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Doctoral Thesis

Child Health and Mortality in Ethiopia: Insights from Leverage Statistical Models

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List of Acronyms

GLMMs Generalized linear mixed models
ART Acute respiratory infection
DHARMa Diagnostics for Hierarchical Regression models
PMA-ET Performance Monitoring for Action Ethiopia
AIC Akaike's information criteria
DHS Demographic and health surveys
EAs Enumeration areas
GLMs Generalized Linear models
EDHS Ethiopian Demographic and Health Survey
WHO World Health Organization
MCMC Markov chain Monte Carlo techniques
GAM Generalized additive model
MRFs Markov random field priors
DIC Deviance information criteria
IMR Infant mortality rate
MDGs Millennium Development Goals
GDP Gross domestic per capita
SEM Structural equation modelling
BCG Calmette-Guérin bacillus

Acknowledgments

"Live as if you were going to die tomorrow.
Learn as if you were to live forever."
Mahatma Gandhi (1869–1948).

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Chapter 1

General Introduction

1.1 Introduction to the topic of the PhD thesis

Ethiopia continues to have a serious public health problem with persistent malnutrition, which is like the larger problem that children under five experience throughout Africa (Amare et al., 2020). There is a high likelihood of co-occurring pediatric comorbidities with this sensitivity to malnutrition and alarmingly high rates of underweight, especially among those who live in locations with minimal resources (S. H. Mohammed et al., 2020; Raru et al., 2022), which exacerbates health issues (S. H. Mohammed et al., 2020). The country also has an alarmingly high infant mortality rate, which is a result of the high rates of underweight and co-morbidity that follow (Central Statistical Agency [CSA] ICF International, 2019).

Ethiopia's high rates of underweight children, comorbidities, and neonatal mortality further impede the country's development. These interconnected issues put considerable pressure on the healthcare system and obstruct economic progress (Krishna Luhila, 2022). According to research by Grantham-McGregor et al. (2007), children who suffer from malnutrition and co-morbidities are more likely to develop long-term health problems that will affect their productivity and academic performance in the future. The high infant mortality rate causes a horrible number of deaths as well as a lot of mental distress for families and communities (Bryce et al., 2006). These problems must be fixed to safeguard the wellbeing of Ethiopian children and pave the way for the nation to enjoy more prosperity in the future.

Despite Ethiopia's progress, the country faces obstacles to achieving optimal child health outcomes. The infant mortality rates in Ethiopia are still higher than average according to the World Bank's data for that year. These disparities are exacerbated by differences in healthcare access and facilities across regions, impacting child health outcomes differently based on location, as highlighted by the World Banks report from 2024 (USAID in 2023; World Infant Mortality Rate 1950-2024). Research conducted by Seale et al., (2022) underscores these inequities by illustrating how the uneven distribution of resources among regions leads to child illnesses. Children are vital to the future of any nation, so ensuring their wellbeing is crucial. Thus, it is important to understand the factors that affect child health to continue moving forward, and these concerning literatures highlight the pressing need for efficient interventions to deal with the various issues obstructing Ethiopian children's health.

Our thesis on child health and mortality in Ethiopia utilizes a multifaceted approach. We employ a generalized linear mixed model (GLMM) to explore child comorbidity determinants using 2019 PMA-ET data, and a Bayesian semiparametric geoadditive model (2016 EDHS data) to investigate geographic and sociodemographic factors influencing underweight in children under five. Finally, structural equation modeling based on World Bank data from 2000-2019 examines causal relationships between indicators and infant mortality rates. This multi-model approach offers a comprehensive understanding of child health determinants in Ethiopia.

1.2 The content of the PhD thesis and limitations of existing researches

This dissertation explores the complex issue of child health and mortality in Ethiopia by employing slice-edge statistical analysis. The exploration utilizes three distinct projects labeled in three chapters, each focused on a specific aspect of this critical child health concern.

Chapter 1: Neighbourhood-level heterogeneity of child comorbidity in a generalized linear mixed model: Based on the Performance Monitoring for Action Ethiopia (PMA-ET) community survey

While significant exploration exists on childhood morbidity in Ethiopia (Asresie et al., 2023; Susuman, 2012a; Takele et al., 2019a; Teklemariam et al., 2000a; Yohannes et al., 1992a), these studies primarily concentrate on factors associated with individual health conditions, and focusing on single conditions fails to capture the full picture of nonage health challenges in Ethiopia. By solely fastening on individual conditions, experimenters miss the opportunity to explore these interrelated factors that contribute to the complex reality of nonage comorbidity in Nigeria (M. Ezeonwu, 2014; Starfield, 1992). Existing studies also frequently fail to account for implicit variations in health outcomes between different groups of children, and children in different regions or communities may face distinct health pitfalls due to varying factors like environmental conditions, access to healthcare installations, and socioeconomic differences. This lack of consideration for implicit clustering goods within the data can lead to inaccurate or deficient understandings of childhood morbidity patterns.

Therefore, our exploration design investigates the factors impacting the socio-occurrence of multiple health conditions (comorbidity) in children across different Ethiopian neighborhoods. We employ a generalized linear mixed model (GLMM) to account for both individual-position variations or between-subject variations and variations in regions of child comorbidity (e.g., the child's ID and region). The analysis utilizes data from Performance Monitoring for Action Ethiopia (PMA-ET), a rich resource covering different

aspects of child health and ménage characteristics. Then, you might examine more closely at (Zimmerman et al.,2020a), who highlighted the importance of PMA-ET dataset for Ethiopian public health research. In terms of parameter estimation, a likelihood-based approach with Akaike's information criteria serves as a tool for model selection (Akaike, 1973a). Moreover, we generate easily interpreted scaled (quantile) residuals for fitted GLMMs using a simulation-based method with the DHARMa package in R for the fitted model (Hartig, 2018). Our analysis of advanced current methodologic approaches with a recent data set of interest will provide robust information for the best possible planning of health services as well as a better understanding of the state of children's health. The article is developed in collaboration with Prof. Kasahun Alemu, Maria Gabriella Campolo and Prof. Angela.

Chapter 2: Bayesian semiparametric geoadditive modeling of underweight among under-five children in Ethiopia.

In the second study, we utilize a Bayesian semiparametric geoadditive model that incorporates both social and demographic factors alongside the spatial effects on underweight prevalence. The model leverages data from the 2016 Ethiopian Demographic and Health Survey (EDHS) and employs statistical techniques like P-splines and Gaussian processes to model non-linear relationships and spatial trends. Our approach is informed by the work of Brezger Lang, (2008a); Dong Harris, (2015a); Eilers Marx, (1996a); and Kammann Wand (2003a) who have made significant contributions to the development and application of these methods.

Thus, even though a substantial body of research exists on child malnutrition in Ethiopia (Agho et al., 2019; Fenta et al., 2020; Hébuterne et al., 2014; Liben et al., 2016; Mulugeta et al., 2010; Rowhani et al., 2012; Tesema et al., 2021; Workie et al., 2020), a closer examination reveals limitations in the methodologies employed. Frequentist approaches often assume linear relationships between anthropometric measures factors (e.g., mother and child age, mother's BMI) and underweight, neglecting potentially non-linear patterns. Additionally, existing studies that utilize Bayesian-Gaussian regression to explore sociodemographic influences (Bacha Tadesse, 2019; S. Mohammed Asfaw, 2018a; Takele, 2013) often fail to analyze anthropometric, geographical, and sociodemographic effects simultaneously. This creates an incomplete picture, as spatial effects can significantly influence malnutrition rates. These limitations leave a critical gap in our understanding of how underweight manifests in Ethiopia. To address this, we propose a novel Bayesian geoadditive regression model that captures non-linear relationships between variables, allows for simultaneous analysis of spatial and sociodemographic effects, and provides a more flexible framework for modelling the complex interplay of factors contributing to underweight in Ethiopian children under five. The article is developed in collaboration with Prof. Maria Gabriella Campolo and Prof. Angela Alibrandi, and the article is under review at international journal of Public health.

Chapter 3: The causality of infant mortality in Ethiopia: A Structural Equation Modelling Approach

Finally, we employ structural equation modelling (SEM) to examine the causal relationships between various indicators and infant mortality rates. Understanding the complex interplay of factors contributing to the infant mortality rate burden is crucial for designing effective interventions. Furthermore, previous studies often fall short of capturing these intricate causal relationships. Thus, our research addresses this gap by employing SEM, a powerful multivariate technique, and allows for the simultaneous analysis of multiple variables and causal paths, enabling the estimation of both direct and indirect effects on IMR (Meydan Şeşen, 2011). This approach surpasses other statistical methods by providing a more comprehensive understanding of the mechanisms underlying the relationships between various factors influencing IMR (Nelson et al., 2020). We utilize data from the World Bank's Health Nutrition and Population Statistics spanning the period 2000-2019, enabling us to assess progress and identify potential areas of intervention. Our work builds upon the call by Tu, (2009), for increased use of SEM in epidemiological research, and leverages the advancements made in software like AMOS, EQS, and Mplus (Byrne, 2013). Furthermore, SEM offers a confirmatory approach, allowing researchers to test pre-established hypotheses about the causal structure (Hair Jr. et al., 2021).This is a significant advantage over exploratory methods that simply identify relationships within the data without necessarily revealing the causal direction. The article is developed in collaboration with Prof. Maria Gabriella Campolo and Prof. Angela Alibrandi, and it is published in MDPI, Children 2023, 10(2), 397; https://doi.org/10.3390/children10020397

1.3 Research questions

Research questions act as a roadmap for addressing a specific knowledge gap within any investigation (Polit Beck, 2008). Crucially, precisely formulated research inquiries help to offer novel insights by addressing unresolved issues in the current scholarly discourse (Rudolph, 2015). Thus, we tried to develop the appropriate research questions that guide the entire structure of this PhD thesis and provide a framework for data analysis, discussion of findings, and the formulation of conclusions.

The first chapter of our study dissects child comorbidity in Ethiopia. It asks how much a child's characteristics and their region influence comorbidity risk and to what extent the failure to examine interconnected factors contribute to our understanding of childhood comorbidity in Ethiopia. This explores the interplay between individual and regional factors by analyzing national data. The research will inform interventions targeting specific regional needs and child vulnerabilities.

Similarly, the second chapter of our research tackles childhood malnutrition in Ethiopia by addressing limitations in prior studies. Existing research often overlooks potential

non-linear relationships between anthropometric measures and underweight, and neglects geographical variations. This creates a gap in understanding the interplay between these factors and underweight prevalence in children under five. Therefore, this study aims to answer key questions like: 1) How do sociodemographic factors and geographical location interact to influence underweight prevalence? 2) Are there non-linear relationships between specific anthropometric variables and underweight that haven't been captured by previous models? 3) Does underweight show spatial clustering across Ethiopian regions? This study with a Bayesian geoadditive regression model allows for a more thorough understanding of the factors that lead to underweight and, in turn, helps to inform targeted interventions to improve nutritional status and address regional disparities.

Finally, our last research project employed structural equation modeling (SEM) from World Bank datasets (2000-2019) to investigate the causal relationships between various health, nutrition, and population indicators and the infant mortality rates (IMR) in Ethiopia. The core question was: What are the direct and indirect causal influences of these indicators on IMR? By elucidating these causal relationships, the study seeks to provide valuable insights for policymakers to develop targeted interventions for reducing infant mortality in Ethiopia.

1.4 Overall Significance

These three projects collectively contribute to a deeper understanding of the factors influencing child health and mortality in Ethiopia. By employing advanced statistical methods, this research provides valuable information for policymakers, stakeholders, and public health professionals working to improve child health outcomes in the country. The findings can ultimately guide the development of more effective strategies for ensuring the well-being of children in Ethiopia.

1.5 Conclusion

This dissertation has undertaken a thorough investigation of Ethiopian child health and mortality, making use of comprehensive statistical models to the light on important variables affecting these vital outcomes. According to the findings, child health and mortality in this rapidly changing country are complicated and varied.

The first study, employing a Generalized Linear Mixed Model (GLMM), delved into the intricate child comorbidity of Ethiopia in children under one year old. This approach proved invaluable in unravelling the hierarchical nature of the data, where socioeconomic background and healthcare use varied based on each child. The analysis revealed a clear association between a child's health and factors such as cooking fuel type, wealth level, maternal education, and access to electricity. Notably, the study identified a crucial role for maternal socioeconomic standing, suggesting that targeted interventions aimed

at uplifting mothers' economic and educational opportunities can significantly reduce the prevalence of childhood illnesses. Despite initial results suggesting minimal overall variation, including random effects helps the model account for potential regional and individual differences. This leads to a more comprehensive understanding of how child illness patterns might vary across locations and for specific people. These findings contribute to the global discussion on the importance of social determinants of health, highlighting their profound impact on child well-being even in early infancy.

The second study, leveraging a Bayesian geoadditive model, tackled the pressing issue of underweight in children under five. This flexible model excelled in capturing the intricate relationships between various factors and a child's weight status. The analysis yielded crucial insights, pinpointing significant associations between a mother's education level, a child's history of diarrhea or anemia, and access to electricity. Interestingly, the study found minimal influence of the child's household head's sex, prompting further investigation into the specific dynamics within Ethiopian households that may influence child health outcomes. The anthropometric variables like mother and child age, and mothers BMI have a nonlinear relation with underweight. Furthermore, the model identified geographic hotspots of underweight, emphasizing the need for targeted interventions in these areas. The study advocates for the development of comprehensive social programs designed to address childhood underweight, focusing specifically on areas with the highest burden. These results support national efforts to improve development in young children and show that focusing interventions on specific areas is crucial for addressing public health issues.

Finally, a structural equation model path analysis delved into the complex web of factors influencing infant mortality rates (IMR) in Ethiopia. This powerful approach facilitated the examination of both direct and indirect effects on IMR, providing a more holistic understanding of the underlying mechanisms. The study revealed a significant impact of maternal mortality ratio, national fertility rate, and GDP per capita on IMR. Notably, a higher GDP was associated with a lower IMR, highlighting the critical role of economic development in improving child health outcomes. This finding aligns with existing literature on the association between economic prosperity and child well-being. However, the research yielded surprising results regarding the limited influence of government health expenditure and the BCG vaccination. These unexpected findings warrant further investigation and exploration of alternative explanations. Based on the study's comprehensive analysis, the study argues that the most crucial strategies for lowering IMR in Ethiopia are reducing national fertility rates, enhancing the quality of maternal care, and increasing GDP. These recommendations provide valuable guidance for policymakers and healthcare professionals in Ethiopia, directing resources and efforts towards the most impactful interventions.

While this dissertation has yielded significant advancements in our understanding of

child health and mortality in Ethiopia, limitations remain. Data availability for periods before 2000 presented a challenge, restricting the analysis of historical trends. Additionally, the studies focused on a specific set of factors. Future research can significantly benefit from incorporating a broader range of variables, including environmental factors, paternal health, and access to specific healthcare services. By expanding the scope of investigation, researchers can create an even more comprehensive picture of the factors shaping child health and mortality in Ethiopia.

In closing, this dissertation has utilized advanced statistical models to unveil the complexities of child health and mortality in Ethiopia. The findings provide valuable insights for policymakers, healthcare professionals, and researchers. By addressing the identified limitations and continuing research efforts, we can contribute to a brighter future for children in Ethiopia, ensuring their optimal health and well-being. Future research holds the promise to further refine our understanding and inform targeted interventions, leading to a healthier and more vibrant future for all Ethiopian children.

Chapter 2

Neighborhood-level heterogeneity of child comorbidity in a generalized linear mixed model: Based on the Performance Monitoring for Action Ethiopia (PMA-ET) community survey

Abstract

Child morbidity affects a child's development, growth, and the overall well-being of society. This study aimed to examine the comorbidity of children in a sample of Ethiopian children based on the Performance Monitoring for Action Ethiopia community survey (PMA-ET), as well as the existence of child-specific, regional variation in children's comorbidity and its relationship to socioeconomic and demographic variables in families. We enrolled 2581 children suffering from different illnesses from six different regions of the country. Maximum likelihood estimates in Generalized linear mixed models (GLMMs) were used to assess children's comorbidity status. We used the Diagnostics for Hierarchical Regression models (DHARMa) package in R to provide readily interpretable scaled residuals and test functions for typical model misspecification problems for the fitted GLMMs. GLMMs with two random intercept models show the presence of child morbidity variations. Cough, fever, and diarrhea were found to be the most frequent types of children's illnesses among the main illness categories that were recorded. Cooking fuel, wealth quartiles, mothers' marital status, mother age, parity, residence, mother's education status, and availability of electricity were significantly associated with children's morbidity. These data show that variations in children's comorbidity were associated with both regional and child-specific characteristics. Thus, general principles for designing policies and interventions are required to reduce child comorbidity.

KEYWORDS

AIC; Children Comorbidity; DHARMa; GLMMs; Laplace Approximation; Random Effect

2.1 Introduction

Child morbidity remains a major health challenge, and its rate of decline is dawdling [1]. According to [29], approximately five million children under the age of five passed away. These deaths were mostly from preventable and treatable causes. The leading causes of such child deaths include preterm birth difficulties, congenital abnormalities, injuries, and non-communicable illnesses such as Acute respiratory infection (ART), acquired heart disorders, diarrhea, cough, fever, pediatric cancers, malaria, vomiting, diabetes, and obesity diseases [23, 55, 6]. Furthermore, even in 2016, 15,000 children lost their lives each day globally, amounting to 5.6 million annually. Although this marks a significant decline from the 35,000 daily deaths (12.6 million annually) in 1990, there is still much progress to be made to achieve target 3.2 of the Sustainable Development Goals, which aims to reduce under-5 mortality to fewer than 25 deaths per 1,000 live births in all countries. Many of these children suffer from preventable or treatable conditions such as fever, diarrhea, and malaria. [68]. Although Ethiopia's infant mortality rate fell from 34.010 deaths per 1000 live births in 2020 to 29.524 deaths per 1000 live births in 2023 [49], child morbidity was still significant, particularly among children under the age of one [25, 63].

Therefore, to successfully formulate a national policy for childhood morbidity intervention, it is necessary to identify determinants in a local context. Hence, several earlier studies suggested that environmental, socioeconomic, demographic, and health-associated factors lead to childhood morbidity globally [1, 31, 34, 41, 61, 66]. Some of the following variables, for instance, mother's age, mother's education, children's food status, family wealth, handwashing, sanitation, gender of the child, child's anemia level, husband's education level, mother's employment status, mother's marital status, breastfeeding status, and exposure to morbidity information have been found to have an impact on child morbidity [5, 15, 19, 18, 24, 31, 36, 41, 44, 38, 65, 71]. Two-parent families have more stable family structures and stronger social support networks for their children to improve their child health [7, 46, 74]. Likewise, the rate of children's illness also differs across geographical regions, their residence, and high-parity-births, and availability of electricity [57, 1, 34, 37, 65, 77, 69].

Furthermore, previous studies in Ethiopia have identified a wide range of risk factors, including socioeconomic, environmental, demographic, and other elements that influence childhood morbidity [3, 18, 52, 67, 70, 77]. Due to the lack of access to healthcare and the low socioeconomic conditions of Ethiopian households, children in Ethiopia typically have various health issues. However, most researchers focused on predicting the characteristics of a single health condition. Besides, understanding the cause and expected outcome of morbidity in children will be insufficient if the focus is on specific diseases or categories of illnesses [27, 66]. Moreover, previous studies also did not account for potential variation among clusters of individuals or groups. Thus, to account for this source of variability, we propose a generalized linear mixed model (GLMMs) that can be used to analyze data

collected from multiple subjects within different clusters or clustered data and handle random effects that are used to model the variability in the response variable due to the grouping structure of the data [47] . In comparison to pure time series or cross-sectional data, GLMMs are more efficient, include more information, have more variability, and can represent both common and individual behaviours [20].

Therefore, in this paper, we specifically focus on studying within-subject variation and between-subject effects in GLMMs considering the presence of two or more health conditions or diseases simultaneously in a child and potential variation among clusters of individuals or groups to understand how child comorbidity varies within and between subjects by considering the child's id and region as random effects and identifying the factors associated with this heterogeneity in Ethiopia. The study also utilizes diverse potential predictors for comorbidity sourced from the 2019 Performance Monitoring for Action Ethiopia Performance Monitoring for Action Ethiopia (PMA-ET) community survey datasets. These datasets systematically gather information on child health and household characteristics, drawing from a nationally representative sample of households. It's worth noting that this dataset captures valuable information that is presently underutilized by other extensive surveys, such as Demographic and health surveys (DHS) [78]. In terms of parameter estimation, a likelihood-based approach is often recommended, with Akaike's information criteria (AIC) serving as a tool for model selection in likelihood-based estimation [4]. Moreover, we generate easily interpreted scaled (quantile) residuals for fitted GLMMs using a simulation-based method with the DHARMa package in the R for the fitted model [35]. Our analysis of advanced current methodologic approaches with a recent data set of interest will provide robust information for the best possible planning of health services as well as a better understanding of the state of children's health.

2.2 Materials and methods

The study makes use of data from the 2019 Ethiopian Performance Monitoring for Action (PMA-Et) community survey, which collects details on mothers' characteristics and child health from a nationally representative sample of households.

