005 (13.0)	680 (13.0)	3712
5th order	1667 (39.0)	3760 (35.0)	2727 (35.0)	1836 (36.0)	10,225
Child Spacing (within 3 years)					
<2 child	3305 (78.0)	8572 (80.0)	6413 (82.0)	4209 (82.0)	22,950
≥2	934 (22.0)	2160 (20.0)	1439 (18.0)	951 (18.0)	5697
Wealth Index					
Poorest	1490 (35.1)	2971 (27.7)	1913 (24.4)	1098 (21.3)	7604
poor	1103 (26.0)	2581 (24.0)	1810 (23.1)	1193 (23.1)	6871
Middle	756 (17.8)	2002 (18.7)	1502 (19.1)	1180 (22.9)	5609
Richer	529 (12.5)	1793 (16.7)	1409 (17.9)	921 (17.8)	4755
Richest	361 (8.5)	1385 (12.9)	1218 (15.5)	768 (14.9)	3808
Under-nutrition					
Stunting					
Not	1448 (34.2)	4442 (41.4)	3300 (42.0)	2383 (46.2)	11,747
Yes	1217 (28.7)	2750 (25.6)	2010 (25.5)	1266 (24.5)	7356

Table 3.3: *Cont.*

Covariates	Low N (%)	Average N (%)	Large N (%)	Very Large N (%)	Total N
Wasting					
Not	2831 (54.9)	1639 (38.7)	5064 (47.2)	3869 (49.3)	13,616
Yes	818 (15.9)	1026 (24.2)	2128 (19.8)	1441 (18.4)	5487
Underweight					
Not	2230 (52.6)	6196 (57.7)	4672 (59.3)	3274 (63.4)	16,630
Yes	435 (10.3)	997 (9.3)	639 (8.1)	375 (7.3)	2475
Diarrhea in last 2 weeks					
No	3079 (72.6)	8556 (74.7)	6315 (80.4)	4096 (79.4)	22,372
Yes	512 (12.1)	915 (8.5)	694 (8.8)	499 (9.7)	2645
Fever in last 2 weeks					
No	2988 (70.0)	7991 (74.0)	5898 (75.0)	3856 (75.1)	21,039
Yes	604 (14.0)	1482 (14.0)	1100 (14.2)	735 (14.0)	3965
Cough in last 2 weeks					
No	3089 (72.9)	8426 (78.5)	6182 (78.7)	4007 (77.7)	22,011
Yes	502 (11.8)	1042 (9.7)	810 (10.3)	568 (11.0)	2965
Antenatal					
No	1028 (24.3)	3005 (28.0)	2380 (30.3)	1776 (34.4)	8256
Yes	237 (5.6)	938 (8.7)	777 (9.9)	530 (10.3)	2500
Vitamin A					
No	2219 (52.0)	5175 (48.0)	3678 (47.0)	2404 (47.0)	13,591
Yes	433 (19.0)	1354 (10.0)	1234 (13.0)	959 (16.0)	4011
Iron/Syrup Supplementation					
No	1656 (39.0)	3115 (29.0)	2180 (28.0)	1388 (27.0)	8420
Yes	1003 (24.0)	3479 (32.0)	2775 (35.0)	1970 (38.0)	9295

Table 3.4: Descriptive statistics of the continuous covariates used in the model by Four-way category of birth size in 2008 NDHS.

Covariates	Four-Way Category of Child Birth Weight			
	Low	Average	Large	Very Large
Child weight at birth (in kg)	2.57 (0.58)	3.09 (0.59)	3.42 (0.68)	3.79 (0.79)
Mother's age at first birth (in years)	18.7 (4.06)	19.1 (4.19)	19.4 (4.28)	19.2 (4.20)
Mother's body mass index	21.7 (3.69)	22.3 (3.80)	22.6 (3.91)	22.8 (3.97)
Mother's age, (in years)	27.1 (7.31)	27.2 (7.00)	27.5 (6.91)	27.4 (6.91)
Mother's height (in cms)	157 (6.50)	158 (6.52)	158 (6.79)	158 (7.06)
Mother's weight, (in kg)	53.9 (10.3)	55.6 (11.6)	56.5 (11.2)	57.3 (11.5)

3.2 The Model formulations

This study utilizes data obtained from the 2008 Nigeria Demography Health Survey (NDHS). The data set on child birth was generated with the aim of assessing the influence of some covariates on the response (child birth size) as reported in the health surveys. NDHS data set contains several other variables, but only those that are related to child birth weight and those similar to the ones considered in the descriptive analysis were selected. The children involved in the survey had ages ranging between 1–59 months and the respondents (mothers) are in reproductive age range of 15–49 years.

The response variable of interest (child birth weight) is classified as

$$\textbf{Model A} \quad y_{i1} = \begin{cases} 1 & \text{if the child birth weight is} \leq 2500 \text{ g} \\ 0 & \text{otherwise} \end{cases}$$

A multi-categorical representation of the response variable is coded as

$$\textbf{Model B} \quad y_{i2} = \begin{cases} 1: & \text{small, if the cbw is} < 2500 \text{ g} \\ 2: & \text{average, if the cbw is} \geq 2.5 \ \& < 3.2 \text{ kg} \\ 3: & \text{large, if the cbw is} \geq 3.2 \ \& < 4.0 \text{ kg} \\ 4: & \text{very large, if the cbw is} \geq 4.0 \text{ kg} \end{cases}$$

where y_{i1} is binary response outcome and y_{i2} is an ordered categorical response outcome. The present study intends to apply a flexible regression model to quantify the effects of fixed and non-linear factors as well as geographical variations of the child birth size based on the response variables, y_{i1} and y_{i2} defined above.

3.2.1 The Geoadditive Models

We propose a structured additive regression model to examine the impact of different types of covariates on low child birth weight as a binary logit model. The parameters in the model are estimated in a unified regression model. We employ *BayesX 2.0.1* version software for fitting the structured additive regression models, which were developed by Brezger et al. (2003) via penalized maximum likelihood (PMLE) methods. With a varying combination of covariates, we formulate three different model specifications:

A.1 $\eta = \text{Spatial} + \text{random effects (No Covariates)}$

A.2 $\eta =$ Linear effects ONLY (Linear model)

A.3 $\eta =$ Non-linear + linear effects + spatial effects (Geoadditive model).

Our model selection strategy is as follows. To convey the potentially extreme distributions, we consider:

- a baseline model (A.1) without covariate, which includes only the spatial effect components

- a purely linear model (A.2), all covariates are categorical fixed effects and

- and a full model (A.3) includes the spatial effects, categorical and continuous covariates.

The continuous variables are modeled using P-smooth splines, the categorical covariates using dummy variables,(1) represents a factor level , (-1) as reference, 0 other levels) and the geo-spatial components are modeled by a Markov random field. This contrasts with whereas model II, which includes only fixed (categorical) effects.

3.2.2 Multinomial Logit Models

In recent decades, there has been growing interest in the application of an ordinal logistic regression model, and its transformation into a latent variable model, as has been described in Agresti (2003); Liu & Agresti (2005) and Tutz (2003). Such regression models, which are based on multi-categorical outcomes, are sometimes called cumulative regression models. Their distributional form had been previously investigated in the literature, by for instance McCullagh et al. (1973) and Fahrmeir & Lang (2001). The models can be motivated from latent variables such that the response variable Y, which could be birth weight, can be written as a categorical ordered response of a continuous latent (utility) variable, thus:

$$Z = \eta + \varepsilon \tag{3.1}$$

where η is a predictor depending on covariates and parameters and ε is the error term. The two variables Y and Z are linked by $Y = j$ if and only if

$$\theta_{j-1} < Z \leq \theta_j, \quad j = 1, 2, \ldots k. \text{ In our case, k=4.} \tag{3.2}$$

with thresholds $-\infty < \theta_0 < \theta_1 < \ldots < \theta_k = \infty$.
In a multinomial logit model setting, the error variables, ε in (3.1) are independent

across the categories and assumed to be standard extreme value distributed with function F. Hence, it follows that Y obeys a cumulative model. The predictor is then defined as

$$Pr(y_i \leq j|\eta) = F(\theta_j - \eta) \tag{3.3}$$

For identifiability, the linear combination does not contain an intercept term γ_0, otherwise one of the thresholds must be set to zero. The probability of a child born with the jth birth category as against the reference category, say, very large birth size, is expressed in the multinomial logit model given by

$$Pr(y_i = j|y_i \geq j, \eta_j) = \frac{\exp(\theta_j - \eta_j)}{1 + \exp(\theta_j - \eta_j)} = F(\theta_j - \eta_j) \tag{3.4}$$

Consider a set of regression observations $(y_i, x_i, s_i, v_i), i = 1, 2, \ldots, n$, where y_i is either binary or categorical response variable, x_i represents a metrical (continuous) covariate such as mother's age at time of child's birth, her body mass index, and the spatial conponent, $s_i \in [1, \ldots, 37$ of the district (states in Nigeria). A further vector $\mathbf{v} = (v_{i1}, \ldots, v_{iq})$ represents the categorical covariates. For a model as specified above, we propose a semiparametric predictor as suggested by Tutz (2003), which is defined as

$$\eta_j = \theta_i^j - (f(x_i) + f_{spat}(s_i) + v_i'\gamma) \tag{3.5}$$

where, $f(x_i)$ and $f_{spat}(s_i)$ represent the estimates of the unknown non-linear smoothing effects of the metrical covariates x_i and the spatial effect respectively, while v_i is a vector of the fixed (categorical) effect. The spatial component, $f_{spat}(s_i)$ captures the random effects of the area $s_i, s \in \{1, \ldots, 37\}$, where the woman i resides in Nigeria. The spatial component, $f_{spat}(s_i)$ is further split into two components:$f_{str}(s_i)$ and $f_{unstr}(s_i)$ as spatially structured (correlated) and unstructured (uncorrelated) random effects respectively.

We also formulate three different model specifications for the semiparametric multinomial logit model with the following predictors:

B.1 $\eta = $ Spatial + random effects (No Covariates)

B.2 $\eta = $ Linear effects ONLY (Linear model)

B.3 $\eta = $ Non-linear and Linear effects + Spatial effects (Geoadditive model)

The model composition is the same as defined in Section 3.2 above for the low birth weight (binary data structure).

Generally, we adopt the appropriate re-parameterization to estimate the model parameters. The semiparametric multinomial logit model (5.4) can be rewritten as a

mixed effect formulation, as suggested in Fahrmeir et al. (2004); Kneib (2006) and Wood (2011) of the predictor. The estimation was done via a penalized likelihood approach, where the smooth functions and model parameters are then estimated simultaneously. Inference is therefore achieved with the empirical Bayes approach based on generalized linear mixed model (GLMM) methodology, by partitioning the coefficients into a penalized and unpenalized part yielding a variance component model, as suggested in Fahrmeir et al. (2004) and Ruppert et al. (2003). Based on the GLMM formulation approach, regression and variance components can be estimated using the iterative weighted least squares and restricted maximum likelihood (REML) developed for GLMM by Fahrmeir et al. (2004) and Brezger (2005). The statistical inference is based on Markov chain Monte Carlo (MCMC) simulation technique as discussed in Fahrmeir et al. (2004) and implemented on a mixed model approach using restricted maximum likelihood (Fahrmeir et al., 2013; Kneib, 2006). All model parameters were estimated in the BayesX software package developed by Brezger et al. (2003).

Akaike information criterion (AIC)and the Bayesian information criterion (BIC) are adopted as measures for model selection. The measures are commonly used in the choice of model within a maximum likelihood framework (Fahrmeir & Lang, 2001). The AIC is defined as AIC $= -2l(\hat{\boldsymbol{\theta}}) + 2edf$, while the BIC $= -2l(\hat{\boldsymbol{\theta}}) + \log(n)edf$, where l is the conditional log likelihood given the penalized parameters, edf is the effective degree of freedom, which are estimated by the trace of the matrix that maps the un-penalized estimates on the penalized estimates corrected for the smoothing parameters uncertainty as defined in Wood et al. (2016). In addition to the two model fit criteria, we also considered the generalized cross validation (GCV) by Golub et al. (1979), which measures the ability of the selected model to predict the data. The GCV is calculated as $GCV = \frac{||y_i - \boldsymbol{\mu}^{-i}||^2}{[n - tr(A)]^2}$, where $\boldsymbol{\mu}$ denotes the prediction of $E(y_i)$ obtained from the model fitted to all data except y_i observation and $tr(A)$ is the trace of the influencing matrix with the leading diagonal element $A = \{A_{ii}\}$.

3.3   Statistical Analysis and Results

We fitted six semi-parametric logit models, three binary models and three multinomial models, for the two data structured response variables, We fitted six semi-parametric models for the two data structured response variables (3 binary logit models and three multinomial (birth size)) with different combination of covariates, and the geographical components. The models A.1 and B.1 had the geographical components as the only predictors (No covariates). The models A.2. and B.2 consist of purely linear predictors on binary and multinomial ordinal responses respectively.

The models can be described as simple linear models with all covariates exerted fixed effects on the response variable, which may not be true in real life situation by considering the complex nature of survey data structure .

For the models A.3 and B.3, we fit a spatial semi-parametric model consisting of spatial effects, linear and non-linear covariates. The categorical covariates (environmental factor, mother and child characteristics) as fixed effects and geographical location of the woman were simultaneously estimated on the response variable (birth weight). The outputs of the analysis are presented in the form of Tables, non-linear graphics and spatial maps.

Table 3.5: Model comparison values based on Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC) for the three specified models together with the marginal log-likelihood (LL) and Generalized cross validation(GCV)

Model	-2LL	df	AIC	BIC	GCV
			Binary logit model		
A.1	22,449.4	35.7	22,520.7	22,814.6	0.804
A.2	4990.1	26.0	5042.1	5221.5	0.685
A.3	6052.1	55.5	6163.2	6557.6	0.676
			Multinomial logit model		
B.1	72,630.1	38.0	72,630.0	73,019.5	2.530
B.2	19,103.4	26.0	19,155.4	19,334.8	2.587
B.3	23,533.1	67.1	23,667.2	24,144.3	2.453

For the AIC, on the binary logit model, we found that the model without covariates (A.1) gave 22520.7, the linear model (A.2) gave 5042.1, while the full model (A.3) gave 6163.13. On the cumulative multinomial logit model, for the model without the covariates (B.1) we obtained AIC = 72630.0, and for the geoadditive model (B.3) AIC =23667.2. In both models A and B, we conclude that the model with the co-variates effects is a better model than the one without any covariate. AIC relies on model likelihood from the data and the number of parameters estimated in the model. How- ever, the models A.1 & B.1 and model A.3 & B.3 included spatial structured effects of the geographical location of the woman (respondent), and such

models are more complex than the linear models A.2 and B.2. Therefore in addition to the Akaike Information Criterion (AIC), we also investigated the values of the Bayesian Information Criterion (BIC), which put emphasis on the model complexity rather than the simplicity. Furthermore, the generalized cross validation (GCV) criterion emphasizes the model optimality rather than simplicity or complexity, as explored by the former two criteria. The researcher is left with varying options in selecting the preferred model.

The models A.2 & B.2 can be described as simple linear models with all covariates exerting fixed effects on the response variables. The spatial heterogeneity random effects (structured and unstructured effects) are completely ignored or excluded in the model B.2, so therefore the variability inherent as a result of geographical location of the respondents could not be captured in the model. Therefore, this model (B.2) could not account for larger proportions of the variation in the data and the model yielded the lowest values for the AIC, BIC and GCV for all options of model specification.

3.3.1 Fixed Effects of socio-demographic factors

Table 3.6 presents the posterior estimates of fixed effects of the socio-demographics variables considered in the binary logit model. A careful interpretation of the coefficient signs of these estimates and the 95% confidence interval is observed. Whenever the 95% credible intervals include zero, it will be interpreted as the effect is not statistically significant at the 5% probability level. A factor with negative coefficient indicates that the effect of the variable (factor) would reduce (lower) the chance low birth weight (LBW) outcome compared to the reference factor, while a positive coefficient would raise(increase) the probability low birth weight prevalence. The signs of the coefficient estimates for the models A.2 (linear) and A.3 (geoadditive) look similar. Model A.2 can be described as a sub-model of A.3. The interpretation would be based on model A.3.

The results showed that there was evidence of significant association with higher probability of low birth weight in the North East compared with the North Central region. This indicates that women in the North East had higher odds of low birth weight prevalence or higher risk of low birth size than the North Central zone. All other geopolitical zones indicate lesser likelihood of low birth size but these were not statistically significant. Marginally, the Southern Zones had lower prevalence of low birth sizes when compared with the Northern parts of Nigeria.

The results from Table 3.6 (column models A.2 and A.3) further showed that a male child had a higher probability of being born with low birth weight and a child born

from a multiple (twin) birth also had higher odds of low birth weight than a single birth. This implies that a male child had a higher odds of being born with a low birth weight compared to a female counterpart. That is the odds of LBW for males was 18% higher than that for female children (i.e. male =exp(0.171) refer model A.3 column, Table 3.6). A child born from a multiple birth would increase the odds of LBW by 45% compared with a single birth. A short birth interval (2 or more children within 3 years) also showed a significantly higher probability of low birth weight compared with a well spaced birth interval, indicating that a short birth interval increases the odds of LBW by 17%, OR = 1.17, 95% CI (1.07, 1.29) compared with a single child birth.

In addition, environmental factors include poor household, death of sibling 2 and 4, firewood/ dung method of cooking and mother smoking, disease morbidity during pregnancy were found to contribute to the higher odds of LBW, although they were not statistically significant at 5% probability level. That is children born to mothers, who suffered from disease morbidity would increase the odds of giving birth to very low birth weight (i.e. there are positive association between LWB and diseases morbidity). There was significant association between childhood under-nutrition (wasting) and low birth weight. The result showed that children born of low birth weight would suffer of growth restriction in their early life(infancy).

Table 3.6: Summary of three binary logistic models based on 2008 Nigeria DHS

Variable	Model A.1		Model A.2		Model A.3	
	Est.	95% CI	Est.	95% CI	Est.	95% CI
Intercept	−1.956	(−2.156, −1.756)	−1.400	(−1.656, −1.143)	−1.193	(−1.962, −0.424)
Geopolitical zones						
North central (ref)	-	-	0		0	
North East (NE)	-	-	0.188	(0.023, 0.354)	0.448 *	(0.004, 0.892)
North West (NW)	-	-	−0.143	(−0.343, 0.056)	0.265	(−0.153, 0.682)
South east (SE)	-	-	0.049	(−0.162, 0.261)	−0.165	(−0.653, 0.324)
South South (SS)	-	-	−0.226	(−0.426, −0.026)	−0.390	(−0.051, 0.062)
South West (SW)	-	-	0.137	(−0.027, 0.301)	−0.228	(−0.680, 0.223)
Child's sex						
Female (ref.)	-	-	0		0	
Male	-	-	0.1797*	(0.106, 0.254)	0.171*	(0.104, 0.237)
Type of birth						
Singleton (ref.)	-	-	0		0	
Multiple birth	-	-	0.206	(−0.023, 0.434)	0.377	(0.162, 0.592)
Place of residence						
Rural (ref.)	-	-	0		0	
Urban	-	-	−0.0114	(−0.104, 0.0810)	−0.038	(−0.124, 0.049)
Mode of cooking						
Cook gas, kerosene (ref.)	-	-	-	-	0	
Firewood/dung	-	-	-	-	0.068	(−0.158, 0.295)
Not literate (ref.)	-	-	0		0	
literate	-	-	−0.184	(−0.272, −0.096)	−0.118	(−0.199, −0.037)
Not (ref.)	-	-	-	-	0	
Smoke	-	-	-	-	0.474	(−0.233, 1.180)
No sibling dead (ref.)	-	-			0	
dead 2	-	-	-	-	0.181	(−0.034, 0.396)
dead 3	-	-	-	-	−0.369	(−0.679, −0.058)
dead 4	-	-	-	-	0.144	(−0.166, 0.454)

Table 3.6: *Cont.*

Variable	Model A.1		Model A.2		Model A.3	
	Est.	95% CI	Est.	95% CI	Est.	95% CI
Child spacing last 3 years						
<2 birth (ref.)	-	-	0		0	
≥2 birth	-	-	0.200	(0.091, 0.308)	0.159	(0.063, 0.254)
stunted	-	-	0.047	(−0.040, 0.134)	-	-
wasted	-	-	0.226	(0.134, 0.318)	-	-
Micro-nutrient supplement.						
No (ref.)	-	-	0		0	
Prenatal iron/Syrup	-	-	−0.282	(−0.375, −0.189)	−0.234	(−0.318, −0.150)
Supplementation						
No (ref.)	-	-	0		0	
Postnatal vitamin A	-	-	0.010	(−0.073, 0.093)	−0.059	(−0.137, 0.019)
No (ref.)	-	-	0		0	
Antenatal	-	-	−0.083	(−0.183, 0.017)	−0.045	(−0.139, 0.050)
Poorest (ref.)	-	-	0		0	
Quintile 1	-	-	0.020	(−0.138, 0.178)	0.030	(−0.117, 0.178)
Quintile 2			−0.121	(−0.265, 0.023)	−0.081	(−0.216, 0.053)
Quintile 3	-	-	0.005	(−0.144, 0.154)	0.007	(−0.129, 0.143)
Quintile 4	-	-	0.051	(−0.141, 0.241)	−0.077	(−0.290, 0.137)
No (ref.)	-	-	0		0	
diarrhea	-	-	0.090	(−0.022, 0.203)	0.062	(−0.043, 0.167)
fever	-	-	−0.019	(−0.131, 0.093)	0.021	(−0.083, 0.125)
cough	-	-	0.059	(−0.058, 0.176)	−0.022	(−0.132, 0.089)

−= indicates the corresponding variable was not included in the model;
CI = confidence intervals; ∗ significant at 5 percent significance level.

There was evidence that micro-nutrient intervention such as iron syrup supplementation during pregnancy would reduce the chance of low birth weight. Also, postnatal vitamin A to children would boost the growth of child born of low birth weight, although vitamin A supplementation was not significant. Iron supplementation to pregnant mother was significant and reduced the odds of LBW by 21%, i.e. OR=0.79, 95% CI (0.73, 0.86), compared to those mothers, who did not take iron syrups. Mother, who attended antenatal also had lesser likelihood of giving birth to a child with low birth weight.

The results further showed a strong impact of mother characteristics for improving her birth outcome. Maternal literacy (ability to read or write), urban resident mother, mother belonging to richer household would likely reduce their chances of giving birth to a child with low birth weight. A literate mother would have a lesser probability (reduce odds) of giving birth to a child with LBW by 11%, i.e. OR=0.89, 95% CI(0.82, 0.96) compared to an illiterate mother. The result showed a significant

evidence that mothers who used firewood/dung/agricultural residues method for cooking would significantly reduce the relative odds of the larger birth size to low birth size over gas/kerosene cooking (refer Model A.3 column, Table 3.6, though not significant in our case.

We further present the estimates of Model B.3 in Table 3.7, iron syrup supplementation to mothers during pregnancy would significantly increase the relative odds of larger birth size than those mothers with no supplements. Mother literacy was found to be significant with relative higher likelihood of large size to low birth size for non-literate mother. Similar influence of categorical covariates were observed from the multinomial model as presented in Table 3.7. The model B.2(linear model) can be considered as a sub-model of model B.3. Furthermore, the parameter signs of the variables in the models are similar. From the results on model B.3, the estimates of the threshold parameters θ_1, θ_2 and θ_3 are presented along with the categorical variables estimates. We interpret threshold(cut point) parameters as a negative(positive) value corresponds to less (higher) likelihood of the birth weights category shifting left(right) on the latent scale. For instance, a negative sign of θ_1 and θ_2 signifies a shift on the latent scale to the left side, yielding a lower probability for category of low birth weight and average size categories respectively.

This can also be interpreted in terms of the relative odds of shifting from very low category to higher birth outcome. For instance the relative odds of shifting from very low to low birth size per unit increase in the predictors is given by θ_1, shifting from low birth to normal size for θ_2, shifting from normal to large birth size for θ_3. This can be stated as the relative odds of a low birth size to an normal birth size(about 2500 grams) , $(\exp(-1.76) = 6.12)$, i.e. about 2 times lesser chances of shifting form low birth category to normal birth size, and $(\exp(1.812) = 1.42)$, 1.41 times odds of getting normal(average) birth size to large birth weight (refer Table 3.7, column Model B.3). Similarly, a positive sign of θ_3 signifies a shift on the latent scale to the right side, yielding a higher probability of moving for the larger category to very large.

Table 3.7: Summary of of three Multinomial logit models based on 2008 Nigeria DHS

Variable	Model B.1		Model B.2		Model B.3	
	Est.	(95% CI)	Est.	(95% CI)	Est.	(95% CI)
Threshold						
θ_1: low birth size	−1.899	(−1.940 , −1.860)	−1.089	(−1.614, − 0.564)	−1.760	(−1.970, −1.550)
θ_2: average size	0.096	(0.070, 0.120)	−0.513	(−1.037, 0.011)	0.350	(0.140, 0.550)
θ_3: large size	1.551	(1.52, 1.58)	1.037	(0.513, 1.562)	1.812	(1.61, 2.020)
NC (ref.)						
NE	-	-	−0.140 *	(−0.240, −0.039)	0.301	(−0.165, 0.770)
NW	-	-	−0.176 *	(−0.287, −0.065)	**0.473**	**(0.020, 0.930)**
SE	-	-	0.249 *	(0.133, 0.365)	**−0.630**	**(−0.618, 0.280)**
SS	-	-	0.100	(−0.004, 0.203)	0.054	(−0.422, 0.530)
Female (ref.)						
Male			0.040	(−0.002, 0.081)	−0.178 *	(−0.221, −0.130)
Single birth (ref.)						
Multiple birth			0.078	−0.081, 0.238)	**−0.271**	**(−0.434, −0.110)**
No (ref.)		0		0		
stunted	-	-	0.025	(−0.025, 0.076)	−0.014	(−0.066, 0.040)
wasted	-	-	−0.001	(−0.057, 0.056)	−0.176 *	(−0.235, −0.121)
underweight	-	-	-	-	-	-
Birth Order 1 (ref.)			0		0	
Order 2	-	-	-	–	0.017	(−0.075, 0.110)
Order 3	-	-	-	–	0.040	(−0.053, 0.130)
Order 4	-	-	-	–	0.001	(−0.098, 0.100)
Order 5	-	-	-	–	0.032	(−0.049, 0.110)
Rural (ref.)			0		0	
Urban			0.057 *	(0.005, 0.109)	−0.035	(−0.090, 0.02)
Children last 3 years						
< 2 birth (ref.)	-	-	0		0	
≥ 2 birth			−0.056	(−0.123, 0.011)	−0.103 *	(−0.173, −0.03)

−= indicates the corresponding variable was not included in the model;
CI = confidence interval; ∗ significant at 5% significance level.

Table 3.7: *Cont.*

Variable	Model B.1		Model B.2		Model B.3	
	Est.	(95% CI)	Est.	(95% CI)	Est.	(95% CI)
No supplement(ref.)				0	0	
Iron/Syrup during pregnancy			0.090 *	(0.031, 0.148)	0.052	(−0.010, 0.11)
Postnatal Vitamin A	-	-	−0.064 *	(−0.110, −0.018)	0.096 *	(0.048, 0.14)
No antenatal (ref.)			0		0	
Antenatal	-	-	−0.025	(−0.079, 0.028)	0.016	(−0.041, 0.07)
Poorest(ref.)						
Quintile 1 (poor)	-	-	−0.010	(−0.103, 0.084)	−0.026	(−0.123, 0.07)
Quintile 2 (middle)	-	-	−0.057	(−0.138, 0.024)	0.037	(−0.047, 0.12)
Quintile 3 (rich)	-	-	0.022	(−0.061, 0.106)	0.048	(−0.039, 0.13)
Quintile 4 (richest)	-	-	0.053	(−0.050, 0.156)	0.074	(−0.039, 0.19)
No disease (ref.)			0		0	
diarrhea	-	-	−0.052	(−0.121, 0.017)	0.029	(−0.042, 0.10)
fever	-	-	−0.004	(−0.068, 0.060)	−0.046	(−0.111, 0.02)
cough	-	-	−0.007	(−0.075, 0.060)	0.019	(−0.051, 0.088)
Not smoking (ref.)			0		0	
Mother smoke	-	-	−0.087	(−0.581, 0.407)	-	-
Not literate (ref.)						
literate	-	-	0.0112	(−0.038, 0.0609)	0.083 *	(0.030, 0.140)

−= indicates the corresponding variable was not included in the model;

CI = confidence interval; ∗ significant at 5% significance level.

Remarks: It is interesting to note that two models gave compelling opposite effects in Table 3.7. For instance, factors (NW,SE, multiple birth, postnatal vitamin A, antenatal etc) had the signs of the coefficient opposing between the linear model (B.2) and multinomial ordinal model (B.3) for the same data set. Theoretically, it should not be surprising when different types of models (binary, ordinal and multinomial logistic regression for categorical outcomes) can be fitted on a common data set, however the linear models can not fit well when the outcome variable is categorical.

From equations (3.3) through (3.5), that the very important minus signs are included before the linear predictor, η_j. This flips the estimate of all the coefficients other than the intercept. We therefore propose a new model and provide a brief theoretical framework at the end of this chapter as addendum and derivation of probability distribution of a binary logit model is provided in the Appendix A 7.1.

3.3.2   Non-Linear Effects of mother's age and mass index

The non-linear components of the model investigate the effects of metrical (continuous) covariates using the binary logit model A.3(corresponds to Table 3.6) are displayed in Figure 3.2, while for model B.3 in Figure 3.3. Each figure is made up of 5 trend lines with the center line being the smooth estimate of the posterior mean bounded by the two inner lines which are the 95 % credible intervals and outer lines which give the 80% credible intervals.

Figure 3.2(a) depicts that there is an inverse linear relationship between between mother's weight (kg) at birth and the low birth weight, while Figure 3.2 b shows a linear relationship between the mother's body mass index ($weight/height^2$) and the child birth weight as estimated from the binary logit model. The graph depicts that an increase in mother's weight would reduce the chance of the woman giving birth to a low weight child. We noticed also a consistent linear association between the smooth effect of the mother weight increases as shown in the Figure 3.2 (a). That is the heavier mother the lower the possibility of giving birth to a baby with low birth weight. A critical point was observed at about 58 kg, it seems that the majority of women in study population had averaging weight at birth of 58 kg.

Further results from the binary logistic analysis, Figure 3.2b showed the effect of mother's body mass index (mbmi)= $weight(kg)/height^2(m)$ on the child low birth weight. The graph depicts a discernible relationship between 'mbmi' and her baby birth weight as linear relationship. A critical point was noticed at around value 24 (which is normal body mass index). A threshold points can be invoked according to the World health Organization (WHO). The graph can be factored into 3 parts: below 18.5, (as underweight mother) would increase the odds of low birth size(underweight child) and $mbmi$ above 24.5, overweight mother would increase the likelihood of large size babies, their babies are at the risk of obesity. Figure 3.2c illustrates the plot of the effects mother's age on the low birth weight. The plot depicts a $U-$ shape function. The graph can be segmented into 3 parts (thresholds) as the effect of mother's age below 20 years,(teenage mother) would increase the likelihood of low birth child weight(downward trend), mother age between 20-40 years (matured mothers) with no significant effect on poor birth outcomes, and mother

age above 40 years(older mothers) would raise the likelihood of giving birth to babies with low weight.
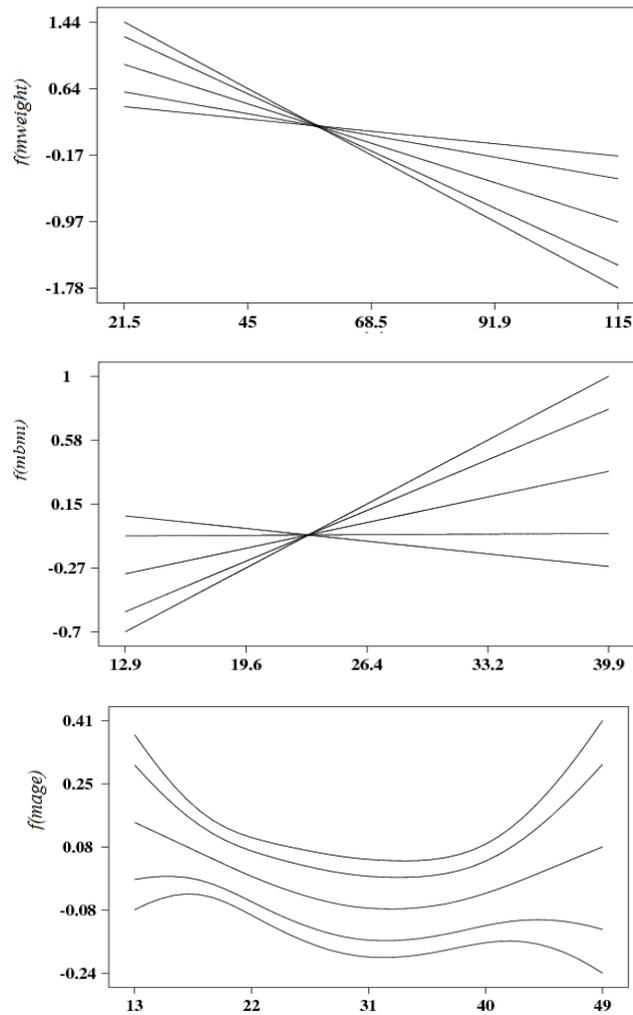
Figure 3.2: Binary logit model results: non-linear effects of (a) Mother's weight, (b)mother's body mass index, and (c)mother's age at birth **Model A.3**

The non-linear effects of (a) mother's weight, (b)mother's body mass index, and (c) mother's age at birth (years) of multinomial model B.3 are shown in Figure 3.3. Figure 3.3a show the non-linear effect of mother's weight showing inverted U-shape. This implies mother's weight on her that for birth size outcome can be described as an inverted $U-$ shape . Figure 3.3b depicts an $S-$ shape function, known as sigmoid function. This represents the impact of mother's nutritional status on her child birth weight. Figure 3.3 (c) shows the effect of mother's age at birth on her child birth weight. The resulting trend lines possess a discernible non-linear relationship. In other words, one can deduce that a teenage mother (< 20 years) resulting in a down-
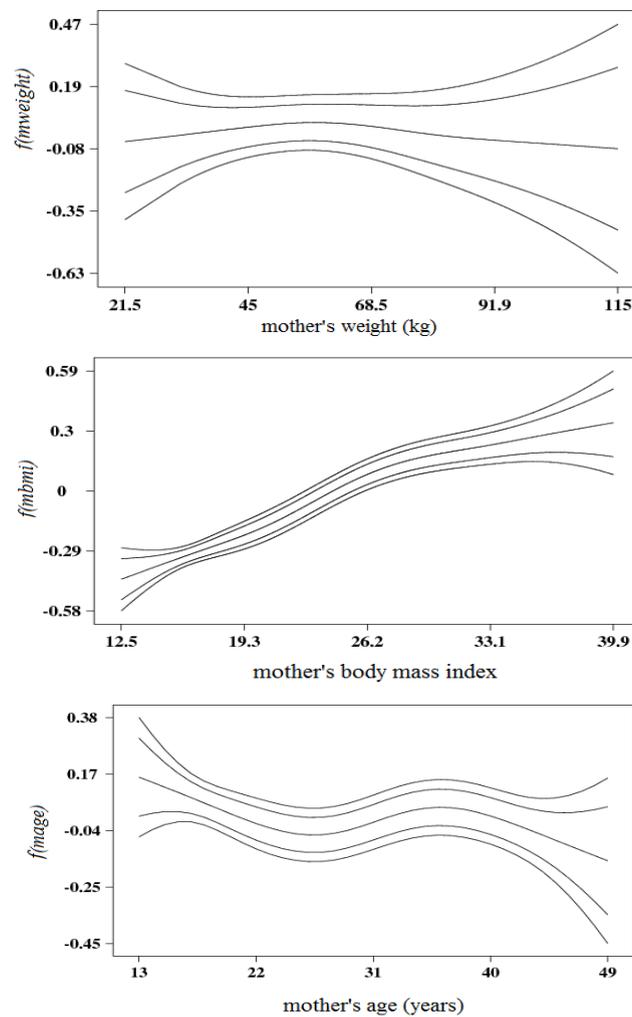
Figure 3.3: Cumulative multinomial model results: non-linear effects of (a) mother's weight, (b)mother's body mass index, and (c) mother's age at birth for **Model B.3**

ward trend on her baby's birth weight, mother's age between 20-40 years would lead to an upward concave curve and mother above 40 years would result into a downward concave on her baby's birth weight. Thus, the optimal mother's age could be obtained on the curve minimal turning point of the graph, say, 31 years, to achieve the lowest risk of poor birth outcome.

The spatial effects from the analysis of two geoadditive binary logistic models are presented in Figures 3.4 (a-d) and two(2)cumulative multinomial model 3.5(e-h). The maps represent the posterior means *(left panel)* and the maps showing the 95% credible intervals *(right panel)* are used to determining the significance level. Using 95% credible intervals, regions(states) with white (black) colours are associated with significantly high (low) prevalence corresponding to regions lie in the positive(negative) sides, while the grey colour depicts the estimates are not significant

among states.
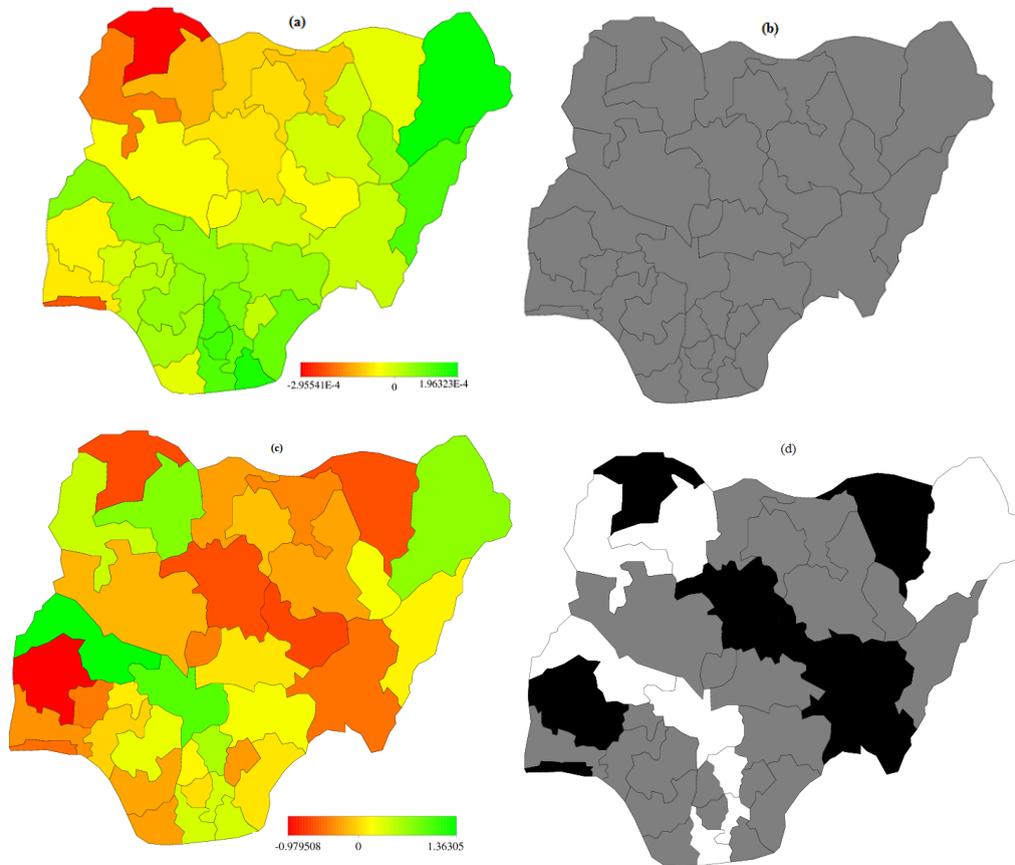
Figure 3.4: Binary logit model results: the posterior mean (a) and corresponding 95% credible intervals (b) of spatial effects for Model A.1 and the posterior mean (c) and corresponding 95% credible intervals (d) of spatial effects for Model A.3

The posterior mean from model A.1 and model A.3 are shown in Figure Figure 3.4 a & c respectively. The geographical variation of low birth prevalence in Nigeria varies from -2.155×10^{-4} to 1.963×10^{4} for the model A.1 (no covariate factors except the geographical component) and from -0.980 to 1.363 for the model A.3 (Geo-additive logistic model). Figure 3.4 b is the corresponding 95% credible intervals to Figure 3.4 a, with all the regions(states) showing "grey" colour depicts that there were no significant difference in the low birth prevalence across the states. For model A.3, Figure 3.4 c is the corresponding 95% credible intervals to Figure 3.4 c, there were evidence of spatial variations in the low birth outcomes across the states/regions in Nigeria after adjusting for some confounding factors. In Figure 3.4(d), districts (states) with black colour are strictly negative, indication of a low birth prevalence in those states. The states include Lagos, Oyo, Sokoto, Kaduna, Plateau, Taraba and Yobe. The 'white coloured' regions are strictly positive, this means that the districts (states) are associated with high prevalence of low birth weight. The states

are; Rivers, Abia, Enugu, Kogi, Kwara, Kebbi, Zamfara and Borno. The high prevalence of low birth weight can be found states(districts), where child bearing women engaged in manual labour and farming activities and arid environment, river communities (areas) with pollution (water or air). Figure 3.5 represents the spatial com-
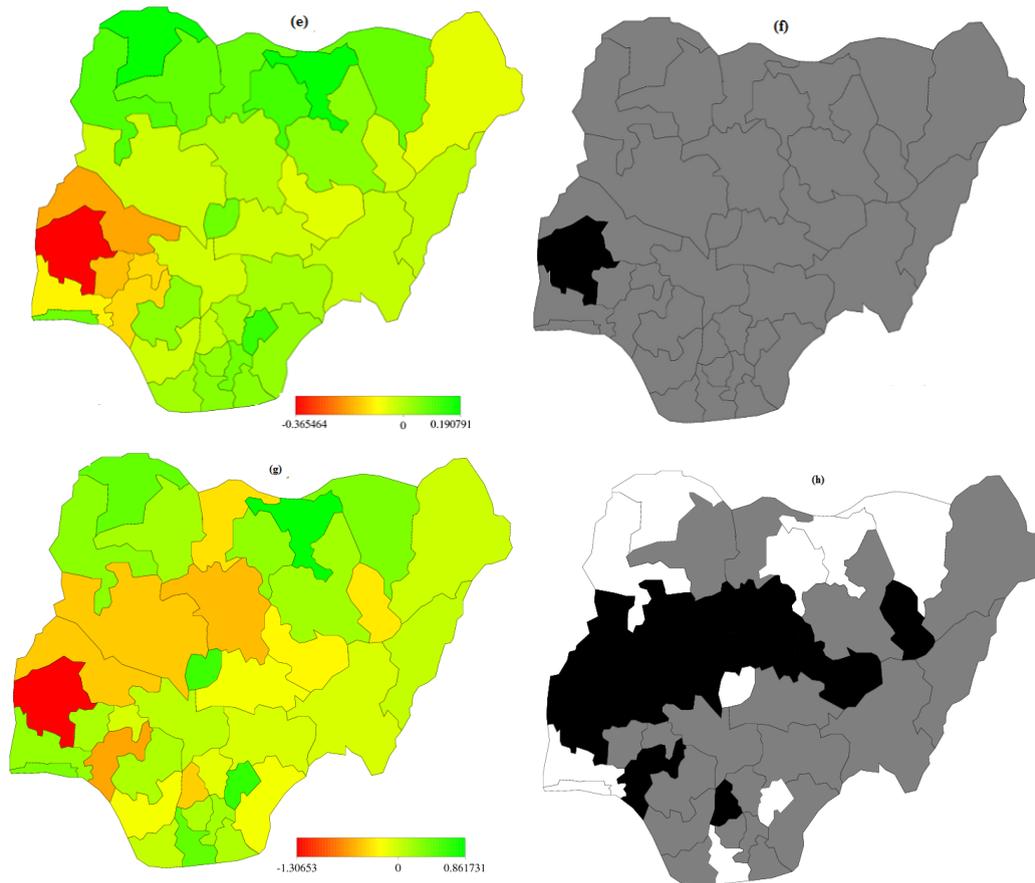
Figure 3.5: Cumulative multinomial models results, the posterior mean(e) and corresponding 95% credible intervals (f) of spatial effects for Model B.1 and the posterior mean (g) and the corresponding 95% credible intervals (h) of spatial effects B.3

ponents of the multinomial models (B.1 and B.3). The left panels (e & f) are the posterior means of residual spatial effects showing evidence of spatial variation. Using Figures (f) & (h) to determine the significance level, Figure 3.5(f) shows that Oyo state with black colour had a relative low probability of low birth weight. Other states with grey coloured had no significant variation. As displayed in Figure 3.5(h) (Full model), the black coloured regions are associated with relative low probability (strictly negative)of low birth weight. The states are: Ondo, Oyo, Kwara, Niger, Kaduna, Plateau and Gombe. The white coloured regions are associated with high likelihood of low birth size (low birth sizes). These records are observed at states: Lagos, Ogun, FCT-Abuja, Rivers, Ebonyi, Kebbi, Sokoto, kano, Jigawa and Yobe. Perhaps, this trend might be attributed to growing population in cities coupled with

over stretched facilities. Other regions with grey coloured are not significant.

3.4   Discussion

This study investigates the social and demographic (environmental, maternal and child characteristics) impacts on the child birth weight. The study simultaneously estimated small area geographical disparities and underlying determinant factors of child birth outcomes in the context of a developing country, Nigeria. Our fixed effect estimates of regression models look reasonable after controlling for contextual factors. The descriptive analysis showed that the percentage of low birth outcomes decreases with increase in birth orders. The percentage of low birth weight was inversely related to the household wealth index. The summary statistics showed that a large proportion of women had not attended antenatal check ups and they had not given postnatal vitamin A supplements to their children.

From the binary logit analysis, the findings revealed that maternal literacy and prenatal iron syrup supplementation had significant association with a lower probability for a low birth weight. Other variables include urban residence and antenatal attendance, which also had a strong influence on low chances of low birth weight, but they were not significant in our case.

The findings also revealed that later childhood undernutrition and morbidity had significant association with the low birth weight. Studies have shown that the genetics are inherent, as well as environmental factors has significant influence on child birth defects. A recent study reported by Kodzi & Kravdal (2013). Our findings corroborates this study. A meta-analysis study by Grunau et al. (2004) gives further evidence that the children born of underweight mothers tend, in later life, to have cognitive disabilities and a lower intelligence quotient. Levitt et al. (2000) have also reported that those children who survived the misfortune of low birth weight had a higher risk of high blood pressure, diabetes and heart diseases at adulthood.

The finding of our study revealed that cooking with firewood or dung or agricultural crop residues is a critical risk factor of low birth weight. This result complements earlier work conducted in other sub-Saharan Africa countries. In a study conducted in Zimbabwe, Mishra et al. (2004) found that low birth weight was not only associated with lack of socioeconomic resources but also with by the use of inferior energy sources for indoor cooking and by with air pollution. Studies in other countries reported in Skokić et al. (2011) and Chen et al. (2007) had enunciated that early childbearing (teenage pregnancies), inadequate access to prenatal health services and economic disadvantage led to a higher prevalence of low birth weight. In addition, poverty and low maternal education contribute significantly to poor birth

outcomes. Our findings corroborate the those by Han et al. (2011) and Shahnawaz et al. (2015) concerning the effect of poor maternal socio-economic factors on increasing incidence of low birth weight in India and sub-Saharan Africa.

The results in this study gave a strong indication that iron syrup supplementation during pregnancy yields improvement in child birth weight, while postnatal vitamin A intervention would boost growth among those children born with low birth weight. This parallels the wealth of evidence of that zinc supplementation reduces diarrhea morbidity and respiratory infections among children. For instance, see reports from Peru and Bangladesh, which show that zinc supplementation during antenatal resulted in an improvement in foetal growth and birth weight (Caulfield et al., 1999; Osendarp et al., 2003) and other similar studies by Osendarp et al. (2001) and Haider & Bhutta (2006). Similarly, a study conducted in Tanzania by y Kawai et al. (2011) found a strong association between multiple micro-nutrient supplementation and reduction in the risk of perinatal mortality. Mixed results are given in a report from China by Zeng et al. (2008) on multiple micro-nutrient supplementation during pregnancy, which indicates that it improved birth weight, but had no effect on neonatal mortality. A contrary result was reported from Nepal by Christian et al. (2003) and Hambidge (2000) that multiple supplementation by the addition of zinc to iron and folic acid formulations nullified the beneficial effect of iron and folic acid on birth weight.

It was more obvious that the continuous covariates of mother's weight, mother's age, and her body mass index showed discernible associations with the child birth weight. Our findings are consistent with established theory and even strengthen the empirical results from similar studies. Our result revealed that an underweight mother with body mass index ($mbmi < 18.5kg/m^2$, mother) possesses a higher risk of a low birth weight child, an correspondingly, an obese mother (mbmi $\geq 26kg/m^2$) had a higher probability of giving birth to an overweight baby, with the child also being at risk for obesity later in life. This result is consistent with that from a recent study by McDonald et al. (2010). Previous studies have identified that the mother's nutritional status or body mass index has a strong relation to reproductive health outcome.

Epidemiological studies reported in Vangen et al. (2002) and Johnston et al. (2002) have also estimated that environmental impacts contribute about 25% of birth weight variance and genetic influences accounted for between 38 to 80% birth weight variance. In our analysis of the 2008 Nigeria DHS data, we have established substantial evidence of geographical variations in the birth weight of babies across states. This output on regional variation has corroborated the finding in similar research studies. Recent studies by Wardlaw (2004) and Ngwira & Stanley (2015) have shown remarkable variations in the prevalence of low birth weight according to geographical pat-

terns. Wardlaw (2004) had reported that over 20 million infants, constituting 15.5 per cent of all global births, are born with low birth weight. Of these, 95.6% were born in developing countries, which was attributed to high poverty levels in developing countries (16.5%) that are more than double the level in developed regions (7%). In addition half of all babies of low birth weight are born in South Central Asia, where about a quarter (27%) of the infants weigh less than 2,500 grams at birth (Ngwira & Stanley, 2015). The low birth weight levels in sub-Saharan Africa are found to be around 15% which is only slightly more than the 14% occurring in the Caribbean, while in Central and South America low birth weights comprise about 10% (Hosain et al., 2006; Esimai & Ojofeitimi, 2014). A significant geographical variation that characterized the prevalence of low birth weight in Europe, with lower rates in the more northerly countries is reported in recent study by Skokić et al. (2011).

3.5 Limitations and Concluding remarks

Birth weight is an important health parameter for obstetricians and gynecologists. It is a good health indicator of a child-bearing mother and a strong predictor of infant morbidity and mortality. For simple epidemiological convenience, the newborn's weight can be considered intuitively as being categorical in nature, and the thresholds can be put on a continuous scale. In survey reporting, the mothers frequently estimate their infant's birth weight and make a classification in ordinal categories (low, normal, large) instead of citing actual birth weight.

The study fits a multinomial regression model to analyze the relationships between the polytomous response and different kind of covariates in a unified manner. The fixed effects of bio-social covariates were estimated in a parametric way and the non-linear effect modeled using P−splines. The spatial component was modeled using a conditional auto-regressive prior. A penalized maximum likelihood estimation was performed to estimate the model parameters.

The cross sectional data on 28,647 children born between 2003 and 2008 were extracted from the 2008 Nigeria Demographic and Health Survey. The results identified risk factors that were significantly associated with low birth weight, which include multiple birth, short interval between births, death of a sibling, disease morbidity, mother's smoking, firewood or dung for cooking and household poverty. The findings further showed spatial patterns, which are not captured by the underlying determinants, and produced probability predictive maps of the spatial residual effects. In addition to the statistical relevance of our method, the generated spatial maps identified areas where low birth weight was highly endemic, which can assist government agencies in channeling scarce health resources. A comprehensive ap-

proach which institutes a combination of interventions to improve the overall health care of the women is needed.

This is cross-sectional data and is self-reported, as is the case for much survey data. Thus, the causal relationship may be difficult to establish with non-response cases. In addition, the self-reported nature of the data means that there is tendency of recall-bias, especially with issues relating to age, birth size or actual birth weight of the children. The birth weight of new baby is a good indicator of health status of the child at birth but many babies in sub-Saharan Africa are not weighed at birth. Thus, the mother's report on the size of the baby at birth is used as a proxy. Furthermore, the analyzed sample is not representative of the populations considered, as birth weights were recorded for a relatively small fraction of births in these populations. Our attempts to understand the implications of the selective nature of the birth weight data on our estimates suggest that we may have overestimated the adverse effects of very high parities and underestimated the advantage of something? at more moderate levels. In spite of these limitations, it is our belief that the findings from this study will facilitate targeted interventions and strategic policies.

The present study explored a robust and flexible methodological approach to analyze the birth weights of under-five children in 2008 Nigeria Demographic and Health Survey data. The multivariate analysis takes into account the influence of unobserved factors related to individual households on child birth weight . Such health outcomes can facilitate effective policy for maternal and child health care. Having controlled the confounding factors in the analysis, the method produced predictive spatial maps of the residual effects that could not been captured by the underlying factors in a classical regression setting, which would have been neglected. The statistical significance of the variables discussed in the fixed effect table can be used to formulate appropriate policy concerning intervention programmes. The spatial plots highlight hot-spots that can assist government to channel resources in an efficient manner.

3.6   Addendum

In the ordinal regression analysis carried out in this chapter, the estimates of the regression coefficients were not stable, this violates the independent assumption (parallelism of separate binary logistic regression). This addendum is necessary to augment the gap noticed.

In the ordinal logistic regression analysis above, the outcome variable is ordered, and has more than two levels. For example, a child birth weight is ordered from very low to large birth size; maternal highest educational level attained is scored

from level 1 to 4; and a response scale of a survey instrument is ordered from no formal education to graduate level. One appealing way of creating the ordinal variable is via categorization of an underlying continuous variable, see Hosmer (2000); Archer et al. (2007) and O'Connell (2006) for further readings.

The present study used the child birth size as an ordinal outcome variable, which is coded 1 = very low; 2 =low, 3=normal and 4 = large) , which is based on categorization according to World Health Organization and DHS report. The child birth weight are categorized in the very low birth size if the birth weight is less 1.5 kg, those with between 1.5 and 2.49 kg are categorized in the low birth size; between 2.5 kg and 3.19 are categorized as normal (average) birth size and child's birth weight of 3.2 kg or more are categorized in the large size. The distribution of child birth weight is highly positively skewed. The violation of the assumption of normality makes the use of ordinary multiple regression analysis inappropriate. Therefore, the ordinal logistic regression is the most appropriate model for analyzing the ordinal outcome variable in this case.

The ordinal logistic regression model can be expressed as a latent variable model discussed in the literature such as Agresti (2003); Wooldridge (2010) and Ananth & Kleinbaum (1997). Assuming a latent variable structure, by creating an auxiliary variable, Z such that

$$Z = \mathbf{X}\beta + \varepsilon \tag{3.6}$$

where $\mathbf{X}$ is a row vector $(k \times 1)$ containing no constant, $\boldsymbol{\beta}$ is a column vector $k \times 1$ of structural coefficients, and ε is random error with standard normal distribution i.e. $\varepsilon \sim N(0,1)$.

Let Z be divided by some cut points (thresholds) such that $\theta_1, \theta_2, \ldots \theta_k$, and $\theta_1 < \theta_2 < \ldots < \theta_k$. Considering the observed child birth size is the ordinal outcome, y, ranging from 1 to 4, where 1= very low, 2 = low , 3-normal and 4 = large (combining large and very large from DHS data),

$$Y = \begin{cases} 1 & \text{if} & z \leq \theta_1 \\ 2 & \text{if} & \theta_1 < Z \leq \theta_2 \\ 3 & \text{if} & \theta_2 < Z \leq \theta_3 \\ 4 & \text{if} & \theta_3 < Z \leq +\infty \end{cases}$$

Therefore, the probability of a woman giving birth to a child falling in j birth size category, can be computed by

$$
\begin{aligned}
P(y=1) &= P(z \leq \theta_1) \\
&= P(\mathbf{x}\beta + \varepsilon \leq \theta_1) \\
&= F(\theta_1 - \mathbf{x}\beta) \\
P(y=2) &= P(\theta_1 < Z \leq \theta_2) \\
&= F(\theta_2 - \mathbf{x}\boldsymbol{\beta}) - F(\theta_1 - \mathbf{x}\boldsymbol{\beta}) \\
P(y=3) &= P(\theta_2 < Z \leq \theta_3) \\
&= F(\theta_3 - \mathbf{x}\boldsymbol{\beta}) - F(\theta_2 - \mathbf{x}\boldsymbol{\beta}) \\
P(y=4) &= P(\theta_3 < Z \leq +\infty) \\
&= 1 - F(\theta_3 - \mathbf{X}\boldsymbol{\beta})
\end{aligned}
$$

The cumulative probabilities can also be computed using the form:

$$
P(Y \leq j) = F(\theta_j - \mathbf{X}\boldsymbol{\beta}), \text{where } j = 1, 2, \ldots J-1 \tag{3.7}
$$

In a binary logistic regression model, the response variable has two levels, with $1 =$ low birth weight $< 2.5kg$, and $0 = cbw \geq 2.5$kg. The probability of a woman giving birth to a child with low birth weight is predicted on a set of predictors. The logistic regression model can be expressed as:

$$
\begin{aligned}
ln(Y') &= \frac{\text{logit}[\pi(x)]}{1 - \text{logit}[\pi(x)]} \\
&= ln\left(\frac{\pi(x)}{1 - \pi(x)}\right) \\
&= \theta + \sum_{j=1}^{p} x'\beta
\end{aligned}
$$

Therefore, the probability of low birth category (i.e success) can be defined by $\pi_i = F(x_i'\beta)$.

Similar to logistic regression, in the proportional odds model, it is easier to work with the logit, or the natural log of the odds. To estimate the ln (odds) of being at or

below the jth category, the PO model can be rewritten as

$$\text{logit}(\pi(Y \leq j|\mathbf{X})) \qquad (3.8)$$
$$= ln\left(\frac{\pi(Y \leq j|x_1, x_2, \ldots, x_p)}{\pi(Y > j|x_1, x_2, \ldots, x_p)}\right)$$
$$= \theta_j + (-\beta_1 X_1 - \beta_2 X_2, \ldots, -\beta_p X_p)$$

Thus, this model predicts cumulative logits across $J - 1$ response categories. By transforming the cumulative logits, we can obtain the estimated cumulative odds as well as the cumulative probabilities being at or below the jth category. The proportional odds (PO) model, also known as cumulative odds model, see Agresti (2003); Lee (1992) and O'Connell (2006) for further readings.

From the analysis above, the independent assumption of covariate factors could not be sustained in our cumulative logit model above. The study then propose a model, which would allow the marginal utilities to vary at the individual level:

$$Z_{ij} = X'_{ij}\beta_i + \epsilon_{ij} \qquad (3.9)$$

where the ϵ_{ij} are again independent of everything else, and of each other, either extreme value, or normal. We can also write this as

$$Z_{ij} = X'_{ij}\bar{\beta} + \vartheta_{ij} \qquad (3.10)$$

where

$$\vartheta_{ij} = \epsilon + X_{ij} \cdot (\beta_j - \bar{\beta}) \qquad (3.11)$$

The expression in equation (3.11) is no longer independent across categories. The key ingredient is the vector of individual specific risk factor parameters β_i, for example, preceeding birth intervals, severity of household poverty or multiple birth could vary for each category in each binomial logit model and the situation is now been represented by the random term, ϑ_{ij} introduced in equation (3.10) to salvage the situation noted in the results above. The is a formulation of a random multinomial logit (probit) model. For a general discussion of such models and their properties in approximating general choice patterns, see McFadden & Train (2000) and Heckman & Singer (1984).

One possibility assume the existence of a finite number of types of individuals, similar to the mixture models suggested by Heckman & Singer (1984) in duration set-

tings: $\beta_i \in \{\beta_1, \beta_2, \ldots, \beta_k\}$ with

$$Pr(\beta_i = b_k | X_i) = \pi_i \tag{3.12}$$

Or equivalently

$$Pr(Y_i = j | X_i) = \frac{\exp(\mathbf{X}_{ij}\beta)}{1 + \sum_{i=0}^{J} \exp(\mathbf{X}'_{ij}\beta)} \tag{3.13}$$

co-variables, $\mathbf{X}$ are not fixed effect covariates as applicable in the analysis above and they can include unobserved heterogeneity random effect factors. To estimate the parameters in the equation (3.12), one can appeal to the Gibbs sampling with the unobserved β_i as additional unobserved random variables may be an effective way of doing inference. Alternatively, one can use EM algorithm due to Dempster et al. (1977). See Heckman & Singer (1984) and Boxall & Adamowicz (2002) for more discussions on random utility models.

Chapter 4

A Hierarchical Bayesian Modeling of Geographical Pattern of Childhood Mortality in Nigeria

Despite remarkable growth recorded by many economies in the last two decades, many developing countries have failed to attain the target Millennium Development Goals (MDGs 1) four(4), the (reduction of under-five mortality by two-thirds between 1990 and 2015) and seven (7), the targets for water and sanitation in urban. Five countries accounted for half of the global infant mortality with Nigeria being the third largest contributor to the under- five mortality rate among children in sub-Saharan Africa (Black et al., 2003; Ayoola et al., 2005). In 2013, the mortality rates for the five countries were: India (24%), Pakistan (10%) , Nigeria (9%), the Democratic Republic of Congo (4%) and Ethiopia (3%) as reported in You et al. (2013). According to a World Bank report, the prevalence of high child mortality in Africa is concentrated in the four sub-Saharan countries of Malawi, Nigeria, Tanzania and Zambia. In 2003, the mortality rates among children less than five years old were estimated at 187 per 1000 live births for Malawi, 183 for Nigeria, 165 for Tanzania, and 202 for Zambia, which are among the highest in the world (Bryce et al., 2003).

Globally, about a billion people still lack access to improved drinking water and approximately 2.5 billion lack improved toilet facilities, which are major causes of diarrhoea infections(Montgomery & Elimelech, 2007; Bartram & Cairncross, 2010). The unimproved hygiene during food preparation, contaminated water, open defecation

and improper faeces disposal could also result in diarrhoea among children, which globally accounts for approximately 1.4 million child deaths each year (Bartram & Cairncross, 2010; Oloruntoba et al., 2014). In a study recently conducted by Black et al [8], it was reported that an estimated 8.8 million children died worldwide from infectious diseases and about 68% (5.970 million) death was caused by diarrhoea. However, Aiello et al. (2008) previously reported that access to improved water and sanitation can lead to a reduction in cases of child diarrhoea and childhood mortality rates.

The major contributory cause of child mortality is attributed to individual family poverty levels or poor household's environments, highly concentrated in rural areas or slums in big cities (Wagstaff, 2002; Fotso, 2006; Isunju et al., 2011). The household poverty and poor environments could exacerbate the problems of poor health and disease prevalence among children, and hence, the high mortality risks. It has been suggested that health inequalities not only reflect the poor health of the most disadvantaged people, but also the apparently limitless health benefits associated with rising socioeconomic status (Oloruntoba et al., 2014; Pearce et al., 2010).

A good number of studies have investigated the health inequality of sub- populations from the perspective of geography, epidemiology, and public health showing that where people live significantly affects their health outcomes are well detailed in the literature (Pearce et al., 2016; Pearce & Dorling, 2006; Curtis, 2004). Some studies commonly employ disease mapping models and applications. A wide range of these studies include sudden infant death syndrome by Lawson et al. (2000), lip cancer in Scotland by Clayton & Kaldor (1987), child mortality by Marshall (1991), and stomach and bladder cancers in Missouri by Tsutakawa et al. (1985). Other studies have found significant associations between proximity to industrial sites and leukemia and lymphoma as reported in Ramis et al. (2009). Recently, a study conducted by Martuzzi et al. (2010) on congenital anomalies and total cancer mortality has shown that the diseases were found to be associated with waste-related environmental pollution.

The challenge of the geographical analysis of health is that it has to deal with methodological uncertainties as well as social and political issues. Methodological uncertainties are caused by issues of ecological fallacy, scale, modifiable areal unit problems (MAUP) and spatial autocorrelation (Shaw et al., 2001; Wong, 2009). The statistical challenge may arise while making inference about a sub-population or area characteristics regarding an individual within the population. These statistical issues are commonly evolved during analyzing the small area estimation of aggregated data, which also require taking local spatial correlation into account (Cressie, 1993; Bernardo et al., 2003). For example, Beale et al. (2008) also recognized another challenge of data sparseness, which is a major problem in small area analysis, es-

pecially when it involves rare diseases. A small number of observed and expected disease occurrences at health unit, district or regional level can lead to unstable risk estimates or unusual relative risk estimates Ancelet et al. (2012). To handle the problem of over-dispersion and sparsity, random effect models are commonly formulated to deal with the problems arising from high varying population sizes for count data within a hierarchical framework as suggested in the literature (Wakefield, 2006; Lee & Durbán, 2009; Fong et al., 2010).

This study therefore used an exploratory method to estimate the SMR of each state (district) in Nigeria and mapped it onto the geographical regions to highlight unusual clusters of low (high) child mortality in the country. The study then proposed Bayesian hierarchical models to capture the unmeasured random heterogeneity effects in child mortality data and estimated the geographical inequalities of the under-five mortality prevalence across the districts (states). The statistical inference was performed within a full Bayesian framework.

The paper is structured in the following order. In Section 2, the study describe the study design, data exploration, and the disease mapping models, including exploratory data analysis. Section 3 described the Bayesian hierarchical models within generalized linear mixed models. In Section 4, the proposed models are applied to under-five mortality rates from the 2013 Nigeria DHS. Section 5 presents the discussion and the concluding remarks of the present study.

4.1 The Data Exploration

The common sources of data for cause-specific mortality include vital registration systems, sample registration systems, nationally representative household surveys and sentinel Demographic Surveillance Sites for epidemiological studies. With an exception of a few countries, such as South Africa, reliable and functioning vital registration systems have been presented a challenge in supporting attribution of causes of child death in many low-middle income countries, particularly in sub- Saharan Africa (Osterbauer et al., 2012; Rudan et al., 2005).

The main source of data for researchers to guide policy makers in a developing country such as Nigeria is the National DHS conducted by the Data Measure program. The United States Agency for International Development (USAID) has provided the technical assistance and funding to conduct surveys in several developing countries, thereby promoting global understanding of public health. The DHS program collects survey data nationally on a variety of socio-demographic and health related issues. The survey collected information about the background of the respondents,

specifically collected information on fertility levels, marriage, fertility preference, awareness and use of family planning methods, child mortality and child nutrition. Detailed information and procedures about the data collection, and questionnaires have been published elsewhere by Nigeria Demographic and Health Survey (2014). The 2013 NDHS survey conducted by the DHS measure used a multi-stage cluster design consisting of 40 320 households in 904 clusters with 372 in urban areas and 532 in rural areas. The survey successfully interviewed 38,948 women occupied in 38 520 households nested in 886 clusters. This yielded a household response rate for women of 99 %. Data extracted from the 2013 NDHS for the present study are: the number of children born between 2008 and 2013, the number of children alive and counts of child deaths at the time of the survey, the proportion of poorest and poor households, the number of cases (children) experiencing diarrhoea two weeks prior to the survey, the number of households using solid cooking fuels such as, coal, charcoal, fire wood, cow dung and agricultural crop residues.

For the purpose of the present study, Figure 4.2 shows the geographical map of Nigeria showing 36 states (districts) and the Federal Capital Territory, Abuja. Nigeria comprises of six geopolitical regions; North-East, North-West, North-Central, South-East, South-South, and South-West which are sub-divided into 36 administrative states and the Federal Capital Territory (FCT). The population groupings within the geopolitical regions and states are relatively homogeneous. Also, the people's cultural beliefs such as the demographic characteristics, arid environment factors and socio-cultural structures are considered similar within the geopolitical zones and states.

Figure 4.1: Map of Nigeria showing 37 districts(36 states and Federal Capital Territory (FCT), Abuja)

In disease mapping, the first step is the removal of the effect of the confounding

factors on the risk estimate in the study population through distribution standardization. Standardization of mortality rates(SMR) or disease incidence is a basic tool in both demography (Rothman et al., 2008) and epidemiology (Rothman et al., 2008; Woodward, 2013). The most frequently used method in epidemiology is the traditional method for estimating the relative risk is the internal standardization method, which calculates the expected disease counts as functions of the observed numbers of cases. Lawson et al. (2003) had earlier detailed the Bayesian methodology for estimating SMR, its inherent challenge and procedure for the application. Recently, Wang et al. (2019) has investigated that mathematical expressions such as SMR are regarded as incoherent and not generative in probability theory, because the observed count appears on both sides of the equation.

For the purpose of present study, we first applied the internal standardized method to the Nigeria DHS data. Consider the death counts, Y_k aggregated data over a state (district), say, 37 ($k = 1, \ldots, 37$) states , where the mother, k resides in Nigeria. The SMR is calculated as

$$SMR_k = \frac{Y_k}{E_k} \tag{4.1}$$

where, E_k is computed by

$$E_k = n_k \left(\frac{\sum_{i=1}^{37} Y_k}{\sum_{k=1}^{37} n_k} \right).$$

In equation (4.1), Y_k is a random variable representing the number of observed cases (under-five deaths) in each kth state (district) and n_k represents the number of children at risk(the number of under-five children in each state, k). In addition, SMR_k is calculated as the ratio of observed number of child death cases to the expected number of cases in the kth state, representing the risk of each kth small area . Whenever the value of SMR is greater (lower) than one (1), it indicates that the area (state) k has a higher (lower) than the average disease risk of the whole region. For example, for $SMR_k = 1.25$, it can be said that the area k has a 25% higher risk of the disease (childhood morbidity). These quantities, SMR_k are plotted as a crude map. This estimator is unbiased, and is frequently used by epidemiologists. However, this estimate is based only on a sample size of one and hence it is not really statistically useful because it is a saturated model. Some of the advantages and disadvantages of a crude map of SMR have been highlighted in Lawson et al. (2000).

In recent years, the attempts to map incidence and mortality from diseases such as cancer have been explored. Such maps usually display either relative rates in each district or province, as measured by a SMR or similar index. The standard models are detailed in the literature on the empirical methods and its applications can be found in Lawson et al. (2003) and Lesaffre & Lawson (2012). According to Wakefield

(2006), the mean of the estimator,$\hat{\theta}_k$ and its variance, which will be large if the expected number of incidence cases is small. This is one of the disadvantages of using the SMR. Other disadvantages are discussed by Lawson (2013), showing that the SMR is based on a ratio estimator.

Figure 4.2(a) displays the map of observed mortality counts across the 37 states (districts) in Nigeria, Figure 4.2 (b) is the map of SMRs of the child mortality. Table 4.1 presents the summary statistics of observed death counts and SMR estimates, with SMRs having their mean value of 0.920 (standard deviation: 0.306). Although, the evidence of observed lower SMRs were recorded in the southern regions of the country, the geographic variation in child mortality with clustering of high mortality observed in the northern states with a relatively low mortality prevalence (isolated area) of Borno state is apparent. No clear spatial pattern emerges from the map.

Figure 4.2 (a) displayed the crude mortality counts, and clusters of child mortality

(a) observed death counts

(b) crude standardized mortality ratio SMR

(c) proportion of poor households

(d) smoothed relative risk RR map from independent Poisson-Gamma distribution
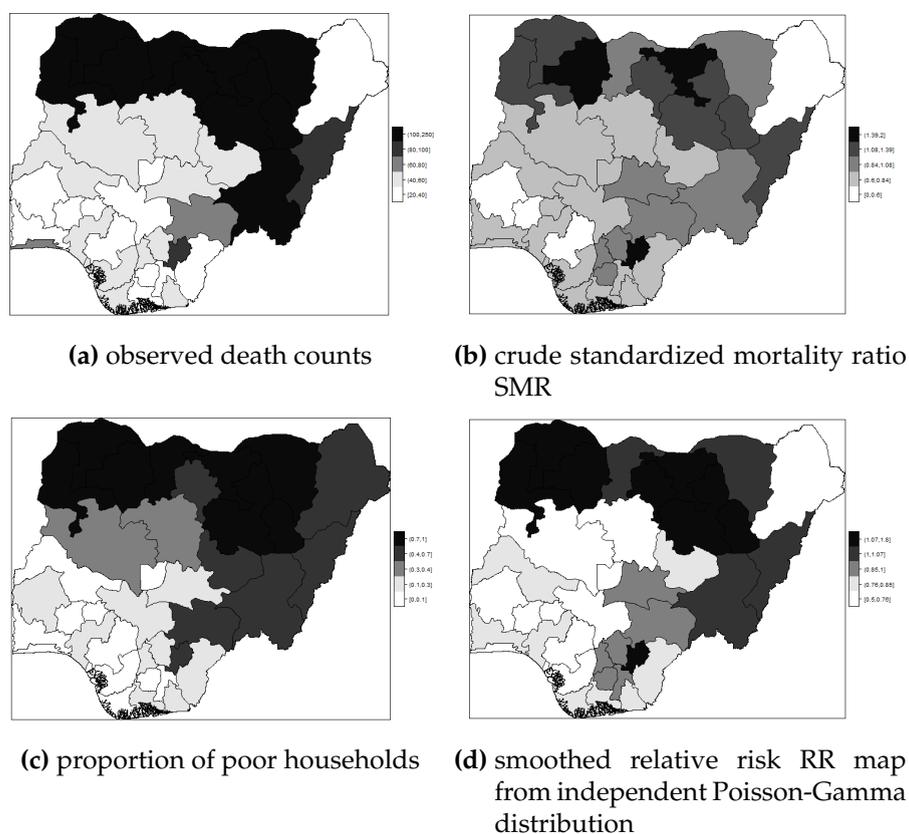
Figure 4.2: Descriptive summary maps: (a) observed death counts (b) crude standardized mortality ratio, SMR (c) proportion of poor households (d) smoothed relative risk RR map from independent Poisson-Gamma model based on 2013 Nigeria DHS

could be observed on the black regions and concentrated in the northern part of the country. Figure 4.2 (c) shows that most northern states are darker, while the south-

ern regions are lighter signifying the severity of household poverty from light (least materially deprived) to darkest (most materially deprived). This indicates that there are more economically deprived households in northern Nigeria than in the southern regions. Figure 4.2 (b) represents the map of SMR, showing that some states were observed with an unusual low (high) mortality prevalence, while such districts (states) are bordered /surrounded by relatively high mortality states. For instance, a case of isolated low prevalence could be seen at state, like Borno State, which is surrounded by states with relatively high child mortality. There was also evident of geographical disparities with no clear patterns. Another scenario can be seen in a state like Zamfara (with a high mortality rate (130)), which shares boundaries with states in the north -western region with other relatively high mortality states such as Kebbi (109.55) and Sokoto (124.77). These scenarios may better be handled by a spatial random effect model or the BYM model, which exhibit borrowing strength that the close neighbours to each other share similarities in their spatial prevalence. The summary statistics, standardized mortality ratios (SMR), model parameters es-

Table 4.1: Descriptive Statistics of the death counts, the expected counts and standardized mortality ratios(smr) on under-five deaths by State for 5-year period(2009-2013) in Nigeria DHS 2013

	Min.	1st Qu.	Median	Mean	3rd Qu.	Max.
Observed counts (y)	21	39	51	78	104	229
Expected Counts (E)	43.73	51.15	61.42	78.00	98.64	197.80
SMR	0.4105	0.7251	0.8303	0.9097	1.0770	1.7570

timates and predictive probability maps of under five mortality are presented in this section. The first step of the analysis is to compute SMRs and plots of SMR maps to examine the geographical patterns of child mortality. Figure 4.2(a) displayed the map of observed mortality counts across the 37 states(districts) in Nigeria, while Figure 4.2(b) is the map of standardized mortality ratios (SMR) of the child mortality and corresponding Table 4.1 with SMRs vary widely around their mean, 0.920, (standard deviation: 0.306). Although, the evidence of observed lower mortality risk(SMRs) occurred in the South- Western states of Nigeria and some parts of the South-East and South-South regions of Nigeria, it is apparent to notice the geographic variation in under-five mortality with clustering of high mortality observed in the northern states with a relatively low mortality prevalence (isolated area) of Borno state. No clear spatial pattern emerges from the map Figure 4.2a on the observed counts.

Figure 4.2 displays the map of (a) the observed child death counts, (b) crude standardized relative risk and (c) Poverty index(d) smoothed relative risk RR map from independent Poisson- Gamma distribution. From Figure 4.2 a, (the raw death counts

map), the cluster of child mortality is found to be concentrated in the north part of Nigeria. With reference to Figure 4.2c, most northern states are more darker than the southern regions. This indicates that there were more economically deprived households living in the northern Nigeria than the southern regions.

Figure 4.2 b displayed the map of crude SMR, showing few districts (states) were observed with an elevated usual(low or high) mortality incidence, while such districts(states) are bordered /surrounded with relatively high counts. For instance, the observed (isolated) low incidence of mortality in Borno State, which was surrounded by states with relatively high death counts was an evidence of geographical disparities with no clear patterns. Another scenario ocan be seen in a state like Zamfara (with high mortality count (130)), which shares boundaries with states in the North - West region with relative high mortality risk states such as Kebbi (109.55) and Sokoto (124.77) (refer to Table 4.2. These scenarios may be better handled by spatial random effect model or the BYM model, which exhibit borrowing strength i.e. regions close to each other share similarity .

Table 4.2 presented in the appendix. The Table displayed the number of child deaths, total births, expected deaths, and relative frequency distribution. The study involved 31482 children born between 2009 and 2013. Out of which 2886 children died before reaching age five years. Zamfara recorded the highest child mortality and relative frequency of 229(7.96) and the second highest occurred at Kano 221(7.66) both states are found in the north-west regions of Nigeria. The lowest under-five mortality was recorded in Osun state of 21(0.73).

Figure 4.2 (b) also depicts the empirical estimation of SMR of child mortality. The geographical patterns of child mortality distribution are similar for both Figures (a)raw mortality count map and the crude SMR (b). The clusters of high mortality or concentrated mortality found in the northern regions could be attributed to partly unobserved heterogeneity and environmental factors. The smooth SMR map (d) shows evidence of localized spatial smoothness and neighbouring states exhibited similar patterns of mortality risk, while other neighbouring regions far apart showed different series of risks. From Figures 4.2 (b) & (d) , the SMR with RR greater than one indicates significantly excess(higher) mortality and they are regions coloured black. The light coloured regions are states signifies low prevalence(RR less than one) of child mortality, while the grey coloured regions are not significant.

Furthermore, the smooth maps (d) was obtained from the independent Poisson model and relative risk (RR mean) was shown in Figure 4.2 (d). The map depicts that such model could not capture the geographical variation in the spatial pattern of the actual mortality count data. For instance, discontinuities can be seen in some states with clustering of high mortality rates in the north when compared with the

Table 4.2: Descriptive distribution of observed counts, total births, expected counts, relative frequency of under-five deaths by state in 2013 NDHS

State	Observed Deaths	Expected deaths	Relative frequency	SMR	Total child birth
Abia	39	46.57	1.35	0.43	508
Adamawa	100	91.76	3.47	1.89	1001
Akwa ibom	43	52.80	1.49	0.87	576
Anambra	46	49.23	1.59	0.35	537
Bauchi	180	131.92	6.24	2.39	1439
Bayelsa	58	75.26	2.01	0.96	821
Benue	61	60.50	2.11	1.11	660
Borno	28	55.00	0.97	0.58	600
Cross river	36	48.22	1.25	0.57	526
Delta	46	63.44	1.59	0.70	692
Ebonyi	92	66.00	3.19	1.69	720
Edo	27	54.55	0.94	0.56	595
Ekiti	33	48.59	1.14	0.63	530
Enugu	46	52.62	1.59	1.00	574
FCT-Abuja	28	45.93	0.97	0.26	501
Gombe	126	105.88	4.37	2.88	1155
Imo	40	43.73	1.39	0.30	477
Jigawa	194	131.64	6.72	2.41	1436
Kaduna	51	80.40	1.77	0.26	877
Kano	221	197.83	7.66	1.65	2158
Katsina	136	133.57	4.71	1.24	1457
Kebbi	152	109.55	5.27	3.39	1195
Kogi	27	44.83	0.94	0.43	489
Kwara	46	62.61	1.59	0.53	683
Lagos	64	87.00	2.22	1.07	949
Nasarawa	58	60.05	2.01	0.66	655
Niger	57	87.64	1.98	1.16	956
Ogun	37	49.14	1.28	0.62	536
Ondo	48	59.40	1.66	0.94	648
Osun	21	51.15	0.73	0.35	558
Oyo	36	60.60	1.25	0.59	661
Plateau	51	61.42	1.77	1.04	670
Rivers	39	49.23	1.35	0.31	537
Sokoto	163	124.77	5.65	1.43	1361
Taraba	123	114.22	4.26	1.25	1246
Yobe	104	98.64	3.60	0.80	1076
Zamfara	229	130.36	7.93	0.08	1422
Total	2886	2886	100	37.4	31482

SMR= standardized mortality ratios=$\frac{Y}{E}$; relative frequency=$\frac{y_i}{\sum n_i} * 100$

observed counts map. Katsina State recorded the highest expected counts and posterior mean RR, but the Poisson model (no random effect) would classify Katsina lower than the actual mortality level. Another scenario in the spatial disparities was also observed in Ekiti State in south-west Nigeria with small expected counts, but the state was an elevated high risk. This dispersion can be attributed to small population size. The independent Poisson sometimes under-estimates the mortality risk such as Balyesa and Lagos, perhaps these could result from a high expected value (denominator).

A careful inspection of the expected counts in Figure 4.2 reveals that higher child mortality risk were detected in some states of the north regions resulting from empirical computation of SMR i.e. Kano, due to large expected count of 197.827 (divisor)(see Table 4.2). Four other states were considered with expected counts of 46.569, 51.153, 61.420 and 131.64 corresponding to approximate percentile values of 0th, 25th, 50th and 90th of the expected counts respectively. It is worth mentioning that the

choice of unusually low relative risk values (SMR) (10th percentile of the expected counts) would establish an epidemiological importance. Interestingly, some districts (states) had unusual low death rates surrounded by neighbouring states with a fairly high mortality risk. The expected mortality rates of the four states correspond to the percentiles (10th - 90th percentile): Abia (46.57), Osun (51.12), Plateau Sate (61.42) and Jigawa (131.64). For example, Plateau state (61.42) had relative low prevalence but is surrounded by states had relatively high mortality. In such cases, the mortality rates in those neighbouring states may have a substantial influence on the smoothing effects on states that share borders. This scenario can be handled by the spatial conditional auto-regressive (CAR) model.

To conclude this section, in comparing the smooth maps of the SMR map and the PG map, it shows that there was no clear difference in the smooth risk maps from both estimates. The empirical approach makes epidemiological sense and provides better understanding of mortality prevalence across the regions in Nigeria. These maps are primarily used as a tool for identifying regions with unusually (low) high risk area, so that further attention can be given to these priority districts (states).

4.2   The Statistical Models

The mapping of mortality rates or disease incidence could provide important information in many epidemiological studies for resource allocation and disease management. To estimate and map crude mortality rates, particularly rare disease aggregated at the administrative unit or regional level can be statistically challenging if the high variability of population sizes over a small area is not taken into account. To mitigate the problem, an exploratory data analysis was carried out by mapping the SMR as suggested by Leroux et al. (2000). The following four models are explored to capture the effects of spatial dependence and overdispersion in the data.

Model 1: Poisson-Gamma Model

The Poisson-Gamma model is sometimes used to model the relative risk of the number of child mortality in a district (state). The relative risk combines with the Poisson likelihood function for the death counts and Gamma prior distribution to yield a Gamma posterior distribution for the relative risk (Clayton & Kaldor, 1987; Lawson et al., 2003).