2.2.1 Data sources, sampling, and study design settings

Data from the Performance Monitoring for Action Ethiopia project, a national survey conducted from August 2019 to September 2020, were used. It measures key reproductive, maternal and newborn health (RMNH) indicators. Pregnant women through one year postpartum are collected in the cohort of 2019 in five large, predominantly agrarian regions: Tigray, Oromiya, Amhara, and Southern Nations, Nationalities, and Peoples' Region, and one urban region, Addis Ababa. We receive a permission to download PMA-Et 2019 data from https://www.pmadata.org/data after making a reasonable request.

Using multistage stratified sampling, PMA-ET selects households in sampled clusters or enumeration areas Enumeration areas (EAs) based on a probability proportionate to their size within strata. Women between the ages of 15 and 49 were all screened, and those who were pregnant or had just given birth were eligible to participate in the survey. By interviewing the required number of women for each EA, PMA-ET was able to produce a sample that was representative at the national and regional levels. During the interview, women were asked about the socioeconomic characteristics of their households and the health status of their children. You may find additional details on the informed consent processes as well as other information on the PMA-ET survey at [78]. We consider a total of 2581 children under the age of one among 2871 mothers in six sample regions.

2.2.2 The variables

Our study includes a range of potential predictors of child comorbidity (see Table 2.1), such as the mother's age, educational background, parity, region, residence, types of cooking fuel, sanitary classification, availability of electricity, and wealth. To identify the most significant associations with childhood illnesses, we utilized data from the 2019 women's survey. The outcome variable considered is binary, taking a value of 1 if a child developed at least one complication (namely cough, fever, diarrhea, vomiting, eye infection, skin rash, poor feeding, difficulty breathing, etc.) during the postpartum interview. Otherwise, it takes a value of 0:

$$y = \begin{cases} 1 & \text{if the child suffers from at least one major complication} \\ 0 & \text{otherwise} \end{cases}$$

Considering the random effect data utilized in this study: Children's identification, labeled as "Child_ID," represents between-subject variation or interclass correlation. It captures variation in child comorbidity due to differences between individual children and is not shared by any other children. The region represents within-subject variation or intraclass correlation. It captures the variation in child comorbidity due to differences in the slope of the relationship between child comorbidity and fixed effects for each child. We grouped samples by six different regions in the country: Afar, Amhara, Oromia, Tigray, SNNP, and Addis Ababa. Each region contributes to child morbidity due to the random slope effect, which is shared by all observations within each child.

In the GLMM model of a categorical variable, one of the categories is used as a reference category, and the other categories are then measured against the reference category [47]. Besides, region and child_ID are uniquely labeled; we can specify random effects as (1|region) and (1|child_ID).

Table 2.1: Sociodemographic covariates and their labeling for child comorbidity study

Variable	Labelling
Cooking fuel	Electricity = 1, kerosene = 2, charcoal = 3, and wood = 4
Wealth	Lower quartiles = 1, middle quartiles = 2, and higher quartiles = 3
Sanitation classification	Improved but not shared facilities = 1, shared facilities = 2, non-improved facilities = 3, and open defecation = 4
Residence	Urban = 1, and rural = 2
Education	Never attended = 0, primary education = 1, secondary education = 2, and above secondary education = 3
Marital status	Married or with a partner = 1, widowed or divorced = 2, and never married = 3
Age	Age between 15 and 24 = 1, age between 25 and 34 = 2, and age above 34 = 3
Parity	Zero parity = 0,parity between 1 and 2 = 1,parity between 3 and 4 = 2, and parity above 4 = 3
Electricity availability	No = 1, and yes = 2
Region	Addis Ababa, Tigray, Afar, Amhara, Oromia, and SNNP

2.2.3 Methods

2.3.1 Generalized Linear mixed models and the specification of the models

Generalized Linear mixed models (GLMMs) combine the features of Generalized Linear models (GLMs) (which handle non-normal response variables) and mixed-effects models (which account for random effects due to clustering or data from different study sites). It effectively incorporates three essential elements: the linear predictor, which combines fixed and random effects; the exponential family, which symbolizes the dependent variable's distribution (e.g., normal, binomial, Poisson); and the link function, which connects the linear predictor to the expected response value [11, 50, 79]. Random effects in the context of cluster data capture unexplained variability beyond what fixed effects account for. Each cluster (e.g., subject or study site, in our case, regions) has its own unique random effect, allowing for subject-specific or study area-specific variation [14, 16],[30]. GLMMs are used for fully parametric, subject-specific inference for clustered or repeated measurement responses in the exponential family [33]. These models are powerful tools for analyzing complex data structures and are commonly used in various fields of research and statistical analysis. It is particularly useful in biomedical studies as they can account for the correlation between observations that arise from the hierarchical structure of the data. In recent years, GLMMs empower biomedical researchers by providing a unified framework for modeling complex data, capturing subject-specific variation, and addressing correlation structures. Their flexibility and interpretability make them a valuable tool for advancing medical knowledge [76, 58].

Model specification of GLMMs

Let y_{ij} be the binary response measure for the i-th cluster, where $i = 1, 2, \ldots, N$ and

$j = 1, 2, \ldots, n_i$. The vector x_{ij} represents the i-th row of the matrix for the fixed effect. The vector y_i is an n_i-dimensional vector of all measurements available for the i-th child, conditional on the random vector b_i with q dimensions. It is supposed to be drawn independently from a distribution belonging to the exponential family. Furthermore, b_i captures unobserved factors specific to each cluster that affect child comorbidity and is assumed to be drawn independently from a normal distribution with mean zero and variance σ_b^2, i.e., $b_i \sim N(0, \sigma_b^2)$, where σ_b^2 represents the population distribution variance and indicates the degree of subject heterogeneity [17, 47, 53].

Thus, the probability density function of the response y_{ij}, which is independent of the distribution of y_i, is given by:

$$f_i(y_{ij}|b_i, \beta, \phi) = \exp\left(\frac{y_{ij}(\theta_{ij} - \Psi(\theta_{ij}))}{\phi} + C(y_{ij}, \phi)\right) \tag{2.1}$$

Here, θ_{ij} is the linear predictor ($\theta_{ij} = x_{ij}^T\beta + z_{ij}^T b_i$), $\Psi(\theta_{ij})$ is the link function, ϕ is the parameter for dispersion, and the normalizing constant is $C(y_{ij}, \phi)$.

The function $g(\mu_{ij})$ is the inverse of the link function $\Psi(\theta_{ij})$. The relationship between $g(\mu_{ij})$ and $f_i(y_{ij}|b_i, \beta, \phi)$ is given by the following equation:

$$g(\mu_{ij}) = \int f_i(y_{ij}|b_i, \beta, \phi)\, dy_{ij} \tag{2.2}$$

Using Laplace approximation, equation (2) approximates to the function:

$$g(\mu_{ij}) = g[\epsilon(y_{it}|b_i)] = x_{ij}^T\beta + z_{ij}^T b_i \tag{2.3}$$

The function $g(\cdot)$ is a known link function that belongs to the GLMM framework, used to map the expected values of the response variable to the linear predictor x_{ij}.

The function $g(\cdot)$ is a known link function that belongs to the GLMM framework. It is used to map the expected values of the response variable to the linear predictor. Here are the relevant terms: x_{ij} is the i-th row of the matrix of fixed effects, z_{ij} is the i-th row of the matrix of random effects associated with b_i, β is the parameter vector of unknown fixed effects and ψ is the scale parameter or cumulant generating function.

Under this GLMMs settings, the logit link function is defined as:

$$g(\mu_{ij}) = \text{logit}(\mu_{ij}) = \log\left(\frac{\mu_{it}}{1 - \mu_{ij}}\right) = \eta_{ij} = x_{ij}^T\beta + z_{ij}^T b_i \tag{2.4}$$

In vector or matrix terms, we can rewrite it as,

$$\begin{bmatrix} \text{logit}\,(\mu_{ij}) \\ \text{logit}\,(\mu_{i2}) \\ \cdot \\ \cdot \\ \cdot \\ \text{logit}\,(\mu_{ini}) \end{bmatrix} = \begin{bmatrix} \text{logit}\,\epsilon(y_{i1}) \\ \text{logit}\,\epsilon(y_{i2}) \\ \cdot \\ \cdot \\ \cdot \\ \text{logit}\,\epsilon(y_{ini}) \end{bmatrix} = \begin{bmatrix} x_{i1}'\beta + z_{ij}'b_i \\ x_{i2}'\beta + z_{i2}'b_i \\ \cdot \\ \cdot \\ \cdot \\ x_{ini}'\beta + z_{ini}'b_i \end{bmatrix} = \begin{bmatrix} x_{i1}' \\ x_{i2}' \\ \cdot \\ \cdot \\ \cdot \\ x_{ini}' \end{bmatrix} \beta + \begin{bmatrix} z_{ij}' \\ z_{i2}' \\ \cdot \\ \cdot \\ \cdot \\ z_{ini}' \end{bmatrix} b_i$$

and can be simplified as

$$= x_i\beta + z_i b_i \tag{2.5}$$

Note that $\mu_{ij} = \frac{e^{x_{ij}'\beta + z_{ij}'b_i}}{1+e^{x_{ij}'\beta + z_{ij}'b_i}}$ is is a conditional probability on b_i. In this case, the conditional expectation equals the conditional probability of a response given the random effects(and covariance values), i.e., $\mu_{ij} = \epsilon(y_{ij}|b_i, x_i) = P(y_{ij}|b_i, x_{ij})$. The model can be expressed as:

$$P(y_{ij}|b_i, x_{ij}, z_{ij}) = g^{-1}(\eta_{ij}) = g^{-1}(x_{ij}^T\beta + z_{ij}^T b_i) \tag{2.6}$$

Where the inverse link function $g^{-1}(\eta_{ij})$ is the logistic cumulative distribution function (CDF), which is used to quantify the binary response, namely:

$$g^{-1}(\eta_{ij}) = \frac{1}{1+e^{\eta_{ij}}} \tag{2.7}$$

In GLMMs, the logistic distribution can facilitate the process of estimating the distribution's parameters by maximum likelihood estimation or other techniques and has the advantage of making a straightforward parameter estimation [32].

Estimation

Likelihood-based approaches rely on the likelihood function to estimate the parameters in Generalized Linear Mixed Models (GLMMs). This provides opportunities such as consistent and efficient estimates of fixed and random effects, likelihood-based inference methods, model comparison with different assumptions and links, and prediction of random effects and conditional responses. With this model, the joint distribution of both the vectors of response and the vectors of random effects is fully specified. We can use similar methods to estimate these models [10, 42, 50]. Given the above model specification for the GLMMs based on the assumption that the binary responses y_{ij} (conditioned on the random effects b_i) are conditionally independent, the joint probability of the response vector (y_i) and the random effect vector (b_i) for the distribution of the ith random effect can be explained as follows:

$$f(y_i, b_i) = f(y_{ij}|b_i)f(b_i) = f(y_{i1}|b_i)f(y_{i2}|b_i)\ldots f(y_{in_i}|b_i)f(b_i) \tag{2.8}$$

Then the likelihood function of the parameters β and σ_b^2 is given by :

$$L(\beta, \sigma_b^2) = \prod_{i=1}^{n} f(y_i) = \prod_{i=1}^{n} \int f(y_i, b_i), db_i = \prod_{i=1}^{n} \int f(y_i|b_i) f(b_i), db_i = \prod_{i=1}^{n} \int \prod_{i=1}^{n} f(y_{ij}|b_i) f(b_i), db_i \tag{2.9}$$

Since y_{ij} is a binary response, having a value of 0 or 1, a logit link function links the conditional mean of y_{ij} to the linear predictors. Consequently, for every $i = 1, 2, \ldots, 2581$ and every $j = 1, 2, \ldots, n_i$, the linear predictor of equation (4) was equivalent to:

$$\eta_{ij} = x'_{ij}\beta + z'_{ij}b_i = x'_{ij}\beta + b_i \tag{2.10}$$

Thus, equation (8) can be put in the form of:

$$L(\beta, \sigma_b^2) = \prod_{i=1}^{n} \int \exp\left(\beta \sum_{i=1}^{n} y_{ij}x'_{ij} + y_i b_i\right) \prod_{j=1}^{n} \frac{1}{1 + \exp(x'_{ij}\beta + b_i)} \frac{1}{\sqrt{2\pi\sigma_b^2}} \exp\left(-\frac{1}{2\sigma_b^2} b_i^2\right), db_i \tag{2.11}$$

The values of β and σ_b^2 that maximize this likelihood function are the Maximum Likelihood (ML) estimates of β and σ_b^2. However, from equation (11), it is not possible to use the entire likelihood function since there are no closed-form solutions. Thus, it is necessary to employ estimates of the probability function to find a solution for this problem. Laplace's approximation methodology serves as the basis for several likelihood-based statistical procedures. When estimating parameters for Generalized Linear Mixed Models (GLMMs), the 'glmer' function from the 'lme4' package in R is used to estimate the likelihood. This approach enables us to make informed inferences about the model parameters [9, 72].

Laplace's approximation

The Laplace approximation is a quadrature method for estimating integrals of this kind was developed by Laplace and published in 1774,

$$\int_a^b f(t)e^{\lambda g(t)}dt \tag{2.12}$$

Where both $g(t)$ and $f(t)$ are continuous smooth functions, $f(t)$ is nonzero at t_0, and $g(t)$ is a twice-differentiable function on (a, b) with a maximum in the interval (a, b). The underlying principle of Laplace's approach is that, for large λ, the integral's bulk will come from the integral's contribution around a certain point, t_0. That resulting integral may be proven to represent the kernel of a normal distribution, which can then be integrated using second-order Taylor series expansions for $g(t)$ and $f(t)$. The integrand in the function is comparable to the likelihood of Generalized Linear Mixed Models (GLMMs), which contains exponential functions from the exponential family of probability distributions, as can be seen by examining the form above [9, 72].

Akaike information criterion

The Akaike information criterion (AIC) is a widely used likelihood-based model criterion. The model that minimizes the AIC is considered the best model. It is frequently employed in combination with the Bayesian Information Criteria (BIC) and the Deviance Information Criteria (DIC), as noted by Akaike (1973b). For data set $D = \{(y_i, x'_{ij})\}$, where y_i is the outcome vector and x'_{ij} is a set of fixed effects, and for the maximum likelihood estimator $\hat{\beta}$ under the computing model for (p) dimension of β, the AIC can be formulated as:

$$AIC = -2L(\hat{\beta}, D) + 2p \tag{2.13}$$

The likelihood ratio test for variance component in GLMMs

GLMMS are used to describe responses from an exponential family with a combination of fixed and random effects, and the variance component of GLMMs comes from random effects. (Sinharay and Stern, 2003). This is equivalent to testing that the variance component equals zero and the hypothesis of interest is:

$$H_0 : \sigma_b^2 = 0 \quad \text{Vs} \quad H_1 : \sigma_b^2 > 0$$

For the maximized log-likelihood under the null hypothesis l_1 and the variance component estimated l_0, the test statistics for variance components of the likelihood ratio test are given by:

$$G^2 = 2(l_1 - l_0) \tag{2.14}$$

Here G^2 follows a chi-square distribution with 1 degree of freedom. Thus, if the null hypothesis (simpler model) is correct, we can use the chi-square distribution to calculate the likelihood of finding a value of G^2 as severe as the one we computed [75].

2.3 Results and Discussion

2.3.1 Results

3.1.1 Explanatory Data Analysis

Our exploratory analysis of clustered data aims to identify characteristics of random variation that differentiate individual children or patients, as well as patterns of systematic variation across geographical variation in the geographical location of children. 2871 women from 6 survey regions were interviewed, and 2581 children (0–1 year old) were considered. Their morbidity status and information about the disease pattern were collected based on the PMA 2019 survey [78].

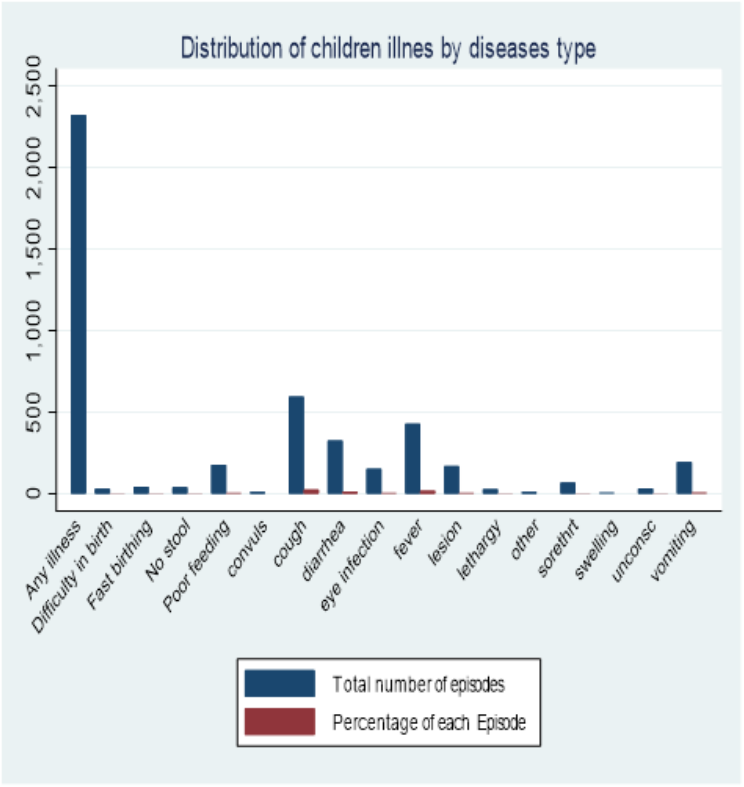
Considering broad category distributions of illness among children (see Table 2.2), cough, fever, and diarrhea were found to be the most frequent types of children's illnesses, with percentages of 25.67, 18.52, and 14.08, respectively. Moreover, fast birthing, no stool, difficulty in birth, and swelling occurred at all lower rates under one year of age. A total of 2322 episodes of any illness, in which children reported having at least one illness, were noted among the children who were considered in the PMA 2019 survey.

Table 2.2: Distribution of the broad categories of illness among children

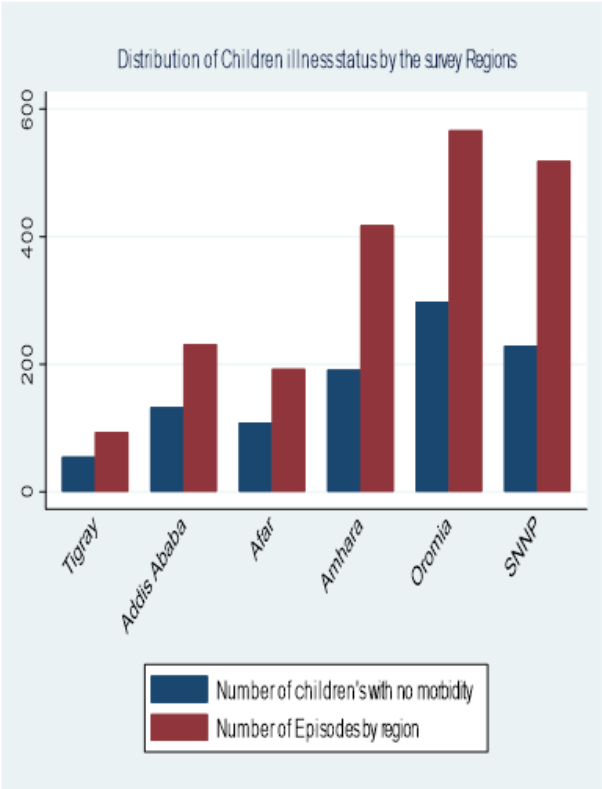
Broad Illness Category	**Total Episodes**	**Percentage of Episodes**	**Mean**	**S. D.**
Any illness	2322	1.148	0.036	
Cold/cough	596	25.67	0.43	0.136
Fever	430	18.52	0.322	0.109
Diarrhea	327	14.08	0.244	0.082
Vomiting	195	8.4	0.139	0.043
Difficulties feeding/unable to suck	178	7.67	0.131	0.043
Skin rash/skin lesion	170	7.32	0.122	0.038
Red eye/passage of pus from eyes	153	6.57	0.121	0.045
Sore throat/Tonsillitis	68	2.93	0.046	0.013
Fast birthing	42	1.81	0.033	0.012
No stool	40	1.72	0.032	0.012
Unconscious	32	1.38	0.005	0.02
Difficulty in birth	31	1.34	0.02	0.005
Reduced alertness (lethargy)	29	1.25	0.025	0.01
Convulsion	11	0.47	0.009	0.004
Abdominal/body swelling	9	0.39	0.007	0.003
Other	11	0.47	0.008	0.003

Furthermore, the subsequent graph (see Figure 2. 1) displays the distribution of illnesses by disease types (left side of plots) as well as children's disease status by survey region (right side of plots). Oromia, SNNP, and Amhara regions account for the highest frequency of morbidity illness episodes in the country, followed by Addis Abeba, Afar, and

Tigray, based on the PMA 2019 survey. Furthermore, cough, fever, and diarrhea were the most common types of disease in the country that were seen during the survey. The



A Distribution of disease type for children illness



B, Distribution of children illness by survey region

Figure 2.1: The distribution of illness types among children (see plot A) and the distribution of illness among survey regions (see plot B)

density of residuals and distribution of responses give insight into how the responses and predictors are related to one another [48, 60]. The distribution of responses is shown on the bottom right of Figure 2.2, whereas the density of residuals is shown on the bottom left (refer to Figure 2.2). With these distributions, non-normally distributed responses are possible accommodated, including non-linear links between the mean of the child morbidity and the predictors, as well as some form of correlation in the data. Thus, GLMMs with logit link functions are an ideal method of detecting child morbidity for the given datasets.