Let y_i and E_i; $i = 1, \ldots, n$, denote the observed and expected number of death cases in district (state). We also assume the death count that $y_i \sim \text{Pois}(E_i \Psi)$, where Ψ is

the unknown relative risk and Poisson mean, μ is modeled as

$$\textbf{Model 1:} \ \ \mu = E_i \Psi. \tag{4.2}$$

where $i = 1 \ldots 37$ in our study n=37 districts (states). Hence, the Likelihood function for y_i is given as

$$P(y|E_i \Psi) = \prod_{i=1}^{n} \frac{(\Psi_i E_i)^{y_i}}{y_i!} exp(-\Psi_i E_i). \tag{4.3}$$

The prior distribution of the relative risk Ψ_i denoted by $p(\Psi_i)$, which takes a gamma distribution with parameters a and $b > 0$ i.e. $\Psi_i \backsim \Gamma(a, b)$ with mean is $E(\hat{\Psi}_i) = \frac{a}{b} = \mu$ and the variance is $var(\Psi_i) = \frac{a}{b^2} = \sigma^2$. Thus, the prior distribution is denoted by

$$P(\Psi_i|a, b) = \frac{b^a}{\Gamma(a)} \Psi_i^{a-1} \exp(-b\Psi_i) \tag{4.4}$$

Using the equation (4.4), the resulting posterior distribution is given by

$$P(a, b, \Psi|y) \propto \prod_{i=1}^{n} \frac{(\Psi_i E_i)^{y_i}}{y_i!} \exp(-\Psi_i E_i) \prod_{i=1}^{n} \frac{b^a}{\Gamma(a)} \Psi_i^{a-1} \exp(-b\Psi_i) \tag{4.5}$$

The parameters a, b and Ψ are estimated using MCMC via Gibbs Sampling. The Poisson-gamma posterior conjugate can be derived since $P(\Psi_i|a, b, y_i) = Gamma(a + y_i, b + E_i)$. Thus, the posterior mean of Ψ_i is equal to Ψ

$$E(\Psi_i|y_i, a, b) = \frac{a + y_i}{b + E_i} = w_i SMR_i + (1 - w_i)\frac{y_i}{E_i} \tag{4.6}$$

where $w_i = \frac{b}{(b+E_i)}$, represnts a weighted average that indicates on how much the observed SMR_i is shrunk towards the individial expecation, E_i of the posterior mean as explained in Lesaffre & Lawson (2012).

One advantage of the Poisson-gamma model is that it provides a simplified way to accommodate over-dispersion in the model as shown in 4.6, especially since a closed form can be derived to express the relationship between the mean and the variance structures in a simplied version. A drawback is that Poisson-gamma model does not permit the inclusion of covariate(s) in the model according to Best et al. (2005) and Lawson et al. (2003).

Other approach of modeling mortality counts is based on the Poisson cluster model that incorporated the spatial random effects via generalized linear mixed models

suggested in literature, see Lawson (2013) and Mariella & Tarantino (2016) among others. The following spatial random effect models (Poisson-Log-normal, conditional auto-regressive and the BYM models) are also explored below.

Model 2: Poisson Log-Normal model

Clayton & Kaldor (1987) first proposed a Poisson log normal model that combines the relative risk and a normally distributed random variable. The model includes the area-specific random effects or spatially unstructured random effects, v_i and the overall relative risk, Ψ. The model was based on assumptions that the logarithm of relative risk follows a multivariate log-normal distribution with normal prior hyperparameters μ and σ_v^2. The spatially unstructured random effects were modeled as using the normal prior with a zero mean Gaussian prior distribution and its variance, σ_v^2, such that $v_i \backsim N(0, \sigma_v^2)$.

From equation (4.2) above, $\mu_i = E_i \Psi$, the log normal model for the relative risk becomes

$$\textbf{Model 2:}\quad \log(\mu) = \log(E) + \log(\Psi) = \log(E) + \eta. \tag{4.7}$$

where the linear link function $\eta = \log(\Psi) - X'\beta + v_i$.

where v_i is the spatially unstructured random effects that were modeled as using the Gaussian prior distribution with a zero mean and the variance, i.e. $v_i \sim N(0, \sigma_i^2)$, where σ_i^2 represents specific area variance. X is a vector of covariates (such as proportion of poor households, unimproved source of drinking water, unprotected toilet, children having diarrhoea, the proportion of mothers using solid fuels (coal, wood, agricultural residues cow dung etc) as cooking method. Thus, the relative risk provides a more flexible alternative to the independent Poisson model, as stated in Lawson (2013).

Model 3: Conditional Autoregressive (CAR) Model

The conditional autoregressive (CAR) model has been widely used for the analysis of spatial data in different areas, such as demography, geography and epidemiology. This model was introduced by Besag et al. (1991) as a spatial methodology to estimate disease risk, which assumed a region shares spatial similarity with its neighboring regions. In general, the CAR model is a class of Gaussian Markov random fields characterized by a conditional probability density function and is used to capture areas or regions that are highly related in spatial associations to a specific area as discussed in Cressie (1993) and Besag (1974).

Here, u_i is used to represent the spatially structured (correlated) random effects, which is been modeled using the conditional autoregressive prior distribution as proposed by Besag et al. (1991).

Using equation (4.2) above, $\mu_i = E_i\Psi$, the CAR model for the relative risk becomes

$$\textbf{Model 3:} \quad \log(\mu) = \log(E) + \log(\Psi) = \log(E) + \eta. \tag{4.8}$$

and the linear link function becomes $\eta = \log(\Psi) = X'\beta + u_i$.

where u_i is modeled by conditional autoregressive prior distribution due to Besag et al. (1991) defined as $\left(u_i | u_j, j \neq i, \sigma_u{}^2\right) \backsim N\left(\sum_{j \neq i} \frac{w_i u_j}{w_{ij}}, \frac{\sigma_u{}^2}{w_{ij}}\right)$, where area $i \sim j$ are adjacent (neighbours), $w_{ij} = 1$ and zero if they are not. X is a vector of covariates as defined above.

Model 4: Baseg, York and Mollie (BYM) Model

BYM model was introduced firstly by Clayton & Kaldor (1987) and later extended by Besag et al. (1991). BYM model facilitates the splitting of the spatial random effects into two components: spatial random and heterogeneity components. One component represents the spatial structured random effects, denoted by u_i, which accounts for the effects that vary in space (clustering or correlated heterogeneity) and the spatially unstructured effects v_i that takes model the effect of area specific area level.

$$y_i \backsim Poisson(\Psi_i E_i) \tag{4.9}$$

The log relative risk are modeled through

$$\hat{\Psi}_i = \exp(\alpha + u_i + v_i) \tag{4.10}$$

thus, by taking logarithm, we obtain

$$\eta = \alpha + u_i + v_i \tag{4.11}$$

where α is the overall relative risk (intercept). Besag et al. (1991) assumed that the two random effects are independent and requires a specification of independent priors. The prior distribution model for the spatially unstructured v_i are assumed to follow a normal distribution and it is given as $v_i \backsim N(0, \sigma_v^2)$ and the prior distribution for the CAR model as defined above. The variance component parameters $\sigma_u{}^2$ and $\sigma_v{}^2$ control the variability of u_i and v_i respectively as stated in Lawson et al.

(2003). In a full Bayesian analysis, prior distributions are specified for those parameters. We considered gamma distributions for both parameters, as suggested by Bernardinelli & Montomoli (1992).

With the observed death count, $y_i \sim Poisson(E_i \exp(\eta_i))$ and $\mu_i = E_i \exp(\eta_i)$ is the mean of the Poisson distribution, then the BYM model for the relative risk becomes

$$\textbf{Model 4:} \quad \log(\mu) = \log(E) + \log(\Psi) = \log(E) + \eta. \tag{4.12}$$

with the linear link function becomes $\eta = \log(\Psi) = X'\beta + u_i + v_i$. and the log relative risk $log(\Psi_i) = \eta_i$.

Fitting a generalized linear mixed model with the log link function, we have

$$\log(\mu_i) = \log(E_i) + X'\beta + u_i + v_i \tag{4.13}$$

where $y, \mathbf{X}, \beta, E$ and μ are observed cases, X, the vector of covariates, the associated parameters, the expected number of cases, and the relative risks of child mortality prevalence respectively.

4.3 Parameter Estimation

Defined the likelihood function as

$$l(\boldsymbol{\beta}, \boldsymbol{u}, \boldsymbol{v} = \prod_{i=1}^{n} \frac{(E_i \exp(\eta_i))^{y_i} \exp(-E_i \exp(\eta_i))}{y!} = P(\boldsymbol{y}, \boldsymbol{E}, \Psi | \boldsymbol{\beta}, \boldsymbol{u}, \boldsymbol{v}) \tag{4.14}$$

The prior distribution for $\boldsymbol{\beta}$ with inverse variance(precision, $\tau_\beta = 1/\sigma_\beta^2$)

$$p(\beta) = \left(\frac{1}{2\pi}\right)^{p/2} \left(\frac{1}{\tau_\beta}\right)^p \exp\left(-\frac{1}{2} \sum_{p=0}^{p} \frac{\beta_p^2}{\tau_\beta^2}\right) \tag{4.15}$$

prior distribution for the area -specific random effect v_i is defined by

$$p(\boldsymbol{v}) = \left(\frac{1}{2\pi}\right)^{n/2} \left(\frac{1}{\tau_v}\right)^n \exp\left(-\frac{1}{2} \sum_{i=1}^{n} \frac{v_i^2}{\tau_v^2}\right) \tag{4.16}$$

and prior distribution for the CAR structure u_i is defined by

$$p(\boldsymbol{u}) = u_i | u_j, j \neq i, \tau_u^2 \sim N\left(\sum_{j \neq i} \frac{w_{ij} u_j}{w_{ij}}, \frac{\tau_u^2}{w_{ij}}\right) \sim CAR(0, \tau_u^2) \qquad (4.17)$$

Putting (4.14) through (4.17) together, the posterior distribution is obtained as

$$p(\boldsymbol{\beta}, \boldsymbol{u}, \boldsymbol{v}, \tau_\beta^2, \tau_u^2, \tau_v^2 | \boldsymbol{y}, \boldsymbol{E}, \boldsymbol{\Psi}) \propto p(\boldsymbol{y}, \boldsymbol{E}, \boldsymbol{\Psi} | \boldsymbol{\beta}, \boldsymbol{u}, \boldsymbol{v}, \tau_\beta^2, \tau_u^2, \tau_v^2) p(\boldsymbol{\beta}, p\boldsymbol{u}, p\boldsymbol{v}) \qquad (4.18)$$

Therefore yields

$$
\begin{aligned}
p(\boldsymbol{\beta}, \boldsymbol{u}, \boldsymbol{v}, \tau_\beta^2, \tau_u^2, \tau_v^2 | \boldsymbol{y}, \boldsymbol{E}, \boldsymbol{\Psi}) = & \prod_{i=1}^{n} \frac{(E_i \exp(\eta_i))^{y_i} \exp(-E_i \exp(\eta_i))}{y!} \\
& \times \left(\frac{1}{2\pi}\right)^{p/2} \left(\frac{1}{\tau_\beta}\right)^{p} \exp\left(-\frac{1}{2} \sum_{p=0}^{p} \frac{\beta_p^2}{\tau_\beta^2}\right) \\
& \times \left(\frac{1}{2\pi}\right)^{n/2} \left(\frac{1}{\tau_v}\right)^{n} \exp\left(-\frac{1}{2} \sum_{i=1}^{n} \frac{v_i^2}{\tau_v^2}\right) \\
& \times \left(\sum_{j \neq i} \frac{w_{ij} u_j}{w_{ij}}, \frac{\tau_u^2}{w_{ij}}\right)
\end{aligned}
\qquad (4.19)
$$

The hyperprior distribution for the precision parameters τ_u^2, τ_v^2 and τ_β^2 are $\tau_u^2 \sim$ Gamma$(0.5, 0.005)$, $\tau_v^2 \sim$ Gamma$(0.5, 0.005)$ and $\tau_\beta^2 \sim$ Gamma$(0.5, 0.01)$ respectively. A noninformative normal disribution is assumed on the fixed effect, i.e. $\beta \sim N\left(0, \sigma_\beta^2\right)$. The τ_v^2 reflects the amount of extra Poisson variation in the data according to Lawson (2013). The precision parameters τ_u^2 and τ_v^2 control the variability of u and v respectively. The parameter estimation was executed via Bayesian Markov Chain Monte Carlo (See Section 2.3.1). Convergence of the MCMC was reached at 15000 iteration after a burn-in period of 5,000 sample and thinning of every 90th element of the chain. Posterior statistics of the UH, CAR and the BYM model are presented in Table 4.4

The model performance was investigated via deviance information criterion (DIC) which is due to Spiegelhalter et al. (2002b) given as $DIC = \bar{D} + pD$, where $\bar{D}$ is the posterior mean of the deviance by $\bar{D} = E_{\boldsymbol{\theta}|\boldsymbol{y}}(D)$, which measures the goodness of fit defined as $D(\bar{\theta}) - 2\log L(\text{data}|\boldsymbol{\theta})$, where $L(\text{data}|\boldsymbol{\theta})$ is the likelihood function for the observed data and $\boldsymbol{\theta}$ is the vector of model parameters. The pD is the effective number of model parameters and it is computed as the difference be-

tween the deviance posterior mean and the parameters posterior mean evaluated by $pD = E_{\theta|y}(D) - D(E_{\theta|y}(\boldsymbol{\theta})$, which represents a measure of model complexity and penalizes over-fitting. The model with lower Deviance, $\bar{D}$ indicates good fit and lower value of pD indicates a parsimonious model. Therefore, the model having smaller value of DIC is the most preferred model as it has achieved a more optimal combination of fit and parsimony.

The parameter estimation was done using Bayesian Markov Chain Monte Carlo via Gibbs Sampling. The convergence of the MCMC was achieved at 15,000 iteration after a burn-in period of 5,000 sample and thinning of every 90th element of the chain. The hyper-prior distributions assumed for the precision parameters $\tau_u^2, \tau_v^2,$ and τ_β^2 are $\tau_u^2 \sim \Gamma(0.05, 0.005), \tau_v^2 \sim \Gamma(0.05, 0.005)$ and $\tau_\beta^2 \sim \Gamma(0.05, 0.01)$ respectively. The coefficients of the covariates of the regression model are assumed to be normally distributed given as , $\boldsymbol{\beta} \sim \mathcal{N}(0, \tau_\beta^2)$. All model analyses were carried out in WinBUGS after Spiegelhalter et al. (2002a).

4.4 Data Analysis and Results

Table 4.3 presents the estimates of the parameters and goodness of fit for the hierarchical models discussed in the previous section. The non-spatial methods (P-Gamma model) does not account for autocorrelation in the residuals, although they appear to perform reasonably well overall. Although the CAR model and BYM model each provides important information about clustering of the childhood mortality relative risk pattern, one would recommend that the BYM is the best fitted model for Nigerian child mortality data, since it yielded the lowest value of the DIC = 285:310 and with a lower pD = 25.04. The CAR model had DIC = 286.40 and pD=(24.16) as the goodness of measure, as the CAR model competes closely with the BYM model. However, the BYM model is the most preferred one due to its robustness and at the same time one can evaluate the proportional of variation that can be attributed to spatial dependence (clustering) and the variation due to random heterogeneity effect structure of the mortality prevalence.

Table 4.4 presents the posterior statistics of the fitted hierarchical models. It can

Table 4.3: *Deviance information criteria (DIC) and the model goodness of fit based on 2013 Nigeria DHS*

Model	$D(\bar{\theta})$	pD	DIC
PG	256.885	31.217	288.101
UH	261.426	25.278	286.704
CAR	262.233	24.163	286.396
BYM	260.267	25.043	285.310

be observed that the posterior mean of P-G model is 0.923: 95% CI (0.826, 1.030), which is approximately the same as the mean of the SMR of 0.920 and standard deviation, 0.306. The overall population parameters, a = 10.310, (6.232, 15.970) and b = 11.20 (6.680, 17.350) from the Poisson Gamma model. The Poisson-log normal model yielded a precision variance, τ_v^2 of 41.76 with a standard deviation of 0.168. This indicates that the relative risk of child mortality at any given state is similar (less heterogeneous) to that of its neighbours. The CAR model's precision variance, $\tau_u^2 = 14 : 34; (5,006 to 35.47)$ and standard deviation of 0.291, which indicates that the geographic patterns of under five mortality exhibits more of clustering across the selected administrative units (states)in Nigeria. The precision variance parameter of the BYM model has CAR precision variance, τ_u^2 is equal to 56.98; 95% CI (6.104, 339.0) and stadard deviation of 0.291, which indicates that the geographic patterns of under-five mortality in the country exhibits more of clustering across the selected administrative units (states) in Nigeria. The variance parameter from the BYM model with CAR precision variance, $\tau_u^2 = 56.98$; 95% CI (6.104, 339.0) and $\sigma_u = 0.291$. The spatial heterogeneity component of variation in BYM model are precision variance, $\tau_v^2 = 330.60$; $95\%(23.84 - 2101)$ and $\sigma_v = 0.0991$. From BYM model, one can deduce the proportion of the variation that is due to clustering as $\alpha = \frac{\sigma_u}{\sigma_u + \sigma_v} = 69.06\%$ and the proportion of variability attributed to the heterogeneity random effect is $1 - \alpha = 30.93\%$.

The results revealed that the geographic pattern of under-five mortality at the administrative unit(states) level across the country exhibits more clustering than the spatial heterogeneity variation, as evidenced from the estimates. The geographic pattern of variation of the under-five mortality can be attributed to clustering from the exposure to local environmental factors, underlying ecological indices or severity of poverty index at local- community level.

Further more, the risk factors were presented along with posterior statistics in Table 4.4. The results revealed that the estimated overall(intercept) relative risk effect of the models are: PLN $\beta_0 = -0.137, (-0.209, -0.075)$, CAR $\beta_0 = -0.137$, 95% CI (-0.182 , -0.092), and BYM model $\beta_0 = -0.138$, 95% CI $(-0.200, -0.080)$. The risk effect of these models were significantly different from zero and negative. These models (CAR and BYM) consolidate the result of UH model that overall child mortality risk. A negative coefficient overall indicates that a decreasing relative risk of childhood mortality by keeping the (fixed covariates) determinant factors of under-five mortality constant. The household poverty variables were significant and positive for all the models (UH,CAR and the BYM) with parameter estimates UH: 1.653, 95%CI (0.773 to 2.491), CAR : 2.088 95%CI (1.088, 3.165), BYM: 2.003, 95%CI (1.101, 3.006). The results showed that the the household poverty would increase the relative risks

Table 4.4: Posterior estimates of the model parameters and ecological covariates

Pars.	PG Est.	PG 95%CI	UH Est.	UH 95%CI	CAR Est.	CAR 95%CI	BYM Est.	BYM 95%CI
β_0	—		−0.136	(−0.209, −0.065)	−0.137	(−0.182, −0.093)	−0.138	(−0.196, −0.080)
a	10.310	(6.232, 15.970)	—	—				
b	11.200	(6.680, 17.350)	—	—				
μ	0.923	(0.826, 1.030)	—	—				
σ^2	0.088	(0.052, 0.142)	—	—				
β_1			0.052	(-0.460, 0.614)	0.130	(-0.452, 0.698)	0.173	(-0.372, 0.730)
β_2			0.350	(-0.149, 0.875)	0.362	(-0.100, 0.857)	0.353	(-0.190, 0.851)
β_3			-0.095	(-0.650, 0.427)	-0.247	(-0.762 − 0.291)	-0.226	(-0.771, 0.333)
β_4			1.653	(0.773, 2.491)	2.088	(1.088 − 3.165)	2.003	(1.101, 3.006)
β_5			-0.306	(-1.066, 0.520)	-0.491	(-1.383, 0.350)	-0.516	(-1.591, 0.430)
τ_u^2					14.34	(5.006, 35.47)	56.98	(6.104, 339.0)
σ_u					0.291	(0.168, 0.447)	0.221	(0.054, 0.405)
τ_v^2			41.760	(16.75, 100.5)			330.6	(23.84, 2101)
σ_v			0.168	(0.100, 0.244)			0.099	(0.022, 0.205)

The covariate parameters in Table 4.4 are designated as follows:
β_1 =the proportion of children, who had diarrhoea two weeks prior to the survey,
β_2= proportion of children, whose households used unprotected
latrine and open defecation (unhygienic toilet /poor sanitary facility),
β_3= proportion of households, who did not have access to improved
drinking source or water pipe borne, β_4= proportion of poor household and
β_5= proportion of households, who used solid fuels cooking sources
(coal, charcoal, crop residues) based on 2013 Nigeria DHS.

of under five mortality among the children of (less endowed households) most economically deprived households. Other covariates in the model were not significant for the childhood mortality. However, the childhood diarrhea and unhygienic toilet/ sanitation showed positive association with the under five child mortality, although they were not significant in this case. The household cooking with solid fuels (charcoal, crops residues etc.) and drinking from unprotected water were negatively associated with child mortality, and the effects were not significant.

4.4.1 Geographic variation and Classification of relative risk

Figure 4.3 presents the maps for posterior mean and the relative risk (RR) value for the UH model, which is used for the classification of the states according to the relative risk (RR) value and significance probability ($\text{RR} > 1$) for UH model. The geographical variation in the relative risk values range from 0.421 to 1.928 corresponding to Osun (minimum) and Zamfara (maximum) state respectively. The relative risk above 1, ($\text{RR} > 1$) indicates that the under-five mortality prevalence are higher in those states than the overall relative risk .

Table 4.5 presents the results of the Poisson log-normal model with districts (states), which classified states according to the relative risk values. The results showed that 6 states had a high significant relative risk above 1. The higher prevalence of under-five mortality risk detected in the five northern states and one isolation case, Ebonyi state found in of south east region of the country. The relative risk probability map

displayed in Figure 4.3 for the UH model, classifying from the lowest risk value to high relative risk areas. The geographic variation could be accounted for the unobserved heterogeneity factors, that could not be captured by the measurable covariates (underlying risk factors).

Table 4.6 presents the posterior risk estimates and 95% CI for results of the CAR
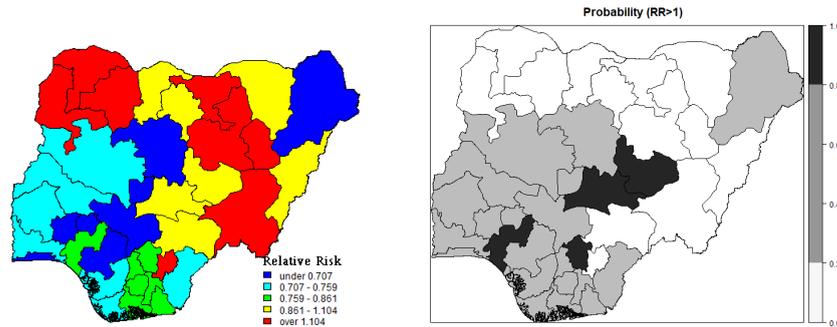
Figure 4.3: Relative Risk of childhood mortality prevalence and significance probability (RR > 1) for UH model based on 2013 Nigeria DHS.

Table 4.5: Relative risk estimates and corresponding 95% credible intervals (CI) for the UH model and states grouped by RR from low to high risk of under-five mortality based on 2013 Nigeria DHS

RR : < 0.05 significant Low risk		RR : 0.500 − 0.999 not significant		RR > 1 significant high	
Osun	0.574 (0.421 , 0.747)	Imo	0.786 (0.604 , 1.014)	Ebonyi	1.275 (1.049 , 1.539)
Edo	0.578 (0.435 , 0.731)	Anambra	0.795 (0.619 , 1.018)	Sokoto	1.289 (1.114 , 1.476)
FCT-Abuja	0.642 (0.484 , 0.822)	Enugu	0.807 (0.633 , 1.016)	Kebbi	1.351 (1.158 , 1.561)
Kogi	0.672 (0.512 , 0.854)	Ondo	0.814 (0.648 , 1.003)	Bauchi	1.360 (1.177 , 1.548)
Ekiti	0.683 (0.522 , 0.865)	Nasarawa	0.893 (0.717 , 1.110)	Jigawa	1.441 (1.259 , 1.640)
Kaduna	0.696 (0.546 , 0.853)	Plateau	0.938 (0.736 , 1.148)	Zamfara	1.710 (1.502 , 1.928)
Lagos	0.698 (0.544 , 0.870)	Benue	1.023 (0.831 , 1.246)		
Borno	0.706 (0.524 , 0.900)	Adamawa	1.057 (0.881 , 1.252)		
Kwara	0.710 (0.559 , 0.892)	Katsina	1.057 (0.900 , 1.221)		
Delta	0.720 (0.575 , 0.891)	Yobe	1.097 (0.910 , 1.292)		
Niger	0.721 (0.578 , 0.876)	Kano	1.098 (0.963 , 1.243)		
Oyo	0.721 (0.559 , 0.904)	Taraba	1.105 (0.932 , 1.287)		
Ogun	0.751 (0.579 , 0.943)	Gombe	1.174 (0.992 , 1.376)		
Cross river	0.757 (0.589 , 0.949)				
Bayelsa	0.758 (0.606 , 0.927)				
Abia	0.759 (0.592 , 0.966)				
Rivers	0.774 (0.608 , 0.969)				
Akwa ibom	0.778 (0.617 0.969)				

model. The states are classified by the credible intervals from significant low, not significant and significantly high. The geographical variation in the relative risk values range from 0.438 to 1.910. Six (6) states are found to have a significantly higher prevalence with relative risk above 1(RR >1.000). This indicates that a higher mortality prevalence occurred in those states than the overall relative risk. The lowest estimated risk value occurred at Osun state: 0.0.572 (0.438, 0.714) and highest risk

recorded at Zamfara state with risk value of 1.680 (1.479 to 1.910). The probability map corresponding to the Table 4.6 was displayed in Figure 4.4, showing relative low risk of under-five mortality detected on the map in the south west states and the high prevalence found in the north regions of Nigeria.

Table 4.7 presents the results of the BYM model with districts(states) categorized ac-
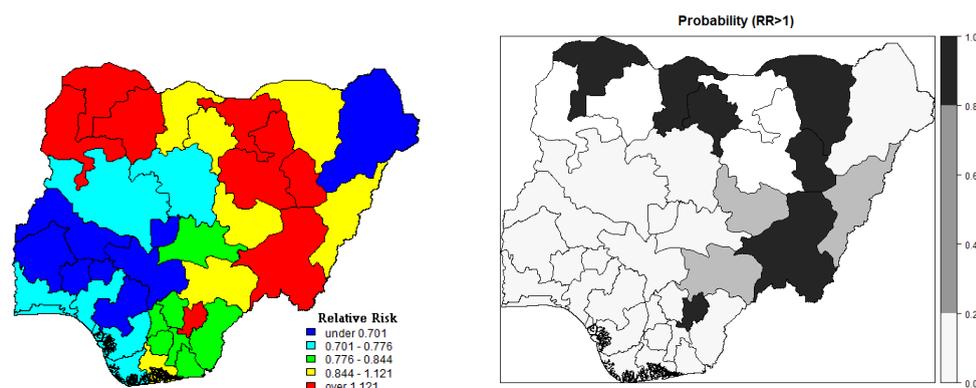
Figure 4.4: Relative Risk of childhood mortality prevalence and significance probability (RR > 1) for for CAR model

Table 4.6: Relative risk estimates and corresponding 95% credible intervals (CI) for CAR model and states grouped by RR from low to high risk of under-five mortality based on 2013 Nigeria DHS

RR : < 0.05 significant low		RR : 0.500 − 0.999 Not significant		RR > 1 significant high	
Osun	0.572 (0.438, 0.714)	Akwa ibom	0.807 (0.640, 1.006)	Sokoto	1.316 (1.133 , 1.515)
Edo	0.591 (0.454, 0.736)	Enugu	0.813 (0.646, 1.013)	Ebonyi	1.326 (1.101 , 1.586)
FCT-Abuja	0.625 (0.469, 0.806)	Imo	0.842 (0.646, 1.076)	Bauchi	1.352 (1.178 , 1.548)
Kwara	0.635 (0.506 , 0.791)	Rivers	0.846 (0.681, 1.033)	Kebbi	1.362 (1.160, 1.594)
Ekiti	0.645 (0.501 , 0.815)	Plateau	0.939 (0.752, 1.142)	Jigawa	1.439 (1.252, 1.645)
Borno	0.680 (0.511 , 0.860)	Adamawa	1.018 (0.843, 1.218)	Zamfara	1.680 (1.479, 1.910)
Kogi	0.680 (0.557 , 0.820)	Benue	1.063 (0.876 , 1.264)		
Oyo	0.699 (0.531 , 0.876)	Katsina	1.083 (0.926, 1.249)		
Kaduna	0.708 (0.576, 0.850)	Kano	1.088 (0.951, 1.231)		
Ogun	0.708 (0.554, 0.885)	Yobe	1.105 (0.926, 1.300)		
Lagos	0.711 (0.552, 0.881)	Taraba	1.125 (0.964, 1.294)		
Delta	0.726 (0.591, 0.881)	Gombe	1.139 (0.969, 1.327)		
Niger	0.736 (0.606 , 0.875)				
Bayelsa	0.762 (0.596, 0.960)				
Ondo	0.773 (0.631 , 0.936)				
Anambra	0.781 (0.633 , 0.964)				
Abia	0.793 (0.651 , 0.964)				
Nasarawa	0.801 (0.647 , 0.984)				
Cross river	0.801 (0.635 , 0.994)				

cording to their range of relative risk. The geographical variation ranges from 0.420 to 1.922. Out of the 37 districts(states), six (6) states had significantly higher relative risk (RR >1.000) than the overall risk. The relative risk estimates for under-five mortality ranges from lowest Osun state: 0.566(0.42, 0.73) to the highest prevalence

at Zamfara: 1.696 (1.491, 1.992). . The corresponding probability risk map is displayed in Figure 4.5 representing the smooth map for the BYM model. The relative risk value greater than 1, is an indication of high prevalence of under-five mortality prevalence.
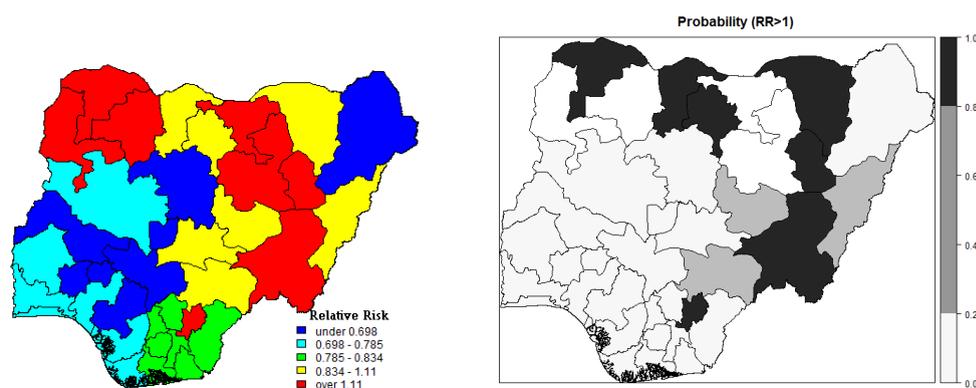

Figure 4.5: Relative Risk of childhood mortality prevalence and significance probability (RR > 1) for for BYM model, 2013 Nigeria DHS

Table 4.7: Relative risk estimates and corresponding 95% credible intervals (CI) for BYM model and states grouped by RR from low to high risk of under-five mortality based on 2013 Nigeria DHS

RR : < 0.05 significant low		**RR : 0.500 − 0.999** Not significant		**RR > 1** significant high	
Osun	0.566 (0.42 , 0.73)	Anambra	0.801 (0.637 , 1.008)	Sokoto	1.310 (1.127 , 1.511)
Edo	0.580 (0.44 , 0.73)	Akwa ibom	0.802 (0.628 , 1.001)	Ebonyi	1.321 (1.092 , 1.590)
FCT-Abuja	0.633 (0.47 , 0.82)	Enugu	0.817 (0.641 , 1.015)	Bauchi	1.358 (1.181 , 1.547)
Ekiti	0.659 (0.51 , 0.84)	Rivers	0.826 (0.648 , 1.024)	Kebbi	1.362 (1.166 , 1.576)
Kwara	0.662 (0.52 , 0.83)	Imo	0.832 (0.636 , 1.068)	Jigawa	1.443 (1.257 , 1.640)
Kogi	0.669 (0.53 , 0.83)	Nasarawa	0.835 (0.666 , 1.042)	Zamfara	1.696 (1.491 , 1.922)
Borno	0.672 (0.50 , 0.86)	Plateau	0.926 (0.738 , 1.135)		
Kaduna	0.697 (0.56 , 0.85)	Adamawa	1.030 (0.854 , 1.224)		
Oyo	0.703 (0.54 , 0.88)	Benue	1.042 (0.839 , 1.252)		
Lagos	0.709 (0.55 , 0.88)	Katsina	1.067 (0.910 , 1.234)		
Niger	0.718 (0.58 , 0.87)	Kano	1.091 (0.960 , 1.231)		
Delta	0.724 (0.58 , 0.89)	Yobe	1.102 (0.918 , 1.302)		
Ogun	0.724 (0.56 , 0.92)	Taraba	1.112 (0.948 , 1.284)		
Bayelsa	0.762 (0.60 , 0.95)	Gombe	1.151 (0.982 , 1.339)		
Ondo	0.785 (0.63 , 0.97)				
Abia	0.786 (0.63 , 0.97)				
Cross river	0.787 (0.62 , 0.98)				

4.5 Discussion

In this study, a Bayesian hierarchical model was employed to assess the child mortality risk and potential risk factors such as socio-cultural and environmental factors

for under-five mortality in Nigeria. The strength of the approach is the ability to incorporate high over-dispersion, spatial structure and covariates into the models.

The result shows that household poverty is significantly associated with under-five mortality in Nigeria. In other words, an economically deprived household has higher likelihood of childhood mortality. This finding corroborates what has been established in previous studies. These have shown that people's living conditions and household poverty influences virtually the totality of the demographic structure and health indices, including life facilities, and even human capital development as reported in Adeyemi et al. (2009); González et al. (2010) and Gwatkin et al. (2007). A similar study conducted in Nigeria by Akinyemi et al. (2013) using data from 1990-2008 found that household wealth had a strong association with not only under-five mortality, but also with the other house members life expectancy, maternal mortality and morbidity, fertility, contraceptive use and the use of health-care.

The results also reveal that poor toilet sanitary conditions and unimproved sources of drinking water are positively associated with childhood mortality, although these factors are not significant. In contrasts, a previous study conducted by de Sherbinin (2011), who introduced similar biophysical/geographical variables into their model of child malnutrition, found that these factors are significantly correlated with child malnutrition: drought prevalence, the percentage of households with piped water, and diarrhoea disease prevalence.

Furthermore, the probability risk maps reveal that there are clusters of high mortality risk concentrated in the northern regions of Nigeria. These outcomes can be attributed to the complexities such as cultural factors, socio-demographics, severity of household poverty, climate and drought, lack of access to portable water, open toilets, house structure and individual household environments. The findings are in complete agreement with the study conducted in Mozambique by Macassa et al. (2006).

The results in Table 4 showed further that there are no significant relationships between drinking water sources and under-five mortality. However, the separate findings from other studies conducted by Omariba et al. (2007) and Masangwi et al. (2009) have demonstrated the positive impact of access to clean water as significant for under-five mortality, while the problem of unsafe drinking water, inadequate water for food and personal hygiene, and insufficient access to sanitation have been identified as partly responsible for about 88 % of child deaths from infectious diseases, and mostly repeated diarrhoea in children globally, as reported in Organization et al. (2009) and Lanata et al. (2013). Other studies have established that a high proportion of children deaths in low-middle income countries can be attributed to diseases resulting from poor housing conditions, unsafe water supply, inadequate sanitary facilities, unhygienic behaviour and household air pollution from

solid cooking fuels - wood, charcoal, and agricultural residues (Cheng et al., 2012; Lim et al., 2012).

The probability risk maps presented in this study highlight geographic disparities and relative high mortality risk among young children in Nigeria, mostly found in the northern parts of the country. The results corroborate the findings from previous studies conducted in Nigeria by Adegboye (2010), who used a scan statistic method and by Uthman (2008a), who used an exploratory spatial analysis. The persistent high risk of child mortality found in the northern regions can be related to environmental factors, neighbourhood structure, education and economic deprivation. Our findings are in tandem with a study conducted by Van Bodegom et al. (2012) in other West African country, Ghana, where the method detected non-random patterns or clusters of high child mortality at village level with a large concentration of polygamous population or nuclear family settings.

The statistical issues relating to disease mapping and modeling of aggregated data of rare disease have been extensively discussed in Lawson (2013), while Dedefo et al. (2016) had earlier investigated the small area clustering of under-five mortality in Ethiopia. Previous studies have explored mixture models, for example, the study conducted by Neyens et al. (2012), where researchers combined a convolution model and Poisson-Gamma model to account for both over-dispersion and spatial correlation in the modeling of kidney and prostate cancer data. A wide range of distributions have been derived with Poisson distribution because of its positive parameter value, see (Congdon, 2005; Kang et al., 2009) for more discussion.

This present study consolidates the existing literature such as (Curtis, 2004; Smith, 2010), reported that the health impacts of climate change, geography and the local environment where people live had significant association with their health outcomes. Furthermore, health inequalities are partly a reflection of social inequalities, which are more widely defined among sub-populations even in developed countries, according to the studies by Marshall (1991) and Martuzzi et al. (2010). A comprehensive assessment of the health impacts of climate change and geography scale was discussed in Smith (2010); Curtis & Oven (2012) and Simandan (2010). In their study, they emphasized that complex processes operating at various geographical scales linking global health with the local and individual characteristics made a significant contribution to health determinants. The findings from the present study can assist health-care givers and government agencies to address the geographic disparities in the mortality prevalence and design needed interventions.

In recent decades, the disease-mapping methods provide ways to extend the Poisson model for count data to deal with the spatial structure in the data and the occurrence of overdispersion. Overdispersion means that the variability in the data is not equal to the mean as prescribed by the Poisson distribution. For example,

Neyens et al. (2017) observed that the non-inclusion of important covariates, and often referred to as non-spatial variability can result into overdispersion. Another prominent problem is the spatial dependency, which means that areas that are close in distance are more similar than in areas further apart, and this is referred to as spatially structured variability. An overview of the different forms of extra-variability in disease-mapping models is documented (Lawson, 2013), while the Bayesian techniques discussed in Besag et al. (1991) and Clayton & Kaldor (1987) that facilitates the incorporation for both spatially structured and non-spatial variability.

4.6 Summary and Concluding remarks

The proposed models and the results reveal that there are apparently geographical inequalities of child mortality prevalence across the states in Nigeria. The maps highlight clusters of high under-five mortality prevalence in the northern states and in an isolated case of Ebonyi state for the study period. Therefore, these states (regions) are in need of urgent attention and interventions. However, a relatively low prevalence of childhood mortality was observed in the south-western parts of Nigeria. The findings can guide in evidence-based allocations of scarce health resources in the sub-region with the aim of improving the chance of child survival. Our methodology was motivated by two specifications, the first of which assessed spatial dependence by borrowing strength from neighbouring states (districts) to identity clusters of child mortality in Nigeria. Secondly, the model investigated the impact of spatial heterogeneity, as a way of evaluating geographical disparities in child mortality prevalence across the regions in Nigeria.

In epidemiological study, disease mapping models are commonly used to estimate the spatial (or temporal) pattern in disease risk and identify high-risk clusters, allowing health interventions and allocation of resource. The study aimed was to present and implement a Bayesian disease mapping approach to estimate child mortality risks and detection of regions with unusual (low) high child mortality incidence. The study proposed the hierarchical Bayesian modeling approach to simultaneously handle the over-dispersion and spatial auto-correlation in the presence of heterogeneity among the population sizes across the country.

The generalized Poisson regression model with random effects were formulated to estimate the mortality risk and explored the relationships between child death counts and the regional risk factors. The random effects are formulated to captured the potential tendency of neighboring regions that share similar risk patterns and/or specific area-level heterogeneity. By applying tradition approach, standardized mortality ratio (SMR) was used to estimate the relative risk of child mortality

and mapped to highlight the unusual patterns in the prevalence at the district (state) level. A full Bayesian inference was implemented in WinBUGS via Markov chain Monte Carlos (McMC) simulation techniques. The results showed that economically deprived households, 2.088: 95% CI(1.088, 3.165) were significantly associated with childhood mortality, while unhygienic sanitary and unimproved water source were not significant. The geographical patterns of under-five mortality exhibited clustering prevalence of about 70% and spatial heterogeneity of 30% among the under-five children in Nigeria. The predicted probability maps identified clusters of high mortality prevalence majorly in the northern states and a relative low prevalence occurred in southern state of the country. The result demonstrates the flexibility in the approach by exploring the regional characteristics as potential risk factors of child mortality and provide a better understanding of the regional variations of mortality risks. Nonetheless, both representations can help provide information for initiating public health interventions.

In this study, an elevated low(high) child mortality prevalence could be attributed to geographical variation in the states with big cities than in states/regions that are made up semi-urban and rural areas, majorly found in the northern region of Nigeria. However, a geographical analysis of the incidence of childhood mortality may be hindered by the large variability in the cases between areas, as well as the excessive number of areas with zero childhood mortality or low mortality cases (less than 5). Yaya et al. (2017) used the same Nigeria DHS data and studied the prevalence of childhood mortality in Nigeria using Zero-inflated negative binomial (ZINB) regression and found a higher incidence of childhood mortality, which was associated with age of the female partners and the partner's location, who resided in the rural areas. Recently, specific studies about temporal trends in childhood mortality in other developing countries have been documented, Anyamele et al. (2015); Morakinyo & Fagbamigbe (2017) had investigated child mortality trends in Nigeria, in South Africa (Argeseanu, 2004) and in Bangladesh(Mondal et al., 2009), while a scenario- based projection of childhood mortality for 2030 in sub-Saharan Africa was undertaken by Alkema et al. (2016).

In the modeling of rare disease cases, previous studies have suggested further extension of the Poisson model to allow for accommodation of the occurrence of extra zeros and other variability caused by an excessive number of zeros. For example, in the work of Lambert (1992) and Greene (1994), they investigated the zero-inflated models for studying univariate count data and other extensions that accommodates overdispersion in the hierarchical setting. A good theory and application on mixture of distributions for count data analysis can be found in the literature (Kassahun et al., 2014; Neyens et al., 2012). On a good understanding of the theory, which facilitates the incorporation of clustering and overdispersion through two separate sets

of normal and gamma random effects, we therefore provide the addendum below to address the shortcoming realized in the analysis of under-five mortality from Nigeria DHS data.

4.7 Addendum

In this chapter, child death count was modeled at aggregated district (state) level in Nigeria using generalized Poisson gamma mixture models commonly used in disease mapping context. This addendum is required; perhaps it would provide better alternative distributions due to clustering patterns observed from our analysis.

4.7.1 Negative Binomial distribution

From the analysis of the Nigeria DHS data, it is not uncommon that a single family may record several child deaths; that is, the death counts are clustered within a single family or a certain family structure, such as the nuclear or extended family system typically found in northern Nigeria. If the number of deaths per family follows a logarithmic distribution, and the number of child deaths over the time interval (i.e. non-overlapping area e.g. a state as a defined administrative unit in our case) follows a Poisson distribution, then the total number of deaths for the time interval or administrative unit can be modeled with the negative binomial distribution.

The basic property of negative binomial distribution is now investigated below.