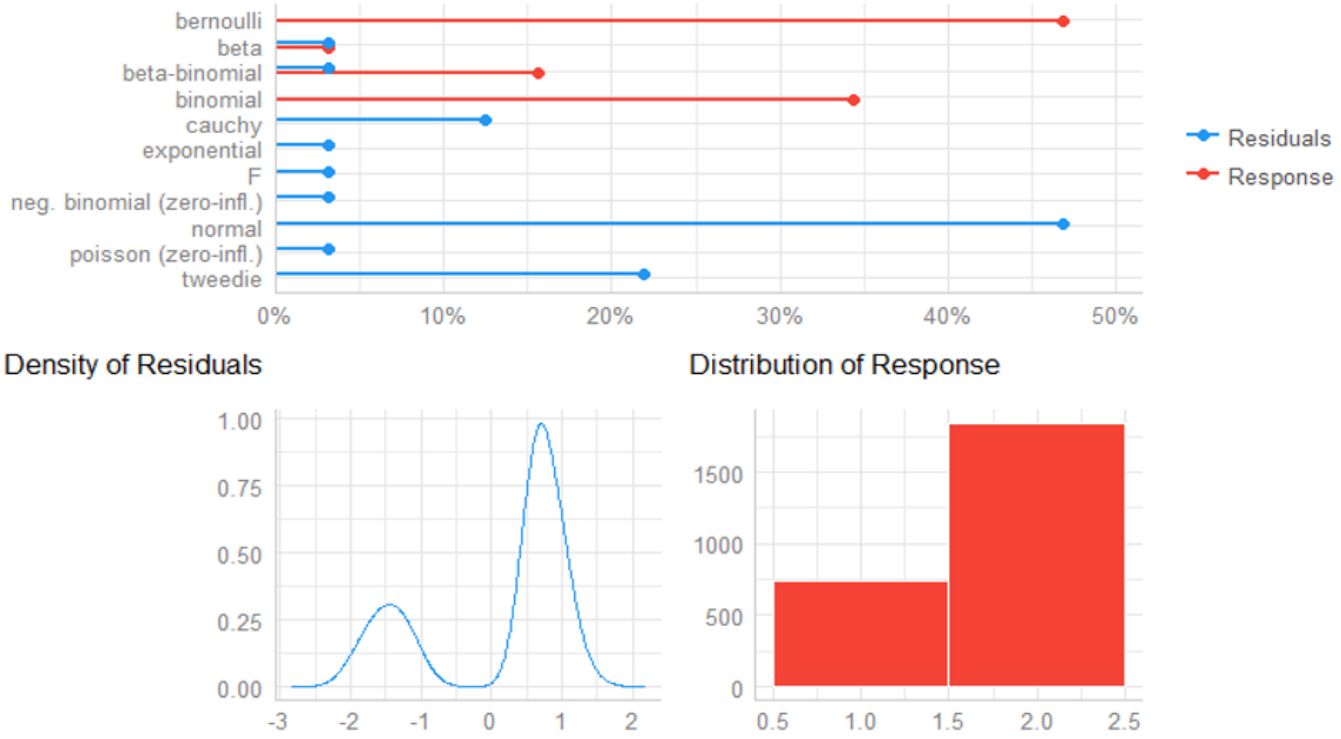


Figure 2.2: Predicted distribution of residuals and response for child comorbidity study

A bivariate study using Pearson's chi-squared test has been carried out to examine the association between a few chosen variables [21]. The following table (see Table 2.3) represents the contingency table of the morbidity status of children, along with Pearson's chi-square value to determine if a particular regression coefficient is significant. Mother's age is the only variable that is not significantly (p-value = 0.632) related to child morbidity among all the factors that were taken into consideration. According to this table, cooking fuel, marital status, education, place of residence, sanitation classification, wealth quartiles, electricity availability, and parity were strongly related to childhood morbidity at the 5% significance level.

Furthermore, morbidity is predominant among children whose mothers use charcoal for fuel (37.16%), never attended education (30.20%), live in rural areas (47.77%), and have lower quartiles of wealth (32.70%). Compared to children from lower-quartile families, children from middle- and upper-quartile households had reduced rates of childhood illness, and comparable situations were also observed for other covariates.

Table 2.3: Characteristics of the study participants by morbidity and their mother's sociodemographic status

Characteristics	W.Freq	No, n (%)	Yes, n (%)	χ^2 (p-value)
Cooking Fuel				
Electricity	435 (16.85)	203 (7.87)	232 (8.99)	$\chi^2(3) = 104.06$, $p < 0.001$
Kerosene	10 (0.39)	1 (0.04)	9 (0.35)	
Charcoal	1764 (68.35)	408 (15.81)	1356 (52.54)	
Wood	372 (14.41)	130 (5.04)	242 (9.38)	
Mothers' Marital Status				
Married or with partner	442 (17.13)	155 (6.01)	287 (11.1)	$\chi^2(2) = 10.36$, $p = 0.006$
Widowed or divorced	810 (31.38)	217 (8.41)	593 (22.9)	
Never married	1329 (51.49)	370 (14.34)	959 (37.16)	
Mothers' Age				
15-24	877 (33.98)	264 (10.23)	613 (23.75)	$\chi^2(2) = 1.19$, $p = 0.632$
25-34	1312 (50.03)	369 (14.30)	943 (36.54)	
35+	392 (15.19)	109 (4.22)	283 (10.96)	
Mothers' Education				
Never attend	986 (38.20)	205 (7.94)	781 (30.20)	$\chi^2(3) = 83.80$, $p < 0.001$
Primary	924 (35.8)	258 (10)	666 (25.8)	
Secondary	393 (15.23)	158 (6.12)	235 (9.10)	
Higher or TVET	278 (10.78)	121 (4.69)	157 (6.08)	
Residence				
Urban	1001 (38.78)	395 (15.30)	606 (23.48)	$\chi^2(1) = 90.22$, $p < 0.001$
Rural	1580 (61.22)	347 (13.44)	1233 (47.77)	
Wealth Quartiles				
Lower quartile	842 (32.62)	148 (5.73)	844 (32.70)	$\chi^2(2) = 101.79$, $p < 0.001$
Middle quartile	400 (15.50)	99 (3.84)	694 (26.89)	
Higher Quartile	1339 (51.88)	495 (19.18)	301 (11.66)	
Parity				
0	518 (20.17)	192 (7.44)	326 (12.63)	$\chi^2(3) = 49.08$, $p < 0.001$
1-2	1031 (39.95)	326 (12.63)	705 (27.31)	
3-4	566 (21.93)	132 (5.11)	434 (16.82)	
5+	466 (18.06)	92 (3.56)	374 (14.49)	
Sanitation Classification				
Improved or shared	119 (4.61)	52 (2.01)	67 (2.60)	$\chi^2(3) = 70.64$, $p = 0.006$
Shared Facility	416 (16.12)	180 (6.97)	236 (9.14)	
Non-improved facility	1170 (45.33)	311 (12.05)	859 (33.28)	
Open defecation	876 (33.94)	199 (7.71)	876 (33.94)	
Electricity Availability				
No	1386 (53.70)	310 (12.01)	1076 (41.69)	$\chi^2(1) = 59.53$, $p = 0.012$
Yes	1195 (46.30)	432 (16.74)	763 (29.56)	

W.Freq = Weighted Frequency,NO= no Morbidity status, Yes= yes Morbidity status

Furthermore, in the GLMMs, the plots of fixed effects an outcome variable can offer important information about how the predictors and the result are related [20, 51], and it supports the direction of the coefficients and the significance of the effects. From Figure 2.3 of the following sample plots, we can see that the fixed effects of residence, marital status, and parity are positively associated with child comorbidity, while the mother's wealth index is negatively associated with morbidity.

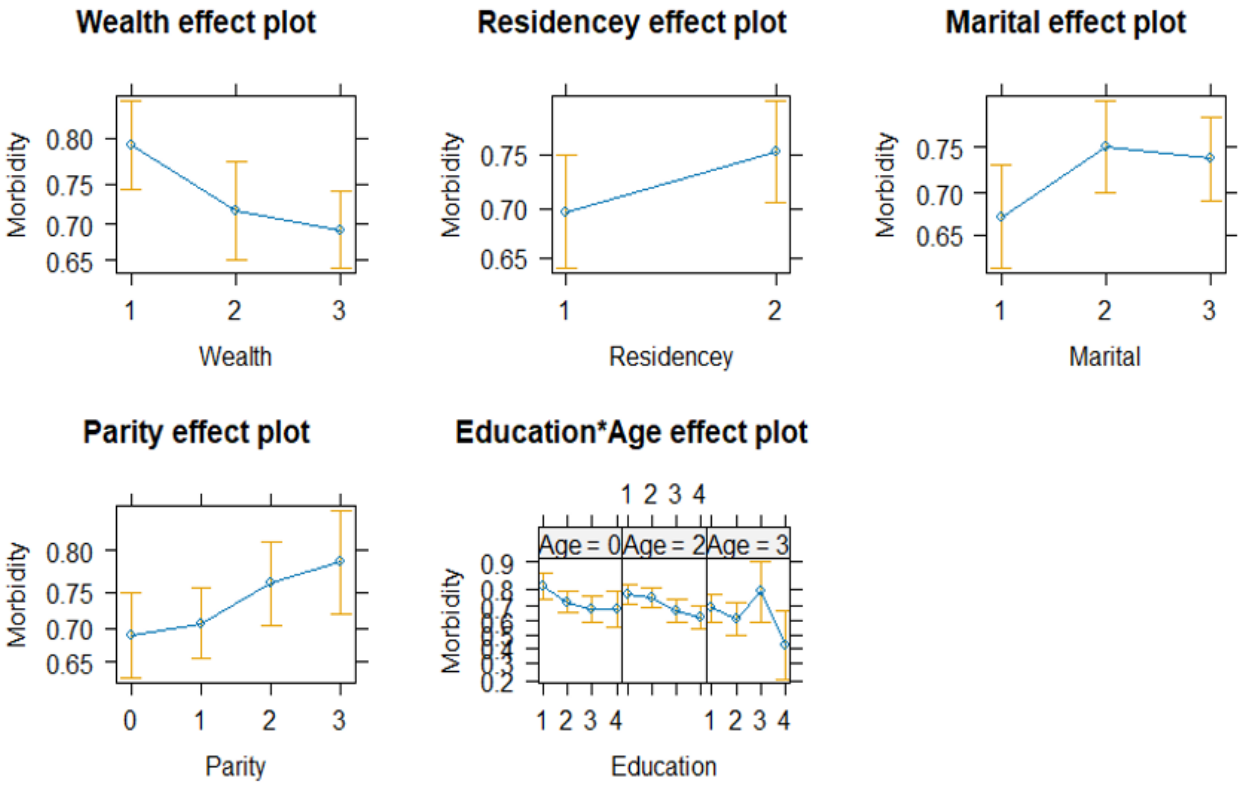


Figure 2.3: Interaction and Fixed Effects Plots in Child Morbidity Study

General linear mixed model analysis

Type III test for fixed effects

In GLMMs, Type-III tests are applied to evaluate each term's significance while taking into consideration the effect of every other term. Type III tests rely on each predictor's main effect, in contrast to Type I or Type II tests, which consider the predictors' order of entry. A significant Type III test indicates that the fixed effect has a statistically significant influence on the response variable [45].Table 2.4 of the Type III analysis of the likelihood ratio test of all the fixed effects (except sanitation class) significantly affects child morbidity.

Table 2.4: Type III tests of fixed effects from GLMMs of child morbidity

Fixed Effects	**DF**	**F-values**	**Pr(¿F)**
Cooking fuel	3	18.8098	0.0005***
Wealth	2	19.3282	0.0003***
Sanitation Class	3	0.6979	0.812
Residence	1	4.8428	0.0044**
Mother Education	3	6.9747	0.0016**
Marital status	2	4.4209	0.0119*
Mother's Age	2	1.9634	0.018*
Parity	3	2.7513	0.044*
Electricity Availability	1	6.0684	0.014*
Mother education: mother's age	6	1.4051	0.209

Tables 2.3 and 2.4 provide the regression estimates of the child comorbidity model using the 'glmer' function of the 'lme4' package in R [8]. The model formula in 'lme4' syntax for sets of fixed effects, interaction effects, and random effects was as follows:

$$\begin{aligned}\text{Logit}(\mu_{it}) =& \beta_0 + \beta_1 \text{Cooking fuel}_{ij} + \beta_2 \text{Wealth}_{ij} + \beta_3 \text{Sanitation Classification}_{ij} \\ &+ \beta_4 \text{Residence}_{ij} + \beta_5 \text{education}_{ij} + \beta_6 \text{marital}_{ij} + \beta_7 \text{age}_{ij} \\ &+ \beta_8 \text{parity}_{ij} + \beta_9 \text{Electricity availability}_{ij} \\ &+ \beta_{10} \text{interaction between education and mother age}_{ij} + \gamma_i + \gamma_{ij}\end{aligned}$$

Where β_1 to β_{10} are cluster odds ratios of children morbidity (unknown regression coefficients of the main and interaction effects for fixed effects), while γ_i and γ_{ij} are the subject-specific and regional level random intercepts, respectively.

Table 2.5 illustrates the estimates for fixed effects using maximum likelihood estimation in fitted GLMMs (see Table 2.5), and the estimates indicate that a one-unit increase (moving from one category to another) in the predictor would be expected to predict an increase in the estimated log odds of comorbidity equal to one when all other predictors are held constant [26, 40]. Moreover, the log odds are the probability of an event (like comorbidity) occurring expressed as its natural logarithm. The odds ratio is obtained by exponentiating the calculated log odds. In this case, an odds ratio of one denotes no change, but a ratio of more than one shows a rise in the likelihood of comorbidity [11, 2].

Based on the results, wealth status significantly affects the child morbidity status, and it is observed that children from middle quartiles (OR = 0.47, $P = 0.002$; 95% CI: $-0.766, -0.167$) and higher quartiles (OR = 0.62, $P = 0.001$; 95% CI: $-1.05, -0.415$) are less likely to suffer illness than children from lower quartiles. Our study also demonstrated that children from a mother with primary, secondary, and higher education are 41%, 52%, and 51% respectively, less likely to be ill than mothers who never attended school.

Similarly, children who lived in rural areas (OR = 1.66, $P = 0.004$; 95% CI: $0.158, 0.858$) are 1.66 times more likely to get affected by morbidity than children who lived in urban areas, and using wood as a fuel is 1.14 times more likely than using electricity to get child morbidity. Likewise, the absence of electricity (OR = 1.49; $P = 0.014$; 95% CI: $0.079, 0.718$) is more likely for children's illness as compared to children who can access electricity. This study's findings also suggest that a woman with a parity of 3–4 and 5+, never married, and divorced or widowed mothers' marriage statuses are more likely to have comorbidity than their counterparts.

Table 2.5: Estimates of fixed effects from GLMMs for children's comorbidity

Covariates	**Coef.**	**SE**	**Z**	**P> |Z|**	**OR**	**95% CI[Coef.]**
(Intercept)	0.79	0.33	2.4	0.016*	2.21	(0.146, 1.44)
Cooking Fuel (Ref. = Electricity)						
Kerosene	1.62	1.11	1.46	0.144	5.02	(-0.556, 3.78)
Charcoal	0.14	0.20	0.68	0.499	1.14	(-0.258, 0.528)
Wood	0.40	0.16	2.5	0.013*	1.48	(0.081, 0.712)
Wealth (Ref. = Lower Quartile)						
Middle Quartile	-0.47	0.15	-3.1	0.002**	0.62	(-0.766, -0.167)
Higher Quartile	-0.74	0.16	-4.5	0.001***	0.47	(-1.05, -0.415)
Sanitation Classification (Ref. = Improved, Not Shared Facility)						
Shared Facility	-0.11	0.22	-0.53	0.603	0.89	(-0.548, 0.318)
Non-Improved Facility	0.06	0.15	0.40	0.693	1.06	(-0.234, 0.351)
Open Defecation	-0.03	0.19	-0.14	0.891	0.97	(-0.380, 0.338)
Residence (Rural)	0.51	0.18	2.8	0.004**	1.66	(0.158, 0.858)
Mother Education (Ref. = Never Attended)						
Primary Education	-0.52	0.22	2.9	0.001**	0.59	(-0.946, -0.085)
Secondary Education	-0.71	0.25	-2.4	0.018*	0.48	(-1.21, -0.218)
Higher Education	-0.69	0.32	-2. 2	0.033*	0.49	(-1.34, -0.055)
Marital (Ref. = Married/Partner)						
Widowed Or divorced	0.38	0.14	2.8	0.004**	1.46	(0.120, 0.648)
Never Married	0.32	0.13	2.6	0.010*	1.37	(0.073, 0.559)
Mother's age (Ref. = 15–24)						
25-34	-0.27	0.23	-1.2	0.243	0.76	(-0.719, 0.183)
35+	-0.72	0.27	-2.5	0.010*	0.49	(-1.26, -0.169)
Parity (Ref. = 0)						
1-2	0.10	0.13	0.80	0.426	1.10	(-0.146, 0.344)
3-4	0.39	0.17	2.3	0.023*	1.48	(0.055, 0.733)
5+	0.53	0.24	2.5	0.013*	1.70	(0.111, 0.954)
Electricity Availability (NO)	0.40	0.17	2.5	0.014*	1.49	(0.079, 0.718)

Signif.codes: '***'= 0.001, '**' = 0.01, '*' = 0.05, '.' = 0.1 and ''= 1
OR = odds ratio, CI = confidence interval, SE = standard error, and SD = standard deviation.

Interaction effects

The interaction between a mother's age and education can be either synergistic or mitigating, as presented in Table 2.6, which shows the interaction between a mother's education (never attended, primary education, secondary education, or higher education) and a mother's age (between 15 and 24, between 25 and 34, and above 35). As the results indicated, children from mothers above 35 years of age have a lower risk of being ill compared to children whose mother's age is less than 34 for the secondary and higher mother education groups (OR = 2.3, P-value = 0.022, OR = 1.67, P-value = 0.015), respectively. Well-educated elder mothers who combine their experience and health skills can lead to better health outcomes for their children [64].

Table 2.6: Estimates of the two-way interaction effects and the variance parameter of the random effect models

Covariates	**Coef.**	**SE**	**Z**	**P> |Z|**	**OR**	**95% CI(Coef.)**
Education and age (Ref. = Never Attended: Age between 15-24)						
Primary Education: Age Between 25-34	0.39	0.28	1.5	0.149	1.47	(-0.142, 0.923)
Secondary Education: Age Above 25-34	0.24	0.32	0.76	0.449	1.27	(-0.379, 0.854)
Higher Education: Age Between 25-34	0.03	0.36	0.08	0.935	1.03	(-0.692, 0.751)
Primary Education: Age Above 35+	0.24	0.35	0.67	0.501	1.27	(-0.453, 0.926)
Secondary Education: Age Above 35+	1.26	0.55	2.3	0.022*	3.53	(0.176, 2.34)
Higher Education: Age Above 35+	0.43	0.65	1.67	0.015*	1.65	(0.831, 1.69)
Random Effects:	**Variance**	**SD**				
Region	5.318e-02	0.231				
Subject-specific (Child$_I D$)	4.598e-07	0.006				
Residual	0.123	1.045				

Signif.codes: '***'= 0.001, '**' = 0.01, '*' = 0.05, '.' = 0.1 and ''= 1

Note: OR = odds ratio, CI = confidence interval, SE = standard error, and SD = standard deviation.

Model comparison and diagnosis

Comparing the models is an important step in the modeling process to see which ones best fit the data [13, 54]. Akaike information criterion (AIC) is a widely used model selection criteria based on the maximum likelihood estimator [4]. Results of the AIC, log-likelihood likelihood test (LRT), BIC, and other information on the fit of the model are presented in Table 2.7. Accordingly, the model with two random intercepts (the random intercept of region and subject-specific) has a lower AIC (AIC = 2929.9) and is statistically significant ($P < 0.001$) in comparison to one random intercept model (AIC = 2942.6). It is also supported in the log-likelihood ratio test (LRT) with a significance P-value ($P < 0.001$). This suggests that two random intercept models from GLMMs permit data correlation

and provide more effective overall performance compared to one random intercept model.

Table 2.7: The Likelihood-Ratio-Test (LRT) and Akacia information criteria for random intercept models comparison

	Information Criteria for Model Comparison					**Likelihood-Ratio-Test (LRT)**		
	AIC	**BIC**	**loglik**	**deviance**	**Pr(¿Chisq)**	**df**	**Chi2**	**Pr(¿Chisq)**
ONE RIM	2942.6	3106.6	-1443.3	2886.6		28		
TWO RIM	2929.9	3099.7	-1435.9	2871.9	$P < 0.001249 * **$	29	14.72	$P < 0.001 * **$

Signif.codes: '***' = 0.001, '**' = 0.01, '*' = 0.05, '.' = 0.1, and '' = 1
ONE RIM: One random intercept model, TWO RIM: Two random intercept model

In GLMMs, random intercept plots are employed to illustrate the distribution of random effects [14, 73]. Figure 4 displays the diagnostic plots for random intercepts (see Figure 4) corresponding to two random effects, providing a visual representation of regional and subject-specific variability in child morbidity, and allowing for different baseline values (intercepts) for two groups or clusters. From the plots (see plots A and B of Figure 2.4), the dot on the horizontal line shows the estimated random intercept for each level of grouping variable, and the horizontal line represents the overall mean of each random effect [51, 10]. Thus, these plots might tell us about the presence of regional and subject-specific level variability for child morbidity. Therefore, even if the estimated variance in the intercept for each region and the subject-specific effect was found to be quite near zero, including random effects is a good modeling choice as there is a fair amount of variation in the estimations of regional and subject-specific effects.

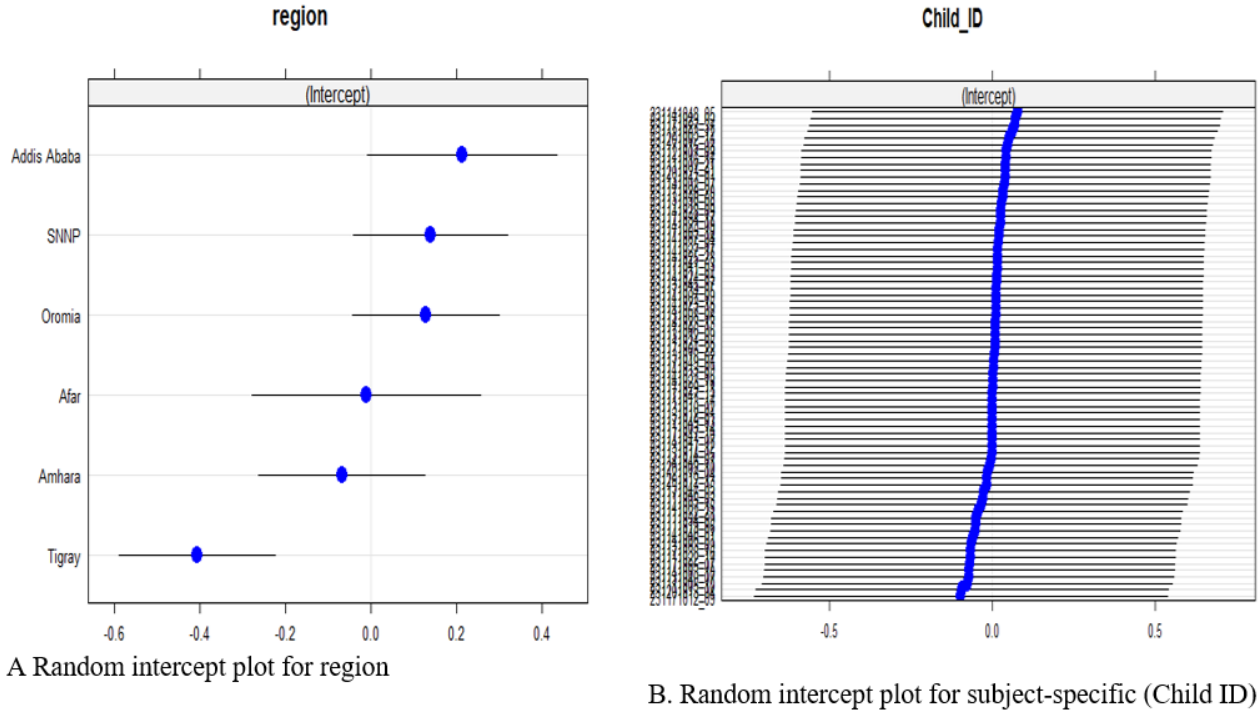


Figure 2.4: Random intercept plots study for regional and subject-specific (Child ID) (see plots A and B), respectively

Residuals diagnosis in GLMMs

Residuals in GLMMs have a coarse structure due to random effects and grouping of data. As a result, these models should not use techniques like QQ plots or Shapiro-Wilk tests to verify residual normality as standard linear models [12, 22, 43]. Therefore, we use the "Diagnostics for Hierarchical Regression models (DHARMa)' package to provide easily interpreted scaled residuals(quantiles) for fitted GLMMs [35] and binned residual plots in dividing the data into bins based on fitted value [30]. Therefore, Figure 2.5 displays the plots of residuals versus fitted values for fitted GLMMs (binned residuals). Hence, most of the residuals fall within the error bound (indicated in blue points), and fewer residuals are outside of the error boundaries (indicated in red points). Thus, most of the binned residual fell within the 95% confidence interval of error bounds, which indicates that the model is a good fit for the data.

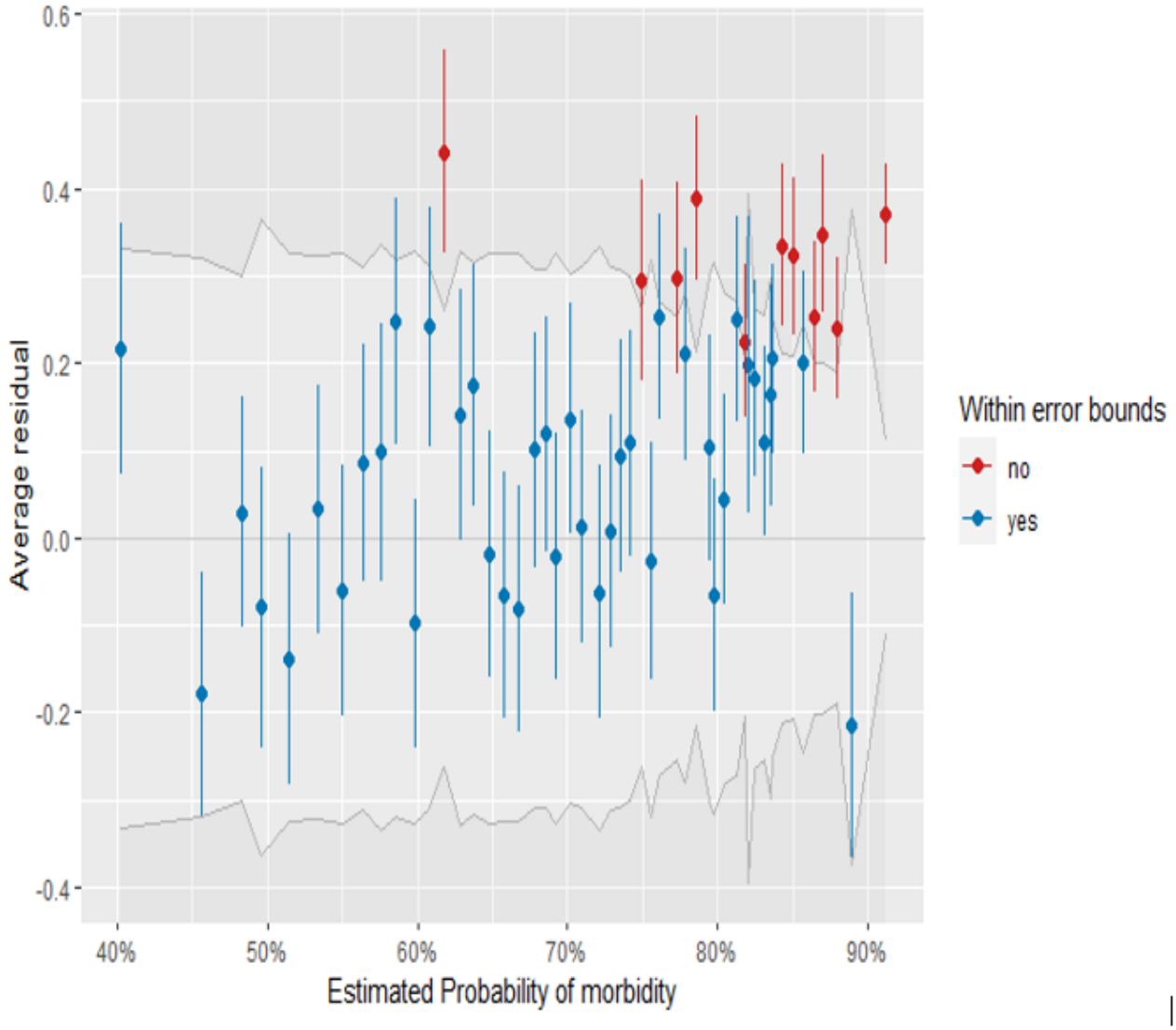


Figure 2.5: Binned residual plot for the children's comorbidity study

Likewise, the DHARMa nonparametric dispersion test evaluates a statistical model's goodness of fit, considering both the fitted model and the simulated values. It is frequently used for count data or other non-Gaussian data. The DHARMa nonparametric dispersion test graph combines a histogram (blue bars) with a kernel density estimate (KDE) plot (red line) which shows how well the model fits the data. The blue bars show the simulated values (perhaps residuals or predicted probability) inside certain bins and each bar's height indicates how frequently the simulated data fall into that category [35, 30]. The frequency (density) is plotted on the y-axis, and the simulated values are plotted on the x-axis between 0.80 and 1.00. In Figure 2.6, the standard deviation of residuals from the fitted model and the simulated values are compared. Therefore, our data exhibits low dispersion and good alignment with the model, as indicated by the p-value of 0.88 (dispersion = 0.99696; p-value = 0.88; alternative hypothesis: two-sided). The high p-value indicates a strong fit.

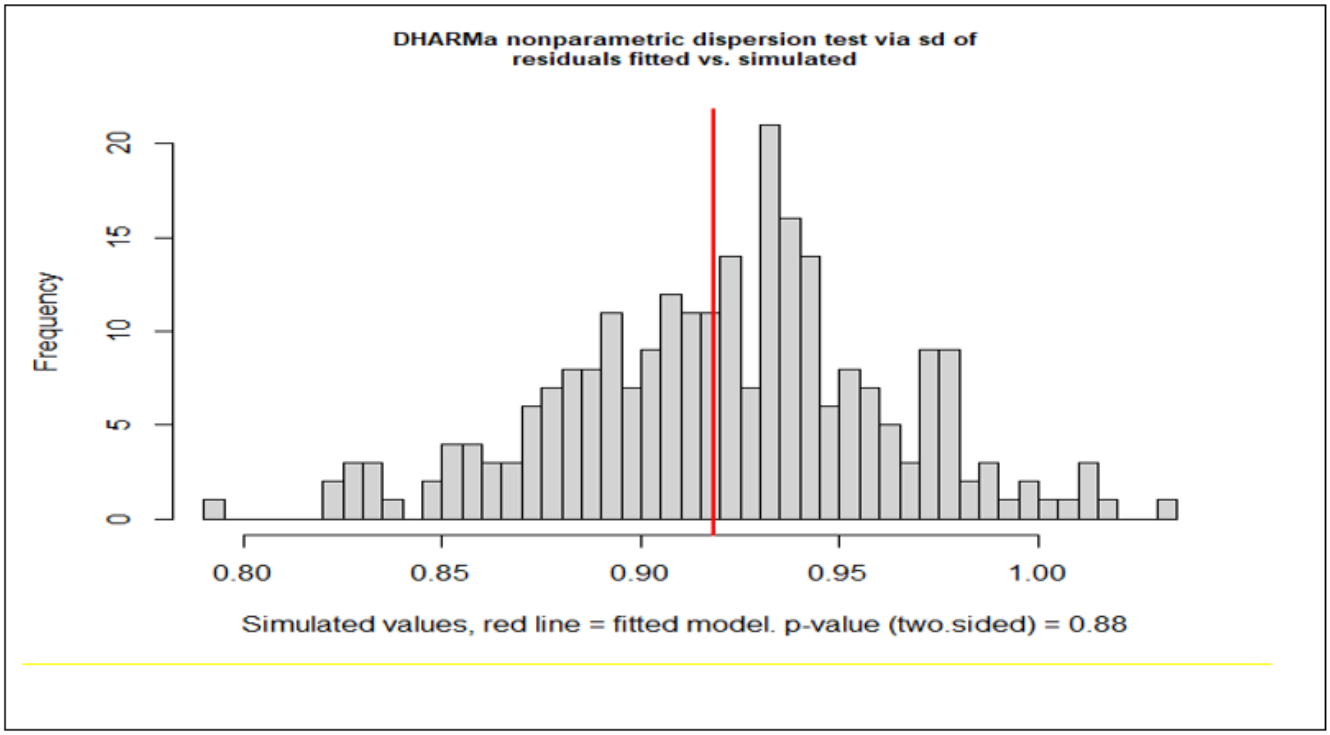


Figure 2.6: DHARMa nonparametric dispersion test with the residuals fitted vs. simulated standard deviation for child comorbidity

Furthermore, in the DHARMa package in R, the QQ plot compares the observed residual to the expected under the assumptions of normality, and the points in the QQ plot fall along a straight line for normally distributed residuals [30, 35]. The plot also displays the Kolmogorov-Smirnov test (KS test), dispersion test, and outlier test [62]. From Figure 2.7, the points on the QQ plot fall along a straight line which indicates that the model can account for the variation in child morbidity, and the model is not systematically overestimating or underestimating child morbidity (see the left of Figure 2.7). Moreover, the insignificant values of the KS test, dispersion test, and outlier test ($P = 0.6764$, $P = 0.88$, $P = 0.82485$, respectively) suggest that the residuals of the model are normally distributed, homoscedasticity variance, and no influential observations in the data. Similarly, the right of Figure 2.7 depicts a plot of the residual against the predicted values. The red solid line at $y = 0.5$ represents the median of the residual, while a dashed red line represents the theoretical median of the residual under the assumption of uniform distribution [35]. Therefore, the two lines are close together at $y = 0.5$ indicating that the residuals are uniformly distributed.

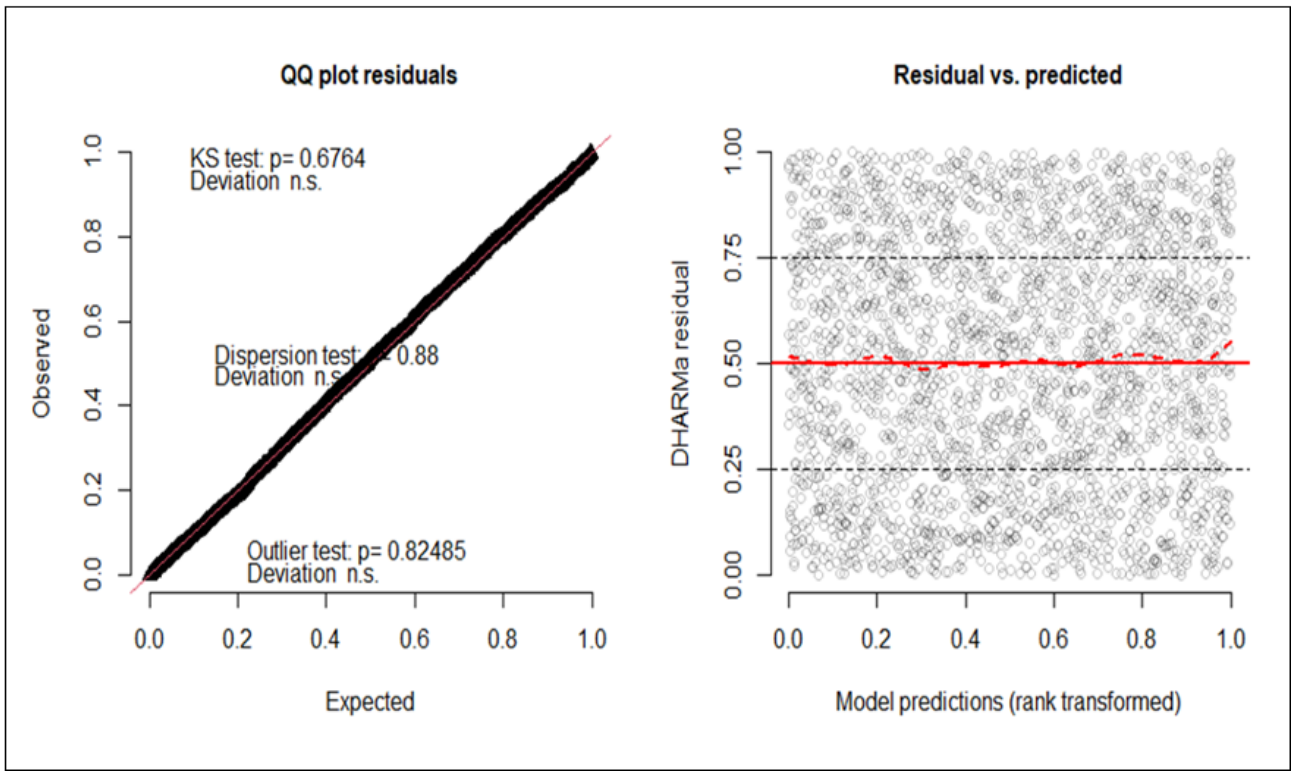


Figure 2.7: DHARMa nonparametric dispersion test with the residuals fitted vs. simulated standard deviation for child comorbidity

2.3.2 Discussion

We tried to check the presence of variability in child morbidity and determine major predictive factors for child morbidity using the GLMMs. We used PMA datasets in STATA-17 and the 4.3.0 version of R for our data analysis. Based on AIC and the likelihood ratio test values, a two-random intercept model was found to be more favorable in illustrating the presence of child morbidity variability between children and within regions. From our study using GLMMs, based on the likelihood chi-square and Type III test, we found that the factors that significantly affect the children's comorbidity were cooking fuel, wealth quartiles, mothers' marital status, mother age, parity, residence mother's education status, and availability of electric city. However, sanitation classification is not influential for the presence of children comorbidity in Ethiopia.

Children from divorced and never-married families are at high risk of suffering illness and experiencing more health problems than children from two-partner families. Like studies carried out [7, 46, 74], our result suggests that a lack of a stable family structure and the absence of one of her or his family members contribute to the negative effects on children's health. Similarly, our findings demonstrated that children with high parity had a higher risk of morbidity than children with low parity, based on PMA-ET datasets. The study found that increased parity is associated with higher odds of child morbidity, and our result is in accordance with [44], and [65] that higher child morbidity is associated with high parity.

Furthermore, the results showed that children who live in rural locations and lack electricity are more likely than their counterparts to experience morbidity difficulty. It demonstrates that living in rural areas and not having access to electricity are positively connected with child morbidity and this result is in accordance with [1, 41, 59]. Moreover, the household wealth index has a negative correlation with morbidity in children and it is a significant socioeconomic determinant influencing children's health in Ethiopia. The lower quartile families had bad nutrition, limited education, poor cleanliness, and poor hygiene. This suggests that compared to children from middle and high quartiles, children from lower households are more likely to experience children's illness. The findings align with those reported by [15, 36], and [69], indicating that an increase in household income is associated with a reduction in the incidence of illness among children.

The results we found also showed a negative correlation between childhood morbidity and the age of the mother. This suggests that children whose mothers were younger than 24 have a higher rate of illness. Our findings support the findings of [39], who noticed that children of mothers 35 years of age and older had lower rates of child morbidity than children of younger mothers. However, our results also contradict those of [56], who found that children of mothers 35 years of age and older had higher rates of child morbidity than children of younger mothers. Another significant risk factor for children's comorbidity is the mother's academic achievement. The risk of morbidity is higher in children whose mothers have not received any education compared to children whose mothers have completed at least primary education. It implies that educated mothers are also more likely to have an income and better access to child health care and have access to information about the health, eating habits, and development of their children, which can enhance the health of their children. These results confirm the results obtained from previous studies [19, 24, 38]. likewise, maternal age is linked to better child health outcomes, especially for mothers with high levels of education Mothers with higher levels of education frequently have increased access to healthcare, are more health-literate, and are more aware of preventative measures. Furthermore, older mothers may make healthier lifestyle choices when pregnant as a result of their experience [28, 38].

In conclusion, according to our result, GLMMs are better suited to handle complex data structures like hierarchical data. This model also offers more precise estimates of random effects on this child comorbidity study to capture heterogeneity and look at how it relates to different variables like socioeconomic status, use of health services, and health outcomes. Cooking fuel, wealth quartiles, mothers' marital status, mother age, parity, residence mother's education status, and availability of electric city were significantly associated with children's morbidity. Improving the socio-economic standings of mothers through socio-economic and education reduces the prevalence of child morbidity.