Let the number of death children per household be a sequence of independently and identically distributed Bernoulli trials before a specific (and fixed) numbers of failures (surviving children) occurs be k. We also denote the fixed number of those alive as $r > 0$, and the probability of a child died is given on each Bernoulli trial as $p \in [0, 1]$, which is a random variable, say, Y. A random variable, Y, thus follows a negative binomial distribution and is denoted by $Y \sim \text{NegBin}(r, p)$. The probability mass function is then given by

$$f(k) = \binom{k + r - 1}{k} \cdot (1 - p)^{r} p^{k}, \quad k \in (0, 1, 2, 3, \ldots). \tag{4.20}$$

Case 1: A negative binomial can be constructed directly from the Poisson and the Gamma distributions. Let the Poisson mean, μ follows a gamma distribution with shape parameter, r and rate parameter, $\beta = \frac{1-p}{p}$ (so $\text{Pois}(\mu)$ mixed with $\text{Gamma}(r, \beta)$), then the resulting distribution is the negative binomial distribution

with the pmf is then given by

$$f(Y = k) = \binom{k + r - 1}{k} \cdot (1 - p)^r p^k, \quad k \in (0, 1, 2, 3, \ldots). \qquad (4.21)$$

Equation (4.21)is an important extension as it allows for r to be any positive real number.

$$f(k, r, p) = \int_0^{+\infty} f_{\text{Pois}(\mu)} \cdot f_{\text{Gamma}}\left(p, \frac{(1 - p)}{p}\right) d\mu \qquad (4.22)$$

$$= \int_0^{+\infty} \frac{\mu^k e^{-\mu}}{k!} \cdot \mu^{r-1} \frac{e^{-\mu(1-p)/p}}{(\frac{p}{1-p})^r \Gamma(r)} d\mu \qquad (4.23)$$

$$= \frac{(1 - p)^r p^{-r}}{k! \Gamma(r)} \int_0^{+\infty} \mu^{r+k-1} e^{-\mu/p} d\mu \qquad (4.24)$$

$$= \frac{(1 - p)^r p^{-r}}{k! \Gamma(r)} p^{r+k} \Gamma(r + k) \qquad (4.25)$$

$$= \frac{\Gamma(r + k)}{k! \Gamma(r)} (1 - p)^r p^k. \qquad (4.26)$$

To prove the basic property, let $Y \sim \text{NegBinom}(r, p)$, then expectation is given by

$$\mathbb{E}(Y) = \frac{pr}{1 - p} \equiv \mu. \qquad (4.27)$$

and its variance is

$$\mathbb{V}(Y) = \frac{pr}{(1 - p)^2} = \mu + \frac{1}{r}\mu^2. \qquad (4.28)$$

Hence, this makes it possible for the variance to account for over-dispersion. Note that as $r \to \infty$, we get the Poisson distribution, $\mathbb{E}(Y) = \mathbb{V}(Y) = \mu$.

Case 2: The negative binomial distribution can be constructed by adding a hierarchical level to the Poisson distribution through a random effect, ε, specifically

$$Y_j | (\varepsilon_j, \mu_j, E_j) \sim \text{Pois}(\varepsilon_j, \mu_j), \quad \varepsilon_j | \Psi \sim \text{Gamma}(\Psi, \Psi) \qquad (4.29)$$

for $y_j = 0, 1, 2, 3, \ldots$, where $\Psi > 0$. The resulting probability distribution function marginal to ε_j is given by

$$Y_j = y_j | (\varepsilon_j, \mu_j, E_j) = \frac{\Gamma(y + \Psi)}{\Gamma(y_j + 1)\Gamma(\Psi} \left(\frac{\mu_j}{\mu_j + \Psi} \right)^{y_j} \left(\frac{\Psi}{\mu_j + \Psi} \right)^{\Psi} \tag{4.30}$$

for $y_j = 0, 1, 2, 3, \ldots$, with $\mathbb{E}(Y_j) = \mu_j$ and $\mathbb{V}(Y_j) = \mu_j + \frac{\mu^2}{\Psi}$.

The negative binomial model also maintains the property of the variance always being greater than the mean and Ψ, the parameter for the extra-Poisson variation, being larger than the variance of the corresponding Poisson distribution. As Ψ approaches zero the distribution of Y_j converges to a Poisson random variable. For more readings, see Wang & Famoye (1997); Famoye et al. (2004); Shmueli et al. (2005) and Denuit et al. (2007).

More recently, the presence of over dispersion in discrete data has led researchers to consider alternative approaches to generalize or extend the Poisson model. For instance, Gupta & Ong (2005) extended the method of mixtures, where the Poisson parameter was allowed to vary as a random variable resulting in a mixed Poisson distribution that accommodates over-dispersion.

4.7.2 Poisson-Inverse Gaussian Distribution

In this chapter, we have adopted gamma distribution to model counts in a spatial context as commonly used in other studies, see Neyens et al. (2012) among others. There is no reason to restrict ourselves to the gamma distribution for modeling the relative risk, Ψ, except perhaps for mathematical convenience. In fact, any distribution with support in the half positive real number is a candidate to model the stochastic behaviour of Ψ. Several authors, (Karlis & Ntzoufras, 2006; Wakefield, 2006; Ngesa et al., 2014) have suggested replacing the normality assumption of the spatially unstructured random effects with other choices such as the Laplace distribution, or student $t-$ distribution. For instance, Ngesa et al. (2014) recently used the generalized Gaussian distribution. The inverse Gaussian distribution can also be an ideal candidate for modeling positive, right-skewed data.

Definition : A random variable, Y, is distributed according to the inverse Gaussian distribution, and denoted as $Y \sim \mathrm{IGau}(\mu, \beta)$, then its probability density function is given by

$$p(y) = \frac{\mu}{\sqrt{2\pi\beta y^3}} \exp\left(-\frac{1}{2\beta y}(y - \mu)^2 \right), y > 0 \tag{4.31}$$

if $Y \sim IGau(\mu, \beta)$ then its mean, $\mathbb{E}(Y) = \mu$ and variance , $\mathbb{V}(Y) = \mu\beta$. The moment generating function is given by

$$M(t) = \int_0^{+\infty} \frac{\mu}{\sqrt{2\pi\beta y^3}} \exp\left(-\frac{1}{2\beta y}(y - \mu)^2 + ty\right) dy \tag{4.32}$$

$$= \exp\left(\frac{\mu}{\beta}\right) \int_0^{+\infty} \frac{\mu}{\sqrt{2\pi\beta y^3}} \exp\left(-\frac{1}{2\beta y}(y - \mu)^2 + ty\right) dy \tag{4.33}$$

$$= \exp\left(\frac{\mu}{\beta}\right) \int_0^{+\infty} \frac{\mu}{\sqrt{2\pi\beta y^3}} \exp\left(-\frac{1}{2\beta y}(y^2(1 - 2\beta t)^2 + \mu^2)\right) dy \tag{4.34}$$

Making the change of variable and replace $\xi = x\sqrt{1 - 2\beta t}$, it yields

$$M(t) = \exp\left(\frac{\mu}{\beta}(1 - \sqrt{1 - 2\beta t})\right) \tag{4.35}$$

Recently, the inverse Gaussian distribution has gained popularity in describing and analyzing right-skewed data. Its most appealing property is its being able to accommodate a variety of shapes. The details of mixed Poisson regressions, such as the Poisson-inverse Gaussian and negative binomial regressions, can be found in Denuit et al. (2007), where they were applied to accommodate over-dispersion in insurance claim data. As well as Rampaso et al. (2016); Lawless (1987) and Cheruiyot et al. (2018). These distributions have elegant property like normal distribution. Hence, in a Poisson mixed regression model with extra-variability parameter, $\Theta \sim Igau(1, \nu)$, that is

$$f_\Theta(\theta) = \frac{1}{\sqrt{2\pi\nu\theta}} \exp\left(-\frac{1}{2\pi\nu}(\theta - 1)^2\right), \ \Theta > 0 \tag{4.36}$$

The probability mass function is then given by

$$P(N = k) = \int_0^{+\infty} \exp(-\lambda d\theta)\frac{(\lambda d\theta)^k}{k!} \frac{1}{\sqrt{2\pi\nu\theta}} \exp\left(-\frac{1}{2\nu\theta}(\theta - 1)^2\right) d\theta \tag{4.37}$$

with mean and variance respectively given as

$$\mathbb{E}(N) = \lambda \text{ and } \mathbb{V}(N) = \lambda + \lambda^2\nu. \tag{4.38}$$

The probability mass function can be expressed using modified Bessel functions of the second kind. Bessel functions have some properties, which are useful in com-

puting the Poisson inverse- Gaussian probabilities and the maximum likelihood estimators. See Hougaard et al. (1997); Gupta & Ong (2005) and Shoukri et al. (2004) for further readings.

Chapter 5

Spatial Analysis of the Risk of Anaemia among Under five Children in Tanzania

5.1   Introduction

Childhood anaemia is a global public health problem, with serious consequences in adulthood. It is a major cause of health problems in children and, as reported in Denny et al. (2006), it adversely affects their cognitive and physical development. It compromises immunity and increases the risk of infections and infant mortality, as contained in the World Health Organization (WHO) (2008) report. According to the WHO, recent reports showed that, globally, prevalence of anaemia among children was 24.8%, with the highest prevalence being in sub-Saharan Africa (67%), followed by south east Asia (65.5%).

Several studies have identified genetic determinants; socio-economic, cultural and dietary factors related to anaemia, using linear and binary logistic regression models (Hadler et al., 2004; Meinzen-Derr et al., 2006; Zimmermann & Hurrell, 2007). A few of these studies have focused on investigating the determinant factors using purely linear effect models for anaemia. For example, Osorio et al. (2004) recently used multiple linear regression model to investigate the determinant factors on Hb concentration among preschool children in Brazil. However, little work has been done to jointly investigate the geographical variations and the underlying determinants

of childhood anaemia. The motivation of this work is to provide a flexible approach that simultaneously estimates linear and non-linear covariate effects, as well as small area geographic patterns across regions (districts) in Tanzania.

Other studies have established that genetic factors, social and demographic determinants, such as a mother's education, wealth index and family size, can affect the asymptomatic prevalence of anaemia in children (Tesfaye et al., 2015; Foote et al., 2013). The studies that examine the prevalence and determinants of anaemia, at a national level in eastern Africa, especially Tanzania, have not been adequately undertaken. Moreover, little attempt has been made to unravel the spatial pattern of hemoglobin (Hb) concentration and anaemic status in children, after taking into account other possible determinants.

Much research work on mapping the distribution of risk of anaemia among children has been conducted in West African countries. There are, however, some limitations to these studies. Magalhaes & Clements (2011) adjusted for nutritional status, parasitic infections and other co-variables among pre- school children, in Burkina Faso, Ghana and Mali. Gayawan et al. (2014) used a structured geo-additive model to map the prevalence of anaemia among under-five children in Nigeria. Other studies have documented a high prevalence of anaemia in children of school age across some regions in Nigeria (Adudu et al., 2011; Ughasoro et al., 2015). Therefore, the current work aims to examine the possible relationship between Hb concentration and severity of anaemia, together with individual and household characteristics of children aged 0-59 months; and the possible geographical variations of the Hb concentration and severity of anaemia at a highly dis-aggregated district level in Tanzania. This study applies a flexible Bayesian geo-additive modeling approach, which allows for joint modeling of fixed effects, nonlinear effects of continuous covariate and spatial effects, while at the same time controlling for the hierarchical nature of the data via random effects.

This study is structured into sections. Section 2 explained model formulation. In section, we applied the models on data from 2010 Tanzania DHS and presents the results and discussion. The conclusion was give in Section 5.

5.2 Model Formulation

In the 2010 Tanzania Demography Health Survey (TDHS) data, a child's anaemia is defined by a measure of hemoglobin (Hb) concentration and such data can be extracted with the aim of assessing the influence of some covariates on childhood anaemia. The TDHS data set contains several other variables, but only those that are related to anaemia and those similar to the ones identified in the literature were

selected. The children involved in the survey have an age range of 0- 59 months and the respondents (mothers) are in their reproductive years 15- 49. Three models are proposed, according to different criteria for the Hb concentration.

Model A: (Gaussian) anaemia is a product of low level of functional hemoglobin(Hb) in the blood. Hence, the concentration of Hb in the blood was considered as a continuous variable, y_{i1} and modeled by assuming a Gaussian distribution.

Model B: (Binary logit) According to WHO, children whose ages range 6-59 months are considered anemic if their Hb concentration levels are below 11.0 g/dl. Thus, a binary response variable ,(y_{i2}) can be created as

$$y_{i2} = \begin{cases} 1: & \text{if Hb concentration level of a child is} \leq 11.0 \text{ g/dl} \\ 0: & \text{otherwise} \end{cases}$$

Model C: (Cumulative logit) The severity level of anaemia in child can vary based on the concentration of Hb level. The WHO further classified Hb level as severe, moderate, mild or normal resulting in a four-ordered category and the response variable, y_{i3} constructed as

$$y_{i3} = \begin{cases} 1: & \text{non-anaemia, if Hb} \geq 11.0 \text{ g/dL} \\ 2: & \text{mild anaemia, if } 10.0 \text{ g/dL} \geq \text{Hb} \leq 10.9 \text{ g/dL} \\ 3: & \text{moderate anaemia, if } 7.0 \text{ g/dL} \leq \text{Hb} \leq 9.9 \text{ g/dL} \\ 4: & \text{severe anaemia, if Hb} < 7.0 \text{ g/dL} \end{cases}$$

where y_{i1} and y_{i2} are univariate responses (continuous, binary response outcome) and y_{i3} is an ordered categorical response outcome.

The present study intends to apply a flexible geo-additive regression model to quantify the fixed and non-linear effects, as well as geographical variations on anaemia level in children as the response variables y_{i1}, y_{i2} and y_{i3} as defined above.

In recent decades, there has been growing interest in the application of an ordinal logistic regression model and its structural tranformation into a latent variable model as contained in (Agresti, 2003) and Tutz (2003). Such regression models based on multi-categorical outcomes are sometimes called cumulative regression models, and its distributional form had been previously investigated in the literature by McCullagh et al. (1973) and Fahrmeir & Lang (2001). The models can be motivated from latent variables such that the response variable y, here, Hb concentration a continuous latent (utility) variable defined by

$$U = \eta + \varepsilon \tag{5.1}$$

where η is a predictor depending on covariates and parameters and ε is the error term. The two variables Y and U are linked by $Y = r$ if and only if

$$\theta_{r-1} < U \leq \theta_r, \quad r = 1, 2, \ldots c \tag{5.2}$$

with thresholds $-\infty < \theta_0 < \theta_1 < \ldots < \theta_c = \infty$, where $c = 4$. In a multinomial logit model setting, the error variables ε in (5.1) are independent across the categories and assumed to be standard extreme value distributed with function Φ. Hence, Y follows a cumulative logit model. The predictor is then defined as

$$Pr(y_i \leq r|\eta) = \Phi(\theta_r - \eta) \tag{5.3}$$

Given a set of observations $(y_i, x_i, s_i, v_i), i = 1, 2, \ldots, n$,(in this case, n=7889, number of children less than five years in 2010 Tanzania survey) where y_i is a continuous, binary or categorical response variable, a vector x_i is the of metrical covariate effects of the mother's age at birth and body mass index, the spatial covariate $s_i \in [1, \ldots, S]$, index of the district(region) where mother i lives in Tanzania and a further vector $\mathbf{v} = (v_{i1}, \ldots, v_{iq})$ of categorical covariates.

According to Tutz (2003), the geoadditive predictor for our model C is then defined as

$$\eta_i = \theta_i^r - (f(x_i) + f_{spat}(s_i) + v_i'\gamma) \tag{5.4}$$

where, $f(x_i)$,$f_{spat}(s_i)$ and γ respectively represent the estimates of the unknown non-linear smoothing effects of the metrical covariates x_i such as mother's age at birth, the spatial effect and a vector of the fixed effect parameters respectively. The spatial component, $f_{spat}(s_i)$ in the model captures the spatial correlation of area $s_i, s \in \{1, \ldots, S\}$, where woman i resides. The spatial component, $f_{spat}(s_i)$ is further split into two components:$f_{str}(s_i)$ and $f_{unstr}(s_i)$ as structured (correlated) and unstructured(uncorrelated) random effects respectively.

All model parameters were estimated in Bayes X version 2.1, a non-commercial software developed by Brezger et al. (2003). We proposed the following models:

Linear: $\quad \eta = v'\gamma$

Random : $\quad \eta = v'\gamma + \phi_{random}(s_i)$

Spatial: $\quad \eta = v'\gamma + \theta_{spat}(s_i)$

Full : $\quad \eta = v'\gamma + f_1(x_{i1}) \ldots + f_p(x_{ip}) + \theta_{spat}(s_i) + \phi_{random}(s_i)$

The model selection is performed via Deviance Information Criteria (DIC) proposed by Spiegelhalter et al. (2002b), as a measure of fit and model complexity. The choice among competing models can be done with the least DIC value. The DIC is de-

fined as $DIC = \bar{D} + pD$, where $\bar{D}$ is the posterior mean deviance. The pD is the difference between the posterior mean deviance and the evaluated deviance at the posterior mean of $\mathcal{Y}$. In fact, pD plays the role of a measure for the effective number of parameters in the model.

5.3 Bayesian estimation and inference

Within a Bayesian framework, all model parameters and non-linear functions are usually taken as random variables and an appropriate prior is needed to be specified for each. For the fixed effects, γ' s, a suitable choice is the independent diffuse prior, i.e. $p(\gamma) \propto constant$.

According to Eilers & Marx (1996), the unknown smooth function f of the continuous covariate x can be approximated by a polynomial spline of degree l on equal intervals k with equally spaced knots $x_j^{min} = \xi_{j0}, \xi_{j1}, \ldots, \xi_{jk} = x_j^{max}$, which lies within the domain of covariate x_j. The study adopted Bayesian P$-$splines, as suggested in the work of Brezger & Lang (2006). These authors used this approach as a flexible alternative to the model of an unknown function of continuous covariate in preference that had been offered by Eilers & Marx (1996). The proposed spline can be constructed as a linear combination of the basis function of $d = l + k$ $\mathbf{B}-$ spline basis function:

$$f_j(x_j) = \sum_{j=1}^{d} \beta_{kj} \mathbf{B}_j(x_j).$$

where $\boldsymbol{\beta} = (\beta_1, \ldots, \beta_d)\prime$ corresponds to the vector of the unknown regression coefficients. The smoothness of function f is achieved by penalizing the differences of coefficients of the adjacent $\mathbf{B}-$ splines as proposed by Marx & Eilers (1998). They suggest a moderate number of knots between 20 to 40 knots and by introducing a roughness penalty on the adjacent regression coefficients that regularize the smoothness to avoid overffiting. The coefficients were later replaced by a flexible first and second order random walk as suggested by Fahrmeir & Lang (2001), defined by

$$\xi_j = \xi_{j-1} + u_j; \qquad \xi_j = 2\xi_{j-1} - \xi_{j-2} + u_j \qquad (5.5)$$

with zero mean Gaussian distributed noise $u_j \sim N(0, \sigma^2)$.

Here, the variance parameter σ_j^2 is equivalent to the inverse smoothing parameter in a frequentist approach and controls the smoothness of j.

Spatial Components

We model the structured spatial effects f_{str} using a Markov random field prior as

proposed by Besag et al. (1991) in spatial statistics . It is given as

$$f_{str}(s)|f_{str}(t), t \neq s, \theta^2 \sim N \left(\sum_{t \in \delta_s} \frac{f_{str}(t)}{N_s}, \frac{\theta^2}{N_s} \right) \tag{5.6}$$

where N_s is the number of adjacent regions and $t \in \delta_s$ denotes that the region t is a neighbour to region s. Thus, the conditional mean of $f_{str}(s)$ is an unweighed average of function evaluations of neighbouring regions t. In the similar manner, θ^2 controls the amount of spatial smoothness. The unstructured spatial effect assumed a normal distribution prior, modeled as $f_{unstr} \sim N(0, \phi^2_{unstr})$.

Furthermore, the smoothing parameters τ^2, θ^2 and ϕ^2 , the smooth functions, and the variance components described above are over-dispersed, but were assigned proper hyperpriors. The variance parameters are assumed inverse gamma distribution with hyper-parameters i.e $\tau_j^2 \sim IG(a_j, b_j)$, with a and b chosen as described below. By assigning a large (small) variance, this leads to less smoothing (smoother) on the curve. The common choices for hyper-parameters are a=1 and b=0.005 or a=b=0.005. For varying choices of a and b, the sensitivity analysis was performed and checked the parameter estimates. The parameters did not change substantially, thus sensitivity analysis was not reported in this work.

5.4   Results and Discussions

The results are presented in terms of tables of fixed effects of categorical covariates, the nonlinear plots of continuous covariates and the residual plots of spatial effects. The model diagnostics and DIC values of the four model specifications each of model A, B and C are presented in Table 5.1. For each of the covariates combination specifications, we realized that the specification IV model had the least DIC values, and are judged the best models with values DIC =6678.05 (Gaussian), 7005.63 (binary) and 8492.22 (cumulative). In addition the pD value, which measures the goodness of fit and model complexity is sometimes used to evaluate the model performance. Thus, the model which consists the linear, non-linear and spatial components with specification IV is the best for prediction purpose and more complex model with a pD value of 56.42.

The posterior estimates of the three models: A(Gaussian model), B(binary logit) and C(cumulative logit) for the fixed effects covariate factors are presented in Table 5.2. Also presented are the 95% confidence intervals, which are used to determine the significance levels.

Table 5.1: Deviance Information Criteria (DIC) and goodness of fit measures for Model selection based on 2010 Tanzania DHS

Model	Diagnostic parameters	Specification			
		Linear	Random	Spatial	Full
A (Gaussian)	Deviance	27389.01	27364.72	6637.15	6602.57
	pD	21.97	26.35	27.68	37.77
	DIC	27432.95	27417.43	6694.51	6678.05
B (Binary)	Deviance	7028.3	7006.06	7004.86	6929.55
	pD	21	24.87	25.45	38.04
	DIC	7070.3	7055.8	7055.77	7005.63
C (Cumulative)	Deviance	8860.22	9394.83	8725.16	8432
	pD	0.647	27.41	49.1	56.42
	DIC	8858.94	9649.41	8823.38	8492.22

For model A (Gaussian model)with regard to the wealth index, the mean Hb was slightly higher in children, who belong to poor and middle class households compared to the poorest(ref.) household, although the effect was not significant for those of the poor, middle and richer households. Mean Hb was higher among the male children than in female children, while also higher among the rural children than those of urban, although not significant. Moreso, the mean Hb concentration is higher in children of mothers, who attained high education compared with not educated mothers. With varying child's age, the mean Hb concentration was significantly higher (better) in the infant of age 6- 11 months, but lower among neonates(1-5 months), 12-23 months old children than in over 24 months (2 years and above) children. The significant impact of maternal wealth having reverse effects on iron deficiency anaemia (IDA) are evidenced among under- five children over the first three years of their life. In early years in life, the children need enough hemoglobin for rapid neural development, biochemical and even for cognitive functioning as reported in Chang et al. (2011) and Grantham-McGregor & Ani (2001).

For the model B and C in Table 5.2, a positive coefficient indicates the factor increases the probability (odds) of anaemia prevalence. In other words, the variable with positive coefficient increases the risk of anaemia, while a variable with negative coefficient reduces the risk of anaemia, while in the the case of model A specification (Gaussian response), a positive coefficient indicates that the factor contributes to increase(improve) the hemoglobin concentration level in the child. For instance, Model A (Gaussian), multiple birth reduces the hemoglobin concentration in a child from a multiple birth compared to a child from single birth. This result agreed with the binary or cumulative ordinal analysis that there would be higher likelihood of anaemia among children from multiple birth than single birth. More so, children of highly educated mothers had better hemoglobin concentration in their blood, agreeing with models B and C that the children of high educated mothers would have reduced risk of anaemia. In summary, model C model would saves computation

time of running 3-4 separate binomial models for each category of anaemia, the results in cumulative model(column) yielded lower standard deviation(error) for each covariate factor signifying better precision. In addition, model model C included cut-off points along with the covariate estimates, as θ_1, θ_2 and θ_3. These values represent the shifting values from non-anaemia to category one(mild anaemia) to category two(moderate anaemia) and category three(severe anaemia).

Table 5.2: Posterior estimates for Gaussian, Binary logit and Cumulative Probit model based on 2010 Tanzania DHS

Variable	A:Gaussian model			B:Binary logit model			C:Cumulative logit model		
	Est.	STD	95% CI	Est.	STD	95% CI	Est.	STD.	95%CI
constant	11.891	0.199	(11.501, 12.279)	-1.268	0.298	(-1.882, -0.712)	—	—	—
Female ref.	0	0	0	0	0	0	0		
Male	0.032	0.023	(-0.011, 0.078)	-0.030	0.029	(-0.030, 0.029)	-0.032	0.030	(-0.091, 0.026)
Sleeping under bednets.	0			0			0		
No bednet	0			0			0		
Bed nets	-0.132	0.029	(-0.186, -0.072)	0.123	0.039	(0.047, 0.198)	0.129	0.038	(0.055, 0.204)
Child's age > 2 yrs ref.	0			0			0		
$1-5$	-0.017	0.047	(-0.106, 0.071)	0.096	0.057	(-0.011, 0.215)	(0.098	0.058)	(-0.016, 0.210)
$6-11$	0.135	0.046	(0.042, 0.223)	-0.141	0.062	(-0.262, -0.018)	-0.163	0.059	(-0.280, -0.048)
$12-23$	-0.195	0.059	(-0.311,-0.081)	0.133	0.075	(-0.012, 0.275)	0.158	0.071	(0.018, 0.297)
Type of birth									
Singleton ref.	0			0			0		
Multiple birth	-0.148	0.077	(-0.299, -0.004)	0.130	0.096	(-0.063, 0.316)	0.133	0.092	-0.048, 0.313
Place of residence									
Rural ref.	0			0			0		
Urban	-0.044	0.041	(-0.123, 0.032)	-0.009	0.050	(-0.109, 0.083)	0.004	0.050	-0.093, 0.101
Mother's education									
No Prim. ref.	0			0			0		
prim	0.156	0.123	(-0.084, 0.389)	-0.028	0.210	(-0.406, 0.447)	-0.056	0.200	(-0.448, 0.336)
sec	-0.405	0.133	(-0.654, -0.138)	0.521	0.216	(0.140, 0.977)	0.466	0.205	(0.064, 0.868)
high	0.543	0.360	(-0.164, 1.256)	-0.963	0.619	(-2.407, 0.118)	-0.858	0.586	(-2.007, 0.291)
Wealth index									
Poorest ref.	0			0			0		
poor	0.037	0.049	(-0.066, 0.132)	-0.055	0.064	(-0.180, 0.070)	-0.075	0.062	(-0.196, 0.046)
middle	0.076	0.048	(-0.014, 0.171)	-0.016	0.063	(-0.144, 0.102)	-0.017	0.061	(-0.136, 0.103)
richer	-0.052	0.048	(-0.146, 0.040)	-0.007	0.060	(-0.129, 0.101)	0.006	0.059	(-0.110, 0.121)
richest	-0.156	0.071	(-0.287, -0.017)	0.192	0.090	(0.015, 0.379)	0.212	0.090	(0.036, 0.388)
Stunting									
No ref.	0			0			0		
severe	0.034	0.050	(-0.065, 0.129)	0.038	0.063	(-0.079, 0.162)	0.041	0.063	(-0.083, 0.164)
moderate	-0.016	0.041	(-0.095, 0.065)	-0.049	0.054	(-0.158, 0.060)	-0.049	0.053	(-0.153, 0.055)
Wasting									
Not ref.	0	0	0	0	0				
severe	-0.094	0.156	(-0.389, 0.206)	0.130	0.199	(-0.288, 0.510)	0.173	0.184	(-0.187, 0.534)
moderate	-0.083	0.105	(-0.289, 0.119)	0.032	0.305	(0.133, -0.227)	0.008	0.125	(-0.235, 0.252)
No disease (2 weeks) ref.	0			0			0		
fever	-0.067	0.056	(-0.179, 0.040)	0.084	0.072	(-0.063 , 0.222)	0.077	0.071	(-0.063, 0.217)
diarrhea	0.105	0.068	(-0.024, 0.237)	-0.132	0.090	(-0.311, 0.050)	-0.124	0.089	(-0.280, 0.050)
Threshold	0			0			0		
θ_1:Severe anemic	—	—	—	—	—	—	4.319	0.323	(3.685, 4.953)
θ_2:moderate anemic	—	—	—	—	—	—	2.110	0.307	(1.509, 2.712)
θ_3:mild anemic	—	—	—	—	—	—	1.148	0.306	(0.549, 1.747)

HAZ: Height-for-age score, WHZ: Weight-for-height score

Figure 5.1 presents the estimates of non-linear smooth function of mother's age(in months) and mother's body mass index for for models A, B and C. Each non-linear graph consists of a center line representing the posterior mean estimate bounded by

95% credible intervals (outer lines) and 80% credible intervals (inner lines). The non-linear effects of mother's age *(left panel)* and mother's body mass index *(right panel)*. *Top panel:* From model A, the plot depicts an inverted $U-$ shape relationship between mother's age and childhood hemoglobin concentration level). This depicts that the Hb concentration would be rise among children of under-aged mothers (teenage mothers: age $<$ 20 years)that with an upward trend in Hb concentration and soon after it stabilizes (no substantial change trend line) Hb concentration for mother's age ranges 22- 38 years and later downward trend for mother's ages above 38. Figure 5.1b describes the effect of mother body mass index(which measures nutritional status of mothers)on the child Hb concentration, which produced similar trend line on Hb concentration value of their children.

 It shows that mbmi below 19 (underweight mother) produced an upward child Hb
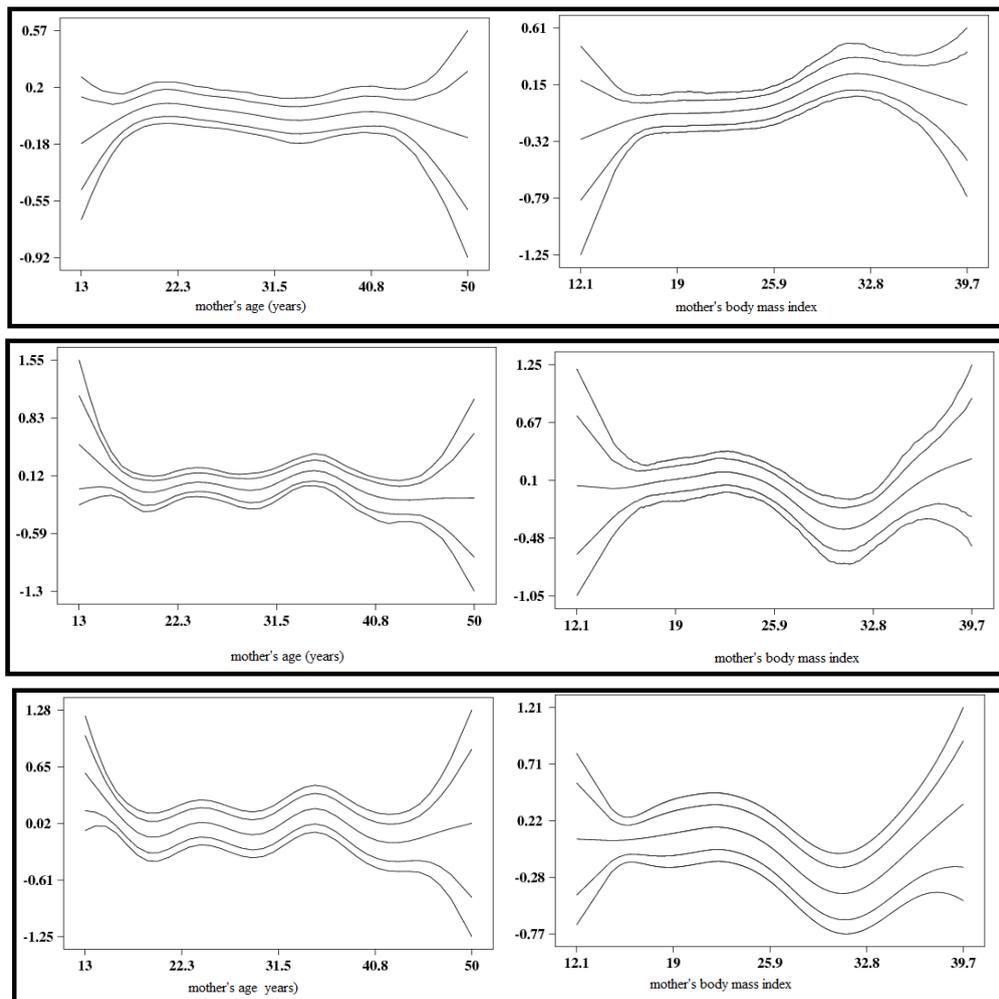
Figure 5.1: Models A, B, and C results : non-linear effects of mother's age (years) *(left panel)* and mother's body mass index (*right panel*

concentration, then little rise (stabilizes) in between 19 - 24, and decline the Hb concentration in children of mbmi above 26 (overweight or obese mothers).

The plots for models B and C showed reversed trend line ($U-$ shape graph)to the model A, but with similar implications. For instance, model B (middle panel), Figure 5.1 which depicts that the smooth curve effect of mother's age on the odds of childhood anaemia in Tanzania, indicating that the there were decreasing odds(risk) of anaemia among the children from the teen mothers (20 year), then it rises slightly and fluctuates among children of mother age from 22 to 40 years and later rises (increased risk). The trend patterns were similar for both models B and C and even for the mother mass index.

Figure 5.2 presents the plots of spatial residual of geographical prevalence of anaemia across Tanzania for models A, B and C displayed in top, middle and bottom panel respectively. Figure 5.2 shows the posterior means *(left panel)* and 80% credible interval *(right panel)*, which is used to determining the significance level. The interpretation of the colour classification from the posterior mean *(left)*, as shown in Figures 5.2 for models A *(top)*, B *(middle)*, and C *(bottom)* respectively. The green colour indicates better outcome (high Hb concentration status) for model A, while green would signify regions a low Hb concentration level for the model B and C. The red colour indicate low mean of Hb concentration level for model A, while red in model B & would mean lower risk of anaemia in those regions.

Using the 95% CI, black colour indicates low prevalence of childhood anaemia, white indicates "colour depicts high prevalence of anaemia, and grey colour means not significant. On the posterior mean plots, the geographical variation in the prevalence for Model A ranges from -0.81 to 0.159, Model B (-0.269 to 0.101) and model C : (-0.265, 0.116).

For moodel A), Figure 5.2 (top panel) represents the posterior mean map, which showed an evidence of spatial variation in anaemia prevalence across the regions (Green colour on the posterior means indicates better outcome (high mean heamoglobin (HB)concentration level i.e. from equation (5.6), value of $\bar{u}_s = \sum_{t \in \delta_s} \frac{f_{str}(t)}{N_s}$). Using the 80% credible intervals, the white colour indicates regions are associated with better Hb concentration level among children in those regions like Kilimanjaro and Tanga (i.e.strictly positive). The remaining regions with grey coloured are not significantly associated with anaemic children.

Figure 5.2 (d) and Figure 5.2 are obtained from the binary or cumulative ordinal model respectively. The black coloured regions indicate strictly negative values from the binary or cumulative logit analysis respectively. The black regions depict low prevalence (risk) of anaemia among under five children in those cities. These re-
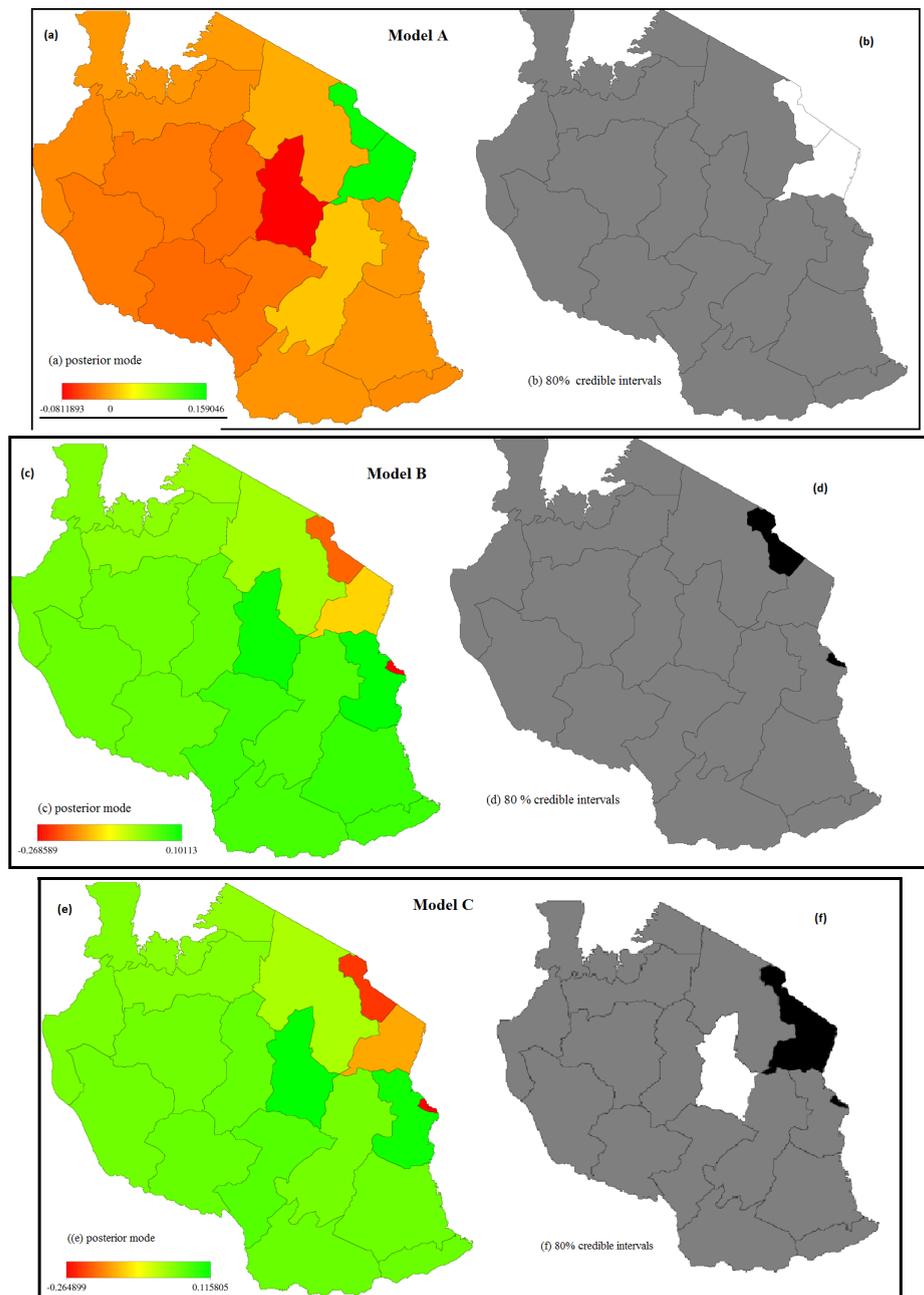
Figure 5.2: Models A, B, and C results: spatial effects with posterior means (a, c, e) *Left panel* and its corresponding 80% credible intervals map (b,d, f) respectively

gions associated with low prevalence of anaemia are Dar Es Salaam, Kilimanjaro and Tanga, while white coloured region (Dodoma) was significantly positive indicating high risk of anaemia in children. The grey coloured are not significantly associated with anaemic children.

In addition to the underlying risk factors presented the table, the significance of the probability predictive maps can be used to assist government agencies for quick in-

terventions. The maps depict that there were relatively high anaemia prevalence among the children population in most regions with similarity patterns (clustering). The findinngs corroborated other previous studies. A study conducted by Hotez & Kamath (2009) reported that neglected tropical diseases, such as hookworm infections were highly prevalent in about a half of poorest people in sub-Saharan African children, which occurred in about 50 million school-aged children and 7 million pregnant women constituted a leading cause of anaemia.

5.5 Conclusion and Summary

Using 2010 Tanzania Demographic and Health (TDHS) data, three different semi-parametric models were fitted to the measurements of anaemia status among under-five children. The measured outcomes belong to three forms of responses (Gaussian, bino- mial, and multinomial response variable) and are assumed belonging to exponen- tial family distributions. On each response outcome, we simultaneously investigate the geographical variation and the risk factor of child anaemia. The spatial com- ponent was modeled using a Markov random field prior. Bayesian inference was performed through Markov chain Monte Carlo (MCMC)simulation techniques to estimate the model parameters and the measures for goodness of fit using Deviance information criterion (DIC). The risk factors found to be significantly associated with anaemia included place of residence, household poverty, childhood malnutrition, and disease morbidity. The non-linear function showed discernible relationships with hemoglobin status among children and continuous covariates (mother's age and mother's body mass index). The results showed that the estimate outputs were similar for binary logit and multinomial ordinal models, but the ordinal model later had smaller standard deviation for corresponding factors. Our method detects spatial effects that may not have been captured by the underlying factors and produce predictive probability maps. The pattern of anaemia prevalence were similar across the regions in Tanzania indicating autocorrelation in the anaemia prevalence among regions and a relative low prevalent found in major cities of the Eastern regions (Kilimanjaro, and Dar es Salaam) of Tanzania. The model output highlights high risk regions that can assist government agency to allocate scarce health resource and effective policy direction.

In conclusion, this chapter presented three models for data on childhood anaemia belonging to a family of exponential models (Gaussian, binary and cumulative ordinal models). Although, the direction (signs) of the statistical inference for the coefficients of covariate (categorical factors) are similar from the three models presented, the cumulative (probit) ordinal model produced better estimate than the ordinary

probit model. As seen from Table 5.2, (posterior estimates), the covariate factors indicated lower standard errors under cumulative (probit) multinomial model than the corresponding ordinary probit model, an indication of better precision under the cumulative model. Moreso, by over-collapsing a response variable from its natural classification into a single binary response variable, perhaps, it might have led to loss of information.

The present study had shown that our the method is flexible and robust, and estimate several effects simultaneously. In addition to the statistical relevance of the output, we produce spatial residual effects which may be neglected in classical regression settings. The spatial residual maps can assist development partners and government agencies to channel the scarce health resource in a more effective manner.

Chapter 6

Multivariate Spatial Joint Modeling of Childhood Anaemia and Malnutrition in sub-Saharan Africa

Children's nutritional status has improved as indicated by the prevalence of stunting and wasting among children having declined significantly in recent decades. According to UNICEF et al. (2014), in 1990 40% (257 million) of the world;s children suffered from stunting whereas in 2013, the proportion had decreased to 25% (161 million). The incidence of wasting also declined over the same period, from 19% (122 million) to 8% (51 million). There has been also a significant reduction in the numbers of underweight (but not wasted) children from 25% (160 million) 15% (99 million). A recent report by WHO (2015) indicates that aenemia is found among a quarter of the global population, including 293 million (47%) children younger than 5 years, women of reproductive age, 42% of pregnant women and 468 million (30%) non-pregnant women, which further indicated that Africa and Asia accounted for more than 85% of the absolute anaemia burden among the high-risk groups.