In our study, the utilization of general linear mixed models (GLMMs) possess the remarkable capacity to delve deeper into the complexities inherent within hierarchical data

structures, where individual children are nested within specific regions. By leveraging the power of GLMMs, researchers gain the ability to conduct more comprehensive analyses that simultaneously account for both individual-level and group-level factors influencing child development. This enhanced analytical approach allows researchers to not only identify significant associations within the data but also to elucidate the potential presence of regional variations in child development across Ethiopia.

Our analysis considered several variables, but factors like health insurance, access to healthcare, and family structure might also significantly influence children's comorbidity. Examining these influences through future empirical research could be valuable. Additionally, a longitudinal study could be particularly interesting to see how children's comorbidity patterns change over time.

2.3.3 Limitations of the study

Although Ethiopia has nine regional states (Afar, Amhara, Benshangul-Gumuz, Gambela, Harari, Oromia, Somali, Southern Nations, Nationalities, and Peoples' Region (SNNPR), and Tigray) and two administrative cities (Addis Ababa and Dire Dawa), the PMA-Et 2019 dataset includes information from only six regions. This limitation in the data presents challenges for conducting a comprehensive comorbidity study across the entire country. The findings derived from the six regions may not be entirely representative of the national context, thereby complicating our ability to draw generalized conclusions and formulate comprehensive recommendations. The exclusion of data from five regions means that critical sociodemographic and environmental variables unique to these areas are not reflected in the study. This geographical limitation could result in an incomplete understanding of the factors affecting child comorbidity across Ethiopia. Furthermore, because longitudinal datasets were lacking, we were unable to stabilize throughout childhood, even though longitudinal studies provide a distinct advantage in understanding childhood morbidity by monitoring changes in health over time, identifying early predictors, and establishing causal relationships between factors and health outcomes.

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Chapter 3

Bayesian semiparametric geoadditive modelling of underweight among under-five children in Ethiopia

Abstract

Children's malnutrition can have long-term and irreversible effects on a child's health and development. This study uses the Bayesian method with spatial variation to investigate the flexible trends of metrical covariates and identify communities at high risk of injury. Cross-sectional data on underweight were collected from the 2016 Ethiopian Demographic and Health Survey (EDHS). The Bayesian geoadditive model is performed. Appropriate prior distributions were provided for scall parameters in the models, and the inference is entirely Bayesian, using Monte Carlo Markov Chain (MCMC) simulation. The results show that metrical covariates like mother age, child age, and body mass index (BMI) affect a child's underweight non-linearly. Lower and higher maternal BMIs seem to have a significant impact on the children underweight. There was also significant spatial heterogeneity and based on IDW interpolation of predictive values, the western, central, and eastern parts of the country are hotspot areas. Our analysis supports the flexible modeling of mother age, child age, and body mass index (BMI) of the mother. In addition to fixed effects and covariates, there is also considerable evidence of a residual influence on underweight.

KEYWORDS:
Spatial distribution; Underweight; Semi-parametric Bayesian analysis; P- splines; BayesX; MCMC; Ethiopia.

3.1 Introduction

The state of malnourished is characterized by an imbalance between the intake and need of nutrients, which leads to cumulative shortfalls of energy, protein, or micronutrients. These deficiencies can have adverse effects on several outcomes, including growth and development [58, 78]. It is often used synonymously with 'undernutrition'. For children under five, malnutrition continues to be a leading cause of illness and death, especially in developing countries [52, 65]. Malnourishment, especially throughout a child's first two years of life, causes nearly irreversible harm to their mental and physical health, poor school performance, decreased future income, frequent illness, and poor cognitive development. It also traps them in a never-ending cycle of disease [45, 41].

According to [73], malnutrition is the largest global health opportunity forfeited, causing serious risks to children's health, particularly in low- and middle-income countries and one of the biggest threats to world health is combating it in all its manifestations. The Global Strategy for Children's Health, to meet the need to eradicate malnutrition, diet-related goals from the 2030 Agenda for Sustainable Development and the Goal Action Plan for the Prevention and Control of Noncommunicable Diseases must be accomplished [55]. Severe acute malnutrition is the most severe form of malnutrition that ends many early childhood lives in countries facing nutrition crises, including Afghanistan, Somalia, Ethiopia, Kenya, Burkina Faso, Mali, Niger, and Yemen [51, 22]. Hence, improvements in the nutritional status of young children require a broad range of nutrition and health interventions [28], and understanding the factors that can lead to malnutrition also guides possible interventions by governments and development partners.

Based on the World Health Organization (WHO) Child Growth Standard, the three manifest indices of undernutrition are wasting, stunting, and underweight [72]. Stunting, which results from chronic or recurrent undernutrition, is characterized as low height for age, whereas wasting is described as low weight for height (severe weight loss). Furthermore, an underweight child may be wasted, stunted, or both. Underweight is described as having a low weight for age [72]. The Z-scores, which show how many standard deviations a child's anthropometric index deviates from the median of the global growth reference population argued by the World Health Organization, are used to quantify these three anthropometric characteristics [89].

For a child i^{th}, the Z-scores (Z_i) for each anthropometric variable (e.g., the weight-for-age) values are defined as follows:

$$Z_i = \frac{AI_i - MI}{\sigma} \tag{3.1}$$

Where: Z_i represents the Z-score for the i^{th} child. AI_i denotes an individual anthropometric characteristic (such as weight at a certain age). (MI) stands for the reference population's median. σ represents the standard deviation.

The World Health Organization (WHO) Child Growth Standard (WCGSM) defines stunted children as those whose height-for-age z-score (HAZ) is less than the negative two-standard deviation (-2SD) from the median. A weight-for-age z-score (WAZ) of lower than two standard deviations (2SD) from the reference median indicates underweight status as well [72]. While both stunting and wasting are indicators of malnutrition, being underweight is often used as a broader measure of overall malnutrition because it considers both chronic and acute forms of malnutrition. It refers to a child who has a low weight for their age, which can be caused by both chronic and acute malnutrition [6]. Underweight children are at risk of stunting, wasting, and other health problems associated with malnutrition [70]. As well, weight can be measured more easily than height or body composition, and weight changes can be observed more quickly [64]. Therefore, underweight has been more widely used in surveys and studies aimed at assessing the prevalence of malnutrition in children [80], and this most visible and immediate recognition of underweight for malnutrition dedicated us to investigate it in our study rather than stunting and wasting.

Approximately 45 percent of child deaths under five globally are caused by undernutrition. It remains an epidemic in many developing countries, especially in sub-Saharan Africa [72, 5]. The prevalence of undernourishment in Ethiopia was 28.8 percent in 2015, and the country's prevalence is still higher than that in the region [1, 5]. Moreover, [75] state that malnutrition is the primary cause of underweight. It was estimated that there would be 101 million underweight children under five (16%) in the globe in 2011. Of those, 26.6% would be found in Africa. Additionally, 24% of Ethiopian children were underweight, according to the 2016 Ethiopian Demographic and Health Survey. Even though the past 15 years' chronic malnutrition trends indicate an improvement, 28 percent of child deaths in Ethiopia are associated with undernutrition [84]. One of the sustainable development objectives in the post-2015 development agenda is Goal 2, which is based on the idea of ending hunger. Despite this, target 2.2 of malnutrition—which aims to end all forms of malnutrition, including achieving targets on stunting, underweight, and wasting in children under the age of five, as well as address the nutritional needs of adolescent girls, pregnant and lactating women, and older persons—has not yet reached the end values for 2030 [68]. This high prevalence is an indication for us to investigate possible factors that could affect children underweight with a suitable statistical methodology yet.

There are many more causes of malnutrition than only dietary deficiencies. Poverty, political upheaval, altered weather patterns, dietary practices, sickness, the COVID-19 pandemic's effects on markets, services, and human movement, contaminated water supplies, poor sanitation, and a host of other complicated challenges are all part of the illustration [49]. Underweight malnutrition of under-five children in Ethiopia could be attributed to many different factors [65, 78, 52, 48]. Researchers also realized a significant correlation between the prevalence of under-five malnutrition in communities and metrical covariates like mother age, child age, and other body mass index. None of

these seems reassuring in terms of the nonlinear (flexible) influence of metrical variables on underweight. Furthermore, it was discovered that sociodemographic characteristics and geographic location were also strongly linked to child malnutrition For this reason, nutritional interventions need to be carefully tailored to the residential location of the patients [48, 52] Favorable socioeconomic circumstances contribute to a decrease in urban malnutrition, which in turn results in improved child and mother care practices [78].

Based on the different pieces of literature that have been done on malnutrition before, most of these researchers use a frequentist approach (e.g., generalized linear models (GLMs) and other forms of regression) to determine the associated factors for underweight [50, 89].In consequence, the frequentist approach solely relies on the data to make statistical inferences and neglects prior knowledge about the parameter, which can make more informed predictions about the parameter value than would be possible based on the data alone [86]. According to [86], Bayesian inference works better than frequentist inference because it allows prior experience and expert opinion to be used in formulating a prior distribution. It eschews many of the difficulties encountered with classical inference and is more directly predicated on what one is interested in [34]. Bayesian regression allows for more flexible model specification, including the use of non-linear functions and more complex models that can capture interactions between variables. This flexibility can help to improve the accuracy of the model and provide better insights into the underlying relationships between the variables [40] and a powerful approach to disease mapping [59].

Given the literature we deal with, there are also few works carried out on Bayesian-Gaussian regression analysis of malnutrition. Even though [67, 7, 83] conducted a Bayesian-Gaussian regression analysis of malnutrition in Ethiopia using EDHS data, they did not consider geographical and sociodemographic effects of undernourishment among children simultaneously. Most of the earlier research on malnutrition [4, 33, 61, 69] relied on a frequentist approach for socio-demographic variables and assumed a linear relationship between the socio-economic variables (particularly the metrical covariates) and the outcome of interest. These studies were not flexible enough, and they neglected to simultaneously estimate the geographic association with underweight and the nonlinear effect of some covariates.

However, in the Bayesian semiparametric model, more adaptable additive predictors are employed instead of conventional linear predictors. The added flexibility enables the estimation of nonparametric effects related to metrical variables and spatial effects simultaneously. The significance of this approach is found in its ability to overcome a limitation of parametric models, which calls for strong assumptions on the functional structure of any nonlinear effects that are related to metrical variables [30, 57]. The Bayesian geoadditive model, which fully embraces the Bayesian method and relies on smoothness priors, has several advantages over other statistical models. First, it provides a principal way to incorporate prior knowledge or beliefs into the analysis. Second, it

allows for the propagation of uncertainty from the parameters to the prediction. Third, it provides a way to quantify the evidence in favor of different models or hypotheses [88].

Thus, in this study, we looked into the application of the Bayesian approach to geo-additive regression to analyze the complex factors contributing to underweight malnutrition in children under five in Ethiopia based on the 2016 EDHS database. Unlike previous studies that predominantly employed a frequentist approach, our research incorporates more flexible and adaptive predictors, allowing for a simultaneous examination of geographic and sociodemographic effects within the Ethiopian context. Hence, the present study intends to analyze the spatial distribution at more localized units and also illustrate that assuming a linear effect for metrical covariates is always too rigid and can result in misleading findings in analysing health indicators.. Additionally, the Bayesian geoadditive model enhances the analysis by integrating prior knowledge and expert opinions, providing a robust framework for understanding the multifaceted causes of malnutrition. The smooth function captures the nonlinear relationship between the continuous covariates (metrical covariates) and the response variable, modeled by using a P-spline, and the spatial effect accounts for the spatial correlation, modeled using a Gaussian process [21, 46, 57]. The inference is performed using full Bayesian inference and efficient Markov chain Monte Carlo techniques (MCMC) techniques. The BayesX package in the R programming language is used for the analysis. For better visualization of the nonlinearity of metrical covariates on underweight, we use the Yeo-Johnson transformation, a power transformation method used in statistics to normalize data that may not follow a normal distribution, to improve the accuracy and reliability of models, especially when dealing with non-linear models [30]. Moreover, for the model fit comparison, we employed the deviation information criterion [79]. Overall, the Bayesian approach enhances our understanding of Ethiopia's patterns of malnutrition by combining statistical rigor with geographical context, and it also guides policymakers and stakeholder groups looking for solutions and effective nutritional interventions in the country

3.2 Materials and Methods

3.2.1 Study variables, data sources, and geography of Ethiopia

The study was conducted in Ethiopia using the 2016 Ethiopian Demographic and Health Survey data. EDHS 2016 was conducted from January 18, 2016, to June 27, 2016, based on a nationally representative sample that provides estimates at the national and regional levels as well as includes urban and rural areas. EDHS 2016 contains detailed information on the background characteristics of the respondents, fertility, marriage, and sexual activity, awareness, use of family planning methods, child feeding practices, nutritional status of women and children, and adult and childhood mortality. We receive permission to download EDHS 2016 data from https://dhsprogram.com/Data/ after making a

reasonable request.

in EDHS 2016, valid geographic coordinates, sociodemographic, and anthropometric data were collected. In each selected household, mothers aged 15 to 49 were interviewed, and anthropometric measurements were taken on all children under the age of five in any family. 10,641 children with full anthropometric measures of underweight are included in 645 clusters from 11 regions. A report on the comprehensive methodology of the 2016 EDHS survey could have been found elsewhere [29]. Our study aims to develop a comprehensive model that considers statistical uncertainty and the geographical setting to better understand and manage underweight issues in early infants in Ethiopia. We employed flexible regression approaches to predict the effects of several factors of underweight. The geographic variance based on the child's place of residency was also considered, and the results indicate both nonlinear and linear relationships between these variables. Underweight status was assessed using standard Z-scores.

Malnutrition in children can result from several causes. Based on earlier research, we embarked on our study by looking at a wide range of covariates, including socioeconomic, demographic, health, and environmental characteristics of childhood malnutrition (see Table 3.1). One typical method in statistical modeling is to classify variables using deviation codes and it provides insights into the effects of categorical variables and makes it easier to include them in our models. Furthermore, one of the levels of the categorical variable is chosen as a baseline level, and the remaining levels are coded as the deviation from the baseline level [16]. Furthermore, to increase the quantity of data that was available, underweight was employed as a continuous variable.

Ethiopia is a federal republic consisting of nine regional cities (Afar, Amhara, Benshangul-Gumuz, Gambela, Harari, Oromia, Somalia, South African nationalities, and people (SNNP), Tigray), and two administrative towns (Addis Ababa and Dire Dawa). The capital city of the federal territory is Addis Ababa. The country, which has a total land area of 1,104,300 km^2 (426,372 m^3), is in the Horn of Africa. Its longitude ranges from 33° to 48°, while its latitude is between 3° and 14.8°. The locations of each region are shown in Figure 1 for those unfamiliar with Ethiopia's terrain [29].

Table 3.1: Socioeconomic and Demographic Characteristics of childhood underweight in Ethiopia

Covariates	**Description**
Child's age (Cage_month)	Child's age in months
Region	Region where mother lives
Mother_BMI	Mother's Body Mass Index (BMI)
Mother's age (Mother_age)	Age of mother in years during childbearing age
Mother education	Mother's education level (categories: no education, primary, secondary, higher)
Child's sex	Child's sex (categories: male, female)
Availability of electricity	Availability of electricity (categories: yes, no)
Sex of household headed	Sex of the household head (categories: male, female)
Diarrhea level	Child's diarrhea level (categories: yes, no)
Anaemia level	Anaemia level of child (categories: severe, moderate, mild, not anemia)
Place of residence	Place of residence (categories: urban, rural)

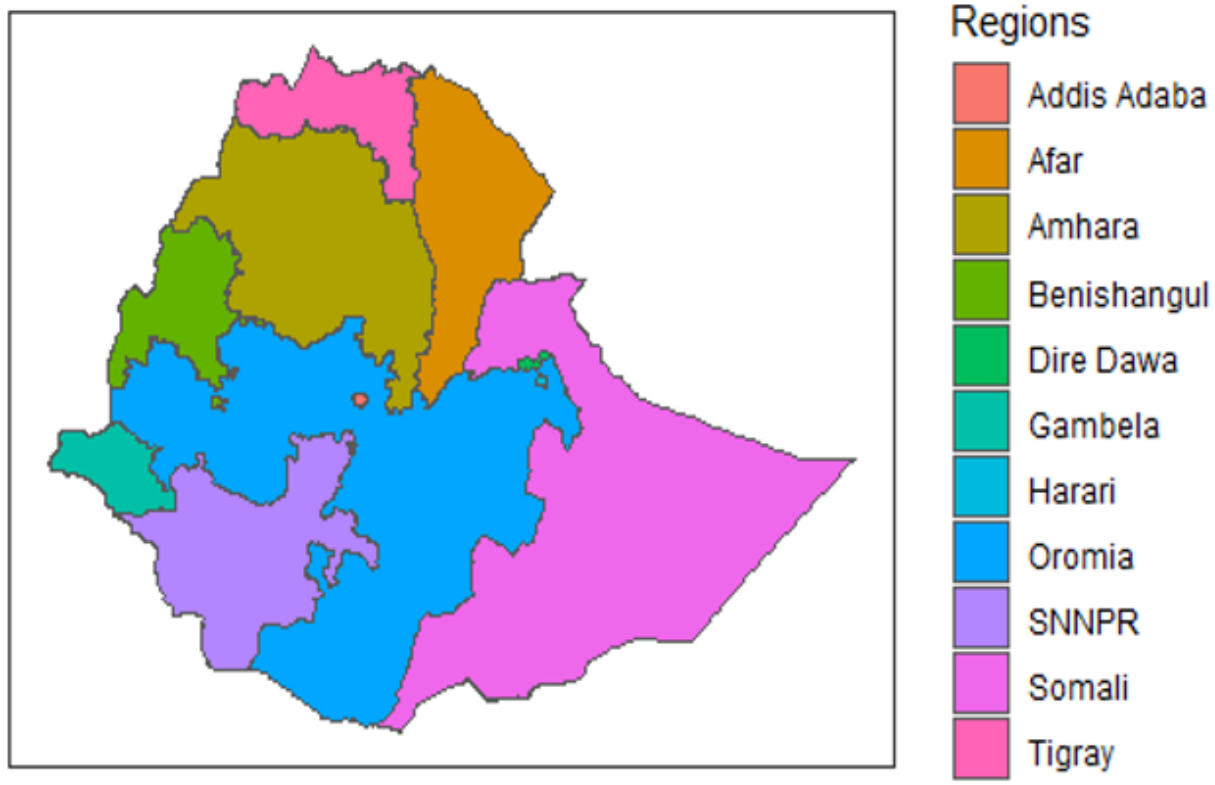

Figure 3.1: Maps of Ethiopia with its Regions

3.2.2 Bayesian Geo-additive Models

Bayesian geoadditive hierarchical models can combine Bayesian inference with geostatistical methods to model spatially varying relationships between a set of variables and a response variable [11, 25]. In this model, all unknown parameters are treated as random variables, and prior distributions are assigned to them based on available prior knowledge or beliefs. Then, given the data set, the posterior probability distribution of the parameter is updated using Bayes theorem [30], More settings for geoadditive models within mixed models have been introduced by [46]. In addition, one of the main fields of statistical research for modeling the non-linear components of a Generalized additive model (GAM) is Bayesian geoadditive models. These models are required nowadays for a variety of applications that can handle both nonlinear spatial effects and the nonlinear effects of continuous covariates at the same time.[91].

Conventionally, the impact of sociodemographic variables on the response variable is modeled using the linear model as follows:

$$\eta_i = X_i\beta + \omega_i\gamma \tag{3.2}$$

With a vector of categorical variables, $\omega = (\omega_i, \ldots, \omega_p)$, and metrical covariates, $X = (x_i, \ldots, x_p)$, for each of the values of $i = 1, \ldots, 10641$. The nonlinear impacts of metrical factors and categorical covariates on underweight childhood malnutrition were considered in our analytical investigation, and the categorical variables were coded by deviation coding.

Thus, for a set of observations (y_i, x_i, v_i), $i = 1, \ldots, n$, on a continuous response y, a vector of continuous covariates (or metrical covariates) $x = (x_1, \ldots, x_p)$, and (v_i) is a vector of further variables associated with each observation and can be expressed as a vector of additional covariates, $\omega = (\omega_1, \ldots, \omega_p)$, the generalized additive model (GAM) for the cross-sectional data, we have an additive predictor η_i for observation i, $i = 1, \ldots, n$, and $j = 1, \ldots, p$ component is given by [43]:

$$\eta_i = f_1(x_{i1}) + \ldots + f_p(x_{ip}) + \omega_i^T\gamma \tag{3.3}$$

Here, the linear combination $\boldsymbol{\omega}_i'\boldsymbol{\gamma}$ corresponds to the typical parametric part of the predictor, including the intercept term. The $f_j(x_{ij})$ is a smooth function of the j^{th} covariates of the (x_{ij}), and $f_1 \cdots f_p$ are unknown smooth p^{th} degree polynomial function of the continuous covariates. We suppose that, given the covariates and unknown parameters, y_i is the Gaussian family distribution, with a common variance σ^2 for all individuals for $i, 1, \ldots,$ 10641. For different function evaluations, the unknown function f_j in equation (2) is represented as $f_j = (f_1(x_{i1}) + \cdots + f_p(x_{ip}))' = \mathbf{X}_j\boldsymbol{\beta}_j$, where $\mathbf{X}_j$ is a design matrix, and vectors of unknown regression coefficients $\boldsymbol{\beta}_j$ can be expressed as such.

$$f_j = (f_1(x_{i1}) + \cdots + f_p(x_{ip}))' = \mathbf{X}_j\boldsymbol{\beta}_j \tag{3.4}$$

From Equation (3), f_j represented a metrical product of a deterministic, non-random design matrix $\mathbf{X}_j$, and a vector of unknown regression parameters $\boldsymbol{\beta}_j$. The unknown function $\mathbf{f} = (f_1, \cdots, f_p)$ denotes the evaluation of the function at observed values of $\mathbf{x}$.

By defining the $n \times M$ design matrix $\mathbf{X}$ with n observations and M covariates, where the element in row i and column p is given by $X_{i,p} = (x_{ij})$, the matrix notation of equation (2) can be rewritten as:

$$\eta_i = x_1\beta_1 + \ldots + x_p\beta_p + \omega_i^T\gamma \tag{3.5}$$

In this case, $\beta_j = \left(\beta_{j1} \ldots \beta_{jm_j}\right)^T$ gives the representation of the unknown regression coefficient vectors, and ω is the matrix for the fixed effects design matrix. Since they are random variables, the unknown parameters for $\beta_j, j = 1 \ldots p$ and γ must be supplied with the proper prior distributions[63]. The means of the unknown functions $f_1 \ldots f_p$ are not identifiable for the pth degree polynomial smooth functions of continuous covariates. Constraining the mean values of smooth functions is necessary for robust statistical modeling to maintain identifiability [3], i.e

$$1/(range(x_1)) = \int_{x_j} f_1(x_j)\, dx_j = 0 \tag{3.6}$$

Throughout each sampler iteration, we center the functions f_j about their means in Bayesian estimation using Markov Chine Monte Carlo (MCMC) to preserve identifiability while estimating the unknown parameters. We then add the subtracted means to the intercept term ($\omega_i^T\gamma$ [95].

To account for the spatial variation in response, we introduce a spatial effect denoted as f_{spat} into equation (2). This modification leads to the development of geoadditive models, as proposed by [46]. In these models, we simultaneously consider the nonlinear effects of metrical, categorical, and spatial covariates (specifically the child's regions of residence) in assessing underweight in childhood. Therefore, by replacing the strictly linear predictive equation (2) with a more flexible geoadditive model, we arrive at the general form of the Bayesian geoadditive model as:

$$\eta_i = f_1(x_{i1}) + \ldots + f_1(x_{ip}) + f_{\text{spat}}(s_i) + \omega_i^T\gamma \tag{3.7}$$

Centered on its application, the spacial effect may be further split into an uncorrelated (unstructured) and a spatially correlated (structured) effect [46] as:

$$f_{\text{spat}} = f_{\text{str}} + f_{\text{unstr}} = X_{\text{str}}\beta_{\text{str}} + X_{\text{unstr}}\beta_{\text{unstr}} \tag{3.8}$$

The function f_{spat} represents the geographical effects of spatial variables $s \in \{1, \ldots, S\}$

indicating regions in a country. The rationale behind for incorporation of the spatial effect functions was to surrogate of several unobserved impacting variables associated with geographic data. While some of these components have a significant spatial structure, others are more localized. To separate these two categories of relevant elements, we estimate the structure and unstructured effect. Using Markov random field priors for the regression coefficients is a typical method for simulating the linked spatial impact f_{str} when working with data seen on an irregular or regular lattice [9].

Prior distribution

The prior distribution in Bayesian geoadditive models serves as a regularization and a mechanism to incorporate prior knowledge or beliefs about the model parameters, which can improve the model's estimation and prediction performance [30]. The unknown parameters, $f_1 \dots f_p$, f_{str}, f_{unstr}, and an uncertain parameter γ and δ^2 are regarded as random variables, hence the proper prior distributions must be added to them[63]. This prior distribution represents information about $f_1 \dots f_p, f_{\text{str}}$. The posterior distribution is then used to guide subsequent conclusions by combining the prior distribution with the probability distribution of the new data. Deciding an appropriate prior distribution is the main issue for a particular application, and for any scenario, there is a prior distribution that is justified by notions from decision theory [35].

Priori for metrical covariatesn

Specifying smoothness priors is important to avoid overfitting and improve the accuracy and interpretability of statistical models. It encourages the model to fit the data in a way that is consistent with prior knowledge or beliefs about the relationship between the covariates and outcome [32].

A range of alternatives have been put out to defining smoothness prior to metrical variables, including autoregressive priors (random walk priors), Bayesian P-splines, and the Bayesian smoothing splines [30, 57, 42]. From these methods of smoothness prior specification, Bayesian P-splines (the P-splines) are a powerful tool for nonparametric regression analysis that offer advantages over other Bayesian methods such as Bayesian smoothing splines and random walk priors in terms of computational efficiency, flexibility, and interpretability [14, 57]. Then, we will focus on P-splines, the most parsimonious parameterization in a Bayesian framework where inference is based on MCMC techniques.

Based on the definition of splines of degree l on the set of knots with equal spacing, the P-splines assume that the unknown smooth function f_j of the metrical variables x_j may be estimated as $\zeta_j \in [\zeta_0, \zeta_r]$ in the domain of x_j, i.e. $(x_{j\min} = \zeta_{j0} < \zeta_{j1} < \ldots < \zeta_{(jr_j-1)} < \zeta_{(jr_j)} = x_{j\max})$. Furthermore, the P-spline or penalized splines assume that the influence of a covariate x may roughly be represented by a polynomial spline expressed as a linear combination of the B-spline basis function. Subsequently, such a spline may be expressed as a linear combination of $M_j = r_j + l$ B-spline basis functions β_{jp}[21].

$$f_j(x_j) = \sum_{t=1}^{m} \beta_{jp}\beta_{jp}(x_j) \tag{3.9}$$

The locally specified basis functions β_{jp} are nonzero only in the domain encompassed by $2 + l$ knots. We take the number of knots in each notation to be the same, $M = M_j$, for each function f_j.

The choice of the number of knots in a P-spline is an important decision that can have a significant impact on the resulting model's performance. If the number of knots is too small, the P-spline may not capture the underlying structure of the data adequately, resulting in underfitting or bias in the model. However, if there are too many knots, the P-spline may overfit the data, resulting in a model that performs well on the training set but poorly on new, unproven data [77]. The knots divide the range of the input variable into a series of intervals, and the spline function is defined by a set of basis functions that are used to model the data within each interval [26] and the number of knots in the P-splines fitting process should be between 20 and 40. This reasonable number of knots is used to guarantee sufficient smoothness of the fitted curve by defining a roughness penalty and overcoming the challenge of regression splines based on first or second-order differences of neighboring B-spline coefficients. Furthermore, knots may be automatically calculated throughout the fitting process, negating the need for the user to specify them directly.

From Equation (4), the unknown regression coefficients Coefficients $\beta_j = \left(\beta_{j1} \dots \beta_{m_j}\right)^T$ and γ can be estimated by penalized likelihood.

$$L = l(y, \beta_1 \dots \beta_p, \gamma) - \lambda_1 \sum_{m=k+1}^{m_j} \left(\Delta^k \beta_1 l\right)^2 - \dots - \lambda_p \sum_{m=k+1}^{m_j} \left(\Delta^k \beta_p l\right)^2 \tag{3.10}$$

.

From this equation, $\lambda_j, \quad j = 1, \dots, p$ is the smoothing parameter, a trend of smoothness and flexibility, and Δ^k is the difference operator of order k. This function should be maximized for unknown regression coefficients $\beta_1 \dots \beta_p$ and γ to find the unknown regression coefficients. However, it becomes challenging to find an optimal solution when the model has a large number of smoothness functions [56].

For a given effect parameter γ, we assume an independent diffusion prior, and for $j = 1, \dots, p$, we assume that $\gamma_j \propto$ constant. Under the assumption of distinct diffusion priors, we allow for independent parameter development, which may lead to more flexible and understandable models [96]. The characterization of priors for the nonlinear function's regression parameter β_j involves substituting the various penalties with their stochastic equivalent in Equation (8). We employ stochastic difference penalties, such as first- or second-order random walks, as priors for regression coefficients in Bayesian modeling. The first-order difference, $\beta_{jm} - \beta_{(j,m-1)}$, penalizes abrupt jumps between consecutive

parameters; the second-order difference, $2\beta_{jm} - \beta_{(j,m-2)}$, penalizes deviations from the linear trend. These random walks give modeling flexibility, and they are specified as:

$$\beta_{jm} = \beta_{(j,m-1)} + u_{jm} \quad \text{or} \quad \beta_{(j,m-1)} = 2\beta_{(j,m-1)} - \beta_{(j,m-2)} + u_{(j,m)} \tag{3.11}$$

Where $p(\beta_{j1})$, $p(\beta_{j2})$ are diffusion errors, u_{jm} is the Gaussian error, which follows $\sim N(0, \tau^2)$, and the α constant denotes for initial values. As a specific situation, first and second random walk processes may be thought of as P-spline degree $l = 0$ [30]. As a product of the conditional densities, the joint distribution of the regression parameter may be simply determined. Thus, in equation (4), the parameter vector $\boldsymbol{\beta}$'s prior distribution may thus be expressed in terms of globally smoothness priors as:

$$P(\beta_j|\tau_j^2) \propto \exp\left(-\frac{1}{2\tau_j^2}\beta_j^T K_j \beta_j\right) \tag{3.12}$$

Where the penalty matrix K_j is used in regularization techniques such as ridge regression and smoothness priors. In many instances, the values of k_j tend to fall short of the expected rank, resulting in partially inappropriate priorities for β_j. This suggests that $\beta_j|\tau_j^2$ adheres to a certain improper Gaussian prior, $\beta_j|\tau_j^2 \sim N(0, \tau^2 K^-)$, where the generalized inverse of the penalty matrix K is denoted by K^-. The inverse smoothing parameter, or variance parameter τ_j^2, regulates the trade-off between the smoothness and flexibility parameters. Smoother solutions are encouraged by the term $\beta_j^T K_j \beta_j$, which penalizes the complexity of the model [23].

We employ Gamma distribution hyperpriors for variance τ_j^2 in complete Bayesian inference. These hyperpriors are weakly informative, so we may keep our flexibility while including prior information. Specifically, we assume that τ_j^2 will have an inverse Gamma distribution with parameters α_j and b_j: $\tau_j^2 \sim \text{IG}(\alpha_j, b_j)$. The normalization's impact is minor when $\alpha_j = b_j$ is set to its default value of 0.001. This decision integrates the effect of the prior with the likelihood of the data, ensuring that the distribution of posteriors incorporates information from both sources. In Empirical Bayes Inference, we use τ_j^2 as an unknown constant instead of defining a hyperprior. The value of τ_j^2 is extracted directly from the data using methods such as restricted maximum likelihood (REML). A more data-driven approach to computing variance is provided by REML estimates, which also take the uncertainty in the fixed effects into account. The penalty matrix K_j is used in regularization techniques such as ridge regression and smoothness priors. $K_j = D^T D$ is the formula for K_j, in which D represents the difference matrix of first or second order. This matrix captures the smoothness or sparsity assumptions on the parameters [36].

Prior Dispersion for the spatial effect

In geoadditive models, the prior can be used to incorporate spatial dependence into the model parameters, where neighboring regions are assumed to have similar effects. The

spatial prior can be based on distance, adjacency, or any other form of spatial relationship, and can help improve the model's estimation accuracy and prediction performance [46]. Prior to the spatial effect of Bayesian spatial analysis, which is based on the Markov random field (MRF), the spatial effect at each location depends on the spatial effects at neighboring locations. Moreover, the advantage of the Markov random field is its flexibility in modeling spatial dependence. The MRF prior is specified using a neighborhood structure, which describes the relationships between neighboring locations. The prior assumes that the spatial effects at neighboring locations are dependent on each other and that the dependence decays with distance. The strength of the dependence is controlled by a spatial parameter, which can be estimated from the data [20].

We selected Markov random field priors Markov random field priors (MRFs) for spatially correlated effects, $f_{\text{str}}(s), \quad s \in \{1, \ldots, S\}$, These priors reflect spatial neighborhood relationships and represent the probability distributions of a variable modeled as a product of local conditional probabilities that depend only on the values of neighboring variables [60]. With spatial analysis and modeling techniques, including spatial clustering, spatial autocorrelation analysis, and spatial regression, two areas, r, and s, are considered neighbors if they have a common border. This is based on the idea that neighboring regions tend to have more similar characteristics and may interact more frequently than non-neighboring regions [39].

Every observation in the random walk model is a function of the one before it and the random error term. However, in a spatial context, observations that are close to each other are likely to be correlated due to spatial dependence. Hence, previous observations and random error terms may not be sufficient to explain the current observation. To account for spatial dependence, the conditional and spatial autoregressive specifications include spatial lag terms in the model. These spatial lag terms capture the influence of neighboring observations on the current observation, and this specification allows for the spatial dependency to be modeled, which can improve the accuracy of the model prediction [24].

The model can be written be as:

$$f_{\text{str}(s)}/f_{\text{unstr}(r)}, r \neq s, \tau^2 \sim N\left(\sum_{r \in \partial s} \frac{f_{\text{str}(s)}}{N_s}, \frac{\tau^2}{N_s}\right) \tag{3.13}$$

Where the conditional means, $f_{\text{str}(s)}$ are an average of the functional evaluation $f_{\text{str}(s)}$ in neighboring regions, τ^2 is the variance that determines the degree of smoothness, and N_s is the sum of adjacent sites. $r \in \partial s$ represents the set of neighbors of site s.

The Gaussian distribution with independent and identical distribution (iid) is a common prior assumption for spatially uncorrelated effects f_{unstr}, because Gaussian (iid) is more flexible and a more popular choice for modeling uncorrelated data [57]. With spatially uncorrelated effects, the model can be written as:

$$f_{\text{unstr}}|\tau^2_{\text{unstr}} \sim N(0, \tau^2_{\text{unstr}}) \tag{3.14}$$

For $j = 1, \ldots, p$, str, and unstr, the variance and smoothness parameter for complete Bayesian inference τ_j^2 are unknowns that are estimated concurrently with the corresponding unknown function f_j. As a result, at the second level of the hierarchy, they are given hyperpriors using a dispersed inverse gamma distribution, $P(\tau^2) \sim \text{IG}(a_j, b_j)$ with known hyperparameters. The conventional values of these hyperparameters are $b = 0.005$ and $a = 1$ or $a = 0.001 = b$, which is close to Jeffrey's noninformative prior [81].

Posterior inferences

The posterior inference in fully Bayesian models, such as Bayesian geo-additive models, is usually carried out through the use of Monte Carlo Markov Chain (MCMC) techniques, which provide a sample based on the posterior distribution [8]. Using Monte Carlo Markov Chain (MCMC) simulations techniques, we may utilize the posterior distribution of the parameter of interest to compute a credible interval in Bayesian geoadditive regression models. The credible interval in Bayesian geoadditive regression models provides a range of values for a model parameter, such as a regression coefficient or a variance component, that is likely to contain the true value of the parameter with a certain degree of probability, typically expressed as a percentage [40]. These credible intervals rely on the posterior distribution of the parameter which considers both the observed data and any prior information [18].

In MCMC sampling, we can compute the statistical properties of a posterior distribution as long as we have a sufficient number of simulated samples from that distribution. i.e.

$$E(f_j)p = \frac{1}{N}\sum_{i=1}^{N}(f_j)^{(i)} \tag{3.15}$$

In this case, f_j is the intended expectation, P is the posterior likelihood distribution of desire, and $(f_j)^{(i)}$ is the ith simulated sample from P

The stationary distribution of the Markov chain is the goal of the posterior distribution in these MCMC techniques, which are iterative algorithms that thrive in a Markov chain. The chain converges to a target distribution once a sufficient number of iterations of the method are performed, and the sample that results may be used to estimate posterior summaries such as the median, mean, quartiles, standard deviation, and credible interval [57].Let τ and α be all unknown parameters in the model and the vector of variance components, respectively (for $\alpha = (f, f_{\text{str}})$). Considering independent conditions, the Bayesian inference relies on a posterior distribution, and it seems like:

$$P(\alpha) \propto L(y, \beta_1, \ldots, \beta_p, f_{\text{str}}, f_{\text{unstr}}, \gamma, \sigma^2) \prod_{j=1}^{p} \left(p(\beta_j|\tau_j^2)p(\tau_j^2)\right) p(f_{\text{str}}|\tau^2_{\text{str}})p(\tau^2_{\text{str}})p(f_{\text{unstr}}|\tau^2_{\text{unstr}})p(\tau^2_{\text{unstr}})p(\gamma)p(\sigma^2) \tag{3.16}$$

The vector $\beta_j = (\beta_{j1}, \ldots, \beta_{jm_j})'$ corresponds to unknown regression coefficients vectors for

f_j. For all unknown parameters, the full conditionals of the vectors f_{str}, f_{unstr}, and fixed effect parameter γ, as well as the full parameter vector conditionals $\beta_1, \ldots, \beta_j$, have known distributions. Since the variance component's marginal probability depends intricately on the data, it lacks a straightforward family of conjugate prior distributions. However, the inverse gamma family is conjugate, and the inverse gamma distribution may be observed in the full conditionals τ_j^2 of $j = 1, \ldots, p$, str, unstr, and δ^2 [36].

Modell goodness of fit criteria

As part of any modeling exercise, it is usually of interest to assess how well a given model describes given data. To this end, several measures have been devised to help in this regard. The first of these is a deviance-based measure called Deviance information criteria (DIC). Second the WAIC or Watanabe Akaike information criteria and posterior predictive loss, and cross validatory measures [37]. The device information criteria (DIC) The device information criteria have been proposed by Spiengel halter et al., (2000), and widely used in Bayesian modeling is defined as

$$\text{DIC} = 2E_{\theta/y}(D) - D(2E_{\theta/y}, (\theta)) \tag{3.17}$$

Where $D(.)$ is the deviance of the model and y is the observed data while DIC is based on a comparison of average deviance $(\bar{D}) = -2\sum_{g=1}^{G} l(y|\theta^g)/G$, and then deviation of the posterior expected parameter estimated posterior distribution $\hat{\theta}$, $\hat{D}_\theta = -2l(y|\hat{\theta})$. For any sample parameter value θ^g, the deviance is $\bar{D}(\theta^g)$. The effective number of parameters (pD) is estimated as $p\hat{D} = \bar{D} - \hat{D}_\theta$.

$$\text{DIC} = \overline{D} + (\hat{p}\hat{D}) = 2\overline{D} - \hat{D}\hat{\theta} \tag{3.18}$$

An estimator $\widetilde{(pD)}$ proposed by [35], is used to calculate the effective number of parameters in the model, and it serves as a proxy for its complexity. This counts the number of factors that influence the models that fit the Bayesian model and can benefit from the use of this estimator. The estimator contains the following terminology:

$$\widetilde{(pD)} = \frac{1}{2(G-1)} \sum_{g=1}^{G} (\theta^g - \overline{D})^2 \tag{3.19}$$

Where G signifies the quantity of chains (samples derived from the posterior distribution), θ^g indicates the values of the parameters in each chain, and $\overline{D}$ is the average deviation over every chain. A higher $\widetilde{(pD)}$ value denotes more complexity as a result of more significant parameters. It improves our comprehension of the model's deviation from a more basic model (one with fewer parameters).

As an alternative, we may compute $\widetilde{(pD)}$ directly from sample output obtained from the chains using tools such as R2WinBUGS. Additionally, DIC is a useful tool for model evaluation and selection since it achieves a compromise between completely informative

and noninformative priors. This estimator can shed light on the parameter contribution and overall model complexity in Bayesian models. Data analysis and graphics were done using R program v4.3.0 in BayesX and R2BayseX packages, and The QGIS v1.8 was used for the generation of maps.

3.3 Results and discussions

3.3.1 Results

3.1.Descriptive statistics

Our study in Bayesian semiparametric geoadditive modeling of underweight among under-five children in Ethiopian attempts to explore demographic and socioeconomic factors, the presence of regional variation in Ethiopia regions, and to contribute result-based nutritional interventions for policymakers and health practitioners to develop effective strategies for the under-five children in Ethiopia. In this study, metrical covariates with a nonlinear trend, covariates with a fixed effect, and spatial effects were considered. All are treated by the Bayesian framework to assign appropriate priors with various forms and levels of smoothness. To summarize the characteristics of the covariates (see Table 3.2), the mean of underweight (weight-for-age z-score) was 1.28 in standard deviation. In our settings, based on the child's and mother's lives, 81.45% of these children live in rural and the remaining 18.55 of them live in urban areas. Among the mothers who were of childbearing age, about 6838 (64.27%) of them had no education, 2678 had completed their primary education, 734 of them had secondary education and 391 of them had completed their secondary education. Besides, from all the children we consider from 2016 EDHS data, 8826 developed diarrheal and the number of children who did have not diarrheal during the survey was only 1090. .