Anthropometric indicators are frequently used to measure malnutrition in children under the age of five (WHO, 1995) and to classify individual as malnourished or of normal nutritional status. Childhood stunting (short height-for-age) is an indica-

tor of linear growth retardation and cumulative growth deficits in children (chronic malnutrition), while wasting (low weight-for-height) measures body mass in relation to height and describes a child's current nutritional status (acute malnutrition). Both anthropometric indices are important public health indicators and they remain global public health problems, with considerable consequences at adulthood.

Research evidence has established that childhood malnutrition in form of stunting or wasting or both constitutes a distinct public health problem. For example, Black et al. (2013) reported that approximately 800,000 global deaths are attributed annually to wasting and over one million are caused by stunting. It has also been estimated that wasting and stunting, respectively, are responsible for the loss of 64.6 and 54.9 million disability adjustment life years (DALY) respectively, accounting for 14.8% and 12.6% of the global DALYs for children (Black et al., 2008). Childhood wasting and stunting are both associated with increased mortality, especially when both are present in the same child (De Onis et al., 2006; Victora et al., 2008). The two forms of malnutrition often share a common cause, which suggests that to decrease malnutrition-related mortality; interventions should aim at preventing both wasting and stunting (Victora, 1992; Martorell & Young, 2012; Black et al., 2013). Childhood malnutrition is reflected in the manifestation in terms of micronutrient deficiencies, anaemia and anthropometric measurements. Consequently, there is a need to identify the risk factors of malnutrition and anaemia in the sub-populations in order to provide a baseline for future public health interventions and to plan an effective intervention and nutritional supplementation programme.

In epidemiology and demography, disease mapping models have long been used in analysing geographical variation of disease rates in order to produce a contiguous map of disease risk and highlight areas of unusual high risk (Richardson et al., 2004; Dreassi, 2007). The spatial mapping models provide decision makers the guide for better allocation of public health resources.

More recently, the research in spatial epidemiology is increasingly growing in area of joint modeling. Besuase the joint disease modeling provides additional information that is useful to identify the common risk factors(similarity) in the case of a variety of diseases. Moreso, the joint modeling approach is more appealing than univariate response analysis. The potential benefits of a multivariate disease mapping include the ease of interpretation, improved precision of the underlying disease pattern estimation, ability to identify shared and specific patterns of risk among different disease prevalence, and improvement in the model precision (Dabney & Wakefield, 2005; Dreassi, 2007; Held et al., 2005). A good review of multiple disease mapping and techniques can be found in existing literature such as Best et al. (2005); Manda et al. (2012) and Downing et al. (2008). The most common approach to analyze multiple disease outcome is done through a shared component model first

proposed by (Knorr-Held & Best, 2001) and the multivariate conditional autoregressive(MCAR)model theory developed by Mardia (1988). Other extensions have been developed, such as the generalized MCAR (GMCAR) for lung and esophagus cancer in Minnesota(Jin et al., 2005), the so-called coregionalized MCAR for multivariate geostatistics(Wackernagel, 2013) and adaptive smoothed ANOVA (SANOVA) for areal data (Zhang et al., 2009). Accordingly, these above mentioned studies have motivated the present study, where we extend the existing conditional autoregressive prior into a multivariate setting. This study therefore explores a multivariate conditional autoreggressive approach to simultaneously model three malnutrition indicators among children less than five years of age.

This study is structured into sections. Section 6.1 explains the procedure to be used, and the sources of data. Section 6.2 outlines model formulation and detail the Bayesian spatial estimation method. Sections 6.3 and 6.4 show the implementation of the method on cross-sectional data obtained from DHS and presents the results. Then Section 6.5 discusses the results, and this is followed by the final section with concluding remarks on the findings.

6.1 The Data Exploration

The data used in this study are extracted from the Demographic and Health Surveys database and in collaboration with Bureau of Statistics of the respective countries. Over the years, the DHS program has provided technical supports and funding to surveys conducted in many developing countries. This thereby promotes global understanding of health and population trends. They developed standard procedures, methodologies and manuals to guide the sampling survey planning, design and data collection processes to obtain quality data to reflect the health and demographic representation of the population comparable among countries. The data used in this study are extracted from the surveys in Burkina Faso INSD (2012), Ghana Statistical Service(GSS) (2008) and Mozambique National Statistics Institute (2011).

Figure 6.1(a)-(c) respectively shows the geographical map of regions(provinces) of the three selected countries: Burkina Faso, Ghana and Mozambique. Burkina Faso is a landlocked country in West Africa. It covers an area of around 274,200 km^2 and it has 13 regions and subdivided into 45 administrative provinces and the nation has a population of about 18.1 million acoording to DeSA et al. (2017). Ghana is situated on the West Africa's Gulf of Guinea with a total land mass of 238 540 km^2. The country is divided into 10 administrative regions. Mozambique is a country in the South-eastern Africa bordered by six countries and the Indian Ocean. According to the 2017 revision of the World Population Prospects, it has a total population of 28.8

million in 2016 according to DeSA et al. (2017) and a total fertility rate of 5.9 according to the Mozambique National Statistics Institute (2011). Mozambique is made of 10 administrative provinces and a total landmass of 799,380 km^2.
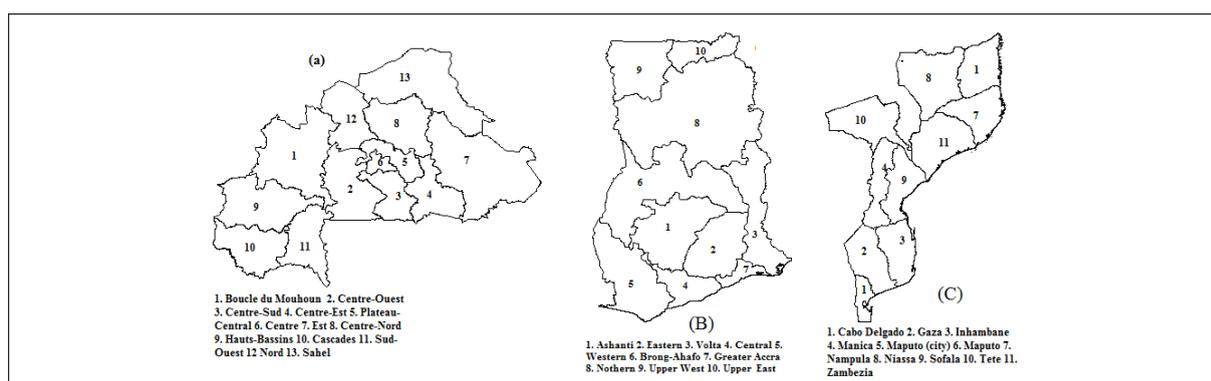
Figure 6.1: Map of (a) Burkina Faso (b) Ghana and (c) Mozambique showing regions / provinces within each country

Data information was assessed and extracted from the Demographic and Health Surveys of the three countries. Anaemia(used as a response variable) is described as a product of low level concentration of functional hemoglobin(Hb) in the blood. According to World Health Organization (WHO), children whose age range from 0-59 months are considered anemic if their Hb concentration levels are below 11.0 g/dl as reported in Stoltzfus (1997). The study adopted the World Health Organization (2006) standard for characterization of child malnutrition: weight-for-height (wasting) and height-for-age (stunting) z-scores as when the child Z− score falls below minus 2 SD, the child is considered as wasting or stunting respectively.

The following variables are also chosen and used in existing empirical studies of childhood malnutrition as suggested in Kazembe (2013) and Smith-Greenaway & Trinitapoli (2014). The factors included in the model are presented in Table 6.1.

The number of malnourished children in the sampled population were tabulated by country and presented in Table 6.2. The overall prevalence of childhood anaemia among the three countries ranges between 30.9− 39.4%, with lowest prevalence recorded in Mozambique and highest recorded in Ghana. The overall prevalence of childhood stunting among the countries ranges between 28.2 − 42.8%, with lowest prevalence in Ghana and maximum in Mozambique. Furthermore, for the prevalence of child wasting, the lowest prevalence of 5.9% was recorded in Mozambique and the highest prevalence of 13.8% in Burkina Faso. In the survey population, there were many missing values, may be the respondent (mother) declined to provide in-

Table 6.1: Variables used in the analysis: variable indicator, Levels and Variable types

Variable indicator	Levels	Variable type
Response variable		
Stunting	stunting =1, 0 otherwise	binary
Wasting	wasting =1, 0 otherwise	binary
Aneamia	anaemic =1, 0 otherwise	binary
Explanatory variable		
child use bed nets	yes= (1), no=0(ref.)	binary
Child age	1-5 months, 6-11 months,	categorical
	12-23 months, 2 years and above (ref.)	
Birth order	1st, 2-3, 4-5, 6 and more	categorical
Gender of child	Female=0(ref.), male=1	binary
birth type	singleton , multiple (twin)	binary
birth order	1st=0(ref.), 2nd , 3rd, 4th or more	categorical
Birth in 3 years	0-1 child, 2 or more children	categorical
diarrhea	Yes, no(ref.)	binary
fever	Yes, no(ref.)	binary
measles vaccination	yes=1, no=0 (ref.)	binary
mother's literacy	read and write =1, not literate=0(ref.)	binary
Mother's age	age at birth (years)	continuous
mother body mass index	mbmi	continuous
antenatal attendance	zero visit, 1-3 visits, 4 and more (ref)	categorical
place of residence	rural=1, urban=0	binary
mother's wealth index	poorest(ref.), poor, middle, richer, richest	categorical
mother's educational level	primary(ref.), secondary, higher	categorical
electricity	yes=1, no=0(ref.)	binary

Table 6.2: Percentage distribution of child's health condition(anaemia, stunting, and wasting by country in total sampled population from the Demographic and Health Surveys

Country	Years of DHS	Population sample(N)	No of children response (n)	No. of stunted children n(%)	No. of wasted children n(%)	No. of anaemic children n(%)
Burkina Faso	2010	15044	6994	2420(34.8)	1084(15.5)	2343(30.9)
Ghana	2008	2992	2083	558(28.2)	176(8.3)	1149(39.4)
Mozambique	2011	11102	10313	4393(42.6)	609(5.9)	3394(30.9)

% = ratio of number of cases children divided by sample population at risk (response)

formation about her child, or the child was not available (not at home) when the interview was been conducted, or the child was sick , or the child was living with a family relation.

Here, we present a brief summary statistics about the survey population of children under -five age involved in the study (Table not available).

From the extracted data, there was a total of 19 390 children aged 0-59 months from the three country surveys, with overall prevalence of stunting in the sample population of 30.0% and 12.4% wasting, and 30.0% of the children were affected by anaemia, with more anemic in infants and younger children than older children.

Majority (81%) of these children were from rural areas, of which there were higher prevalence of stunting among the children, of 30%, but lesser prevalence of anaemia of 22.7% and wasting of 4.7% among rural children than urban. About one-half of the children (54%) were either first-born or at second and third order. While the percentage prevalence of anaemia had a linear association with birth order of birth. Childhood stunting and wasting did not indicate any consistent linear association with the birth order. Majority of these children (97%) were born of single birth, with higher prevalence of anaemia and stunting of 25.9% and 39.1% respectively among children of multiple birth (twin) than the single birth (22.9% and 28.2%) respectively, but lower prevalence of child wasting. About a quarter of these children were born to mothers who attained no formal education(25.5%), some formal education (63.3%), while 11.3% achieved secondary or higher).

Generally, the prevalence of anaemia did not differ significantly among the maternal wealth strata(groups), but the prevalence of childhood malnutrition(stunting and wasting) decline consistently as the maternal wealth move upward the economic strata. Furthermore, it was observed that the prevalence of both childhood anaemia and wasting are linearly related to maternal household wealth strata, but the wealth index was inversely linear associated with child stunting, except for the extreme poor. The higher prevalence of the three malnutrition indicators were found among the children born of mothers with short birth intervals, and those mothers who did not attend antenatal clinic at all or less than 3 visitations.

6.2 The Statistical Models

The joint modeling method known as the shared component model was introduced by Knorr-Held & Best (2001), with subsequent extension of their work to joint disease clusters detection, as described in Lawson (2006). The shared component models have been successfully applied to model jointly the risk of oral and oesophageal cancers by Knorr-Held & Best (2001), of chronic obstructive pulmonary disease (COPD) and lung cancer by Best & Hansell (2009), of infant mortality and causes of death in Austria by Waldhoer et al. (2008), of infant mortality from four cancers by Held et al. (2005) and of gender inequalities in hospital admission for chronic diseases in the work of Ibáñez-Beroiz et al. (2011). Ecological co-morbidity of childhood diseases in Somalia was also carried out by Kinyoki et al. (2017).

Although, as stated in Best & Hansell (2009) and Downing et al. (2008), analysts could use a multivariate normal model approach to assess co-variances and correlation within and between diseases of underlying spatial risk, multivariate spatial models permit the estimation of conditional correlation between two diseases, while

at the same time estimating their spatial dependence with the region. By performing joint modeling analysis on multiple diseases, it has been demonstrated that such models yield better detection and estimates of the underlying risk patterns and so they showed superiority over a separate univariate model analysis of any single disease response (Poletta et al., 2007; Boyd et al., 2004; Green & Richardson, 2002). Details concerning recent developments in the theory and applications of multivariate joint modeling techniques can be found in Assunção & Castro (2004); Dobra et al. (2011) and MacNab (2011). A scale mixture approach was recently proposed by Congdon (2017) as a contribution to the field of multivariate disease mapping models.

The shared component model in the work of Knorr-Held & Best (2001) can be briefly summarized as follows. The case of two diseases is most commonly applied in the literature for a two-component case, where one describes the association in the prevalence of the two diseases and the second component introduced is specific to one of the diseases. The two components are formulated to incorporate the unobserved spatial factors that affect the risk of the disease(s). Let y_{i1} be the observed cases of diseases 1 in region i and E_{i1} be the corresponding expected number of cases for the same disease. Similarly, y_{i2} and E_{i2} are the observed and expected number of cases for the disease 2 in region i. The counts (incidence of cancer) assumed that, $y_{i1} \sim Poisson(E_{i1}\exp(\eta_{i1}))$, $y_{i2} \sim Poisson(E_{i2}\exp(\eta_{i2}))$, and the log of the relative risks were modeled using normal random effects such that $\eta_{i1} \sim \mathrm{N}(\alpha_1 + u_{1i}\delta + u_{2i}, \tau_1)$, and $\eta_{i2} \sim \mathrm{N}(\alpha_2 + u_{1i}/\delta, \tau_2)$. The parameter u_1 is the shared component while u_2 is the component specific to the first disease only. They were assumed to follow Gaussian Markov random fields (GMRF) with precision parameters, τ_1) and τ_2) respectively. The non-negative parameter, δ should be included in the model to allow the two diseases to have different risk gradients in the shared components. They assumed that $\log \delta \sim \mathrm{N}(0, \sigma^2)$, with the value of σ^2 is set to 0.17 as suggested in Knorr-Held & Best (2001).

The joint models in the work of Knorr-Held & Best (2001) was modified to suit the Bernoulli data (i.e. malnourished/nourished outcome) in the present study. Let y_{ijk} be the disease (nutritional status) coded (0/1) of a disease k, where $k = 1$ for stunting, $k = 2$ for wasting, and $k = 3$ for anaemia for individual child i, $i = 1, 2, \ldots, n_j$, in regions j (provinces as the case may be). We further assume that the observed outcomes arise from a trivariate distribution, with p_{ijk} as the probability of disease k occurring in individual in area j. The data generation model is defined as

$$y_{ijk} \sim Bernoulli(p_{ijk}). \tag{6.1}$$

and for each model, the covariates are introduced as stated below.

In the separate analysis, the covariates and random effects are introduced as follows:

$$
\begin{aligned}
logit(p_{ij1}) &= \alpha_1 + \mathbf{X}'\boldsymbol{\beta_1} + u_{1i} + v_{1i} \\
logit(p_{ij2}) &= \alpha_2 + \mathbf{X}'\boldsymbol{\beta_2} + u_{2i} + v_{2i} \\
logit(p_{ij3}) &= \alpha_3 + \mathbf{X}'\boldsymbol{\beta_3} + u_{3i} + v_{3i}
\end{aligned}
$$

where u_1, u_2 and u_3 are modeled using the independent conditional auto-regressive prior, while v_1, v_2 and v_3 are modeled using the independent normal distributions. In a shared component model approach, the covariates and random effects are introduced as follows

$$
\begin{aligned}
logit(p_{ij1}) &= \alpha_1 + \mathbf{X}^T\boldsymbol{\beta_1} + u_1\delta + u_{2i} \\
logit(p_{ij2}) &= \alpha_2 + \mathbf{X}^T\boldsymbol{\beta_2} + \frac{u_{1i}}{\delta}
\end{aligned}
$$

u_1 is the shared component while u_2 is the component specific to the first disease only. These two components are modeled using conditional autoregressive priors with precision, τ_1 and τ_2 respectively.

The multivariate joint modeling approach can be easily generalized to more than two outcomes. In the multivariate setting , the covariates and random effects are introduced as follows

$$
\eta_{ij2} = logit(p_{ij1}) = \alpha_1 + \mathbf{X}^T\boldsymbol{\beta_1} + \phi_i \tag{6.2a}
$$

$$
\eta_{ij3} = logit(p_{ij2}) = \alpha_2 + \mathbf{X}^T\boldsymbol{\beta_2} + \phi_i \tag{6.2b}
$$

$$
\eta_{ij1} = logit(p_{ij3}) = \alpha_3 + \mathbf{X}^T\boldsymbol{\beta_3} + \phi_i \tag{6.2c}
$$

where $\phi = (\phi_1, \phi_2, \phi_3)^T$ is modeled using a multivariate conditional autoregressive prior that is $\boldsymbol{\Phi} \sim \text{MCAR}(1, \Sigma)$, where Σ is the covariance matrix including correlations.

In order to induce spatial correlation structure between the set of binary logistic models, equations (6.2) can be fused together into a multivariate version, and the matrix form explicitly expressed as

$$
\eta = \begin{pmatrix} logit(p_{ij1}) \\ logit(p_{ij2}) \\ logit(p_{ij3}) \end{pmatrix} = \begin{pmatrix} \alpha_1 \\ \alpha_2 \\ \alpha_3 \end{pmatrix} + \mathbf{X} \begin{pmatrix} \boldsymbol{\beta_1} \\ \boldsymbol{\beta_2} \\ \boldsymbol{\beta_3} \end{pmatrix} + \begin{pmatrix} \phi_i \\ \phi_i \\ \phi_i \end{pmatrix} \tag{6.3}
$$

We then model y_{1ij}, y_{2ij} and y_{3ij} via the following tri-variate normal distribution

$$
\begin{pmatrix} y_{1ij}^* \\ y_{2ij}^* \\ y_{3ij}^* \end{pmatrix} |\phi_i \sim \mathrm{N}_3 \left[\begin{pmatrix} \eta_{1ij} \\ \eta_{2ij} \\ \eta_{3ij} \end{pmatrix}, \mathbf{R} \right] = \mathrm{N}_3 \left[\begin{pmatrix} \eta_{1ij} \\ \eta_{2ij} \\ \eta_{3ij} \end{pmatrix}, \begin{pmatrix} 1 & \rho_{12} & \rho_{13} \\ \rho_{21} & 1 & \rho_{23} \\ \rho_{31} & \rho_{32} & 1 \end{pmatrix} \right] \tag{6.4}
$$

where $\alpha_k, k = 1, 2, 3$ in equation (6.3) represents individual specific disease intercept, and the terms $\boldsymbol{\beta} = (\beta_1, \beta_2, \beta_3)^T$ are $p \times 1$ vectors of regression parameters to the set of covariates (fixed effects) (presented in Table 6.4, Table 6.5, Table 6.6) for Burkina Faso , Ghana and Mozambique respectively. $\mathbf{R}$ is a within -subject correlation matrix with diagonal elements set to 1 for identifiability and off diagonal ρ denoting the conditional correlation between $y_{1ij}, y_{2ij},$ and y_{3ij} given $\boldsymbol{\phi_i}$ and $\boldsymbol{\phi_i} = (\phi_1, \phi_2, \phi_3)'$ is a 3×1 vector of spatial dependent random effects for the ith region or province as presented in Table 6.7, Table 6.8 & 6.9) for respective countries: Burkina Faso, Ghana and Mozambique.

Model estimation was carried in a full Bayesian approach by assigning appropriate prior distributions to all the parameters of the models. In addition, the prior distribution was also assigned to the random effects discussed in the models above, non-information priors were assigned to the fixed coefficients. For the intercepts, diffuse priors were assumed, that is, $p(\alpha_k)$, while the fixed effect covariate was assigned a highly dispersed normal prior distribution, that is, $p(\beta) \sim \mathrm{N}(0, 10^4)$.

In a shared component model, the analyst needs to specify an extra parameter δ by allocating a prior as $\log \delta \sim (0, \sigma^2)$, while in the case of multivariate setting, the covariance matrix was assigned an inverse Wishart prior as $\Sigma \sim IW(r, R)$ with R considered to be an identity matrix. All models were fitted using WinBUGS software due to Spiegelhalter et al. (2002b).

There are two ways of proceeding, via the covariance matrix or its reciprocal. One way is to specify the joint distribution (**??**) and assume specific forms for Σ. For further readings, see Gelfand & Vounatsou (2003) and Assunção & Castro (2004).

6.2.1 Univariate Conditional Autoregressive (CAR) Prior

Consider a vector $\boldsymbol{\phi} = (\phi_1, \phi_2, \ldots, \phi_n)^T$ of p components that follow a multivariate Gaussian distribution with mean zero and variance -covariance matrix $\boldsymbol{Q}^{-1}$, where $\boldsymbol{Q}$ is $p \times p$ symmetric and positive definite matrix. It follows that the joint pdf of ϕ is given by

$$
p(\phi) = (2\pi)^{\frac{p}{2}} |\boldsymbol{Q}|^{\frac{1}{2}} \exp \left\{ \frac{1}{2} \phi^T \boldsymbol{Q} \phi \right\} \tag{6.5}
$$

The conditional distribution of one of the components given the remaining ones, in term of the elements of the matrix Q, is given by

$$p(\phi_i|\phi_{-i}) \propto \exp\left\{-\frac{b_{ii}}{2}\left(\phi - \sum_{j \neq i}\frac{-bij}{b_{ii}}\phi\right)^2\right\} \qquad (6.6)$$

Equation (6.6) can be written in short form as $\phi|\phi \sim N(-\sum_{j \neq i}\frac{b_{ij}}{b_{ii}}\phi_j, \frac{1}{b_{ii}})$. Besag (1974) showed the conditions under which the full conditional distributions specified above uniquely defined a full joint distribution, which allows $\frac{-b_{ij}}{b_{ii}} = c_{ij}, c_{ii} = 0$ and $\frac{1}{b_{ii}} = \sigma_i^2$. Then, the elements of the matrix **C** equal to c_{ij} and c_{ii}, while the matrix **M** defined in

$$\mathbf{Q} = \mathbf{M}^{-1}(\mathbf{I} - \mathbf{C}). \qquad (6.7)$$

Then the joint distribution of ϕ is $\text{MVN}(0, \mathbf{M}^{-1}(\mathbf{I} - \mathbf{C}))$. It is worth to note that **C** and **M** must be properly specified so as to ensure symmetry in **B**. The condition $c_{ij}\sigma_j^2 = c_{ji}\sigma_i^2$ guarantees the matrix to be symmetric. The matrix **C** is also specified to show its relationship between neighbours.

A commonly used adjacency matrix for lattice data was due to Besag (1974). He defined the element of matrix **C** as $c_{ii} = 0$ and $c_{ij} = \frac{1}{m_i}$ if j is adjacent to i and zero otherwise. Here, m_j is the number of neighbours of region i. We define another matrix **W** to hold the adjacency and structure, $w_{ii} = 1$ if region i and region j are neighbours and zero otherwise. It then follows that $\mathbf{C}=\mathbf{W}_s$ where $\mathbf{W}_s = diag(\frac{1}{m_i})$. The notation implies the following specification to the matrix **B**; $b_{ii} = \lambda m_i$ and $b_{ij} = -\lambda$ if region j is adjacent to region i and zero otherwise. Two regions are defined to be neighbours if they share a common boundary. It then follows that

$$\mathbf{Q} = \lambda(diag(m_i) - \mathbf{C}). \qquad (6.8)$$

From the equation (6.7), $\mathbf{M}^{-1}(\mathbf{I} - \mathbf{C})$ has to be positive definite for the conditional distributions to give rise to a valid joint probability density function(pdf). Besag (1974) gave the definition of the adjacency matrix leads to an improper joint pdf. This can be overcome by introducing a parameter α into the precision matrix **Q** and it then yields

$$\mathbf{Q} = \mathbf{M}^{-1}(\mathbf{I} - \alpha\mathbf{C}). \qquad (6.9)$$

If $|\alpha| < 1$, then the matrix $\mathbf{M}^{-1}(\mathbf{I} - \alpha\mathbf{C})$ is a diagonally dominant and symmetric matrix. Bernardo et al. (2003) showed that the matrix expressed in equation (6.9) is a symmetric and diagonally dominant matrix, which is positive-definite. The auto-

regressive term in this study is used in the context of space(place) or region. Extensions and variants to these models have led to different approaches such as stated in Gelfand & Vounatsou (2003) and Jin et al. (2005).

Ma et al. (2007) used multivariate approach to model areal wombling for multiple disease boundary analysis

6.2.2 Multivariate Conditional Auto-regressive(MCAR) Prior

The development of the multivariate model is credited to Mardia (1988); as such it is an extension of Besag (1974) results into a multivariate setting. The work, of Mardia (1988) showed the conditions under which the conditional multivariate distribution uniquely determines the corresponding multivariate joint probability density function. Under the same condition, Carlin et al. (2003) extended the MCAR model of Jin et al. (2005) into a generalized MCAR model in the following manner. Let $\boldsymbol{\Phi} = (\phi_1^T, \phi_2^T, \ldots, \phi_n^T)$, where each $\boldsymbol{\phi_i}$ is $n \times 1$ vector. Then Φ is an $np \times 1$ vector. Also let Φ have a multivariate Gaussian distribution with mean, $\mathbf{0}$ and precision matrix $\mathbf{Q}$, it can then be defined by

$$p(\boldsymbol{\Phi}) = (2\pi)^{\frac{np}{2}} |\boldsymbol{Q}|^{\frac{1}{2}} \exp\left\{-\frac{1}{2}\boldsymbol{\Phi}^T \mathbf{Q} \boldsymbol{\Phi}\right\} \tag{6.10}$$

$\mathbf{Q}$ is an $np \times np$ symmetric and positive definite matrix. It is informative to look at $\mathbf{Q}$ as a $p \times p$ block matrix with $n \times n$ block $\mathbf{Q}_{ij}$, hence the full conditional distributions are given by

$$p(\boldsymbol{\phi_i}|\boldsymbol{\phi_j}) \propto \exp\left\{-\frac{1}{2}\left(\phi_i - \mathbf{Q}_{ii}^{-1}\sum_{j\neq i}(-\mathbf{Q}_{ij})\boldsymbol{\phi_i}\right)^T \mathbf{Q}_{ii}\left(\boldsymbol{\phi_i} - \mathbf{Q}_{ii}^{-1}\sum_{j\neq i}(-\mathbf{Q}_{ij})\boldsymbol{\phi_i}\right)\right\} \tag{6.11}$$

This implies that $\phi_i|\phi_j \sim N_{ii}\left(\mathbf{Q}_{ii}^{-1}\sum_{j\neq i}(-\mathbf{Q}_{ij})\phi_j, \mathbf{Q}_{ii}^{-1}\right)$ and the full conditional probability density function is given by

$$p(\boldsymbol{\phi_i}|\boldsymbol{\phi_j}) = \mathrm{N}_{ii}\left(\sum_{j\neq i}\mathbf{C}_{ij}\boldsymbol{\phi_j}, \Sigma_i\right), \ \ i = 1, 2, \ldots, p \tag{6.12}$$

where $\boldsymbol{\Sigma_i}$ and $\mathbf{C}_{ij}$ are $n \times n$ are matrices that are analogue to σ_{ij} and c_{ij} as defined for the univariate case above (6.6). The matrix $\boldsymbol{\Sigma_i}$ is also symmetric and positive definite. The relationship between the matrices $\boldsymbol{\Sigma_i}$ and $\mathbf{C}_{ij}$ can also be written in terms of $\mathbf{Q}$, and the precision matrix of the joint distribution as $\mathbf{C}_{ij} = -\mathbf{Q}_{ii}^{-1}\mathbf{Q}_{ij}$ and

$\Sigma_i = \mathbf{Q}_{ii}^{-1}$. If we set Σ to be a block diagonal matrix with Σ_i blocks and $\mathbf{C}$ as a partitioned matrix with blocks $\mathbf{C}_{ij}$ and $\mathbf{C}_{ij} = \mathbf{0}_{n \times n}$, then

$$\mathbf{Q} = \Sigma^{-1}(\mathbf{I} - \mathbf{C}) \tag{6.13}$$

A parameter, α can be added into the precision matrix into equation (6.12) to yield

$$\mathbf{Q} = \Sigma^{-1}(\mathbf{I} - \alpha\mathbf{C}) \tag{6.14}$$

For a symmetric matrix $\mathbf{Q}$ and smoothing parameter α satisfying the condition such that $\mathbf{C}_{ij}\Sigma_j = \Sigma_i\mathbf{C}_j^T$, the distribution is denoted by $\mathbf{MCAR}(\mathbf{C}, \Sigma)$ according to Banerjee et al. (2014). To carry out the inference, α and Σ are assigned appropriate priors, such as uniform distribution for $\boldsymbol{\alpha}$ and Wishart (ρ, Σ_0) for Σ.

6.2.3 Posterior Distribution

Bayesian inference is based on the posterior distribution and it is carried out using the Markov chain Monte Carlo (McMC) simulation technique. The posterior distribution is obtained by updating the prior distribution with the data likelihood and hence it the distribution of the parameter after the data has been observed. Due to the high dimensionality involved in drawing the samples, McMC method permits direct sampling from the posterior distribution repeatedly and summary statistics such as mean and median are computed from samples drawn. Under of the assumptions of conditional independence between the response variable and the hyper-parameters, the joint posterior distribution for the a spatial bilogit model, is given by

$\pi(\boldsymbol{\beta_1}, \boldsymbol{\beta_2}, \boldsymbol{\alpha_1}, \boldsymbol{\alpha_2}, \boldsymbol{\rho}, \boldsymbol{\Phi}, \boldsymbol{\Sigma}) \propto$

$$\prod_{i=1}^{n}\prod_{j=1}^{n_i} \Big[\Pr\left(y_{1ij}^* < 0, y_{2ij}^* < 0 | \eta_{ij}, \rho\right)^{(1-y_{1ij})(1-y_{2ij})} \Pr\left(y_{1ij}^* > 0, y_{2ij}^* < 0 | \eta_{ij}, \rho\right)^{y_{1ij}(1-y_{2ij})}$$

$$\times \Pr\left(y_{1ij}^* < 0, y_{2ij}^* > 0 | \eta_{ij}, \rho\right)^{(1-y_{1ij})y_{2ij}} \Pr\left(y_{1ij^*} > 0, y_{2ij}^* > 0 | \eta_{ij}, \rho\right)^{y_{1ij}y_{2ij}} \Big]$$

$$\times \exp\left(-\frac{1}{2}\boldsymbol{\Phi'}\left[(\boldsymbol{M} - \boldsymbol{A}) \otimes \Sigma^{-1}\right] \boldsymbol{\pi(\beta_1)\pi(\beta_2)\pi(\alpha_1)\pi(\alpha_2)\pi(\rho)\pi(\Sigma)}\right)$$

$$\tag{6.15}$$

where $\boldsymbol{\eta_{ij}} = (\eta_{1ij}, \eta_{2ij})\prime$ as given in equation (6.4) and the $\pi(.)$ represent the prior distributions for their respective parameters as defined in section 6.2.1 above.

For a case of k $= 3$, let $\boldsymbol{\theta}$ be the vector of all the parameters in the model, $L(y_i|\eta_i)$ is the likelihood of the trivariate model and τ_j^2 is the variance parameter, then the posterior distribution is given by

$$p(\boldsymbol{\theta}|y) \propto L(y|\alpha, \beta, \rho, \phi, \tau^2) \times p(\alpha, \beta, \rho, \phi, \tau^2) \tag{6.16}$$

$$= \prod_{i=1}^{n} L(y_i, \eta_i) \prod_{j=1}^{p} \left[p(\beta_j|\tau_j^2) p(\tau_j^2) p(\phi_u|\tau_u^2) p(\phi_v|\tau_v^2) p(\tau_v^2) \right] \tag{6.17}$$

$$\times \prod_{j=1}^{r} p(\gamma_j) p(\sigma_j^2)$$

where $\beta_j,\ j = 1, \ldots, p$ are the vectors of regression coefficients and all are full conditionals. For the variance components, $\tau_j^2,\ j = 1, 2 \ldots, p, \tau_u^2, \tau_v^2$ and and σ^2 the full conditionals are inverse gamma distributions. All the model analyses were carried out using WINBUGS 14 (Spiegelhalter et al., 2002a). In order to estimate the model parameters, 15000 McMC iterations were performed for each model run with the initial 5 000 iterations were discarded to take care of the burn in period. The McMC convergence was assessed using the remaining 15 000 iterations.

We performed separate independent CAR analysis for each malnutrition indicator to assess demographic risk factors associated with the child malnutrition status. Data cleaning and re-coding was done using the R software (Team, 2014), and the analysis was carried out via a Bayesian approach using the Integrated Nested Laplace Appropriation(INLA) due to Rue & Held (2005). The Shapefile data of the geo-reference coordinates of countries was downloaded at Global Administrative Areas (2012) and the maps of the posterior estimates of the models was done using R-INLA environments. The following model specifications are used to combine the socio-demographic factors.

- M_1: $\eta_i = x'\beta$

- M_2: $\eta_i = x'\beta + f_1(z_i) + v_i$

- M_3: $\eta_= x'\beta + f_1(z_i) + \phi_i$

- M_4: $\eta_= x'\beta + f_1(z_i) + v_i + \phi_i$

where, the set of observations (x_i, z_i, s_i) represents categorical covariates, metrical variable, and index of geographical location for unobserved (unstructured) spatial and structured factors.

- Model (M1), which we denote as the baseline model estimated as fixed effect regression model ,

- model (M2) adds the non-linear terms of mother's age, (x_i) and the unstructured (uncorrelated) spatial random effects.

- Model M3, includes both structured random spatial effects, and categorical variables and the non-linear terms.

- Model M4 includes linear and non-linear covariates, unstructured and structured (correlated) effects.

In order to account for the variability or 'noise',in the data, which are not measurable by the categorical and continuous covariate factors, many different approaches to spatial smoothing have been developed. But the one that has gained wide acceptance and applicability is that of Besag, York and Mollie (the BYM model), which allows for both unstructured heterogeneity and spatially structured random effects according to Besag et al. (1991). For the structured spatial effect, a first-order intrinsic Gaussian Markov Random Field prior as suggested by Rue & Held (2005) and two-dimensional P-spline prior according to Lang & Brezger (2004).
For all functions and parameters, the appropriate prior functions were assigned. To estimate smooth effect functions and model parameters,we used the empirical Bayesian approach, as in Fahrmeir et al. (2013)). For fixed effect parameters, β, a non-informative diffuse prior was assumed such that$p(\beta_k) \sim$ constant
In order to estimate non-linear smooth functions of continous covariates (mother's age (in years), we adopt a Bayesian P-splines prior as suggested in the work of Fahrmeir & Lang (2001), as an extension of polynomial regression splines proposed by Eilers & Marx (1996). The basic assumption behind the P-splines approach is that the unknown smooth function f can be approximated by a spline of degree l defined on a set of equally spaced knots within the domain of x. The spline can then be written as a linear combination of basis function (B-spline), i.e.$f(x) = \sum_{j=1} \beta_j B_j(x)$ where $B_j(x)$ are $B-$splines. Smoothness of the basis function is achieved by a first- or second-order random walk model. We adopted the second-order random walk in this study i.e. $\beta_j = 2b_{j-1} + b_{j-2}$ with Gaussian error $\beta \sim N(0, \tau^2)$.
The model performances were compared via deviance information criterion (DIC) defined in Spiegelhalter et al. (2002b) as $DIC = D(\theta) + pD$ is a model selection criterion according to which the model performance is evaluated as the sum of a measure of fit, the posterior mean of the deviance, $D = E[-2log(f(y|parameters))]$, and a measure of complexity, the effective number of parameters, pD is obtained as the difference between the deviance posterior mean and the deviance evaluated at the parameters posterior mean. Thus, a model is preferred if it shows a lower DIC value.

6.3 Results of independent univariate and multivariate analysis

In this section the results are presented as follows. The section starts with DIC fits into univariate analysis. It then presents the multivariate analyses. The univariate analysis produced three sets of output: tables of posterior estimates of fixed effects covariates, non-linear graphs and maps of spatial structured residual effects based on the outputs from the application of the univariate conditional autoregressive prior model (Model M3). The results of the multivariate analysis are presented after implementing the Multivariate CAR model, which was discussed in Section 6.2.2

6.3.1 DIC Model fits

Table 6.3 presents the deviance information criteria(DIC) of the univariate independent analyses of the malnutrition indicators among under-five children in Burkina Faso, Ghana and Mozambique. Model M2 showed a substantial difference (Δ DIC) over model M1 with in range between 0.5 and 150. For each country, we provide the goodness of fit values for each type of child malnutrition. The best model selected has the lowest DIC. BYM model (M4) and CAR (M3) model are competing models and they are considered the best models overall because they yielded the smallest DIC values. The inference revealed that the spatial random effects were significant determinant factors for the high prevalent of childhood malnutrition .

Table 6.3: Deviance Information Criterion(DIC) of the goodness of fitness for univariate independent analysis

Model	Burkina Faso			Ghana			Mozambique		
	$D(\theta)$	pD	DIC	$D(\theta)$	pD	DIC	$D(\theta)$	pD	DIC
anaemia									
M1	9237.85	23.96	9261.81	3891.12	23.89	3915.01	13461.00	24.00	13485.00
M2	9087.20	36.20	9123.40	3875.64	30.81	3906.45	13335.97	34.66	13370.63
M3	9087.19	35.99	9123.18	3879.00	29.11	3908.11	13336.18	34.74	13370.92
M4	9087.00	36.20	9123.20	3875.25	30.71	3905.96	13334.39	34.67	13369.06
stunting									
M1	7368.49	23.94	7392.43	2384.72	23.75	2408.47	11047.37	23.98	11071.35
M2	7330.61	34	7364.61	2383.76	24.52	2408.28	10891.12	35.29	10926.41
M3	7330.89	33.29	7364.18	2383.80	24.51	2408.31	10891.10	35.14	10926.24
M4	7330.59	33.98	7364.57	2383.08	25.00	2408.08	10891.00	35.30	10926.30
Wasting									
M1	4909.40	23.86	4933.26	1325.71	23.40	1349.11	3176.17	23.69	3199.86
M2	4845.11	34.55	4879.66	1316.32	25.70	1342.02	3113.29	32.58	3145.87
M3	4846.58	34.00	4880.58	1323.96	25.75	1349.71	3113.31	32.70	3146.01
M4	4845.18	34.47	4879.65	1312.43	29.05	1341.48	3113.24	32.59	3145.83

6.3.2   Geographical mapping of childhood anaemia, stunting and wasting

Figures 6.2, 6.3 and 6.4 present the maps of the estimated smooth geographical effects on the childhood malnutrition indicators, anaemia, stunting and wasting, for the respective countries, after controlling for other covariates. The posterior means of the structured residual effects were plotted for the indicators from each country. From the graphics, it is apparent that there were geographical variations aggregated at regional or provincial level. The spatial residual estimates are grouped into five categories with black coloured regions signifying the lowest prevalence and a yellow coloured region denoting the highest prevalence. Individual discussions are given for each country's results below.

Burkina Faso: From the spatial plots shown in Figure 6.2, a high prevalence of anaemia was found in about 15% of the geographical areas of Burkina Faso, whereas high prevalence of stunting and wasting were found in 23% and 46% of the geographical areas, respectively. A visual inspection of the maps indicates that only Sahel region was simultaneously afflicted by high prevalence of all three malnutrition indicators (anaemia, stunted and wasted). The Est region was the only region that experienced high risk of both stunting and wasting. Cascades region had a high prevalence of both anaemia and stunting. Sahel and Cascades both recorded a high likelihood of anaemia among young children; three regions (Sahel, Est and Cascades) had high prevalence of childhood stunting, while two regions (Centre-Nord and Centre-Sud), constituted 30% of regions with higher prevalence of wasting. Two regions (Hauts-Bassins and Sud-Ouest) had the lowest likelihood of children being anaemic and wasted, while Centre-Ouest and Boucle du Mounhoun regions had the lowest prevalence of childhood stunting only while the Sud-Oest and Hauslts-Bassins regions recorded low prevalence anaemia nor wasting.

Ghana : Figure 6.3 shows evidence of geographical variations in the prevalence of the child malnutrition across Ghanaian regions, with high probability detected in many regions. The geographical areas implicated in high prevalence of anaemia, stunting and wasting were 30%, 50% and 50%, respectively. Generally, the maps show that the northern parts of the country experienced a higher prevalence of all the malnutrition indicators than did the southern parts. One can infer from the patterns for anaemia and stunting that the country has a north-south delineation, with the southern regions having lower likelihood of the indicators. The highest prevalence of both anaemia and stunting was detected in the Upper West region. In other words, Northern and Upper West regions experienced high prevalence of all (co-occurrence) three child malnutrition indicators (anaemia, stunting and wasting). The high prevalence and co-existence of both stunting and wasting (child growth failure) were detected in about 50% of the geographical areas, with Greater Accra

and Upper East regions having lower prevalence of childhood wasting. For instance, Brong Ahafo and Volta regions experience at higher prevalence of both stunting and wasting, while Ashanti, Central and Western regions had low prevalence of childhood stunting.