Table 3.2: Description of socioeconomic,demographic and metrica variables for underweight

Factors	**Frequency**	**Percentage**	**Coding**	
Mother education				
No education	6838	64.27	1	
Primary	2678	25.7	2	
Secondary	734	6.89	3	
Higher	392	3.68	4	
Child's sex				
Male	5483	51.53	1	
Female	5158	48.43	2	
residency				
Rural	8667	81.45	1	
Urban	1974	18.55	2	
Availability of electricity				
Yes	2367	22.53	1	
No	8141	77.48	2	
Anaemia level				
Severe	311	3.99	1	
Moderate	2531	32.47	2	
Mild	1849	23.73	3	
Not anaemia	3104	39.83	4	
Diarrhea level				
Yes	8826	89.01	1	
No	1090	10.9	2	
Sex of household headed				
Male	8383	78.78	1	
Female	2258	21.22	2	
Metrical covariates	**Min.**	**Max.**	**Sd**	**Mean**
Child's age in months	0	59	16.65	28.58
Mother age	15	49	29.23	6.65
Mother's BMI	11.73	83.85	3.43	20.73
Underweight	-5.92	4.92	1.28	-1.045

Apart from the descriptive statistics, the Yeo-Johnson transformation (see Figure 3.2) visualizes a range of values that are used to group data into categories for the purpose of detail visualization [90]. For example, by taking the bin visualization between the child's age and underweight, underweight is not constantly decreasing against the child's age (see the left plot of Figure 3.2). The plot undergoing the oscillation moves back and forth from the child's age between 20-40 months and then remains constant to the right. Similarly, as a mother's BMI increases, underweight will improve, but there is a threshold point. Any increase in BMI will worsen underweight. At low levels of maternal BMI, underweight is low (see the right plot of Figure 3.2). Furthermore, underweight will decline when the values of the mother's BMI increase for longer. This is evidence that the effects of

children being underweight are not a constantly increasing or decreasing trend for each value of mothers' BMI. Therefore, from the Yeo-Johnson transformation visualization, the metrical covariates like the mother's BMI, the child's age, and the mother's age at birth are good candidates for the nonlinear effects on children underweight based on the EDHS 2016 dataset.

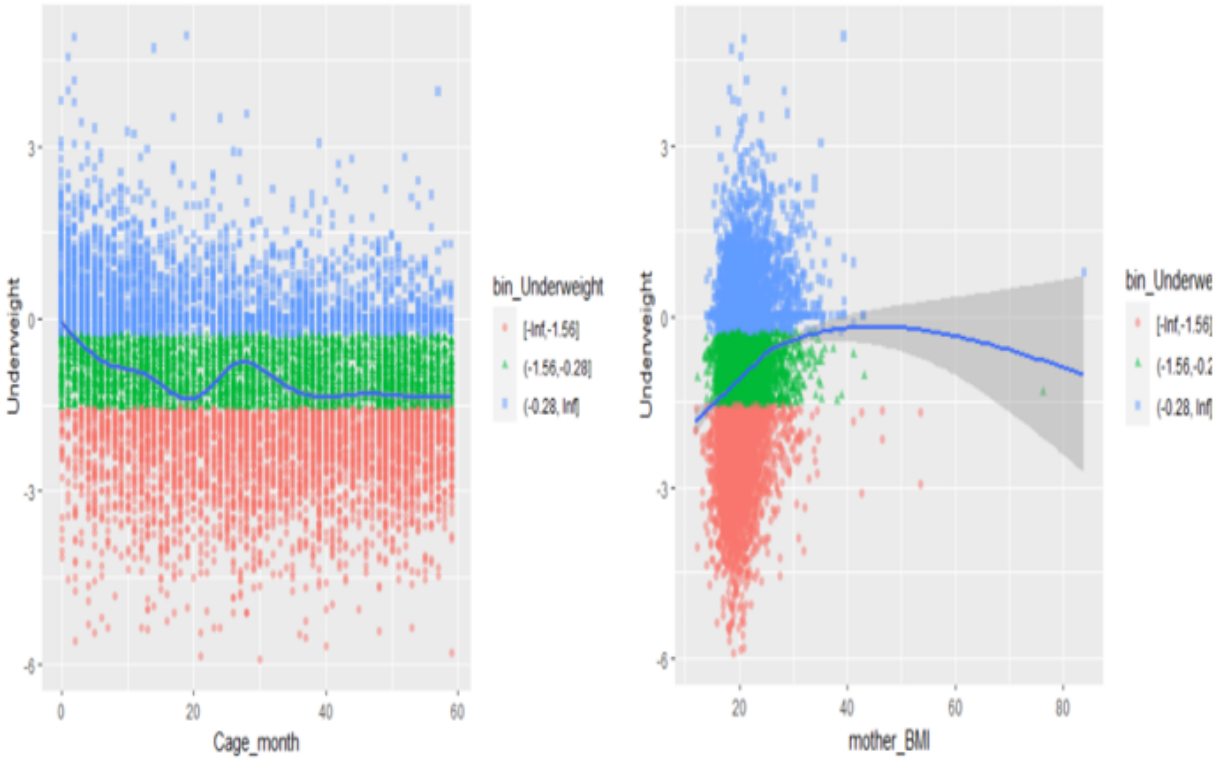

Figure 3.2: Yeo-Johnson transformation visualization of metrical covariates on underweight

In the Bayesian geoadditve model, the histogram of the model parameter estimates is expected to show a smooth and center distribution, while the density plot shows the posterior distribution of the parameter [10]. As shown in Figure 3, the smoothness of the histogram indicates that the model is well-calibrated, and the data is well-represented by the chosen model and the assigned prior's distribution. Besides, the distribution is roughly symmetric, centered around the posterior mean (see Figure 3.3). Hence, these no more discrepancies between the histogram and density plot to the model prediction tell us that the Bayesian geoadditive model is a feasible alternative for our inference.

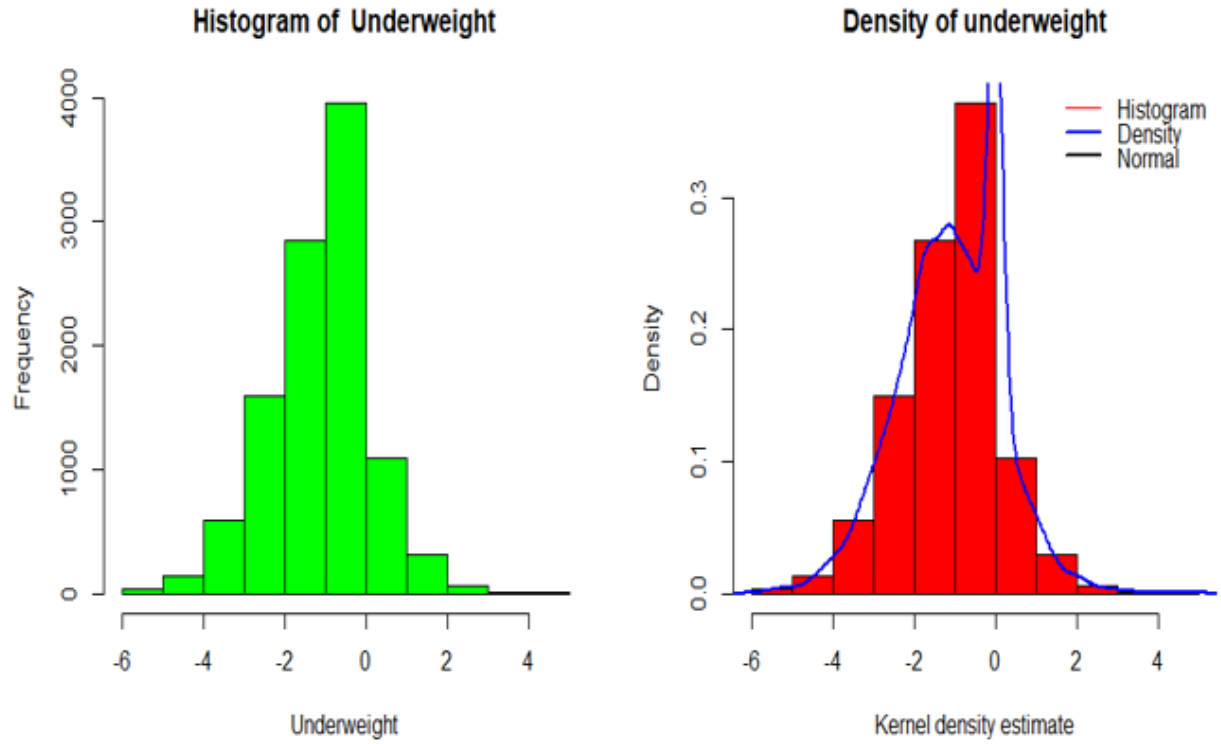

Figure 3.3: Distributions of underweight A) Histogram; B) Kernel Density Estimate

Bayesian geo additive model

We employed the Bayesian geoadditive statistical procedure that worked together with the BayesX stepwise selection method to identify various covariates that have an impact on underweight. The fixed effects and smooth term variance of the geoadditive Gaussian model are given in Table 3-4. From the tables, the value of 50% represents the median or most probable estimate of the coefficient, while the 2.5% and 97.5% quartiles represent the lower and upper bounds of a 95% credible interval [66]. A 95% credible interval can be used to estimate the statistical importance of variables in the Bayesian framework. If a parameter's credible interval excludes zero, then the parameter is likely statistically significant [37].

As shown in Table 3.3, the results of the fixed effect are as expected. Female children, absence of electricity, severe diarrhea, moderate anemia, and primary, secondary, and higher education were statistically significant at the 5% level. However, living in an urban area and being the female head of a household are not statistically significant. Therefore, the findings suggest that female children are at a higher risk of being underweight than male children. Children born to mothers with secondary and higher education levels, who had electricity in their house during their pregnancies are at lower risk of underweight malnutrition compared to children born to mothers with a primary and lower education level and who couldn't obtain electricity.

Furthermore, in comparison to the reference group of male children, the estimated mean of the effect of female children on underweight is 0.0837 with an estimated standard

deviation of 0.023 (see Table 3.3). These results suggest that the effect of the female children on underweight malnutrition is likely positive and relatively precise and estimated to be around 0.0837 units higher than the male children's on average with a 95% credible interval lies between 0.0371 and 0.1293 units. Furthermore, the quantile values of 2.5%, 50%, and 97.5% the posterior distribution of the effects of female children are 0.0371, 0.0840, and 0.1293, respectively. Similarly, the effect of the absence of electricity on underweight is likely negative and is estimated to be around -0.1976 units higher than the reference level (the presence of electricity) on average, with a 95% probability that the true effect lies between -0.2746 and -0.1158 units.

Table 3.3: Posterior estimate of the fixed effect parameter for underweight in Ethiopia

Covariates	**Mean**	**SD**	**2.5% quartiles**	**50% quartiles**	**97.5% quartiles**
Intercept	-1.1037	0.3060	-1.7565	-1.0935	-0.5333
Sex of child (Ref = Male*)					
Female Ⓡ	0.0837	0.0225	0.0371	0.0840	0.1293
Residence (Ref = Rural*)					
Urban	0.0296	0.0451	-0.0610	0.0302	0.1160
Availability of electricity					
Yes*					
No Ⓡ	-0.1976	0.0418	-0.2746	-0.1977	-0.1158
Sex of the household headed					
Male*					
Female	-0.0521	0.0294	-0.1098	-0.0525	0.0074
Diarrhea level					
No*					
Yes Ⓡ	0.2161	0.0317	0.1534	0.2174	0.2780
Anaemia level					
Not anaemic*					
Severe Ⓡ	-0.5319	0.0533	-0.6323	-0.5343	-0.4189
Moderate Ⓡ	0.2998	0.0239	0.2529	0.2988	0.3456
Mild Ⓡ	0.0139	0.0276	-0.0421	0.0146	0.0663
Mother education					
No*					
Primary Ⓡ	-0.0946	0.0265	-0.2796	-0.0941	-0.0424
Secondary Ⓡ	0.1232	0.0375	0.0441	0.1241	0.1939
Higher Ⓡ	0.2277	0.0265	0.2796	0.2272	0.1754

*: reference group, Ⓡ: significance

Likewise, Table 3.4 represents the degree of smoothness of the function being estimated, typically referring to the variance parameters associated with the smooth terms in an additive model of metrical covariates. It shows the estimated mean of the variance, the estimated standard deviation of the variance, the 2.5% and 97.5% quartiles of the posterior distribution of the variance, the estimated the estimated median (50% quartile) of the posterior distributions of the variance for spatial and metrical covariates. Thus, the child's and mother's age, and mother's BMI are significant predictors of being underweight in Ethiopia. The spatial effects are also quite significant suggesting that the socioeconomic variables are unable to account for the consideration portions of this regional spatial effect.

When considering the metrical variables, the estimated mean of the variance, the estimated standard deviation of the variance, and the 50% quantile of the posterior distribution of the variance of children's age are 1.6961, 1.2180, and 1.3796, respectively. Besides, the 2.5% and 97.5% quartiles of the posterior distribution of the variance for the child's age are 0.05 and 0.15, respectively. The estimated mean of the spatial random effect variance (sx (spatial effect)) is 0.3047. This shows that there is spatial variation in underweight that is not explained by the other covariates in the model, and there is a 95% probability that the true variance for the spatial random effect lies between 0.0721 and 0.7589 based on the data and the model. This table also shows the hyperparameter of error variance (Sigma2), which is the amount of variation in the response variable that is not explained by the covariates and the spatial effects. A smaller value of error variance indicates that the model can explain large proportions of the variation in the response variable, while a larger value of error variance indicates that there is more random variability in the response variable that is not explained by the model [13]. In conclusion, this output indicates that the estimated posterior mean value of Sigma2 is 1.3511, with a relatively small standard deviation of 0.0187 and a 95% credible interval. The scale parameter Sigma2 is also likely statistically significant.

Table 3.4: Posterior estimate of the Smooth term's variances and Scale estimate for underweight malnutrition

Smooth term variances	**Mean**	**SD**	**2.5%**	**50%**	**97.5%**	**Min**	**Max**
sx(Child age/month) Ⓡ	1.6961	1.2180	0.4691	1.3796	4.8556	0.3018	14.9313
sx(spatial effect) Ⓡ	0.3047	0.1819	0.0721	0.2690	0.7589	0.0392	1.7381
sx(Mother's age) Ⓡ	0.0114	0.0181	0.0007	0.0055	0.0604	0.0003	0.1588
sx(mother's BMI) Ⓡ	0.4226	0.9056	0.0322	0.1890	2.1381	0.0142	11.4281
Scale estimate							
Sigma2	1.3511	0.0187	1.3154	1.3510	1.3892		

N = 10641, burn in = 2000, method = MCMC, family = Gaussian, Iteriation = 1200, steps = 10, SD = standard deviation Ⓡ= significance

The 95% credible interval indicates the range of values that the true variance is likely to fall within with 95% probability, while the 80% credible interval will give a narrower range of values with 80% probability [37]. The posterior means, together with the credible intervals of metrical covariates (mother's age, BMI, and child's age), of underweight have been shown in Figure 3.4. From these figures (see Figure 3.4), the shaded area for each metrical covariate provides a way to visualize the uncertainty in our predictions for each metrical covariate and the underweight in our analysis. The wider the shaded area, the more uncertain our predictions are, while the steeper the black line, the stronger the relationship between metrical covariates and underweight.

The top left in Figure 3.4 reveals that a child's age has a large nonlinear impact on underweight, particularly between the ages of 0 and 10 months. During this time, the child's underweight gradually worsens, following an almost linear pattern. This indicates that the chance of being underweight increases progressively as the child grows older. However, after 10 months, the tendency changes and stabilizes at a moderate level between the ages of 15 and 25 months. This means that, while the danger of malnutrition remains, it does not grow as fast as it did previously, and the child's nutritional status stabilizes to some extent. This might occur if younger children are more sensitive to underweight malnutrition owing to a lack of access to proper food and healthcare, while older children are more likely to be influenced by social and environmental variables such as poverty and food insecurity [84]

A mother's BMI and her child's weight for age have a nonlinear relationship, as seen in Figure 4's bottom left panel. According to the graph, the association between the mother's BMI and her child's weight-for-height for the z-scores appears to be an inverted U shape. This suggests that when BMI rises above the minimum of 12, the child's weight-for-height in Z-score rises as well (i.e., there is less underweight). However, a higher maternal BMI above 50 seems to have a significant impact on the child's underweight (high underweight). According to our result, maternal BMI between 12 and 50 is an optimal range of maternal BMI (between 12 and 50) that is associated with lower levels of underweight in children. A BMI of less than 20 may have a lower Z-score of weight-for-height for their children compared to mothers with a higher BMI, which could indicate that the child maybe undernourished or not growing properly. Furthermore, a BMI of less than 18.5 is considered underweight, and it indicates acute undernutrition in the mother. This can lead to negative health outcomes for both the mother and the child, such as an increased risk of complications during pregnancy and childbirth, low birth weight, and a higher risk of developmental delays for the child.

The effect of the mother's age is also quite slight (see the top right panel of Figure 3.4). It shows the weight-for-height Z-score is low for mothers aged between 15 and 35 years. The Z-score of weight-for-age decreases (and underweight increases) after the age of 35. After this age, the effects of mother age increase with an almost linear trend on

underweight. It shows that their children are worth their nutrition status as compared with children whose mothers are in the younger age group. This is because as women age gets an increase, they are more likely to have chronic health conditions such as diabetes, hypertension, or heart disease, which can affect the health of the developing fetus and increase the risk of being underweight at birth, and older mothers may be more likely to have unhealthy lifestyle habits, such as smoking, drinking alcohol, or poor nutrition, which can increase the risk of underweight in babies [54, 92]. Moreover, the plotted line corresponds to the average predicted response across the predictor value, and the x-axis tick marks on the plot represent the unique predictor values in the selected dataset.

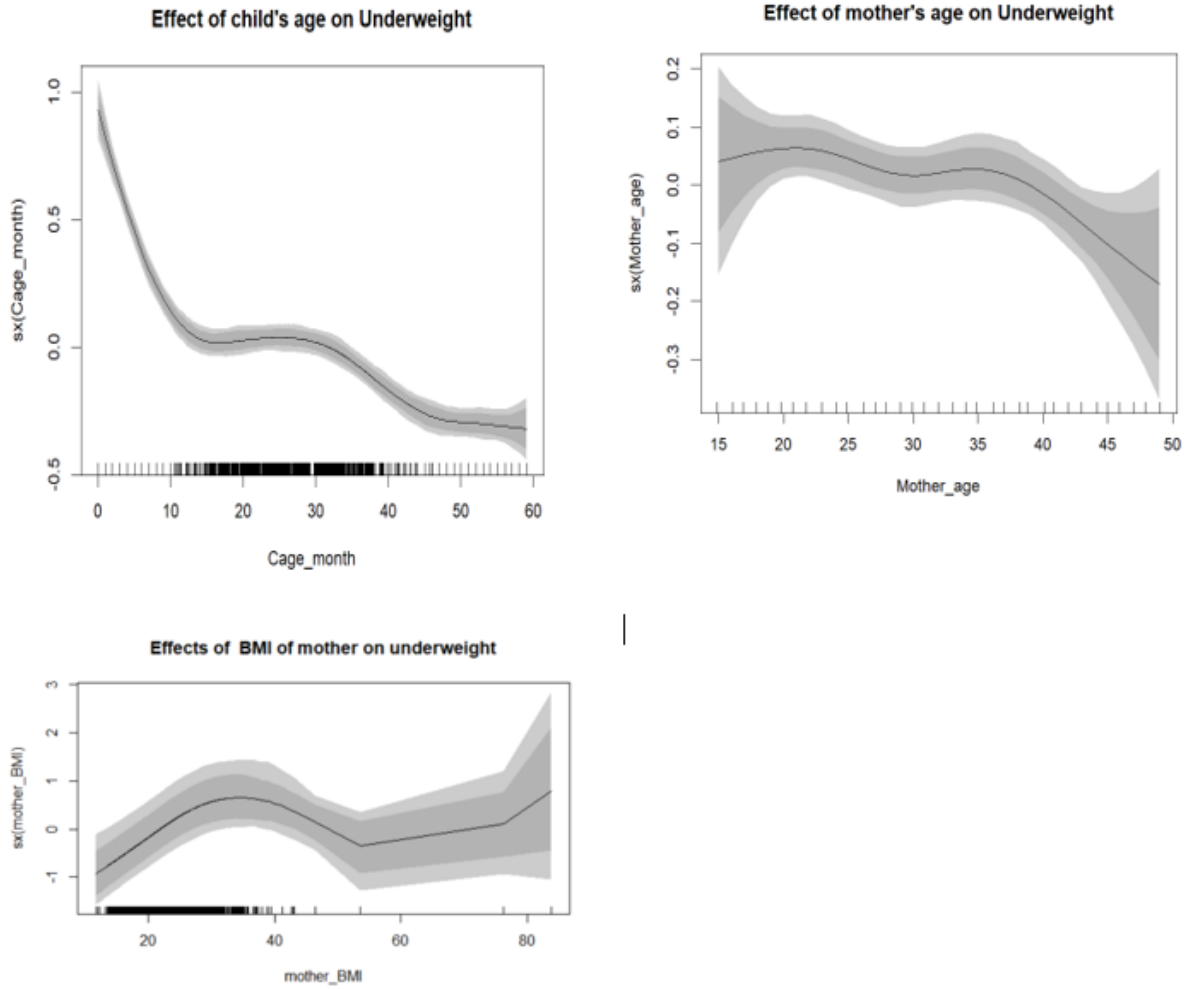

Figure 3.4: Nonlinear effects of metrical factors on underweight in Ethiopia: posterior mean with the 80% and 95% credible interval

The posterior spatial effect in the fitted model is shown in Figure 3.5, and the significance of spatial effects is shown by the posterior probability maps. In this map, the colors red and blue signify significant positive and negative effects on the Z-score, respectively, while grey shows no significance. The findings indicate strong support for incorporating geospatial analysis due to the substantial variation in child underweight observed in the Gaussian model. Moreover, a significant spatial influence on children's underweight was evident across most regions in Ethiopia. Furthermore, in Bayesian geoadditive models, centering spatial effects around zero increases computing efficiency, stability, and interpretation and it ensures that the model converges effectively. Positive values indicate areas where the effect is stronger (higher risk) than the spatial effects around zero (reference), while negative values indicate weaker effects (lower risk) [62, 87, 94].