Mozambique: The spatial residual effects on the child malnutrition status for Mozambique are plotted and displayed in Figure 6.4. It can be seen that about 65% of the areas provinces in Mozambique (the seven provinces of Niassa, Nampula, Tete, Maniza, Gaza, Sofala and Zambezia) were found to have high prevalence of anaemia in young children living there. Furthermore, three provinces (Cabo, Delgado, and Gaza) recorded high prevalence of childhood stunting, and four other provinces had high prevalence of childhood wasting. There were evidence of co-occurrence of two or three of the malnutrition indicators in some provinces. For instance, anaemia and wasting prevalence was found to co-exist in the two provinces of Manica and Nampula, but only Gaza province recorded high prevalence in anaemia and stunting, while Maputo had a high prevalence of both stunting and wasting. The maps generally revealed that higher prevalence of stunting and wasting occurred in about 35% and 37%, respectively, of the provinces.
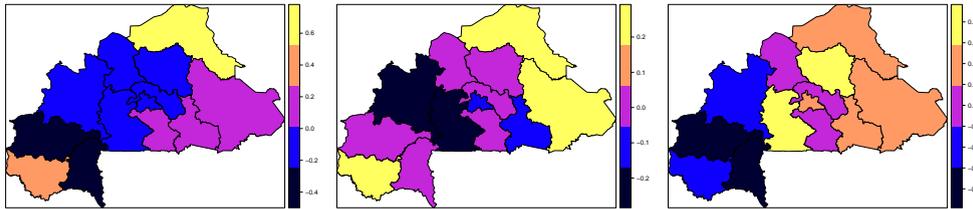
Figure 6.2: Mapping of posterior mean of structured spatial residual effect of childhood (a)anaemia, (b)stunting and (c) wasting showing posterior mean of prevalence in Burkina Faso, DHS2010

6.3.3 Non-linear effects of mother's age on childhood anaemia and malnutrition

In this section, we present the relationship between the effect of mother's age (mage) on the three child malnutrition indicators(anaemia, stunting and wasting.) These are shown in Figures 6.5.6.5 and 6.5 for Burkina Faso, Ghana, and Mozambique respectively. It is notable that all three relationships for all three countries can be depicted as non-linear functions. Each graph highlights critical points, or contours on the curve, that are important to the health professionals. The first graph is each set
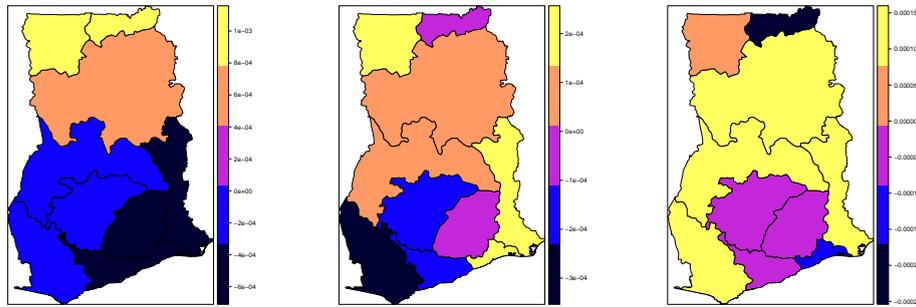
Figure 6.3: Mapping of posterior mean of spatial structured effect of childhood (a)anaemia, (b)stunting and (c) wasting showing posterior mean of prevalence in Ghana,DHS2008.
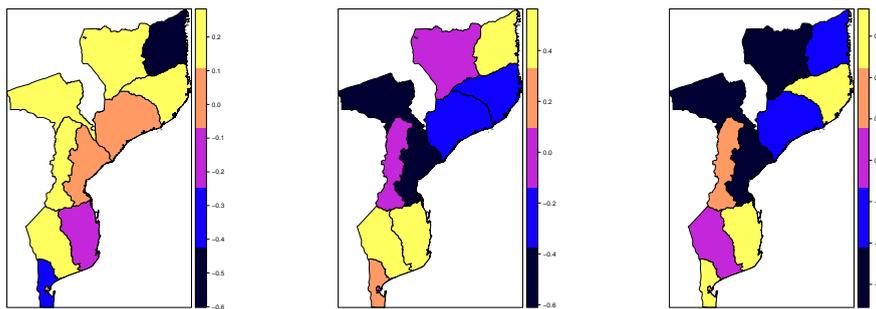
Figure 6.4: Mapping of posterior mean of structured spatial residual effect of childhood (a)anaemia, (b)stunting and (c) wasting showing posterior mean of prevalence in Mozambique DHS 2011.

(left hand panel) represents data from Burkina Faso. The middle panel represents from Ghna and the right hand panel shows the relationship for Mozambique.

Burkina Faso: Figure 6.5 (a) represents the effect of mother's age on her child's anaemia status. The non-linear relationship is represented as a flip S-shape curve, or reversed sigmoid curve. The graph can be segmented into three phases: (under-age (teenage, ≤ 20 years) mothers, young mother (20-32 years) represented by a 'plateau' showing that it stabilizes, and older mother (≥ 32 years). This indicates that among underage mothers the prevalence of anaemia in children declines slowly with the mother's increasing age and flexes at age between $20-30$ years, and soon after 30 years the anaemia prevalence then declines faster among older mothers. Furthermore Figure 6.6 (a) shows the relationship of mother's age with stunting prevalence in Burkina Faso. We can describe the relationship between mother's age and children risk of stunting as a reverse sigmoid-curve, which resembles that for anaemia risk (Figure 6.5 (a) but the but declines at a slower rate. By contrast, Figure 6.7 (a) shows that the relationship between the mother's age and childhood wasting was a power law curve or downward-concave curve, indicating that in Burkina Faso the risk of child-

hood wasting decreases faster among the children of older mothers (≥ 30 years) than it does for younger and teenage mothers.

Ghana: As displayed in Figure 6.5 (b), the graph depicting the relationship between the mother's age (in years) and the child's anaemia can be described as a U-shape curve or J-shape function. This indicates that a reduced prevalence of anaemia was found among children whose mother's age was less than 28 years, and for older mothers it rose exponentially. The lowest predicted of anaemia prevalence among Ghanaian children was found among mothers of age 28 -30 years. As shown in Figure 6.6 (b), a concave upward curve represents the function of the mother's age with stunting among children. The decreasing slope is an indication of slow decline in stunting prevalence with increased maternal age among children of older mothers. However, as shown in Figure 6.7 (b), a power function, or a concave downward curve with an increasing slope relationship is found for the relationship between mother's age and wasting prevalence. There is an indication of increasing prevalence of wasting found among children of older mothers.
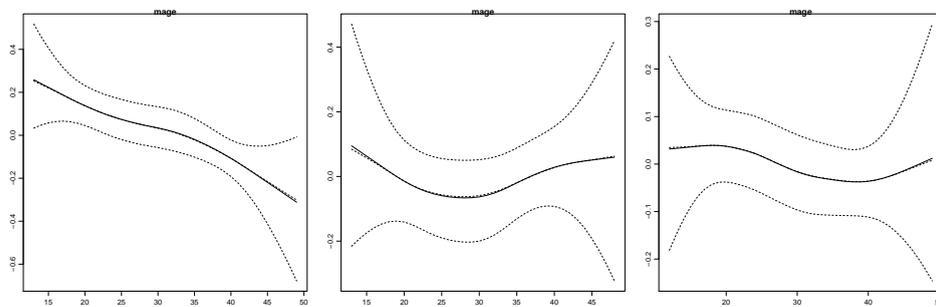
Figure 6.5: Non-linear plot of the effect of mother's age(years) on childhood anaemia, showing posterior mean and 95% credible interval for (a) Burkina Faso, (b)Ghana and (c) Mozambique. In all tests, the mother's age (in years) along the horizontal axis, and relative posterior risk estimate of anaemia on the vertical axis.
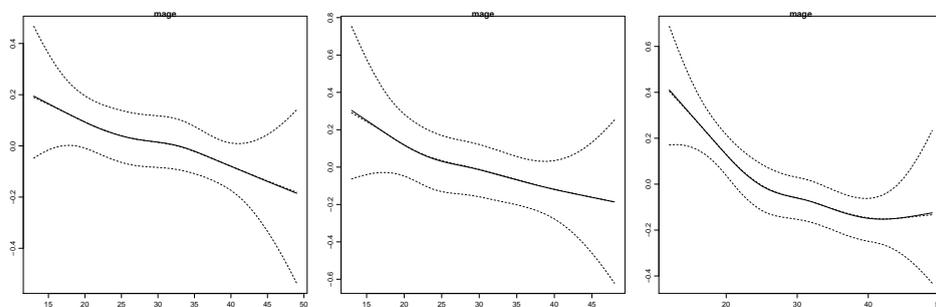
Figure 6.6: Non-linear plot of the effect of mother's age(years) on childhood stunting showing posterior mean and 95% credible interval for (a) Burkina Faso, (b)Ghana, and (c) Mozambique. In all tests, the mother's age (in years) along the horizontal axis, and relative posterior risk estimate of stunting on the vertical axis.
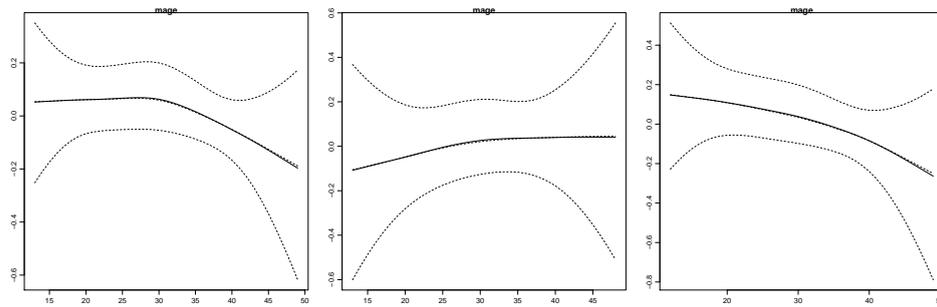
Figure 6.7: Non-linear plot of the effect of mother's age(years) on childhood wasting, showing posterior mean and 95% credible interval for (a) Burkina Faso, (b) Ghana, and (c) Mozambique. In all tests, the mother's age (in years) along the horizontal axis, and relative posterior risk estimate of wasting on the vertical axis.

Mozambique: The relationship between mother's age and anaemia prevalence can be described by a non-linear relation such like a flipped S− shape curve, as shown in Figure 6.5 (c), which is an indication of a small rise of anaemia prevalence with increasing age of the mother in children of teenage mothers (less than 20 years) and the childhood anaemia prevalence decreases until mother's age of 40 and then rises again. The lowest prediction of the anaemia prevalence among the Mozambican children is found among mother's of age 40 years. Furthermore, the relationship between mother's age and the child's stunting status could be described by a truncated exponential function (simply an inverse J-shape) as shown in Figure 6.6 (c), while the relationship with wasting could be described by a power-law function with a constant exponent scaling factor or concave down curve as shown in Figure 6.7 (c).

6.3.4 Fixed effects estimates of risk factors

The relative contribution of each risk factor to anaemia was estimated and presented as prevalence odd ratios derived from geoadditive binomial regression models with anaemia as the outcome. In this section, the fixed effect estimates of the covariates considered to be categorical in the models and the corresponding 95% credible intervals (CI) are presented for the three childhood malnutrition indicators. The interpretation of the binomial response models was done using its odd ratios and corresponding 95% credible intervals. Generally, whenever the odds ratio confidence interval includes one, it indicates that the factor effect is statistically not significant at 95% CI.

Risk factors associated with anaemia Tables 6.4, 6.5 and 6.6 present the results of the fixed-effects parameter estimates for child's anaemia based on Model 3 uni-

variate analysis . Table 6.4 included the odds ratios and 95% credible intervals for the categorical covariate factors such as age of the child, mother attended antenatal clinic , maternal literacy, households SES, iron syrup, birth spacing, breast feeding, morbidity, sleeping under bed-net. Among the Burkina Faso children, eight factors were found to be significantly associated with anaemia prevalence. The children aged 12-24 months had strongest prevalence of anaemia than the reference group (above 2 years old), with higher odds ratio of (OR= 1.19; CI 95%; 1.01, 1.40), but young infants, aged 1-5 months old had lower odds ratio of anaemia, OR=0.836; CI 95% (0.74 to 0.94). Similar result with higher odds ratio of anaemia was found among infants age less than 6 months and 12-23 months for Mozambique. However, older children in Ghana were less anaemic

Furthermore, it was found that children whose mothers attended antenatal clinic session had lower odds ratios with anaemia. That is, children whose mothers did not attend antenatal clinic at all were 3.75 times higher odds ratio of anaemia, however children whose mothers attended at least one antenatal clinic session had lower odds ratio of anaemia (OR=0.544, CI 95%; 0.37 to 0.79).

In addition, children living in families that belonged to a higher wealth index quartile were at lower prevalence for anaemia (unadjusted 0.90; 95% CI, 0.81 – 1.00) for richer household, and lowest anaemia prevalence among children of richest household (OR=0.87, CI 95% ; 0.72. to 1.03), but not significant. Children who suffered from disease morbidity (2 weeks prior to the survey) increased odds ratio of anaemia, particularly diarrhea has contributed to a higher anaemia prevalence among children in Ghana: OR=1.230 (1.047, 1.443), but diarrhea was not significant; Burkina Faso: OR= 1.141 (0.888, 1.46),and Mozambique: OR= 1.084 (0.931, 1.260) and Mozambique : OR= although they were not statistically significant. Also, morbidity such as fever had raised the odds ratios of anaemia prevalence in Mozambique :OR=1.170;95%CI(1.014, 1.348), but not significant in Ghana: OR=1.048 (0.796, 1.377). Also, children whose mother were literate (ability to read and write) had significant lower odds of being anemic compared with children of non-literate, except Ghana which indicates not significant, 0.954 (0.863, 1.055); Burkina Faso (OR=0.84, CI 95% ; 0.77 to 0.91); and Mozambique(OR=0.91, CI 95% ; 0.85 to 0.95) . In Table 6.6 for Mozambique data, more factors were significant associated with the prevalence of anaemia. The direction of the effects of covariates were similar to that of Burkina Faso, child spacing, breastfeeding, antenatal attendance, use bed-nets, mother's literacy and children age 5-11 months were associated with lower likelihood of anaemia. However, young children age 12-23 months and younger, whose mothers took iron syrup during pregnancy and did not attend antenatal sessions at all were found to raise odds ratio of anaemia. Other factors such as morbidity, measles vaccination and urban residence would increase the odds ratio of anaemia,

but the were not statistically significant. Generally, lower prevalence of anaemia was found among male children than the female counterparts and the children living in urban cities, although they were not statistically significant.

Table 6.4: Posterior odd ratios and 95% credible intervals of fixed effect risk factors for anaemia, stunting and wasting among under-five children in Burkina Faso (BDHS2010)

variables	stunting		wasting		anaemia	
	odds ratio	95% CI	odds ratio	95% CI	odds ratio	95% CI
(Intercept)	0.227	(0.177 , 0.289)	0.068	(0.049 , 0.094)	0.330	(0.272 , 0.400)
Sleeping under bednet						
No(ref.)	1.000		1.000		1.000	
bednet	1.044	(0.963 , 1.133)	0.924	(0.832 , 1.026)	0.963	(0.896 , 1.036)
Child age(months)						
1 − 5	1.872	(1.622 , 2.166)	1.163	(0.990 , 1.365)	1.064	(0.957 , 1.184)
6 − 11	1.991	(1.719 , 2.308)	1.770	(1.508 , 2.077)	0.836	(0.742 , 0.941)
12 − 24	0.171	(0.125 , 0.230)	0.626	(0.483 , 0.806)	1.193	(1.014 , 1.403)
> 24 months (ref.)	1.000		1.000		1.000	
Antenatal visits						
No	0.618	(0.267 , 1.422)	1.646	(0.448 , 5.954)	3.578	(1.714 , 7.449)
1 − 3	1.256	(0.820 , 1.926)	0.872	(0.454 , 1.686)	0.544	(0.374 , 0.793)
≥ 4 (ref.)	1.000		1.000		1.000	
Place of residence						
Rural (ref.)	1.000		1.000		1.000	
Urban	0.953	(0.869 , 1.046)	1.077	(0.957 , 1.210)	0.921	(0.846 , 1.002)
Child's sex						
Female (ref.)	1.000		1.000		1.000	
Male	0.906	(0.857 , 0.958)	0.912	(0.847 , 0.981)	0.994	(0.945 , 1.045)
Maternal literacy)						
No (ref.)	1.000		1.000		1.000	
literate	0.936	(0.851 , 1.027)	0.981	(0.869 , 1.104)	0.838	(0.769 , 0.912)
Mother wealth index)						
Poorest (ref.)	1.000		1.000		1.000	
poor	1.093	(0.971 , 1.230)	1.011	(0.863 , 1.183)	1.089	(0.980 , 1.210)
middle	1.177	(1.050 , 1.319)	0.993	(0.853 , 1.154)	1.088	(0.981 , 1.206)
richer	0.985	(0.879 , 1.103)	1.043	(0.899 , 1.208)	0.901	(0.812 , 0.999)
richest	0.658	(0.536 , 0.805)	0.888	(0.683 , 1.146)	0.865	(0.724 , 1.032)
Mother supplement						
No (ref.)	1.000		1.000		1.000	
Iron	0.610	(0.327 , 1.133)	1.265	(0.479 , 3.301)	2.446	(1.418 , 4.210)
Vitamin A	0.955	(0.805 , 1.136)	1.262	(1.014 , 1.585)	0.988	(0.849 , 1.152)
spacing(birth in3 years						
≤ 1 child(ref.)	1.000		1.000		1.000	
≥ 2 children)	1.039	(0.934 , 1.154)	1.057	(0.926 , 1.203)	0.968	(0.888 , 1.054)
Breast feeding						
No (ref.)	1.000		1.000		1.000	
Breastfed	1.058	(0.886 , 1.265)	1.727	(1.376 , 2.177)	0.697	(0.613 , 0.792)
Disease morbidity						
No	1.000		1.000		1.000	
diarrhea	1.230	(1.047 , 1.443)	1.327	(1.095 , 1.605)	0.949	(0.811 , 1.110)
Cough	0.012	(0.000 , 48.790)	2.472	(0.380 , 12.52)	0.931	(0.160 , 4.106)
fever	0.916	(0.790 , 1.062)	1.210	(1.006 , 1.453)	1.108	(0.962 , 1.274)
Vaccination						
No	1.000		1.000		1.000	
Measles vacc.	1.038	(0.958 , 1.126)	0.940	(0.847 , 1.045)	0.982	(0.913 , 1.057)
Access to electricity						
No	1.000		1.000		1.000	
Yes	0.846	(0.719 , 0.993)	0.907	(0.745 , 1.101)	1.065	(0.934 , 1.213)

Risk factors associated with stunting and wasting Generally, in all the countries under study, male children showed lower prevalence of malnutrition than the female counterpart. This was an indication of significant differentials in the prevalence of

Table 6.5: Posterior odds ratios and 95% credible intervals of fixed effect risk factors for anaemia, stunting and wasting among under-five children in Ghana (GDHS 2008)

Variables	stunting odds ratio	95% CI	wasting odds ratio	95% CI	anaemia odds ratio	95% conf. int.
(Intercept)	0.104	(0.060, 0.173)	0.037	(0.016 , 0.077)	0.781	(0.613 , 0.995)
Use bednet						
No	1.000		1.000		1.000	
Bednet	1.018	(0.898, 1.154)	0.876	(0.731 , 1.052)	0.916	(0.833 , 1.008)
Child's age)						
1−5	2.296	(1.612, 3.389)	0.829	(0.586 , 1.157)	1.216	(1.034 , 1.430)
6−11 months	3.104	(2.220, 4.498)	2.424	(1.847 , 3.184)	0.916	(0.780 , 1.075)
12−23 months	0.047	(0.017 0.116)	0.737	(0.458 , 1.166)	0.917	(0.718 , 1.171)
≥ 24 months (ref)	1.000		1.000		1.000	
Antenatal visits						
No	0.428	(0.102, 1.761)	11.68	(1.115 , 126.4)	1.453	(0.487 , 4.327)
1−3	1.520	(0.758, 3.072)	0.286	(0.087 , 0.921)	0.864	(0.504 , 1.482)
≥ 4 (ref.)	1.000		1.000		1.000	
Place of residence						
Rural(ref.)	1.000		1.000		1.000	
Urban	0.967	(0.826, 1.132)	0.987	(0.782 , 1.243)	0.961	(0.859 , 1.076)
Child's sex						
Female (ref.)	1.000		1.000		1.000	
Male	0.954	(0.863, 1.055)	0.995	(0.858 , 1.154)	0.960	(0.890 , 1.036)
Mother literacy						
No (ref.)	1.000		1.000		1.000	
literate	0.971	(0.849, 1.110)	0.945	(0.771 , 1.152)	1.025	(0.931 , 1.129)
Wealth group						
Poorest (ref.)	1.000		1.000		1.000	
poor	1.429	(1.123, 1.818)	1.136	(0.788 , 1.629)	1.162	(0.967 , 1.397)
middle	1.046	(0.831, 1.311)	0.805	(0.560 , 1.136)	0.993	(0.841 , 1.171)
richer	0.810	(0.609, 1.071)	0.819	(0.531 , 1.241)	1.039	(0.855 , 1.262)
richest	0.651	(0.439, 0.954)	1.050	(0.602 , 1.797)	0.784	(0.604 , 1.015)
Mother Supplement						
No (ref.)	1.000		1.000		1.000	
Iron	0.490	(0.181, 1.31)	6.328	(1.169 , 35.51)	1.258	(0.590 , 2.680)
Vitamin A	1.015	(0.850, 1.217)	0.877	(0.706 , 1.099)	0.959	(0.841 , 1.094)
Spacing (birth in 3 yrs)						
≤ 1 child (ref.)	1.000		1.000		1.000	
≥ 2 children	0.954	(0.791, 1.145)	1.236	(0.978 , 1.552)	1.104	(0.978 , 1.246)
Breast feeding						
Not breastfed (ref.)	1.000		1.000		1.000	
Breasted	1.175	(0.814, 1.751)	1.270	(0.704 , 2.591)	0.899	(0.762 , 1.062)
Disease morbidity						
No (ref.)	1.000		1.000		1.000	
Diarrhea	1.141	(0.888, 1.461)	1.526	(1.090 , 2.126)	1.125	(0.916 , 1.380)
Cough	0.886	(0.675, 1.159)	1.656	(1.149 , 2.372)	0.017	(0.001 , 115.4)
Fever	1.048	(0.796, 1.377)	0.703	(0.463 , 1.053)	1.235	(0.988 , 1.542)
Measles vaccination.						
Not (ref.)	1.000		1.000		1.000	
Vaccinated	0.936	(0.802, 1.094)	0.683	(0.563 , 0.831)	0.998	(0.889 , 1.121)
Access to electricity						
No access	1.000		1.000		1.000	
Access	1.010	(0.865, 1.180)	1.119	(0.878 , 1.423)	1.161	(1.031 , 1.307)

malnutrition among the under-five children of both sexes. Children belonging to upper socio-economic status(SES) had a lower likelihood of being undernourished (stunting and wasting) compared to the most economically deprived household . Children, age below 12 months (infants) were at higher odds of child malnutrition (adjusted height-for-age and weight-for-height) than the older children (over 2 years

Table 6.6: Posterior odds ratios and 95% credible intervals of fixed effect risk factors for anaemia, stunted and wasting among under-five children in Mozambique (MDHS2011)

Variables	stunting		wasting		anaemia	
	odds ratio	95% CI	odds ratio	95% CI	odds ratio	95% CI
(Intercept)	0.318	(0.270 , 0.374)	0.020	(0.014 , 0.029)	0.369	(0.322 , 0.422)
Sleeping under bednet						
No(ref.)	1.000		1.000		1.000	
bednet	0.994	(0.934 , 1.058)	1.154	(1.003 , 1.331)	0.930	(0.879 , 0.985)
Child age(months)						
$1-5$	1.351	(1.211 , 1.508)	0.997	(0.790 , 1.254)	1.000	(0.914 , 1.093)
$6-11$	1.866	(1.682 , 2.070)	2.030	(1.669 , 2.467)	0.892	(0.816 , 0.974)
$11-24$	0.298	(0.244 , 0.362)	0.936	(0.654 , 1.324)	1.200	(1.048 , 1.373)
> 24 months (ref.)	1.000		1.000		1.000	
Antenatal visits						
No	1.241	(0.656 , 2.344)	0.983	(0.183 , 5.153)	2.564	(1.460 , 4.496)
$1-3$	0.926	(0.672 , 1.275)	0.996	(0.433 , 2.313)	0.644	(0.485 , 0.855)
≥ 4 (ref.)	1.000		1.000		1.000	
Place of residence						
Rural (ref.)	1.000		1.000		1.000	
urban	0.994	(0.929 , 1.063)	0.855	(0.727 , 1.000)	1.055	(0.993 , 1.120)
Child's sex						
Female (ref.)	1.000		1.000		1.000	
Male	0.922	(0.881 , 0.965)	0.902	(0.815 , 0.998)	0.996	(0.956 , 1.038)
Mother literacy						
No (ref.)	1.000		1.000		1.000	
literate	0.895	(0.843 , 0.950)	0.819	(0.711 , 0.941)	0.905	(0.857 , 0.954)
Mother wealth index)						
Poorest (ref.)	1.000		1.000		1.000	
poor	1.133	(1.015 , 1.263)	0.913	(0.710 , 1.171)	0.926	(0.837 , 1.026)
middle	0.993	(0.896 , 1.099)	0.958	(0.755 , 1.210)	0.973	(0.886 , 1.069)
richer	0.978	(0.887 , 1.078)	0.867	(0.684 , 1.090)	0.957	(0.877 , 1.044)
richest	0.705	(0.581 , 0.855)	0.897	(0.566 , 1.410)	1.033	(0.877 , 1.217)
Mother supplement						
No (ref.)	1.000		1.000		1.000	
Iron	1.179	(0.748 , 1.857)	1.417	(0.425 , 4.657)	2.030	(1.357 , 3.035)
Vitamin A	0.917	(0.842 , 0.999)	0.896	(0.760 , 1.061)	0.997	(0.922 , 1.079)
Spacing(birth in 3 yrs						
≤ 1 child(ref.)	1.000		1.000		1.000	
≥ 2 children)	0.977	(0.905 , 1.056)	0.998	(0.842 , 1.174)	0.917	(0.857 , 0.980)
Breast feeding						
No(ref.)	1.000		1.000		1.000	
Breastfed	0.894	(0.789 , 1.014)	1.110	(0.869 , 1.431)	0.725	(0.660 , 0.797)
Disease morbidity						
No	1.000		1.000		1.000	
Diarrhea	1.084	(0.931 , 1.260)	0.914	(0.664 , 1.242)	0.941	(0.811 , 1.091)
Cough	0.258	(0.022 , 1.765)	6.709	(0.961 , 36.64)	1.200	(0.328 , 3.937)
fever	1.170	(1.014 , 1.348)	1.152	(0.851 , 1.544)	1.088	(0.947 , 1.249)
Vaccination						
No	1.000		1.000		1.000	
Measles vacc.	0.983	(0.920 , 1.050)	1.042	(0.909 , 1.200)	1.021	(0.961 , 1.085)
Access to electricity						
No	1.000		1.000		1.000	
electricity	0.901	(0.817 , 0.994)	1.127	(0.895 , 1.412)	0.958	(0.881 , 1.042)

of age).

The present data demonstrated a peculiar case, where children whose mothers took iron syrup supplementation during pregnancy had higher odds ratios for the prevalence of one or both stunting and wasting in all the three countries. For instance, the iron supplementation had respectively higher odds ratios for stunting and wasting in Burkina Faso as: OR= 1.265 (0.479, 3.301) and 2.446 (1.418, 4.210); Ghana : OR=6.328 (1.169, 35.51)and OR=1.258 (0.590, 2.680) , and Mozambique OR= 1.417;(0.425, 4.657) and OR=2.030 (1.357, 3.035)

There were consistent association in the influence of childhood morbidity with childhood stunting and wasting. Children, who had experienced one disease morbidity or the other (two weeks before the survey) had higher probability of being either stunting, wasting or both. For instance, in Burkina Faso (see Table 6.4)children who suffered diarrhea: OR= 1.327 (1.096, 1.605) and cough 1.210 (1.01, 1.453) contributed to higher odds ratios for wasting, and diarrhea OR=1.23 (1.017, 1.443) raised the odds ratio of stunting , but in Ghana (Table 6.5)diarrhea OR=1.53(1.09, 2.13) and cough OR=1.66 (1.15, 2.37) had raised the odds ratios of childhood wasting. In Mozambique (see Table 6.6), children who suffered disease morbidity such as fever:OR=1.17 (1.014, 1.348) was found to have raised the prevalence of stunting but not wasting.

6.4 Results of Multivariate Conditional Autoregressive (MCAR) Analysis

The results presented in this section include the estimation of parameters from the equation 6.4 via multivariate conditional auto-regressive (MCAR) prior model discussed in section 6.2.2 and implemented in WinBUGS version 14. After adjusting for the e factors, the covariates such as child's age(categorical), maternal wealth index(soio-economic deprivation) and birth intervals found to be significant under univariate analysis. The Table 6.7, Table 6.8 and Table 6.9 contain the posterior estimates for the fixed effect risk factors, structured spatial effects and variance-component parameters.

Burkina Faso: Table 6.7 presents the posterior estimates from the multivariate conditional autoregressive (MCAR) model analysis, which included estimates of some fixed effects, the regional (spatial random effects), and conditional correlations between childhood malnutrition prevalence with the regions in Burkina Faso. The posterior estimates showed that there were significantly strong associations between the child malnutrition prevalence with the regions in Burkina Faso. Out of 13 regions in Burkina Faso, six regions were estimated with high prevalence of stunting and seven regions recorded significant low prevalence. Regions with high prevalence of stunting are:Boucle de Mouhoun, Cascades, Centre, Nord and Plateau Central. In contrast, childhood wasting and anaemia prevalence were significantly low in those regions, but high prevalence of stunting. Surprisingly, the results revealed that wasting and anaemia recorded significantly high prevalence in those regions with low stunting prevalence. Seven regions were found with high childhood wasting and anaemia prevalence are :Centre-Est,Centre-Ouest, Centre-Sud,Est,Hauts Basins, Sahel and Sud-Ouest. There were strong negative correlation between stunting and

wasting;$\rho_{12} = -0.998$; 95% CI (-1.000, -0.984), and a perfect negative correlation ($\rho_{13} = -1$) between stunting and anaemia, but a significant positive correlation between wasting and anaemia i.e. $\rho_{23} = 0.997$; 95% CI (0.9778 to 1.0000) (refer to Table 6.7).

Ghana: Table 6.8 presents the posterior estimates of the conditional correlation between the malnutrition status and regions in Ghana. There was significantly low prevalence of wasting among under-five children in the Upper east with posterior estimate; -0.499; 95% CI(-1.016, -0.018), but a significantly high prevalence of wasting was detected in the Upper West 0.441; 95% CI(0.100 to 0.778). Six regions were predicted with high anaemia prevalence, although they were insignificant. There was a significantly low prevalence anaemia in Volta region of the country with value -0.369; 95% CI(-0.671 to -0.090). The results further revealed that there were weak and insignificant correlation between stunting and wasting: $\rho_{12} = 0.184$; 95% CI (-0.475,0.728), stunting and anaemia $\rho_{13} = -0.038$; 95%CI (-0.621, 0.582), and a weak negative correlation between wasting and anaemia, $\rho_{23} = -0.037$; 95%CI (-0.65, 0.595). The geographical variations were significant for stunting:σ_{u1}^2=0.7549 (0.4693, 1.264); wasting σ_{u2}^2=0.9197; (0.535, 1.591) and anaemia: σ_{u3}^2=0.7335, (0.4606, 1.214) for the prevalence among under five children in Ghana.

Mozambique: The multivariate analysis results presented in Table 6.9 for Mozambique children data. Three provinces were associated with high stunting prevalence. Children living in Niassa, Tete, and Sofala showed significantly higher prevalence for stunting, while five other provinces(Cabo delgado, Nampula, Zambezia, Gaza and Maputo provincia) were found with significantly low stunting prevalence. A significantly high prevalence of childhood wasting was detected in Niassa, Tete, Manica, and Sofala. Five other provinces were associated with significantly high prevalence of anaemia: Niassa, Cabo Delgado, Zambezia, Maputo provincia and Maputo cidade. Table 6.9 also presents the estimates of random effects for the provinces, and the regional variances (geographical variation) of each malnutrition. There was a strong positive correlation between stunting and wasting; 0.986; (0.899, 1.000), which was significant at 95% CI. There were significantly negative correlation between stunting and anaemia:-0.720, (-0.934, -0.308) and between wasting and anaemia :-0.640; (-0.903 -0.174). The results further indicated that the geographical variations in the prevalence pattern of stunting: 1.427 (0.914, 2.268), wasting: 1.751 (1.117, 2.803) and anaemia 0.556 (0.280, 0.979) were significant across the provinces in Mozambique.

In summary, multivariate mapping modeling provides the epidemiologists and health practitioners a tool to study the disease etiology and jointly quantify the conditional spatial correlation and geographical variations of each outcome across regions and association among the indicators within the region (area).

Table 6.7: Posterior estimates and 95% credible intervals of multivariate conditional association between childhood malnutrition indicators and regions parameters among under-five children in Burkina Faso

variables	parameters	stunting			wasting			anaemia		
		mean	2.50%	97.50%	mean	2.50%	97.50%	mean	2.50%	97.50%
intercept	α	-2.074	-199.5	199.8	2.159	-208.6	197.3	0.437	-197.6	192.3
$1-5$ months	β_1	1.471	-195.2	196.0	1.301	-196.4	175.0	2.529	-192.4	196.6
$6-11$	β_2	-0.047	-194.9	191.3	-7.222	-199.5	187.8	0.006	-198.4	192.8
$12-24$	β_3	1.543	-194.8	198.3	-3.824	-208.3	203.0	2.372	-194.4	200.5
$24-36$	β_4	-0.168	-191.6	189.6	0.428	-214.6	200.1	-0.396	-194.0	193.3
poorest	β_5	-0.913	-191.4	193.9	2.334	-186.1	205.7	-1.088	-193.0	190.5
poor	β_6	1.762	-200.0	197.1	-2.205	-196.2	191.1	1.328	-190.6	198.1
middle	β_7	-1.513	-187.4	194.4	2.248	-197.0	199.7	-1.094	-195.7	197.5
richer	β_8	-0.835	-194.6	190.8	-0.774	-191.6	198.2	1.257	-193.2	200.0
>2 children	β_9	0.417	-195.9	199.4	0.733	-185.1	191.9	-0.551	-195.0	190.8
Regions	Random effects									
Boucle de Mouhoun	ϕ_1	2179	2150	2199	-367.1	-373.6	-363.6	-790.9	-875.6	-502.0
Cascades	ϕ_2	3953	3898	3969	-661.8	-670.2	-657.5	-1444.0	-1569.0	-909.2
Centre	ϕ_3	5686	5666	5720	-949.1	-957.3	-938.9	-2213.0	-2393.0	-2070.0
Centre-Est	ϕ_4	-2621	-2662	-2609	441.3	437.8	454.9	953.2	618.9	1037.0
Centre-Nord	ϕ_5	4362	4346	4378	-731.5	-748.0	-727.8	-1643.0	-1727.0	-1426.0
Centre-Ouest	ϕ_6	-564.4	-597.3	-494.2	91.44	86.71	97.93	210.0	-111.0	307.8
Centre-Sud	ϕ_7	-870.5	-883.5	-828.3	144.1	134.0	160.1	362.1	75.10	698.7
Est	ϕ_8	-5412	-5464	-5396	910.9	906.3	924.4	2005.0	1626.0	2088.0
Hauts Basins	ϕ_9	-3748	-3786	-3722	629.5	624.3	631.8	1415.0	1153.0	1544.0
Nord	ϕ_{10}	3046	3027	3096	-511.6	-524.7	-506.3	-1132.0	-1195.0	-920.7
Plateau Central	ϕ_{11}	2988	2961	2999	-503.4	-505.1	-499.7	-1107.0	-1184.0	-880.6
Sahel	ϕ_{12}	-4080	-4097	-4059	685.8	683.1	692.4	1506.0	1055.0	1588.0
Sud-Ouest	ϕ_{13}	-4917	-4987	-4905	821.5	813.5	838.0	1879.0	1505.0	2017.0
spatial variances	σ_{u1}^2	8151	(5573	12320)						
	σ_{u2}^2	—	—	—	1428	(933.3	2196)			
	σ_{u3}^2	-	-	-	-	-	-	3126	(2119	4747)
Correlation	ρ_{12}	-0.998	(-1.000	-0.984)						
	ρ_{13}	-1.0								
	ρ_{23}	0.997	(0.9778	1.000)						

6.5 Discussion

The present study has investigated the risk factors and small-area geographical variations of child health outcomes in the three countries understudy. The present study revealed that in the countries under study, the children who slept under treated bed nets had lower prevalence of anaemia. In other words, the use of bed nets would significantly reduce the odds ratios of anaemia in the young children. Conversely, the use of treated bed net did not have a significant association with child under-nutrition (stunting and wasting) in these countries. Although, the use of bed nets resulted in a reduction of the odds of wasting among children in both Burkina Faso and Ghana, but not stunting, nevertheless in Mozambique, the use of bednets led to reduction in the prevalence of stunting, but this difference was not statistically significant. A similar outcome has been reported in other analyses for other sub-Saharan Africa: insecticide-treated bed nets were very efficacious in reducing the prevalence of anaemia and boosting the mean hemoglobin concentrations in Kenyan

Table 6.8: Posterior estimates and 95% credible intervals of multivariate conditional association between childhood malnutrition indicators and regions parameters among under-five children in Ghana (GDHS 2008)

variables	parameters	Stunting mean	2.50%	97.50%	Wasting mean	2.50%	97.50%	anaemia mean	2.50%	97.50%
intercept	α	-2.529	-2.859	-2.193	-1.469	-1.893	-1.099	-0.609	-0.852	-0.321
$1-5$	β_1	**1.169**	**0.773**	**1.536**	**-1.668**	**-2.179**	**-1.181**	**0.387**	**0.061**	**0.688**
$6-11$	β_2	**1.456**	**1.128**	**1.800**	-0.333	-0.710	0.058	0.100	-0.200	0.393
$12-24$	β_3	**-1.663**	**-2.880**	**-0.292**	**5.276**	**3.822**	**6.629**	-0.727	-1.718	0.335
$25-36$	β_4	**-2.433**	**-3.630**	**-1.519**	**-1.219**	**-1.878**	**-0.626**	0.105	-0.240	0.462
poorest	β_5	**0.420**	**0.213**	**0.640**	0.275	-0.046	0.571	0.072	-0.094	0.246
poor	β_6	0.489	0.285	0.690	0.234	-0.045	0.519	0.097	-0.068	0.266
middle	β_7	0.065	-0.141	0.267	-0.060	-0.426	0.285	0.000	-0.187	0.175
richer	β_8	-0.305	-0.544	-0.067	-0.248	-0.675	0.142	0.052	-0.127	0.239
birth interval (≥ 2)	β_9	0.006	-0.170	0.167	0.067	-0.161	0.281	0.092	-0.031	0.209
Regions	Random effects									
Ashanti	ϕ_1	-0.095	-0.382	0.198	-0.167	-0.631	0.244	0.072	-0.187	0.328
Brong Ahafo	ϕ_2	0.230	-0.064	0.523	0.329	-0.067	0.757	-0.012	-0.287	0.251
Central	ϕ_3	-0.166	-0.508	0.172	-0.262	-0.781	0.218	-0.132	-0.405	0.149
Eastern	ϕ_4	-0.160	-0.458	0.123	-0.339	-0.806	0.104	-0.104	-0.370	0.143
Greater Accra	ϕ_5	0.133	-0.168	0.438	-0.307	-0.802	0.141	0.066	-0.177	0.325
Northern	ϕ_6	0.054	-0.185	0.286	0.186	-0.158	0.536	0.202	-0.004	0.412
Upper East	ϕ_7	-0.131	-0.444	0.137	**-0.499**	**-1.016**	**-0.018**	0.090	-0.161	0.322
Upper West	ϕ_8	0.093	-0.177	0.349	**0.441**	**0.100**	**0.778**	0.145	-0.073	0.366
Volta	ϕ_9	0.251	-0.045	0.542	0.308	-0.086	0.752	**-0.369**	**-0.671**	**-0.090**
Western	ϕ_{10}	-0.208	-0.488	0.083	0.310	-0.079	0.706	0.042	-0.210	0.297
spatial variances	σ_{u1}^2	0.7549	(0.4693	1.264)						
	σ_{u2}^2	–	–	–	0.9197	(0.535	1.591)			
	σ_{u3}^2	-	-	-	-	-	-	0.7335	(0.4606	1.214)
Correlation	ρ_{12}	0.184	(-0.475	0.728)						
	ρ_{13}	-0.038	(-0.621	0.582)						
	ρ_{23}	-0.037	(-0.650	0.595)						

children, as reported in ter Kuile et al. (2003) and Desai et al. (2005).

The present study further indicates a significant association between maternal characteristics and childhood malnutrition. The finding revealed that mother's iron and, vitamin A supplementation and antenatal attendance were found to be associated with the prevalence of anaemia and other malnutrition indicators in children. The children of mothers, who did not attend antenatal clinic session during pregnancy would have an higher probability of being anaemic and malnourished than those children whose mothers had attended four or more antenatal sessions.

Additionally, the study revealed that children from wealthy households were found to be at reduced risk of anaemia and malnutrition than the materially deprived households. This finding is consistent with the studies conducted in other parts of sub-Saharan Africa. For instance, a study carried out in Rwanda by Kateera et al. (2015) indicates that children with a lower socio-economic status (SES) (measured by education status and occupation) were found to be associated with higher prevalence of anaemia, malaria infection and undernutrition. Notwithstanding the complexity in our model, the findings are in accordance with previous studies in Ghana, which indicated that malnutrition prevalence among children from the poorest SES households was as much as twice that of their counterparts from the richest households, as reported in Van de Poel et al. (2007) and Amugsi et al. (2013). A study

Table 6.9: Posterior estimates and 95% credible intervals of multivariate conditional association between childhood malnutrition indicators and provinces(regions) among under-five children in Mozambique from equation (6.3)

variables	Parameters	Stunting			Wasting			anaemia		
		mean	2.50%	97.50%	mean	2.50%	97.50%	mean	2.50%	97.50%
intercept	α	-4.274	-196.4	189.8	-1.283	-203.5	188.3	0.6402	-198.6	189.0
$1-5$	β_1	-1.108	-271.1	222.9	1.459	-198.3	203.0	-1.142	-207.0	205.9
$6-11$	β_2	-1.010	-229.5	231.7	1.090	-250.5	299.2	0.560	-332.0	275.2
$12-24$	β_3	4.426	-208.5	217.2	-1.645	-260.0	247.9	-12.86	-301.4	271.7
$25-36$	β_4	8.502	-194.6	224.3	1.091	-195.6	207.6	-4.077	-206.3	216.5
poorest	β_5	3.988	-220.6	233.4	-4.064	-239.8	220.1	-4.907	-232.2	216.0
poor	β_6	-1.563	-190.2	203.9	3.453	-192.5	202.0	-2.165	-194.7	206.5
middle	β_7	-2.372	-196.9	193.3	1.052	-212.2	213.9	0.825	-202.7	194.1
richer	β_8	1.836	-189.8	198.4	3.511	-198.4	198.3	-1.236	-197.3	191.8
birth intervals ≥ 2	β_9	1.514	-207.5	204.6	1.848	-191.6	204.5	1.546	-202.2	198.5
Provinces	Random effects									
Cabo Delgado	ϕ_1	538.1	302.0	733.5	979.6	843.2	1126.0	383.5	265.2	530.3
Gaza	ϕ_2	-1380.0	-1496.0	-1319.0	-1628.0	-1950.0	-1434.0	512.3	403.8	577.1
Inhambane	ϕ_3	-400.5	-554.0	-221.8	-676.2	-1051.0	-316.8	-122.8	-225.3	-27.72
Manica	ϕ_4	-843.1	-908.5	-799.6	-1002.0	-1202.0	-888.1	322.7	274.6	368.5
Maputo cidade	ϕ_5	962.2	820.3	1147.0	1205.0	970.2	1485.0	-289.3	-345.8	-245.5
Maputo provincia	ϕ_6	2080.0	1430.0	2204.0	2482.0	1920.0	2680.0	-788.2	-886.7	-680.4
Nampula	ϕ_7	788.2	716.6	868.5	906.2	723.8	1138.0	-336.3	-382.9	-283.1
Niassa	ϕ_8	-100.6	-290.7	252.6	-320.8	-542.4	384.0	-257.5	-372.1	-152.4
Sofala	ϕ_9	-557.3	-636.9	-480.0	-721.2	-795.5	-602.0	77.25	-2.95	153.9
Tete	ϕ_{10}	-975.6	-1007.0	-913.4	-1181.0	-1294.0	-995.5	295.4	172.7	412.8
Zambezia	ϕ_{11}	-112.3	-182.6	156.0	-45.5	-164.5	138.5	203.1	174.2	294.4
spatial variances $*10^{-3}$	σ^2_{u1}	1427	(913.6	2268)						
	σ^2_{u2}	−	−	−	1751	(1117	2803)			
	σ^2_{u3}	-	-	-	-	-	-	556	(279.5	978.9)
Correlations	ρ_{12}	0.986	(0.899	1.000)						
	ρ_{13}	-0.720	(-0.934,	-0.308)						
	ρ_{23}	-0.640	(-0.903,	-0.174)						

conducted in regions of Brazil by Osório et al. (2001) similarly found that the determinant factors associated with the risk of anaemia among children included poorer households, inadequate nutritional food intake, poor sanitation, lack of portable drinking water and infectious diseases. Our study revealed that the odds of anaemia decreased with increasing child's age, which conflicts with that one from Brazil, the increasing odds of malaria observed across the same age groups in other studies involving malaria-endemic settings (Ehrhardt et al., 2006; Osterbauer et al., 2012). Furthermore, our results indicated that the child's age group is an important determining factor for the severity of malnutrition, including anaemia. The results revealed that the children of age group 6 - 11 months (young infants) were associated with high odds of anaemia and highest odds of wasting, but lower odds of stunting. This finding corroborated similar studies in sub-Saharan Africa, which showed that malnutrition was also found to be more prevalent among older than younger children (Pongou et al., 2006; Omilola et al., 2010). It is not surprising that our findings revealed a relatively high odds of anaemia among younger infants (age 1-5 months) and highest odds of anaemia among the age group (12-23 months). Studies, such as reported by Chang et al. (2011), have shown that at various stages in the early years of a child's development there were changing heamoglobin requirements for physical and psychomotor functioning and cognitive development. It is worth not-

ing childhood anaemia is seldom investigated or measured among infants below six months of age. Where this study indicates there are high odds of anaemia in young infants aged 1-5 months, it could suggest that child bearing mothers were themselves anaemic during pregnancy.

Our finding have, in addition, revealed that some factors exerted non-linear relationship or curve-like association with child malnutrition status. The S-shaped growth curve (sigmoid growth curve) reveals a pattern of growth in which, in a new environment, the malnutrition prevalence within the sample population started increasing slowly, in a positive acceleration phase; then increases rapidly and approaches an exponential growth rate as in the J-shaped curve; but then decrease in a negative acceleration phase until at zero growth rate as the population stabilizes. For more discussion, see Salkind (2010) on a similar curve-linear growth in a population context.

The spatial distribution of anaemia and child growth failure among young children in the countries under study are presented as smooth maps. This demonstrated the merit of the proposed approach. The output corroborated the findings in similar studies conducted in other sub-Saharan Africa countries. For example, a model-based geostatistics was used by Magalhaes & Clements (2011) to map the risk of anaemia, malaria and helminth prevalence in three neighbouring countries (Burkina Faso,Niger, Mali) in West Africa, where they reported that anaemia prevalence was found among 37% of preschool children, which could have been averted by treating malnutrition and malaria related infections. Other researcher had used Bayesian geostatistical prediction in West Africa to estimate local variations in *Schistosoma haematobium infection* Clements et al. (2009), where a high risk of S. haematobium infections was detected in the north-western part of the Niger River valley. The study also found a clustered high-density of S. haematobium infection in western and central Mali, and the north-eastern region of Burkina Faso. Kandala et al. (2009) applied a Bayesian geoadditive model on DHS data from three sub-Saharan African countries and the method detected a high pattern of childhood malnutrition in eastern and north-eastern Zambia, central Malawi and southern Tanzania.

The usefulness of multivariate disease mapping models has been emphasized in previous studies. For example, Kinyoki et al. (2017) recently used the multivariate approach for modeling the ecological co-morbidity of childhood diseases in Somalia. Assunção & Castro (2004) applied a multivariate model in the study of multiple cancer site incidence. The results from this work will consolidate the applicability and usefulness of multivariate mapping methods for outperforming separate independent analysis. The present study applied multivariate joint modeling and demonstrated the strength of the method in estimating the conditional correlation among malnutrition indicators and co-occurrence within the region. In addition

to the computation the induced correlation between the malnutrition indicators, the approach estimate the geographical variation of each individual malnutrition prevalence across the regions. The findings further identified potential socio-demographic risk factors of childhood malnutrition, which can be used to target specific interventions and even combinations of inventions. Such results could aid policy makers in designing a combination of multiple strategies to optimize the scarce health resources in a more effective manner.

6.6   Summary and Conclusion

In epidemiological studies, several diseases share common risk factors or co-exist spatially. Disease mapping allows health practitioners and epidemiologists to proffer hypotheses pertaining to their etiology and gain better understanding of the geographical pattern of the disease risks. This chapter proposed spatial analysis to jointly investigate two child growth indicators (stunting and wasting) and anaemia (micro-nutrient deficiency) in three Sub-Saharan African countries. Both univariate independent CAR and multivariate analyses were carried out on the malnutrition indicators. The study then explored the multivariate conditional autoregressive error model to jointly model the small area-specific effect of the co-occurrence of malnutrition status across the regions.

There were 19,390 children under the age of five involved from the three countries. In the three countries, the stunting prevalence was highest in Mozambique (42.8%), Burkina Faso had highest wasting prevalence (15.5%), while Ghana had highest anaemia prevalence of (39.4%). While potential socio-demographic risk factors were identified by the univariate analysis, the multivariate joint analysis results revealed that, in Burkina Faso, the spatial correlation between stunting and wasting was negatively correlated ; -0.998; 95% CI (-1.000, -0.984), and a perfect negative correlation of -1 between stunting and anaemia, but a significant positive correlation between wasting and anaemia: 0.997;95% CI (0.978, 1.000). For the Ghanaian data, the variations of the individual childhood nutritional indicators was stunting 0.7549; 95%CI (0.4693, 1.264); wasting 0.9197; (0.535, 1.591) and anemia 0.7335, (0.4606, 1.214). These were significant across the geographical regions of the country, but the correlations between pairs of indicators were all not significant. Among the Mozambican children, there was significantly positive correlation between stunting and wasting; 0.986; (0.899, 1.000); a significant negative correlation between stunting and anaemia:-0.720,95% CI(-0.934, -0.308), and a significantly negative correlation between wasting and anaemia:-0.640; (-0.903, -0.174. The statistical relevance of the

findings and spatial maps identified hot spots or provinces that require appropriate nutritional interventions for pregnant and lactating mothers, as well as their children. and to assist the relevant agencies in the optimum allocation of scarce health resources in a effective manner for child survival.

The central aim of the present study was to jointly and simultaneously analyze three malnutrition indicators in children less than five years. The findings provide reasonable patterns for the co-occurrence in geographic prevalence across regions. The Bayesian multivariate model adopted provides a flexible and robust tool to assess the risk factors in a unified regression model. The proposed method facilitates the estimation of conditional correlation between the multiple disease outcomes (malnutrition status) and spatial dependence within the region and across regions. The results obtained provide a better understanding of the spatial variations in the co-existence and etiological patterns of childhood undernutrition, which would have been neglected with the standard spatial analysis. The findings should inspire public health practitioners, epidemiologists, and policy makers to design a combination of intervention strategies and to allocate more effectively their scarce health resources. Despite the complexity of the methodology, the results are reasonable and consistent with those from univariate analysis. The advantage of the multivariate approach is that it yields more precise estimates and allows easy interpretation of regressions coefficients defined in terms of odds ratios. A potential drawback, however, is the huge computational burden involved in MCMC simulations. The researcher's experience with the univariate models suggests that the two approaches provide similar results with respect to the direction and strength of predictor outcome associations.

Chapter 7

Conclusions

The aim of this thesis was to develop and apply Bayesian model approaches for analyzing spatial data exhibiting unusual spatial patterns and to extend the related methodology. To achieve this aim, four specific research objectives were established:

1. To apply recently developed Bayesian structured additive regression models for analyzing spatial data in order to gain a better understanding of the spatial patterns of child birth weights in a developing country and investigate the risk factors of poor birth outcomes among under-five children.

2. To extend the methodology of generalized linear mixed models to Poisson spatial generalized linear mixed models (GLMM) to analyze spatial data that exhibit a high over-dispersion and spatial association by accounting for extra Poisson variation in death counts among under-five children.

3. To apply the Bayesian approach to model mis-specification and spatial clustering for the child health outcomes

4. To extend the methodology of an conditional autoregressive (CAR) model to a multivariate conditional autoregressive (MCAR) model by analyzing the impact of the spatial dependence and induced correlation of multiple health conditions.

The first research objective was addressed in Chapter 3. The models presented in that chapter could accommodate different kinds of data (categorical, metrical and geographical data) in a unified regression model. The method was able to identify hot-spots for poor birth outcomes. The findings provide useful insight into the geographic disparities across the states (districts) in Nigeria and the underlying risk factors for these outcomes, which include indoor air pollution from cooking with

solid fuels(e.g. charcoal, coal, fire wood and agricultural residues). Other significant risks associated with low birth weight were short birth intervals, under-age mothers and economically deprived households (poor households).

The second objective was addressed in Chapter 4 by implementing a Bayesian hierarchical disease mapping approach to investigate over-dispersion and spatial auto-correlation in child mortality rates in the context of a developing country. Poisson regression models with random effects were formulated to assess the potential risk factors and over-dispersion in the mortality data for under-five year old children. The random effects were specified to reflect the potential tendency of neighbouring regions to sharing unobserved similar risk factors. Through the empirical) method, standardized mortality ratio (SMR) was explored to identify regions with unusually elevated risk and geographical patterns for child mortality prevalence at specific area (state) level. The development of Poisson GLMMs within the Bayesian framework made it possible to identify both individual and geographic factors associated with under-five mortality outcomes across the spatial domain. Furthermore, being able to highlight small areas with elevated risk has implications for health care providers in the detection and management of childhood mortality.

Chapter 5 presented models for analyzing different classes of data structures. Three semi-parametric models were fitted to data for childhood anaemia belonging to exponential family distributions. The models demonstrated the flexibility of the approach in accommodating different data structures as spatial generalized linear mixed models.

The study then considered other extensions of disease mapping models for multiple health outcomes in Chapter 6. In order to account for conditional correlation among multiple diseases and the spatial association within the region, a multivariate conditional autoregressive (MCAR) model was formulated and implemented to identify the geographic patterns of co-occurrence of multiple malnutrition indicators. The chapter modified the popular shared component model originally developed for Poisson count data to suit binary data. The multivariate conditional autoregressive model was proposed and applied to carry out joint spatial analysis to investigate shared and divergent patterns in malnutrition prevalence among under-five children, suggesting possible common risk factors and different patterns in their geographical variation. Shared patterns of spatial variation in health outcomes and different causes of mortality could also be attributed to regional differences in health care awareness and quality of health services and provision available at the sub-national levels.

7.1 Future Research

A recent development in Bayesian disease mapping models Lawson (2013) would be the extension from purely spatial models to models for space-time, and interaction models for disease rates. For instance, the ability to identify the spatial patterns of disease risk that evolve systematically over a period of time and understanding the trend patterns would be beneficial in terms of interpretation and potential for detention of localized excesses.

In the future, it may be relevant to consider how to combine the ideas of space-time analysis of a single disease and multivariate spatial disease mapping to explore the formulation and application of Bayesian models for analysis of multiple health outcomes in space and time.

Appendix A

Appendix A includes two sections: the Derivation of Latent Utility Model and BayeX Codes for Cumulative Multinomial models.

Supplementary Material from Chapter 3

A.1: Derivation of Latent Utility Models

The general latent model is motivated by the original work of McFadden (1974), who stated that the conditional logit model could be extended from a single latent model to multiple choices (categorical selection). Suppose the utility, for say an individual child i birth size falls to child birth category j, having a random utility function of the jth alternative has the form

$$U_j = \bar{\eta}_j + \epsilon_j \tag{7.1}$$

Furthermore, let individual i choose option j (so that $Y_i = j$) if categorical j provides the highest level of utility, or

$$Y_i = j \ \text{ if } U_{ij} \geq U_{il} \ \text{ for all } \ l = 0, \ldots, J, \tag{7.2}$$

Suppose that the ε_{ij} are independent across the birth category and individual child and have type I extreme value distributions. Then the choice Y_i follows the conditional logit model. The type I extreme value distribution has cumulative distribution function is defined by $F(\varepsilon) = \exp(-\exp(-\varepsilon))$, and pdf: $f(\varepsilon) = \exp(-)\exp(-\exp(-\varepsilon))$. For discussion about the assymmetry of the distribution, see Wooldridge (2010) for prove and comparison with normal density function.

Given the extreme value distribution the probability of choice 0 is

$$
\begin{aligned}
Pr(Y_i = 0 | X_{i0}, \ldots, X_{iJ}) &= Pr(U_{i0} > U_{i1}, \ldots, U_{i0} > U_{iJ}) \\
&= Pr(Pr(\varepsilon_{i0} + X'_{i0}\beta - X'_{i1}\beta > \varepsilon_{i0} \ldots, \varepsilon_{i0} + X'_{i0}\beta - X'_{i1}\beta > \varepsilon_{iJ}) \\
&= \int_{-\infty}^{+\infty} \int_{-\infty}^{\varepsilon_{i0} + X'_{i0}\beta - X'_{i1}\beta} \ldots \int_{-\infty}^{\varepsilon_{io} + X'_{i0}\beta - X'_{iJ}\beta} f(\varepsilon_{i0}) f(\varepsilon_{i0}) \ldots f(\varepsilon_{iJ}) d\varepsilon_{iJ} \ldots d\varepsilon_{i0} \\
&= \exp(-\varepsilon_{i0}) \exp(-\exp(-\varepsilon_{i0}). \exp(-\exp(-\varepsilon_{io} - X_{i0}\prime\beta + X'_{i1}\beta) \\
&\times \exp(-\exp(-\varepsilon_{io} - X_{i0}\prime\beta + X'_{iJ}\beta) \\
&= \frac{\exp(\eta_{io})}{\sum_{i=0}^{J} \exp(\eta_{io})} \\
&= \frac{\exp(\eta_{io})}{\sum_{i=0}^{J} \exp(\eta_{io})} \\
&\cdots\cdots\cdots \\
Pr(Y_i = J | X_{iJ}, \ldots, X_{iJ}) &= \frac{\exp(\eta_{iJ})}{\sum_{i=0}^{J} \exp(\eta_{iJ})}
\end{aligned}
$$

In general, the probability of a woman giving birth to a child belonging to a category j birth size, is given by

$$
Pr(Y_i = j | \eta) = \frac{\exp(\eta_j)}{\sum_{i=0}^{J} \exp(\eta_j)}
$$

where $\eta = \mathbf{X}'\boldsymbol{\beta}$ is the linear predictor. That is for a binary variable y_i, the probability of low birth category (i.e success) is given by $\pi_i = \mathrm{F}(X'_i\beta)$

A.2 BayesX Code for Cumulative Multinomial Models

```
# Paper Cumulative logit model
# BayesX analysis
# Loading dataset
dataset d
d.infile , maxobs=100000 using c:\Users\215076528\Desktop\cum\
   cngn2008.txt
d.describe

# Loading map file
```

```
map m
# Empirecal Bayes (No covariate)
m.infile using c:\Users\215076528\Desktop\cum\nigeria.bnd.txt
remlreg r

r.outfile = c:\Users\215076528\Desktop\cum\spatial
logopen using c:\Users\215076528\Desktop\cum\spatial.log
r.regress cat = state(spatial,map=m), family=cumlogit using d
logclose

# Nonlinear models
r.outfile = c:\Users\215076528\Desktop\cum\monlinear
logopen using c:\Users\215076528\Desktop\cum\nonlinear.log

r.regress cat = mbmi(psplinerw2) + fbage(psplinerw2) +
  deadchildren(psplinerw2) + hhsize(psplinerw2) +
  state(spatial,map=m), family=cumlogit using d
logclose

# Spatial Nonlinear models
r.regress cat = csex1 + twin1 + urban + stunted + wasted +
  underwg + poor +   middle + richer + richest + diarrhea +
  fever + cough +   space + Iron +   Ante + maldrug +
  mheight(psplinerw2) + mbmi(psplinerw2) + hhsize(psplinerw2)
    +
 state(spatial,map=m), family=cumlogit using d

# Mother characters model
r.outfile = c:\Users\215076528\Desktop\birth\modacx
logopen using c:\Users\215076528\Desktop\birth\log_modacx
r.regress cat = urban + smoke+ literate + catholic+ christain
    +
  muslim +   poor + middle + richer + richest+ fbage(
    psplinerw2) +
  mbmi(psplinerw2) +   mweight(psplinerw2) + state(spatial,
    map=m),
family=cumlogit using d
logclose
end
```

Appendix B

This section presents the probability distributions and the WinBUGS codes for the analysis in Chpater 4.

Supplementary Material from Chapter 4

B.1   Probability Distributions

The probability distributions used in this book are summarized next:

- **Bernoulli distribution** The Bernoulli distribution with parameter $0 < q < 1$, denoted as $Ber(q)$, has probability mass function mass function

$$p(k) = q^k(1-q), \;\; k \in (0,1) \tag{7.3}$$

- **Binomial distribution** The Binomial distribution with parameters $m \in \{1, 2, 3, \ldots m\}$ and $0 < q < 1$, denoted as $Bin(q)$, has probability

$$p(k) = \binom{m}{k} q^k(1-q), \;\; k \in (0,1,\ldots,m) \tag{7.4}$$

- **Poisson distribution:** the Poisson distribution with parameter $\lambda > 0$, denoted as $Pois(\lambda)$, has probability mass function

$$p(k) = \exp(-\lambda)\frac{\lambda^k}{k!}, \;\; k \in \mathbb{N} \tag{7.5}$$

- the Negative Binomial distribution with parameters $a > 0$ and $\lambda > 0$, denoted as $NBin(a, \lambda)$, has probability mass function

$$p(k) = \binom{a + k - 1}{k} \frac{a}{(a + \lambda)^a} \frac{\lambda}{(a + \lambda)^k} \quad k \in \mathbb{N}; \tag{7.6}$$

- the Normal distribution with parameters $\mu \in \mathbb{R}$ and $\sigma^2 > 0$, denoted as $N(\mu, \sigma^2)$, has probability density function

$$f(x) = \frac{1}{\sigma \sqrt{2\pi}} \exp\left(-\frac{1}{2\sigma^2}(x - \mu)^2\right), \quad x \in \mathbb{R}; \tag{7.7}$$

- the Log Normal distribution with parameters $\mu \in \mathbb{R}$ and $\sigma^2 > 0$, denoted as $LN(\mu, \sigma^2)$, has probability density function

$$f(x) = \frac{1}{x\sigma \sqrt{2\pi}} \exp\left(-\frac{1}{2\sigma^2}(\ln(x) - \mu)^2\right), \quad x \in \mathbb{R}^+; \tag{7.8}$$

- **Gamma distribution:** A random variable $\mathcal{X}$ is distributed according to the two-parameters, $\alpha > 0$ and $\beta > 0$ Gamma distribution, which will henceforth be denoted as $X \sim Gam(\alpha, \beta)$, if its probability density function is given by

$$f(x) = \frac{x^{\alpha-1} \beta^\alpha \exp(-\beta x)}{\Gamma(\alpha)}, \quad x > 0 \tag{7.9}$$

- when $\alpha = 1$, the Gamma distribution reduces to the Negative Exponential distribution (which is denoted as $X \sim \exp(\beta)$ with probability density function

$$f(x) = \beta \exp(-\beta x), \quad x > 0 \tag{7.10}$$

- the Inverse Gaussian distribution, with parameters, $\mu > 0$ and $\beta > 0$, denoted as $IGau(\mu, \beta)$, has probability density function

$$f(x) = \frac{\mu}{\sqrt{2\pi \beta x^3}} \exp\left(-\frac{1}{2\beta x}(x - \mu)^2\right), x > 0 \tag{7.11}$$

B.2   WinBUGS Code for Bayesian Hierarchical Models

```
# Appendix A
#WinBuGS Codes for Chapter 3
#Models
########******** POISSON–GAMMA MODEL********
model
{
for (i in 1:N)
{
  # Poisson likelihood for observed counts
  O[i]~dpois(mu[i])
  mu[i]<-E[i]*theta[i]
  # Relative Risk
  theta[i]~dgamma(a,b)
  SMR[i]<- O[i] / E[i]
  prob[i]<-step(theta[i]-1)
}
# Prior distributions for "population" parameters
a~dexp(0.1)
b~dexp(0.1)
# Population mean and population variance
mean<-a/b
var<-a/pow(b,2)
}
#Data
#Initials
list(a=1, b=1)
list(a=10,b=10)
list(a=10,b=10)

###************LOG–NORMAL MODEL*******#
model
{
for (i in 1 : N) {
  v[i]~ dnorm(0.0,tau.v)
  O[i] ~ dpois(mu[i])
  log(mu[i])<-log(E[i])+alpha0+v[i]
  RR[i] <- exp(alpha0+v[i]) # Area-specific relative risk (
```

```
        for maps)
}
# Other priors:
alpha0 ~ dflat()
tau.v ~ dgamma(0.5, 0.0005) # prior on precision
sigma.v <- sqrt(1 / tau.v) # standard deviation
}


#### Data
#Data


#Initials
list(tau.v = 1, alpha0=0, v=c(0, 0, 0, 0, 0, 0, 0, 0, 0, 0,
    0, 0, 0, 0, 0, 0,
    0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0,
        0, 0))



###********* LOG NORMAL WITH COVARIATES #######
model{
  for (i in 1 : N) {
    v[i]~ dnorm(0.0, precv)
    O[i]    ~ dpois(mu[i])
    log(mu[i]) <- log(E[i])+alpha[1]+alpha[2]*((dr[i]-mean(dr
        []))/sd(dr[])/sqrt(37))+alpha[3]*((sn[i]-mean(sn[]))/
        sd(sn[])/sqrt(37))+alpha[4]*((dw[i]-mean(dw[]))/sd(dw
        [])/sqrt(37))+ alpha[5]*((pr[i]-mean(pr[]))/sd(pr[])/
        sqrt(37)) + alpha[6]*((cc[i]-mean(cc[]))/sd(cc[])/sqrt
        (37)) + v[i]
    SMR[i]<-(O[i]+eps2)/(E[i]+eps2)
    RR[i]<- exp(alpha[1]+alpha[2]*((dr[i]-mean(dr[]))/sd(dr
        [])/sqrt(37))+alpha[3]*((sn[i]-mean(sn[]))/sd(sn[])/
        sqrt(37))+alpha[4]*((dw[i]-mean(dw[]))/sd(dw[])/sqrt
        (37))+ alpha[5]*((pr[i]-mean(pr[]))/sd(pr[])/sqrt(37))
        + alpha[6]*((cc[i]-mean(cc[]))/sd(cc[])/sqrt(37)) + v
        [i])

    #### Excedence Probability
    probability[i]<-step(RR[i]-1+eps)   #probabilty of RR>1
```

```
    }

    # Other priors:
    for (j in 1:6){
       alpha[j]~dnorm(0,0.01)
    }
    eps<-1.0E-16
    eps2~dnorm(0,0.05)
    precv~dgamma(0.05, 0.0005)                    # prior on
       precision of v
    sigmav <- sqrt(1 / precv)             # standard deviation
       of v

}

### DATA
### INITIAL
list( alpha=c(0,0,0,0,0, 0), precv=1, eps2=1,
     v=c
        (0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,(
        )

###*******************************************************************


################## CAR MODEL ##################
model {
  # Likelihood
  for (i in 1 : N) {
    O[i]  ~ dpois(mu[i])
    log(mu[i]) <- log(E[i]) + alpha0+u[i]
    RR[i] <- exp(alpha0 +u[i]) # Area-specific relative risk
       (for maps)
  }
  # CAR prior distribution for random effects:
  u[1:N] ~ car.normal(adj[], weights[], num[], tau.u)
  for(k in 1:sumNumNeigh)
  {
    weights[k] <- 1
```

```
  }
  # Other priors:
  # Other priors:
  alpha0 ~ dflat()
  tau.u ~ dgamma(0.5, 0.0005) # prior on precision
  sigma.u <- sqrt(1 / tau.u) # standard deviation
}

#Data
#Data


#Initials
list(tau.u = 1, alpha0=0, u=c(0, 0, 0, 0, 0, 0, 0, 0, 0, 0,
   0, 0, 0, 0, 0, 0, 0,
  0,  0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0,
    0))


list(tau.u = 1, alpha0 = 0, u=rep(0,37))
list(tau.u = 1, alpha0 = 0, u=rep(0,37))


######*********** CAR COVARIATES***************

model{
  for (i in 1 : N) {
    O[i]  ~ dpois(mu[i])
    log(mu[i]) <- log(E[i])+alpha[1]+alpha[2]*((dr[i]-mean(dr
       []))/sd(dr[])/sqrt(37))+alpha[3]*((sn[i]-mean(sn[]))/
       sd(sn[])/sqrt(37))+alpha[4]*((dw[i]-mean(dw[]))/sd(dw
       [])/sqrt(37))+ alpha[5]*((pr[i]-mean(pr[]))/sd(pr[])/
       sqrt(37)) + alpha[6]*((cc[i]-mean(cc[]))/sd(cc[])/sqrt
       (37)) + u[i]
    SMR[i]<-(O[i]+eps2)/(E[i]+eps2)
    RR[i]<- exp(alpha[1]+alpha[2]*((dr[i]-mean(dr[]))/sd(dr
       [])/sqrt(37))+alpha[3]*((sn[i]-mean(sn[]))/sd(sn[])/
       sqrt(37))+alpha[4]*((dw[i]-mean(dw[]))/sd(dw[])/sqrt
       (37))+ alpha[5]*((pr[i]-mean(pr[]))/sd(pr[])/sqrt(37))
        + alpha[6]*((cc[i]-mean(cc[]))/sd(cc[])/sqrt(37)) + u
       [i])
```

```
  #### Excedence Probability
  probability[i]<-step(RR[i]-1+eps)   #probabilty of RR>1
}

# CAR prior distribution for CH random effects:
u[1:N]~car.normal(adj[], weights[], num[], precu)
for(k in 1:sumNumNeigh) {
  weights[k] <- 1
}

# Other priors:
for (j in 1:6){
  alpha[j]~dnorm(0,0.01)
}

eps<-1.0E-16
eps2~dnorm(0,0.05)
precu~dgamma(0.05, 0.0005)          # prior on precision
    of u

sigmau <- sqrt(1 / precu)          # standard deviation
    of u

}

### DATA

### INITIAL
list( alpha=c(0,0,0,0,0, 0), precu=1, eps2=1,
u=c
  (0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,(
  )

###********CONVOLUTION MODEL (NO COVARIATES) *********

#######Initials *************************
list(tau.u=1, tau.v = 1, beta =0, u=rep(0,37),v=rep(0,37))
list(tau.u = 1, tau.v = 1, beta =0,u=rep(0,37), v =rep(0,37)
```

###********** CONVOLUTION MODEL DATA with covariates

```
model{
  for (i in 1 : N) {
    O[i]   ˜ dpois(mu[i])
    log(mu[i]) <- log(E[i])+alpha[1]+alpha[2]*((dr[i]-
        mean(dr[]))/sd(dr[])/sqrt(37))+alpha[3]*((sn[i]-
        mean(sn[]))/sd(sn[])/sqrt(37))+alpha[4]*((dw[i]-
        mean(dw[]))/sd(dw[])/sqrt(37))+ alpha[5]*((pr[i]-
        mean(pr[]))/sd(pr[])/sqrt(37)) + alpha[6]*((cc[i
        ]-mean(cc[]))/sd(cc[])/sqrt(37)) +  u[i]+v[i]
    SMR[i]<-(O[i]+eps2)/(E[i]+eps2)
    RR[i]<- exp(alpha[1]+alpha[2]*((dr[i]-mean(dr[]))/sd
        (dr[])/sqrt(37))+alpha[3]*((sn[i]-mean(sn[]))/sd(
        sn[])/sqrt(37))+alpha[4]*((dw[i]-mean(dw[]))/sd(
        dw[])/sqrt(37)) + alpha[5]*((pr[i]-mean(pr[]))/sd
        (pr[])/sqrt(37)) + alpha[6]*((cc[i]-mean(cc[]))/
        sd(cc[])/sqrt(37)) + u[i]+v[i])
    v[i]˜dnorm(0,precv)
    probability[i]<-step(RR[i]-1+eps)  #probabilty of RR
        >1
  }

  # CAR prior distribution for CH random effects:
  u[1:N]˜car.normal(adj[], weights[], num[], precu)
  for(k in 1:sumNumNeigh) {
    weights[k] <- 1
  }

  # Other priors:
  for (j in 1:6){
    alpha[j]˜dnorm(0,0.01)
}

eps<-1.0E-16
eps2˜dnorm(0,0.05)
precu˜dgamma(0.05, 0.0005)                          # prior on
    precision of u
precv˜dgamma(0.05,0.0005)                                #
    prior on precision of v
```

```
sigmau <- sqrt(1 / precu)                                    #
    standard deviation of u
sigmav <- sqrt(1 / precv)                                    #
    standard deviation of v


 }

 ### DATA

 ### INITIAL
 list( alpha=c(0,0,0,0,0,0),precu=1,precv=1,eps2=1,
u=c
   (0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,(
    ,
 v=c
    (0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0,0
    )

#####*************COnvolution   model***********######

   #### Alternative model
   model {
     for (i in 1 : N) {
       # Data distribution
       O[i] ~ dpois(mu[i])
       log(mu[i]) <- log(E[i]) + alpha + beta[1]*dr[i]+
         beta[2]*sn[i]+ beta[3]*dw[i] + beta[4]*pr[i]+ +u[i
           ]+v[i]
       # Area-specific residual relative risk (for maps)
       RR[i] <- exp(alpha + u[i]+v[i])
       ## Exchangeable prior on unstructured random effects
       v[i]~dnorm(0.0,tau.v)
     }
     # Probability that RR >1 (excess risk)
     pRR[i] <- step(RR[i]-1)
   }
   # iCAR prior distribution for spatial random effects u[i
     ]
   u[1:N] ~ car.normal(adj[], weights[], num[], tau.u)
```

```
      for(k in  1:sumNumNeigh) {
        weights[k] <- 1
      }
      # Other priors:
      alpha ~ dflat()
      for(i in 1:4) {beta[i] ~dnorm(0.0, 0.0001)}
      tau.v ~ dgamma(0.005, 0.005)
      sigma.v <- sqrt(1 / tau.v)
      tau.u ~ dgamma(0.005, 0.005)
      sigma.u <- sqrt(1 / tau.u)
      }
###### DATA

####*****************Initials**************
list(tau.u = 1, tau.v = 1, alpha=0, beta=c(0, 0,0,0),
   v=c(0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0,
   0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0),
  u=c(0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0,
   0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0))
######## ANALYSIS ENDS HERE *****************************
```

B.2   INLA CODE for Models Chapter 4

```
# Packages required
rm( l i s t =ls ( ) )
# install.packages("faraway") TO MANAGE REGRESSION ANALYSIS
# install.packages("dplyr") for
library ('faraway')
library ("MASS")
library ("lattice")

library ("ctv")

library ("sp")

library (maptools)

library (rgdal)
require (RColorBrewer)

library ("RColorBrewer") # added coloured brewers

library (spdep)
require (INLA)
library (stargazer )
library (xtable )
library (dplyr )

#******************LOADING DATA
   *****************************
data<-read.csv("C:/Users/215076528/Desktop/surv_codes/
   nigeria2013direct.csv",header=T,sep=",")

dim( data )
head ( data )
attach ( data )
table (V113)
table (V116)
state <- table (SSTATE,B5)
write.csv(state , "aliveb.csv")
```

```
table(B0)
str(data)
data$distr.unstruct = data$district



#******************LOADING GEOGRAPGHICAL MAPS
    *****************************
ngn.graph<- readRDS("C:/Users/215076528/Desktop/surv_codes/
    NGA_adm1.rds")# NIGERIA admin 1

#gha.graph<- readRDS("C:/Users/215076528/Desktop/districtal/
    GHA_adm1.rds") # GHANA
#moz.graph<- readRDS("C:/Users/215076528/Desktop/districtal/
    MOZ_adm1.rds") # Mozambique




#******************STARTS TANZANIA SECTIONS HERE
    *****************************
windows(width=7, height=7)
png("bukaa11.png")
#png("buk%5d.png") # By including % you can generate multiple
     plots
#pdf("bukall.pdf")
par(mfrow=c(2,3), mai=c(0.6,0.5,0.1,0.1),mgp=c(2,0.7,0))
#plot(civ3.graph, main="Cote d'Ivoire") #plot(civ2.graph,
    main="Cote d'Ivoire") #plot(bkf.graph, main="Burkina faso
    ")
#plot(civ.graph, main="Cote d'Ivoire") #plot(gha.graph, main
    ="Ghana") plot(moz.graph, main="Mozambique") #Mozambique

plot(ngn.graph, main="NIGERIA")
dev.off()

adjtz<-poly2nb(ngn.graph)#Creates adjacency for ken
adjtz



nb2INLA("ngn.graph",adjtz) #INLA graph file #spdep command
```

NIGERIA

```
names(data)
##*********************TANZANIA ANALYSIS
    ******************************************
table(data$anebin);table(data$stunted);table(data$wasted);
    table(data$mortality);table(data$undered)
table(data$magecat)
# Secere ANEMIA 4
f1<-imm~no + ant + vitA + breast1 + csex1 + twin1 + cbw1 +
    cbw2 + cbw3 + christain +
  muslim + cath + urban + space + prim + sec + high + mage20
      + mage30 + mage40 + abbm1 + abbm3 +
    abbm4 +pn2 + di2 + fe2
f2<-u5mm~no + ant + vitA + breast1 + csex1 + twin1 + cbw1 +
    cbw2 + cbw3 + christain +
  muslim + cath + urban + space + prim + sec + high + mage20
      + mage30 + mage40 + abbm1 + abbm3 +
    abbm4
f3<-y5~no + ant + vitA + breast1 + csex1 + twin1 + cbw1 +
    cbw2 + cbw3 + christain +
  muslim + cath + urban + space + prim + sec + high + mage20
      + mage30 + mage40 + abbm1 + abbm3 +
    abbm4

r1<-inla(f1,family="binomial",data=data,control.compute=list(
    dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
    compute=TRUE))
r2<-inla(f2,family="binomial",data=data,control.compute=list(
    dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
    compute=TRUE))
r3<-inla(f3,family="binomial",data=data,control.compute=list(
    dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
    compute=TRUE))
#control.fixed = list(expand.factor.strategy="inla"),
summary(r1)
summary(r2)
summary(r3)
a<- summary(r1)
b<- summary(r2)
```

```
c<- summary(r3)
ww <-exp(r1$summary.fixed)

w=summary(result0)
summ  <-exp(result0$summary.fixed) # Code for coverting to
    odd
bb <-exp(r1$summary.fixed)
bb1 <-exp(r2$summary.fixed)
bb2 <-exp(r3$summary.fixed)
kk<-round(bb1, digits=2)
kk2<-round(bb2, digits=2)
write.csv(kk,"LINmortality.csv")
write.csv(kk,"Lchildmortality.csv")
write.csv(kk2,"Lu5mortality.csv")




# PLOTTING THE REGRESSION COEFFICIENTS ON GRAPH
table(data$district)
library(ggplot2)
library(plyr)
IID=exp(r1$summary.fixed)
NBSum2DF <- data.frame(IID)
NBSum2DF$var<- row.names(NBSum2DF)
NBSum2DF <- subset(NBSum2DF, var != "(Intercept)")
NBSum2DF <- subset(NBSum2DF, var != "sigma2")
ggplot(data = NBSum2DF, aes(x = reorder(var, X0.025quant),
y = mean,
ymin = X0.025quant, ymax = X0.975quant)) +
geom_pointrange(size = 1.4) +
geom_hline(aes(intercept= 0), linetype = "dotted") +
xlab("var\n") + ylab("\n Posterior odds for infant mortality
    ") +
coord_flip() + theme_bw(base_size = 20)
dev.off()

# UNSTRUCTURED  SPATIAL MODEL
f10 <-imm~no + ant + vitA + breast1 + csex1 + twin1 + cbw1 +
    cbw2 + cbw3 + christain +
```

```
    muslim + cath + urban + space + prim + sec + high + mage20
        + mage30 + mage40 + abbm1 + abbm3 +
    abbm4 + f(district, model="iid")
f20<-u5mm~no + ant + vitA + breast1 + csex1 + twin1 + cbw1 +
    cbw2 + cbw3 + christain +
    muslim + cath + urban + space + prim + sec + high + mage20
        + mage30 + mage40 + abbm1 + abbm3 +
    abbm4 + f(district, model="iid")


f30<-y5~no + ant + vitA + breast1 + csex1 + twin1 + cbw1 +
    cbw2 + cbw3 + christain +
    muslim + cath + urban + space + prim + sec + high + mage20
        + mage30 + mage40 + abbm1 + abbm3 +
    abbm4 + f(district, model="iid")


r10<-inla(f10,family="binomial",data=data,control.compute=
    list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
    compute=TRUE))
r20<-inla(f20,family="binomial",data=data,control.compute=
    list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
    compute=TRUE))
r30<-inla(f30,family="binomial",data=data,control.compute=
    list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
    compute=TRUE))
#control.fixed = list(expand.factor.strategy="inla"),

summary(r10)
summary(r20)
summary(r30)
a<- summary(r10)
b<- summary(r20)
c<- summary(r30)

w=summary(result0)
summ  <-exp(result0$summary.fixed) # Code for coverting to
    odd
bb  <-exp(r10$summary.fixed)
bb1 <-exp(r20$summary.fixed)
bb2 <-exp(r30$summary.fixed)
```

```
kk<-round(bb, digits=2)
kk1<-round(bb1, digits=2)
kk2<-round(bb2, digits=2)
write.csv(kk,"NSIFMortality.csv")
write.csv(kk1,"NSchildmortality.csv")
write.csv(kk2,"NSU5mortality.csv")


rr1<-r30$summary.random$district # residual district effect
   morta
tza<- data.frame(rr1)
rr1<-r10$summary.random$district # residual district effect
   morta
tza<- data.frame(rr1)
tza$district<- row.names(tza)
N     = table(data$district)
mean = rr1[,2],
row.names(N)
sd    = rr1[,3]
##  CREATING STA ERROR
length(data$district)
tt<- data.frame(table(data$district))
tta<-cbind(sd,tt)
se    = tta[,1] /tta[,3]
ggplot(data = tza, aes(x = reorder(district, X0.025quant),
y = mean,  ymin = X0.025quant, ymax = X0.975quant)) +
geom_pointrange(size = 1.4) +
geom_hline(aes(intercept= 0), linetype = "dotted") +
xlab("state\n") + ylab("\n unstructured posterior residuals")
    +
theme_bw(base_size = 20)
+ geom_errorbar(aes(x, ymin=y-se, district, ymax=y+se)
ggplot(data = tta, aes(x = reorder(district, sd)
)
dev.off()


###### Spatai Random effect
# STRUCTURED  SPATIAL MODEL
f11 <-imm~no + ant + vitA + breast1 + csex1 + twin1 + cbw1 +
   cbw2 + cbw3 + christain +
```

```
    muslim + cath + urban + space + prim + sec + high + mage20
        + mage30 + mage40 + abbm1 + abbm3 +
    abbm4 + f(fage,model="rw2") +  f(district, model="besag",
        graph.file="ngn.graph")
f21<-u5mm~no + ant + vitA + breast1 + csex1 + twin1 + cbw1 +
    cbw2 + cbw3 + christain +
    muslim + cath + urban + space + prim + sec + high + mage20
        + mage30 + mage40 + abbm1 + abbm3 +
    abbm4 + f(fage,model="rw2") + f(district, model="besag",
        graph.file="ngn.graph")


f31<-y5~no + ant + vitA + breast1 + csex1 + twin1 + cbw1 +
    cbw2 + cbw3 + christain +
    muslim + cath + urban + space + prim + sec + high + mage20
        + mage30 + mage40 + abbm1 + abbm3 +
    abbm4 + f(fage,model="rw2") + f(district, model="besag",
        graph.file="ngn.graph")


r11<-inla(f11,family="binomial",data=data,control.compute=
    list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
    compute=TRUE))
r21<-inla(f21,family="binomial",data=data,control.compute=
    list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
    compute=TRUE))
r31<-inla(f31,family="binomial",data=data,control.compute=
    list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
    compute=TRUE))
#control.fixed = list(expand.factor.strategy="inla"),
plot(r11)
summary(r11)
summary(r21)
summary(r31)

# converting to odds
summ  <-exp(result0$summary.fixed) # Code for coverting to
    odd
bb  <-exp(r11$summary.fixed)
bb1 <-exp(r21$summary.fixed)
bb2 <-exp(r31$summary.fixed)
```

```
kk<-round(bb, digits=2)
kk1<-round(bb1, digits=2)
kk2<-round(bb2, digits=2)
write.csv(kk,"SSIFMortality.csv")
write.csv(kk1,"SSchildmortality.csv")
write.csv(kk2,"SSU5mortality.csv")

##  CREATING RESIDUAL ERRORs FOR SPATIAL PLOT
res0<-r11$summary.random$district # residual district effect
    structured model (INFANT MORTALITY)
res1<-r21$summary.random$district# residual district effect
    structured model (CHILD MORTALITY)
res2<-r31$summary.random$district# residual district effect
    structured model (U5 MORTALITY)
# y5
res01<-res0$"0.5quant"      # median
res02<-res0$"0.025quant"    # cat 5%
res03<-res0$"0.975quant"    # cat 95%

#### CHILD MORTALITY
res11<-res1$"0.5quant"      # median
res12<-res1$"0.025quant"    # cat 5%
res13<-res1$"0.975quant"    # cat 95%

# UNDER FIVE
res21<-res2$"0.5quant"      # median
res22<-res2$"0.025quant"    # cat 5%
res23<-res2$"0.975quant"    # cat 95%
#

ngn.graph$STR<-res01  ### MEDIAN INFANT
ngn.graph$ABC<-res11
ngn.graph$AAA<-res21




pdf("nigeria_infant2.pdf")
par(mfrow=c(2,2), mai=c(0.6,0.5,0.1,0.1),
    mgp=c(2,0.7,0))
```

```
plot(r11)
spplot(ngn.graph,"STR") # No colour specify
spplot(ngn.graph,"STR",cuts=4, names.attr="Posterior mean of
    infant") # 4 colour specify
spplot(ngn.graph,"ABC",cuts=4) # 4 colour classfication
spplot(ngn.graph,"AAA",cuts=4) # 4 colour specify

dev.off()

sum(data$deadchildren)
##### SPATIAL STRUCTURED WITHout BIRTH WEIGHT
###### Spatai Random effect
# STRUCTURED  SPATIAL MODEL
f101 <-imm~no + ant + vitA + breast1 + csex1 + twin1 +
    christain +
  muslim + cath + urban + space + prim + sec + high + mage20
     + mage30 + mage40 + abbm1 + abbm3 +
  abbm4 + f(district, model="besag",graph.file="ngn.graph")
f201<-u5mm~no + ant + vitA + breast1 + csex1 + twin1 +
    christain +
  muslim + cath + urban + space + prim + sec + high + mage20
     + mage30 + mage40 + abbm1 + abbm3 +
  abbm4 + f(district, model="besag",graph.file="ngn.graph")

f301<-y5~no + ant + vitA + breast1 + csex1 + twin1 +
    christain + muslim + cath + urban + space +
  prim + sec + high + mage20 + mage30 + mage40 + abbm1 +
     abbm3 +
  abbm4 + f(district, model="besag",graph.file="ngn.graph")

r101<-inla(f101,family="binomial",data=data,control.compute=
    list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
    compute=TRUE))
r201<-inla(f201,family="binomial",data=data,control.compute=
    list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
    compute=TRUE))
r301<-inla(f301,family="binomial",data=data,control.compute=
    list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
    compute=TRUE))
```

```
#control.fixed = list(expand.factor.strategy="inla"),


summary(r101)
summary(r201)
summary(r301)


# converting to odds
bb0 <-exp(r101$summary.fixed) # Code for coverting to odd
bb10 <-exp(r201$summary.fixed)
bb20 <-exp(r301$summary.fixed)
kk<-round(bb0, digits=2)
kk1<-round(bb10, digits=2)
kk2<-round(bb20, digits=2)
write.csv(kk,"SSWIFMortality.csv")
write.csv(kk1,"SSWchildmortality.csv")
write.csv(kk2,"SSWU5mortality.csv")


## CREATING RESIDUAL ERRORs FOR SPATIAL PLOT
res0<-r11$summary.random$district # residual district effect
    structured model (INFANT MORTALITY)
res1<-r21$summary.random$district# residual district effect
    structured model (CHILD MORTALITY)
res2<-r31$summary.random$district# residual district effect
    structured model (U5 MORTALITY)
# y5
res01<-res0$"0.5quant"        # median
res02<-res0$"0.025quant"    # cat 5%
res03<-res0$"0.975quant"    # cat 95%




######### ENDS HERE
    ****************************************************


formula<-y5~ cbw1 + cbw2 +cbw3 + mage20   + mage30   + mage40 +
      urban + csex1 +   poor + middle +
    richer + richest + no + ant + vitA +  space +  breast1  +
```

```
        measles   +
f(mage,model="rw2") + f(mbmi,model="rw2") +  f(district,
    model="besag",graph.file="ngn.graph")


result0<-inla(formula,family="binomial",data=data,control.
    compute=list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.
    predictor=list(compute=TRUE))
ww=summary(result0)
summ  <-exp(result0$summary.fixed) # Code for coverting to
    odd
kk<-round(summ, digits=3)
bb <-exp(result0$summary.fixed)
write.csv(bb,"U5MM_model.csv")



res0<-result0$summary.random$district # residual district
    effect structured model anemia
res1<-result1$summary.random$district# residual district
    effect structured model stunted
res2<-result2$summary.random$district# residual district
    effect structured model wasted
res3<-result3$summary.random$district# residual district
    effect structured model underweight
# y5
res01<-res0$"0.5quant"       # median
res02<-res0$"0.025quant"     # cat 5%
res03<-res0$"0.975quant"     # cat 95%


# Stunted
res11<-res1$"0.5quant"       # median
res12<-res1$"0.025quant"     # cat 5%
res13<-res1$"0.975quant"     # cat 95%
#
res21<-res2$"0.5quant"       # median
res22<-res2$"0.025quant"     # cat 5%
res23<-res2$"0.975quant"     # cat 95%


res33<-res3$"0.5quant"       # median
cor1 <-data.frame(res03,res13,res23)
```

```
cor <-cor(cor1)
cov<-cov(cor1)
write.csv(cor,"Corrtza95.csv")
write.csv(cov,"covtza95.csv")
#kk1 <-cbind(res02,res01,res03)


ngn.graph$STR<-res01
ngn.graph$ABC<-res11
ngn.graph$AAA<-res21
ngn.graph$ABB<-res33
#mm <-ngn.graph$STR


pdf("NIGERIA_U5MR.pdf")
par(mfrow=c(2,2), mai=c(0.6,0.5,0.1,0.1),
mgp=c(2,0.7,0))
plot(result0)
spplot(ngn.graph,"STR") # No colour specify
spplot(ngn.graph,"STR",cuts=4, names.attr="Posterior mean of
    Anemia") # 4 colour specify
spplot(ngn.graph,"ABC",cuts=4) # 4 colour classfication
spplot(ngn.graph,"AAA",cuts=4) # 4 colour specify
spplot(ngn.graph,"ABB",cuts=4) # 4 colour specify
dev.off()



#stargazer(bb, type = "text", title="Descriptive statistics",
    digits=2, out="table1.txt")


# Secere Stunted
formula1<-stunted~ bednet + cage5 +    cage11 + cage24 +
    visitno +  visit13  + urban + csex1 + literate + poor +
    middle +  richer +
richest + Zinc + iron + vitA + hospital +       private +
    space +  breast1 + diarrhea + cough + fever + pneu +
    measles +  electricity +
f(mage,model="rw2") + f(mbmi,model="rw2") + f(district, model
    ="besag",graph.file="ngn.graph")


result1<-inla(formula1,family="binomial",data=data,control.
```

```
    compute=list (dic=TRUE,mlik=TRUE,cpo=TRUE) ,control .
    predictor=list (compute=TRUE))
summary( result1 )
sum2  <−exp ( result1$summary . fixed )
kk1<−round (sum2,  digits =3)
write . csv (kk1,"tanstuRR . csv ")
# Secere Wasted
formula2<−wasted~ bednet +  cage5 +       cage11 + cage24 +
    visitno +  visit13  + urban + csex1 + literate + poor +
    middle +  richer +
richest + Zinc + iron + vitA + hospital +       private +
    space +  breast1 + diarrhea + cough + fever + pneu +
    measles +  electricity +
f (mage,model="rw2") +  f (mbmi,model="rw2") + f (district , model
    ="besag" ,graph . file ="ngn.graph")


result2 <−inla (formula2 , family ="binomial" ,data=data , control .
    compute=list (dic=TRUE,mlik=TRUE,cpo=TRUE) ,control .
    predictor=list (compute=TRUE))
summary( result2 )
exp (result2$summary . fixed )
sum3  <−exp ( result2$summary . fixed )
kk3<−round (sum3,  digits =3)
write . csv (kk3,"tawastRR2 . csv ")


formula3<−stunted ~   cage5 +     cage11 + cage24 + visitno +
    visit13  + urban + csex1 + literate + poor + middle +
    richest +
richer + Zinc + iron + vitA +  acessmed + river + space +
    diarrhea + cough + fever + pneu + measles + vaccine  +
    electricity +
f (mage,model="rw2") +  f (mbmi,model="rw2") + f (district , model
    ="besag" ,graph . file ="ngn.graph")
result3 <−inla (formula3 , family ="binomial" ,data=data , control .
    compute=list (dic=TRUE,mlik=TRUE,cpo=TRUE) ,control .
    predictor=list (compute=TRUE))
summary( result3 )
summ2<−exp (result3$summary . fixed )
#control . fixed = list (expand. factor . strategy ="inla ") ###
```

included to handle NULL variable
SBB <− summary (result0)
SUMM <− summary (result0) [3] # LAYERS of RESULT EXTRACTION
write . csv (summ2 , " tanUnder . csv ")
r20<−r2$summary . random$district# residual district effect
UNstructured model anemia
r21<−r20$ " 0.5 quant "   # median

Appendix C

Supplementary Material from Chapter 6

C.1 INLA Code for Univariate models in Chapter 6

```
# Packages required
rm(list=ls())
#install.packages("faraway") TO MANAGE REGRESSION ANALYSIS
library(faraway)
library("MASS")
library("lattice")

library("ctv")

library("sp")

library(maptools)

library(rgdal)
require(RColorBrewer)

library("RColorBrewer") # added coloured brewers

library(spdep)
require(INLA)
library(stargazer)
```

```
library(xtable)

#*******************LOADING DATA
    *****************************
data<-read.csv("C:/Users/Adeyemi/Desktop/regional/tan2010.csv
    ",header=T,sep=",")

data1<-read.csv("C:/Users/Adeyemi/Desktop/regional/moz2011.
    csv",header=T,sep=",")

data2<-read.csv("C:/Users/Adeyemi/Desktop/regional/bkf2010.
    csv",header=T,sep=",")

data3<-read.csv("C:/Users/Adeyemi/Desktop/regional/civ2011.
    csv",header=T,sep=",")

data4<-read.csv("C:/Users/Adeyemi/Desktop/regional/gha2008.
    csv",header=T,sep=",")

head(data)
attach(data)

#data$distr.unstruct  = data$region

head(data1)
attach(data1)

head(data2)
attach(data2)

head(data3)
attach(data3)

head(data4)
attach(data4)
summary(data4$anegg);summary(data4$stunting);summary(
    data4$wasting)

#*****************LOADING  GEOGRAPGHICAL  MAPS***************
```

```
bkf.graph<- readRDS("C:/Users/Adeyemi/Desktop/regional/
   BFA_adm1.rds") # burkina faso


gha.graph<- readRDS("C:/Users/Adeyemi/Desktop/regional/
   GHA_adm1.rds") # GHANA
moz.graph<- readRDS("C:/Users/Adeyemi/Desktop/regional/
   MOZ_adm1.rds") # Mozambique


#*********PLOT GEOGRAPHICAL DISTRIBUTION BY COUNTRIES
   ***********
windows(width=7, height=7)
#png("bukaa11.png")
#png("buk%5d.png") # By including % you can generate multiple
    plots
pdf("GHANAANEMIA.ODDS.pdf")
par(mfrow=c(2,3), mai=c(0.6,0.5,0.1,0.1),
   mgp=c(2,0.7,0))


plot(bkf.graph, main="Burkina faso")
plot(civ.graph, main="Cote d'Ivoire")
plot(gha.graph, main="Ghana")
plot(moz.graph, main="Mozambique") #Mozambique
plot(tz1.graph, main="Tanzania")
dev.off()


# Creates adjacency for Countries
adjgha<-poly2nb(gha.graph)
adjbkf<-poly2nb(bkf.graph)#Creates adjacency for Burkina faso
adjmoz<-poly2nb(moz.graph)#Creates adjacency for ken


adjgha
adjmoz
adjbkf


# Converting for INLA graph file #spdep


nb2INLA("bkf.graph",adjbkf) # #spdep BURKINA FASO
nb2INLA("gha.graph",adjgha) # #spdep GHANA
nb2INLA("moz.graph",adjmoz) #  #spdep mozambique
```

##∗∗∗∗∗∗∗∗∗BURKINA FASO ANALYSIS STARS HERE∗∗∗∗∗∗

```
#inla.update(testing=TRUE)
## Model1
f1<-anebin~ bednet + cage5 +  cage11 + cage24 + visitno +
   visit13  + urban +
  csex1 + literate + poor + middle +  richer +   richest +
     Zinc + iron + vitA +
  space + breast1 + diarrhea + cough + fever +  measles +
     mage

f2<-stunted~ bednet + cage5 +  cage11 + cage24 + visitno +
   visit13  + urban +
  csex1 + literate + poor + middle +  richer +   richest +
     Zinc + iron + vitA +
  space + breast1 + diarrhea + cough + fever +  measles +   f
     (mage,model="rw2") +
  f(region,  model="iid")

f3<-wasted~ bednet + cage5 +  cage11 + cage24 + visitno +
   visit13   + urban +
  csex1 + literate + poor + middle +  richer +   richest +
     Zinc + iron + vitA +
  space + breast1 + diarrhea + cough + fever +  measles +   f
     (mage,model="rw2") +
  f(region,  model="besag",graph.file="bkf.graph")

r1<-inla(f1,family="binomial",data=data2,control.compute=list
   (dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
   compute=TRUE))
r2<-inla(f2,family="binomial",data=data2,control.compute=list
   (dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
   compute=TRUE))
r3<-inla(f3,family="binomial",data=data2,control.compute=list
   (dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
   compute=TRUE))

## MODEL SUMMARY
```

```
summary(r1)
summary(r2)
summary(r3)


formula6<-anebin~ bednet + cage5 +        cage11 + cage24 +
   visitno +  visit13  + urban + csex1 + literate + poor +
   middle +  richer +
  richest + Zinc + iron + vitA +            space + breast1 +
    diarrhea + cough + fever +  measles +
  f(mage,model="rw2") + f(mbmi,model="rw2") + f(region, model
    ="besag",graph.file="bkf.graph")


result6<-inla(formula6,family="binomial",data=data2, control.
   fixed = list(expand.factor.strategy="inla"), control.
   compute=list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.
   predictor=list(compute=TRUE))
summary(result6)


SBB11 <- exp(result6$summary.fixed)
#SUMAB <- summary(result120)[3] # LAYERS of RESULT EXTRACTION
write.csv(SBB11,"bkfaneMAR.csv")


# Secere Stunted
formula12<-stunted~ bednet + cage5 +     cage11 + cage24 +
   visitno +  visit13  + urban + csex1 + literate + poor +
   middle +  richer +
  richest + Zinc + iron + vitA +  space + breast1 + diarrhea
    + cough + fever +  measles +
  f(mage,model="rw2") + f(mbmi,model="rw2") + f(region, model
    ="besag",graph.file="bkf.graph")
result12<-inla(formula12,family="binomial",data=data2,
   control.fixed = list(expand.factor.strategy="inla"),
   control.compute=list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.
   predictor=list(compute=TRUE))
summary(result12)
exp(result12$summary.fixed)
SBB2 <- exp(r3$summary.fixed)
write.csv(SBB2,"MOZAM_AASTUNT_MAR.csv")
```

```
# Secere Wasted
formula13<-wasted~ bednet + cage5 +        cage11 + cage24 +
    visitno +  visit13  + urban + csex1 + literate + poor +
    middle +   richer +
   richest + Zinc + iron + vitA +             space + breast1 +
      diarrhea + cough + fever +   measles +
   f(mage,model="rw2") + f(mbmi,model="rw2") + f(region, model
      ="besag",graph.file="bkf.graph")
result7<-inla(formula13,family="binomial",data=data2, control
    .fixed = list(expand.factor.strategy="inla"), control.
    compute=list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.
    predictor=list(compute=TRUE))
summary(result7)
SBB3 <- exp(result7$summary.fixed)
#SUMAB <- summary(result120)[3] # LAYERS of RESULT EXTRACTION
write.csv(SBB3,"bkfwastMAR.csv")

formula14<-undered ~  cage5 +   cage11 + cage24 + visitno +
    visit13  + urban + csex1 + literate + poor + middle +
    richest +
   richer + iron + vitA +  acessmed + river + space + diarrhea
      + cough + fever + pneu + measles + vaccine  +
      electricity +
   f(mage,model="rw2") + f(mbmi,model="rw2") + f(region, model
      ="besag",graph.file="bkf.graph")
result8<-inla(formula14,family="binomial",data=data2, control
    .fixed = list(expand.factor.strategy="inla"), control.
    compute=list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.
    predictor=list(compute=TRUE))
summary(result8)

#  Extracting residual district effect structured model
res11<-result6$summary.random$region #    anemia
res12<-result12$summary.random$region #     stunted
res13<-result7$summary.random$region #     wasted

#Anemia
res10<-res11$"0.5quant"        # median
res101<-res11$"0.025quant"     # cat 5%
```

```
res102<-res11$"0.975 quant"    # cat 95%


# Stunted
res20<-res12$"0.5 quant"       # median
res012<-res12$"0.025 quant"    # cat 5%
res013<-res12$"0.975 quant"    # cat 95%
# wasted
res30<-res13$"0.5 quant"       # median
res302<-res13$"0.025 quant"    # cat 5%
res303<-res13$"0.975 quant"    # cat 95%


# Extractting the Residuals of posterior estimates


bkf.graph$STR10<-res10
bkf.graph$ABC11<-res20
bkf.graph$AAA12<-res30
bkf.graph$ABB13<-res40


# PLOTING SPATIAL MAPS
pdf("BFAUNDER2010.pdf")
par(mfrow=c(2,2), mai=c(0.6,0.5,0.1,0.1), mgp=c(2,0.7,0))
plot(result8)
spplot(bkf.graph,"STR10") # No colour specify
spplot(bkf.graph,"STR10",cuts=4) # 4 colour specify
spplot(bkf.graph,"ABC11",cuts=4) # 4 colour specify
spplot(bkf.graph,"AAA12",cuts=4) # 4 colour specify
spplot(bkf.graph,"ABB13",cuts=4) # 4 colour specify
dev.off()


##************GHANA NALYSIS STARTS HERE *********
# Anemia
formula<-anebin~ bednet + cage5 +        cage11 + cage24 +
   visitno + visit13  + urban + csex1 +
  literate + poor + middle + richer +   richest + Zinc +
     iron + vitA +
  space + breast1 + diarrhea + cough + fever + measles +
  f(mage,model="rw2") + f(mbmi,model="rw2") + f(region, model
     ="besag",graph.file="gha.graph")
```

```
result0<-inla(formula,family="binomial",data=data4,control.
    compute=list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.
    predictor=list(compute=TRUE))


# Stunting
formula1<-stunted~ bednet + cage5 +      cage11 + cage24 +
    visitno +   visit13  + urban + csex1 + literate + poor +
    middle +   richer +
  richest + Zinc + iron + vitA +    space +   breast1 +
      diarrhea + cough + fever + measles +
  f(mage,model="rw2") + f(mbmi,model="rw2") + f(region, model
      ="besag",graph.file="gha.graph")
result1<-inla(formula1,family="binomial",data=data4,control.
    compute=list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.
    predictor=list(compute=TRUE))


# Wasting
formula2<-wasted~ bednet +  cage5 +      cage11 + cage24 +
    visitno +   visit13  + urban + csex1 + literate + poor +
    middle +   richer +
  richest + Zinc + iron + vitA + space +   breast1 + diarrhea
      + cough + fever   + measles +
  f(mage,model="rw2") + f(mbmi,model="rw2") + f(region, model
      ="besag",graph.file="gha.graph")
result2<-inla(formula2,family="binomial",data=data4,control.
    compute=list(dic=TRUE,mlik=TRUE,cpo=TRUE),control.
    predictor=list(compute=TRUE))


## Extracting summary statistics for posterior of
    categoraical var.
### orresponding Odds ratios
summary(result0)
exp(r3$summary.fixed)
summary(resul1)
summ2<-exp(result1$summary.fixed)
summary(result2)
exp(result1$summary.fixed)


# Residual district effect structured model
```

```
res0<-r0$summary.random$region # anemia
res1<-result1$summary.random$region#   stunted
res2<-result2$summary.random$region#   wasted


## Residual  of Posterior estimates
# Amemia
res01<-res0$"0.5quant"        # median
res02<-res0$"0.025quant"      # cat 5%
res03<-res0$"0.975quant"      # cat 95%
# Stunted
res11<-res1$"0.5quant"        # median
res12<-res1$"0.025quant"      # cat 5%
res13<-res1$"0.975quant"      # cat 95%
#
res21<-res2$"0.5quant"        # median
res22<-res2$"0.025quant"      # cat 5%
res23<-res2$"0.975quant"      # cat 95%


# Creating vector for an the residual  lotts
gha.graph$STR<-res0
gha.graph$ABC<-res0
gha.graph$AAA<-res21
gha.graph$ABB<-res33


# Plotting maps of posterior estimate mean, 95\% and 5\%

pdf("GHANAWAST2.pdf")
par(mfrow=c(2,2), mai=c(0.6,0.5,0.1,0.1),
    mgp=c(2,0.7,0))
plot(result2)
spplot(gha.graph,"STR") # No colour specify
spplot(gha.graph,"STR",cuts=4) # 4 colour specify
spplot(gha.graph,"ABC",cuts=4) # 4 colour specify
spplot(gha.graph,"AAA",cuts=4) # 4 colour specify
spplot(gha.graph,"ABB",cuts=4) # 4 colour specify
dev.off()
spplot(gha.graph,"STR",col.regions=bgy.colors(20)) # 4 colour
    specify
```

```
##**********MOZAMBIQUES ANALYSIS STARTS HERE**

f1<-anemia~ bednet + cage5 +  cage11 + cage24 + visitno +
    visit13  + urban +
  csex1 + literate + poor + middle +  richer +   richest +
     Zinc + iron + vitA +
  space + breast1 + diarrhea + cough + fever +  measles +
     mage

f2<-stunting~ bednet + cage5 +  cage11 + cage24 + visitno +
    visit13  + urban +
  csex1 + literate + poor + middle +  richer +   richest +
     Zinc + iron + vitA +
  space + breast1 + diarrhea + cough + fever +  measles +    f
     (mage,model="rw2")  +
  f(region,  model="iid")

f3<-wasted~ bednet + cage5 +  cage11 + cage24 + visitno +
    visit13  + urban +
  csex1 + literate + poor + middle +  richer +   richest +
     Zinc + iron + vitA +
  space + breast1 + diarrhea + cough + fever +  measles +    f
     (mage,model="rw2")  +
  f(region,  model="besag",graph.file="moz.graph")


r1<-inla(f1,family="binomial",data=data1,control.compute=list
   (dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
   compute=TRUE))
r2<-inla(f2,family="binomial",data=data1,control.compute=list
   (dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
   compute=TRUE))
r3<-inla(f3,family="binomial",data=data1,control.compute=list
   (dic=TRUE,mlik=TRUE,cpo=TRUE),control.predictor=list(
   compute=TRUE))

## MODEL SUMMARY
summary(r1)
```

```
summary ( r2 )
summary ( r3 )

## Posterior estimates Summary of the categorical variable
   and odds ratios
summary ( result5 )
exp ( result5$summary . fixed )

# Secere Stunted
formula6<-stunted~ bednet + cage5 +        cage11 + cage24 +
   visitno +  visit13  + urban + csex1 + literate + poor +
   middle +  richer +
  richest + Zinc + iron + vitA +  space + breast1 + diarrhea
     + cough + fever + measles +  electricity +
  f (mage,model="rw2") + f (mbmi,model="rw2") + f (region , model
     ="besag" ,graph . file ="moz. graph")

result6<-inla (formula6 , family ="binomial" ,data=data1 ,control .
   compute=list ( dic=TRUE, mlik=TRUE, cpo=TRUE) ,control .
   predictor=list (compute=TRUE))
summary ( result6 )
SBB10 <-exp ( result6$summary . fixed )
write . csv (SBB10 ,"mozstuntMAR . csv ")

# Wasting
formula7<-wasted~bednet + cage5 +        cage11 + cage24 +
   visitno +  visit13  + urban + csex1 + literate + poor +
   middle +  richer +
  richest + Zinc + iron + vitA +  space + breast1 + diarrhea
     + cough + fever + measles +  electricity +
  f (mage,model="rw2") + f (mbmi,model="rw2") + f (region , model
     ="besag" ,graph . file ="moz. graph")
result7<-inla (formula7 , family ="binomial" ,control . fixed =  list
   (expand . factor . strategy ="inla") ,  data=data1 ,control .
   compute=list ( dic=TRUE, mlik=TRUE, cpo=TRUE) ,control .
   predictor=list (compute=TRUE))
summary ( result7 )
SBB15 <-exp ( result7$summary . fixed )
write . csv (SBB15 ,"mozswastMAR . csv ")
```

```
# Extract  residual district effect structured model
res5<-result5$summary.random$region # residual district
    effect structured model anemia
res6<-result6$summary.random$region # residual district
    effect structured model stunted
res7<-result7$summary.random$region # residual district
    effect structured model wasted
res8<-result8$summary.random$region# residual district effect
    structured model underweight
# Amemia
res50<-res5$"0.5quant"       # median
res02<-res5$"0.025quant"     # cat 5%
res03<-res5$"0.975quant"     # cat 95%
# Stunted
res60<-res6$"0.5quant"       # median
res61<-res6$"0.025quant"     # cat 5%
res62<-res6$"0.975quant"     # cat 95%
# Wasting
res70<-res7$"0.5quant"       # median
res71<-res7$"0.025quant"     # cat 5%
res72<-res7$"0.975quant"     # cat 95%

moz.graph$STR<-res50
moz.graph$ABC<-res60
moz.graph$AAA<-res70
moz.graph$ABB<-res80
#mm <-tz1.graph$STR

# PLOTS mapping the posterior estimates
pdf("MOZAMEN1.pdf")
par(mfrow=c(2,2), mai=c(0.6,0.5,0.1,0.1),
    mgp=c(2,0.7,0))
plot(result5)
spplot(moz.graph,"STR") # No colour specify
spplot(moz.graph,"STR",cuts=4, names.attr="Posterior mean of
    Anemia") # 4 colour specify
spplot(moz.graph,"ABC",cuts=4) # 4 colour specify
spplot(moz.graph,"AAA",cuts=4) # 4 colour specify
```

```
spplot(moz.graph,"ABB",cuts=4) # 4 colour specify
dev.off()


##*****************ANALYSIS ENDS HERE **********


#******** INLA COD ENDS HERE *************************
```

C.2   WinBUGS Separate CAR Models

```
###############################
########## Separate Analyses ####
###############################
model
{
  #likelihood
  for(i in 1: N)
  {#N=2992

    #for STUNTING ##beta1[9]*vtmn[i] +
    stunt[i]~dbern(p1[i])
    p1[i]<-min(1,max(0,PSTT[i]))
    logit(PSTT[i])<-beta1[1]+ beta1[2]*bbno[i] +
      beta1[3]*bbp[i]+ beta1[4]*bbf[i] + beta1[5]*cage5[i] +
          beta1[6]*cage11[i] +
      beta1[7]*cage12[i] + beta1[8]*cage24[i] +  beta1[9]*vst
          [i] +beta1[10]*literate[i]
    + beta1[11]*poorest[i] + beta1[12]*poor[i] + beta1[13]*
        middle[i] +beta1[14]*rich[i]

    #for WASTING ## +beta2[9]*vtmn[i]
    wast[i]~dbern(p2[i])
    p2[i]<-min(1,max(0,PWTT[i]))
    logit(PWTT[i])<-beta2[1]+ beta2[2]*bbno[i] +
      beta2[3]*bbp[i]+ beta2[4]*bbf[i] + beta2[5]*cage5[i] +
          beta2[6]*cage11[i] +
      beta2[7]*cage12[i] + beta2[8]*cage24[i] + beta2[9]*vst[
          i] +beta2[10]*literate[i]
    + beta2[11]*poorest[i] + beta2[12]*poor[i] + beta2[13]*
```

```
    middle[i] +beta2[14]*rich[i]


  #for anemia ##+beta3[9]*vtmn[i]
  anebin[i]˜dbern(p3[i])
  p3[i]<−min(1,max(0,PAMN[i]))
  logit(PAMN[i])<−beta3[1]+ beta3[2]*bbno[i] +
    beta3[3]*bbp[i]+ beta3[4]*bbf[i] + beta3[5]*cage5[i] +
      beta3[6]*cage11[i] +
    beta3[7]*cage12[i] + beta3[8]*cage24[i] + beta2[9]*vst[
      i] +beta3[10]*literate[i]
  + beta3[11]*poorest[i] + beta3[12]*poor[i] + beta3[13]*
    middle[i] +beta3[14]*rich[i]



}


#Getting Odds ratio from logOdds, by taking exponent of the
    coefficients
for(i in 2:14){ Oddsbeta1[i]<−exp(beta1[i]); Oddsbeta2[i]<−
  exp(beta2[i]);
Oddsbeta3[i]<−exp(beta3[i]) }
#prior
for(j in 1: 14)
{ beta1[j]˜dnorm(0,0.0001); beta2[j]˜dnorm(0,0.0001);
  beta3[j]˜dnorm(0,0.0001) }
for(i in 1: N)
{
  for(j in 1: 10)
  { PH[j,i]<−(PSTT[i])*(equals(region[i],j))
  PHPS[j,i]<−(PWTT[i])*(equals(region[i],j))
  PAM[j,i]<−(PAMN[i])*(equals(region[i],j))
  }
}
for(j in 1: 10)
{
  for(i in 1: N)
  {count[j,i]<−equals(region[i],j) }
  number[j]<−sum(count[j,])
```

```
    PCHV[j]<-sum(PH[j,])/number[j]
    PCHPS[j]<-sum(PHPS[j,])/number[j]
    PAMS[j]<-sum(PAM[j,])/number[j]
  }
}


#DATA
#INITIALS
list(beta1=c(0,0,0,0,0, 0,0,0,0,0, 0,0,0,0),
     beta2=c(0,0,0,0,0, 0,0,0,0,0, 0,0,0,0),
     beta3=c(0,0,0,0,0, 0,0,0,0,0, 0,0,0,0))
```

C.3   WinBUGS Codes for Multivariate CAR Models

```
###########################################
#########Multivariate CAR model##########
###########################################
model
{

#likelihood
for(i in 1:N)
{ #N=2992

  #for STUNTING ## + beta1[9]*VSN[i] +  beta1[10]*VST[i] +
     beta1[11]*LITER[i]
  ###beta1[2]*bbno[i] + beta1[3]*bbp[i]+ beta1[4]*bbf[i] + U[
     region[i],1]
  stunt[i]~dbern(p1[i])
  p1[i]<-min(1,max(0,PSTT[i]))
  logit(PSTT[i])<-beta1[1] +  beta1[2]*cage5[i] + beta1[3]*
     cage11[i] + beta1[4]*cage12[i] + beta1[5]*cage24[i]    +
     beta1[6]*poorest[i] + beta1[7]*poor[i] + beta1[8]*middle
     [i] +beta1[9]*rich[i] + beta1[10]*space[i] +S[1,region[i
     ]]

  #for WASTING ## +beta2[9]*VSN[i] + beta2[10]*VST[i]   +beta2
```

```
        [11]*LITER[i]
   ## beta2[2]*bbno[i] + beta2[3]*bbp[i]+ beta2[4]*bbf[i] + U[
      region[i],2]


   wast[i]~dbern(p2[i])
   p2[i]<-min(1,max(0,PWTT[i]))
   logit(PWTT[i])<-beta2[1] +   beta2[2]*cage5[i] + beta2[3]*
      cage11[i] + beta2[4]*cage12[i] + beta2[5]*cage24[i]   +
      beta2[6]*poorest[i] + beta2[7]*poor[i] + beta2[8]*middle
      [i]   + beta2[9]*rich[i] + beta2[10]*space[i] + S[2,
      region[i]]


   #for anemia ## +beta3[9]*VSN[i] + beta2[10]*VST[i] + beta3
      [11]*LITER[i]
   ### beta3[2]*bbno[i] + beta3[3]*bbp[i] + beta3[4]*bbf[i] +
       U[region[i],3]


   anebin[i]~dbern(p3[i])
   p3[i]<-min(1,max(0,PAMN[i]))
   logit(PAMN[i])<-beta3[1] + beta3[2]*cage5[i] + beta3[3]*
      cage11[i] + beta3[4]*cage12[i] + beta3[5]*cage24[i]   +
      beta3[6]*poorest[i] + beta3[7]*poor[i] + beta3[8]*middle
      [i] + beta3[9]*rich[i] + beta3[10]*space[i] + S[3,region
      [i]]


}


#Getting Odds ratio from logOdds, by taking exponent of the
    coefficients
for(i in 1:10)
{ Oddsbeta1[i]<-exp(beta1[i])
Oddsbeta2[i]<-exp(beta2[i])
Oddsbeta3[i]<-exp(beta3[i])
}
#prior
for(j in 1: 10)
{ beta1[j]~dnorm(0,0.0001); beta2[j]~dnorm(0,0.0001)  ; beta3
    [j]~dnorm(0,0.0001)
```

```
}

# MVCAR prior
S[1:Ndiseases ,1:Nareas]  ~ mv.car(adj[],weights[],num[],omega[
    , ])
for (i in 1:sumNumNeigh) { weights[i] <- 1 }
R[1,1] <- 3; R[1,2] <- 0; R[1,3]<- 0; R[2,1] <- 0; R[2,2] <-
    3
R[3,1] <- 0;  R[3,2] <- 0;  R[3,3] <- 3;  R[2,3] <- 0
# Precision matrix of MVCAR
omega[1 : Ndiseases, 1:Ndiseases] ~ dwish(R[ , ],Ndiseases)

# ****************Covariance matrix of MVCAR**************
sigma2[1 : Ndiseases, 1 : Ndiseases] <- inverse(omega[ , ])

# conditional SD of S[1, ] (Stunting)
sigma[1] <- sqrt(sigma2[1, 1])

# conditional SD of S[2,] (Wasting)
sigma[2] <- sqrt(sigma2[2, 2])

# conditional SD of S[3, ] (Anemia)
sigma[3] <- sqrt(sigma2[3, 3])

### MCAR Ends here *************************************
# within-area conditional correlation
corr12 <- sigma2[1, 2] / (sigma[1] * sigma[2])
corr13 <- sigma2[1, 3] / (sigma[1] * sigma[3])
corr23 <- sigma2[2, 3] / (sigma[2] * sigma[3])

# between Stunting  Wasting and anemia
mean1 <- mean(S[1,]); mean2 <- mean(S[2,]); mean3 <- mean(S
    [3,])
for(j in 1: 13) { S1[j]<-S[1,j]; S2[j]<-S[2,j]; S3[j]<-S[3,j
    ]}

# within-area correlation between unstructured component of
    variation in STUNTING and
# WASTING
```

```
#      corr.Usw <- sigma2.U[1, 2] / (sigma.U[1] * sigma.U[2])


# within-area correlation between unstructured component of
    variation in STUNTING and
# Anemia
# corr.Usa <- sigma2.U[1,3] / (sigma.U[1] * sigma.U[3])
# within-area correlation between unstructured component of
    variation in    # WASTING  and #Anemia
# corr.Uwa <- sigma2.U[2, 3] / (sigma.U[3] * sigma.U[2])


# within-area conditional correlation between total random
    effect
# (i.e. spatial + unstructured components) for STUNTING and
    for WASTING-2
# corr.sum12 <- (sigma2[1, 2] + sigma2.U[1, 2] ) /
#  (sqrt(sigma2[1, 1] + sigma2.U[1, 1]) * sqrt(sigma2[2, 2] +
    sigma2.U[2, 2]))


# within-area conditional correlation between total random
    effect
# (i.e. spatial + unstructured components) for STUNTING and
    for WASTING-2
# corr.sum13 <- (sigma2[1, 3] + sigma2.U[1, 3]) /
#(sqrt(sigma2[1, 1] + sigma2.U[1, 1]) * sqrt(sigma2[3, 3] +
    sigma2.U[3, 3]))


# ##*** Prior

for(i in 1: N)
{
  for(j in 1: 13)
  {
    PH[j,i]<-(PSTT[i])*(equals(region[i],j))
    PHPS[j,i]<-(PWTT[i])*(equals(region[i],j))
    PAM[j,i]<-(PAMN[i])*(equals(region[i],j))
  }
}
#*********
for(j in 1: Nareas)
```

```
{
  for(i in 1: N) { count[j,i]<-equals(region[i],j) }
  number[j]<-sum(count[j,])
  PCHV[j]<-sum(PH[j,])/number[j]
  PCHPS[j]<-sum(PHPS[j,])/number[j]
  PAMS[j]<-sum(PAM[j,])/number[j]
}
}

#DATA
#INITIALS
list(beta1=c(0,0,0,0,0,0,0,0,0,0,0,0,0),    beta2=c
   (0,0,0,0,0,0,0,0,0,0,0,0,0),
     beta3=c(0,0,0,0,0,0,0,0,0,0,0,0,0), S=c
        (0,0,0,0,0,0,0,0,0,0,0,0,0),
     omega=structure(.Data=c(1,1,1,1,1,1,1,1,1), .Dim=c(3,3))
        )

####****MULTIVARIATE WINBUGS CODES ENDS HERE*****
```

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Published Papers

D.1 Published Paper from Chapter Three

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Article

Semiparametric Multinomial Ordinal Model to Analyze Spatial Patterns of Child Birth Weight in Nigeria

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Abstract: *Background*: Birth weight is an important health parameter for obstetricians and gynaecologists. It is a good health indicator of a child-bearing mother and a strong predictor of infant morbidity and mortality. *Methods*: This paper utilizes data on 28,647 children born between 2003–2008 obtained from the 2008 Nigeria Demographic and Health Survey (NDHS). For a simple epidemiological convenience, the occurrence of a newborn weight can intuitively be considered to be categorical in nature and the thresholds can be put on a continuous scale. In survey reporting, the mothers frequently estimate their infant's birth weight and make a classification in ordinal category (low, normal, large) instead of actual birth weight. The study fits a multinomial regression model to analyze the relationships between the polytomous response and different kind of covariates in a unified manner. We estimate the fixed effects of bio-social covariates parametrically and the non-linear effect modeled using P-spline. The spatial component was modeled using conditional autoregressive error. A penalized maximum likelihood estimation was performed to estimate the model parameters. *Results*: We found risk factors that are positively associated with low birth weight, which include multiple birth, short birth interval, death of sibling, childhood diarrhea, fever, mother's smoking, firewood/dung cooking and poor household. Results further showed that iron syrup supplementation, antenatal attendance, mother literacy and household wealth had significant association with low probability of low birth weight. The finding also showed spatial patterns, which are not captured by the underlying determinants, and we produced probability predictive maps of the spatial residual effects. *Conclusions*: In addition to the statistical relevance of our method, the generated spatial maps identify highly endemic areas of low birth weight that can assist government agency to channel scarce health resources. A comprehensive approach which institutes a combination of interventions to improve the overall health care of the women is needed.

Keywords: Nigeria; child birth size; cumulative multinomial model; penalized spline; spatial maps

1. Introduction

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RESEARCH ARTICLE

A Bayesian Hierarchical Analysis of Geographical Patterns for Child Mortality in Nigeria

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Abstract:

Background:

In an epidemiological study, disease mapping models are commonly used to estimate the spatial (or temporal) patterns in disease risk and to identify high-risk clusters, allowing for health interventions and allocation of the resources. The present study proposes a hierarchical Bayesian modeling approach to simultaneously capture the over-dispersion due to the effect of varying population sizes across the districts (regions), and the spatial auto-correlation inherent in the childhood mortality at districts (state) level in Nigeria.

Methods:

This cross-sectional study was based on 31842 children data extracted from the 2013 Nigeria Demographic and Health Survey (DHS). Of these children, 2886 died before reaching the age of five years. A Standardized Mortality Ratio (SMR) was estimated for each district (state) and mapped to highlight the risk patterns of the child mortality. Generalized Poisson regression models were formulated with random effects to estimate the mortality risk and then explored to investigate the relationship of under-five child mortality and the regional risk factors. The random effects are formulated to reflect the potential tendency of "neighbouring" regions to have similar risk patterns and the spatial heterogeneity effect was used to capture geographical inequalities in the mortality outcomes. The models were implemented using a full Bayesian framework. All model parameters were estimated in WinBUGS *via* Markov chain Monte Carlos (MCMC) simulation techniques.

Results:

The results showed that of the economically deprived households, 2.088: 95% CI (1.088, 3.165) were significantly associated with childhood mortality, while unhygienic sanitation and lack of access to improved water sources were positively associated with child mortality, but not statistically significant at 5% probability level. The geographical variation of the under-five mortality prevalence was found to be attributed to 69% clustering and 31% was due to spatial heterogeneity factors. The predicted probability maps identified clusters of high risk mortality in the northern regions and low prevalence of concentrated mortality in the south-west regions of Nigeria.

Conclusion:

The results demonstrated the flexibility of the approach that explored the geographical variation in the potential risk factors of child mortality and that it provides a better understanding of the regional variations of mortality risks. Nonetheless, both representations can help to provide information for the initiation of public health interventions.

Keywords: Child mortality, Poisson mixed model, Health geography, Spatial epidemiology, Geographical patterns, DHS.

1. INTRODUCTION

Despite remarkable growth recorded by many economies in the last two decades, many developing countries have failed

to attain the target Millennium Development Goals (MDGs 1) four(4), the (reduction of under-five mortality by two-thirds between 1990 and 2015) and seven (7), the targets for water and sanitation in urban. Five countries accounted for half of the global infant mortality with Nigeria being the third largest contributor to the under- five mortality rate among children in sub-Saharan Africa [1, 2]. In 2013, the mortality rates for the

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