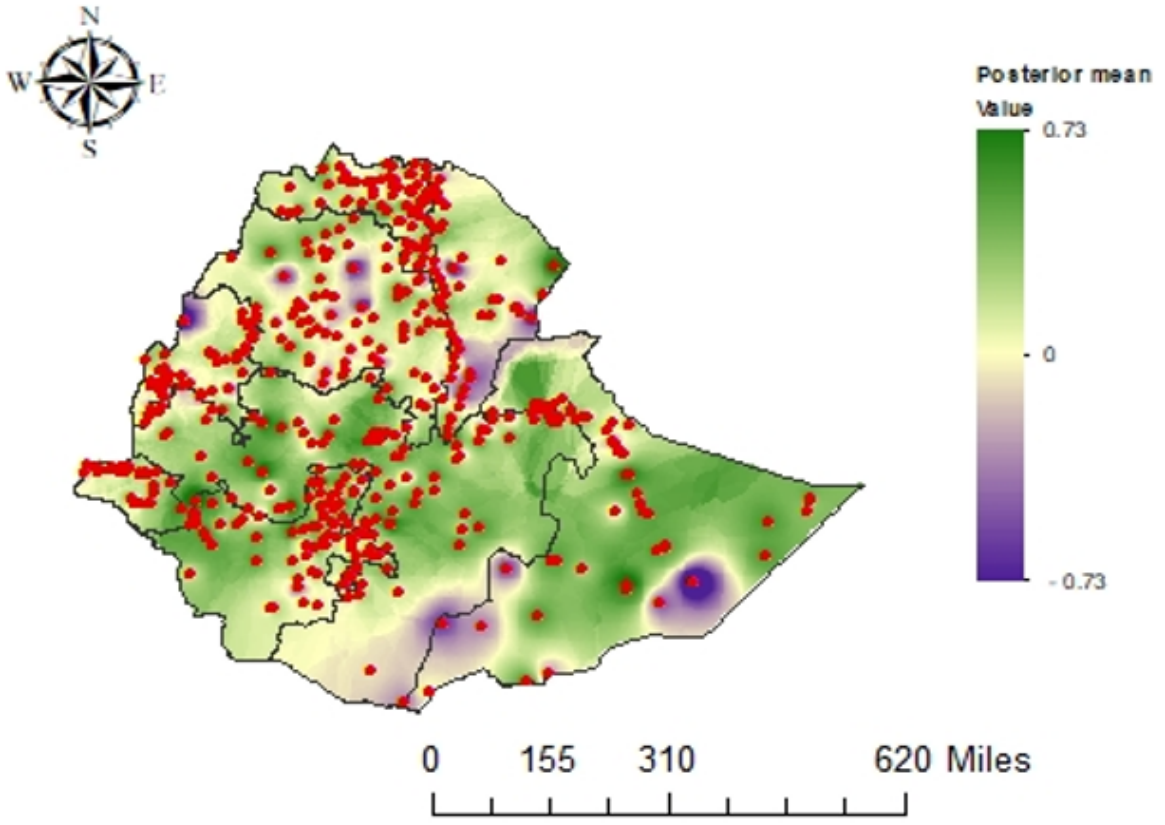

Figure 3.5: he Gaussian model's posterior mean of the spatial effect in underweight

Figure 3.6 displays the residual spatial pattern (left panel) and the IDW-interpolated surface of predicted values (right panel) following a Gaussian model fit. The left panel displays how the model deviates from the observed data, while the right panel uses IDW interpolation to reveal spatial patterns within the predicted values. This observed residual spatial pattern in underweight children may be ascribed to unobserved factors not represented by the covariates in the model and identifying them is an issue of hypothesis. Furthermore, the IDW interpolation of predictive values provides estimates of the underweight, and identifies areas of high, and low risk or abundance at each unsampled lo-

cation, and evaluates the effectiveness of different management or intervention strategies, and identifies areas where further data collection or monitoring is needed. The predicted values are then mapped to make it easier to interpret [85]. Therefore, the yellow color in the IDW interpolation of predictive values in the figure indicates the higher value of underweight and that area is the hotspot area (the left plot). The prediction of this unsampled location was done by using the observed values of the nearest ones.

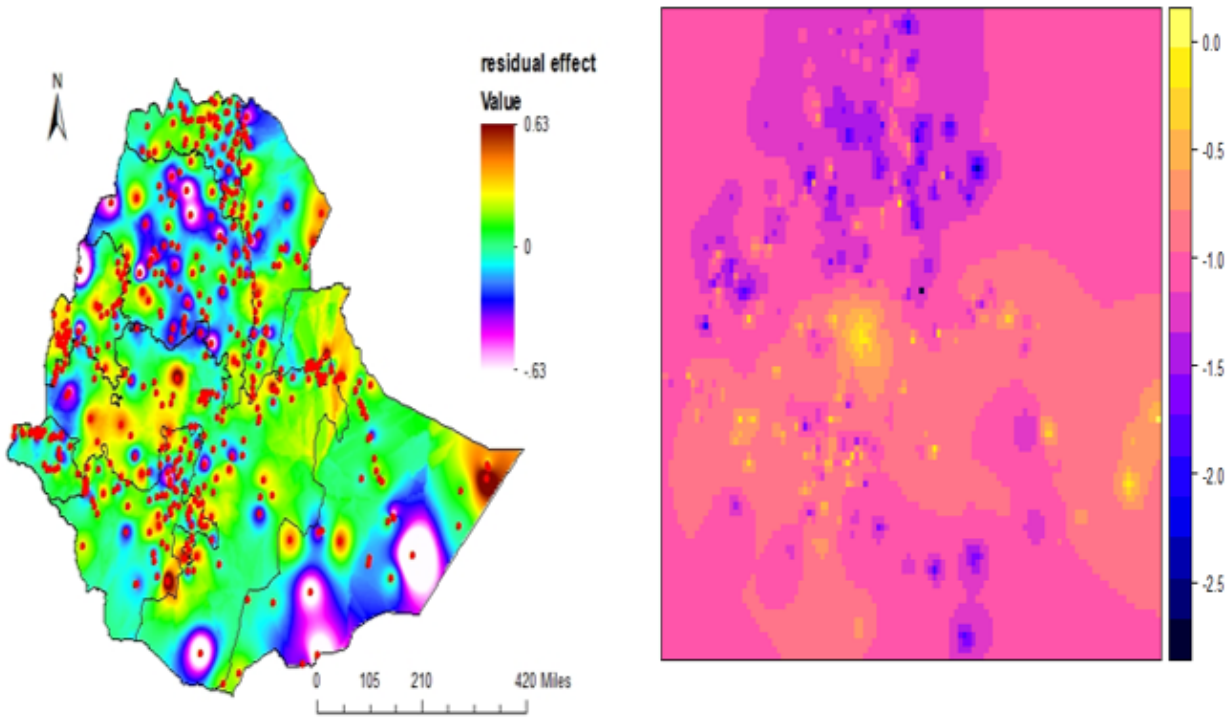

Figure 3.6: Posterior means of the residual spatial effects in underweight for the Gaussian model (left) and IDW Interpolation Predicted Values (right)

The goodness of fit of a Bayesian geo-additive model.
The goodness of fit of a Bayesian geo-additive model can provide insights into the accuracy and precision of the model's predictions, as well as identify potential sources of bias or model misspecification. Tracking plots, autocorrelation plots, and residual plots are important diagnostic tools for Bayesian geo-additive modeling [13].

By using the P-spline penalty, which can be used to control the smoothness of the estimated function, the series of samples from the posterior distribution can be used to obtain estimates of the linear function of our nonlinear covariates (mothers and child's age, BMI of mothers) and the spatial components where the child lives. Each corresponds to a different set of model parameters sampled from the posterior distribution. The functions of a series of samples from the posterior that are generated from a P-spline penalty is to provide estimates of model parameters, a smooth estimate of the function, and a basis for inference on quantities of interest and model comparison [13].

According to Monte Carlo sampling [44], we can take a sampling from a probability distribution and use those samples to approximate the desired quantity. Thus, the following figure, (see Figure 3.7) represents a series of samples generated from the posterior distribution, which can be used to make inferences and predictions. Likewise, the posterior samples obtained from MCMC algorithms are consistent with the observed data and prior information, and these give evidence to estimate the distribution of model parameters as well as measures of uncertainty or credible intervals.

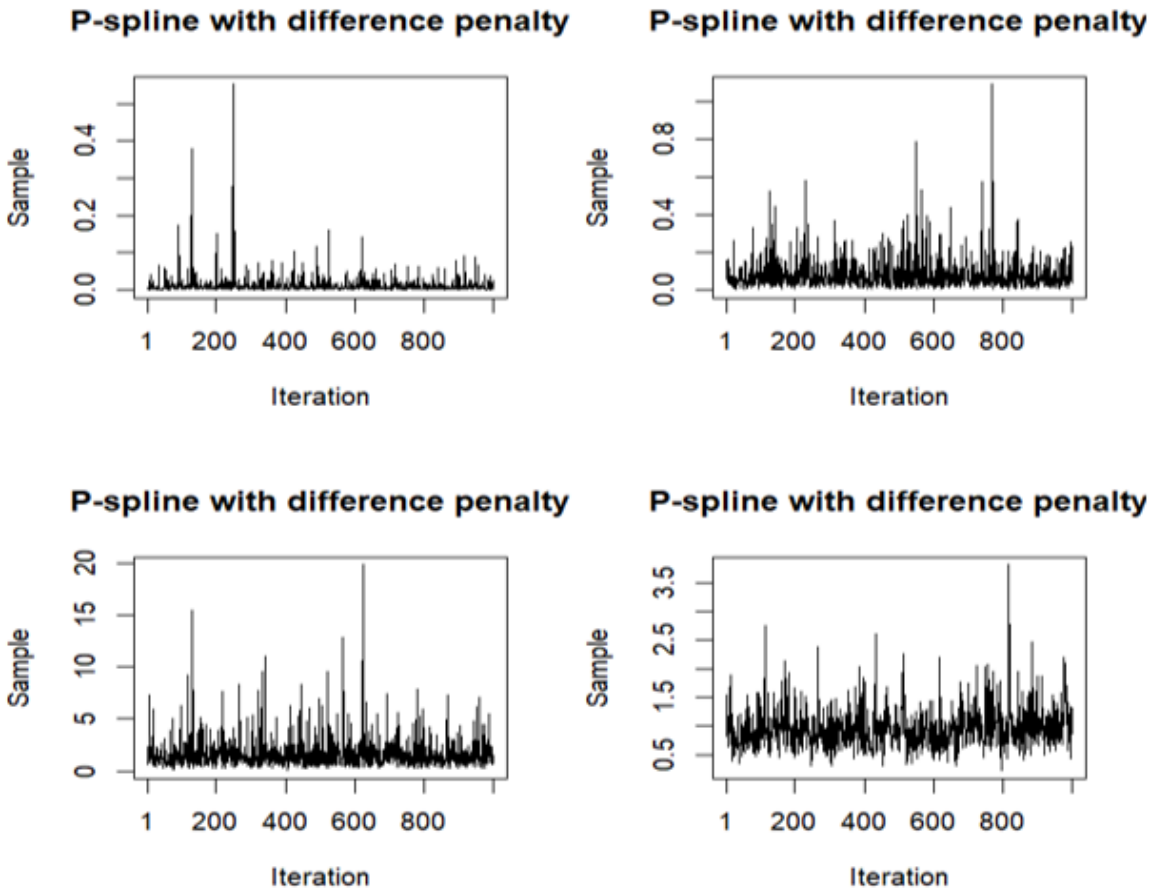

Figure 3.7: Sample posterior distributions from MCMC stimulation

Furthermore, in this Bayesian inference, we employ a tracking plot to visualize the behavior of Markov Chain Monte Carlo (MCMC) simulations. These MCMC chains are derived from sampling a probability distribution and are subsequently used to approximate desired quantities. The tracking plot could help assess the reliability and precision of the estimated quantity as well as spot any problems such a poor convergence or mixing [15]. The x-axis of the plot represents the iteration number, and the y-axis represents the values of the parameter. Thus, the tracking plot of a graphical representation of the MCMC chains (see Figure 3.8) shows the values of the model parameters at each iteration of the MCMC algorithm, and it has stabilized after the first 200 iterations, indicating that the MCMC algorithm has converged to a stationary distribution. Furthermore, the plot suggests that the MCMC algorithm has done a good job of exploring the posterior

distribution and has converted it to a stable distribution for the model parameters.

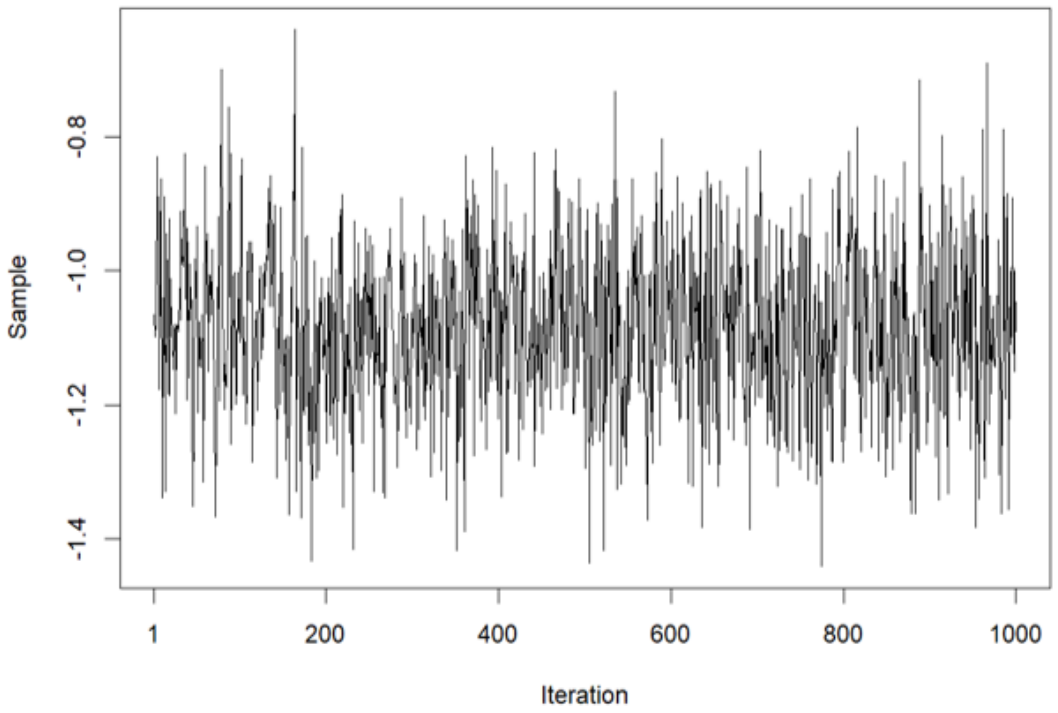

Figure 3.8: MCMC tracking plot of underweight malnutrition in the Bayesian geoadditive model in Ethiopia, P-spline with different penalties

Conjugation with other diagnostic tools, such as tracking plots, autocorrelation can be a useful tool for assessing the goodness of fit of a Bayesian geoadditive model to gain a compressive understanding of the model's performance [19, 46]. It is a measure of the correlation between a parameter value at time t iteration and a parameter value at time t+k iterations [10]. The plot depicted in Figure 3.9 is used to check for autocorrelation in the chain and it shows a rapid decay in autocorrelation as iteration k increases. Hence, a slight autocorrelation is visible in the plot, as evidenced by the slight correlation between the current and previous iterations. However, this is not a major concern as it does not appear to be having a significant impact on the mixing or convergence of the chains. Moreover, the autocorrelation drops off quickly, indicating that the MCMC algorithm is efficiently exploring the posterior distribution. This suggests that the MCMC algorithm is mixing well, and we may run it for a shorter time. This low autocorrelation is an indication of shorter convergence times and unbiased inference, and it is important for achieving efficient and accurate inference in Bayesian geoadditive models [19, 46].

The scale-location plot and residual plot diagnostic tools can also help to identify potential issues with a model's fit and convergence and guide model selection and improvement [10, 46, 76]. The plot presented on the right is a scale-location plot, a diagnostic tool used to assess the goodness of fit of the model. This plot shows the standard residuals (the residual divided by their deviation) against the square root of the estimated variance

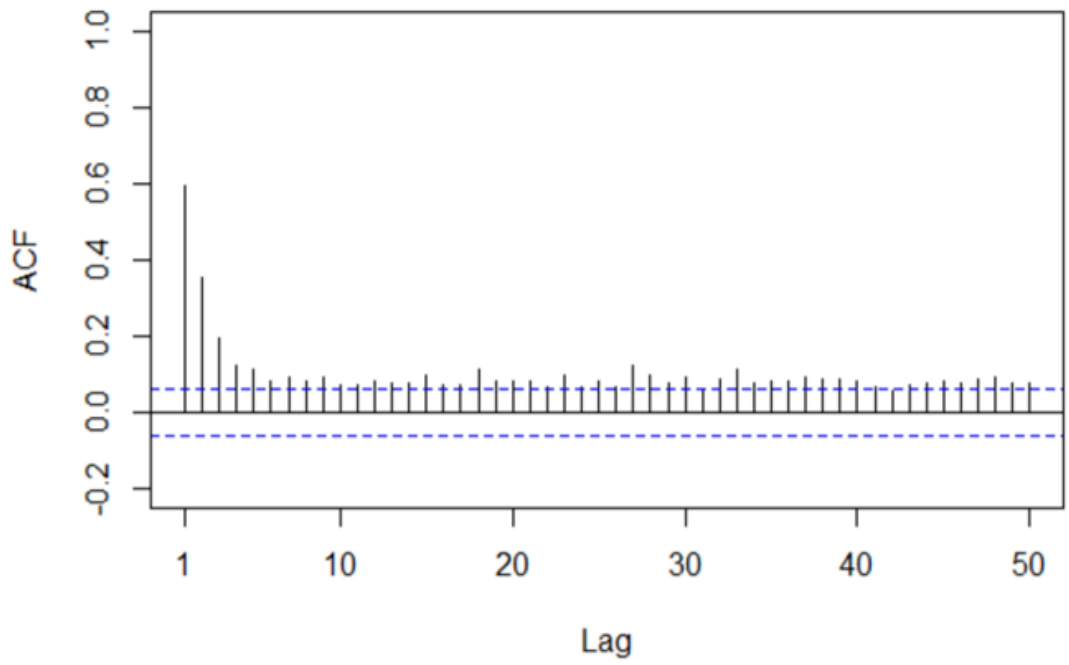

Figure 3.9: Maximum autocorrelation of model parameter for underweight malnutrition

of the response variable, which is also known as the scale parameter. The residuals are randomly scattered around zero (see the left plot of Figure 3.10), and if the spread of the residuals is constant across the range of the response variable, is not systematically above or below zero, or if the spread of the residuals varies across the range of the response variable, then the model is a good fit to the data.

Likewise, the plot depicted on the left is the residual plot or the fitted values versus the residuals. The fitted values represent the predictions made by the model for the response variable at different locations or spatial units, while the residuals represent the differences between the observed and the predicted values (see Figure 3.10). The plot of fitted values versus residuals is a commonly used diagnostic tool to assess the adequacy of the model fit and to identify any patterns or trends in the residuals that may suggest model misspecification or violations of model assumptions. If the model is correctly specified and the assumptions are met, we expect to see a random scatter of points, indicating that the model is capturing the variation in the data adequately. However, if there are any systematic patterns in the plot, this may suggest that the model is mis-specified. For example, if the residuals show a systematic increase or decrease as the predicted values increase, this may suggest that the model is underestimating or overestimating. If the residuals show a U-shape or a curved pattern, this may suggest that the model is missing a nonlinear component [30, 93]. Thus, the residuals (see the right plot from Figure 10), have not experienced any pattern or trend indicating a good fit of the model. There are no visible patterns or trends in the data that would suggest a poor fit.

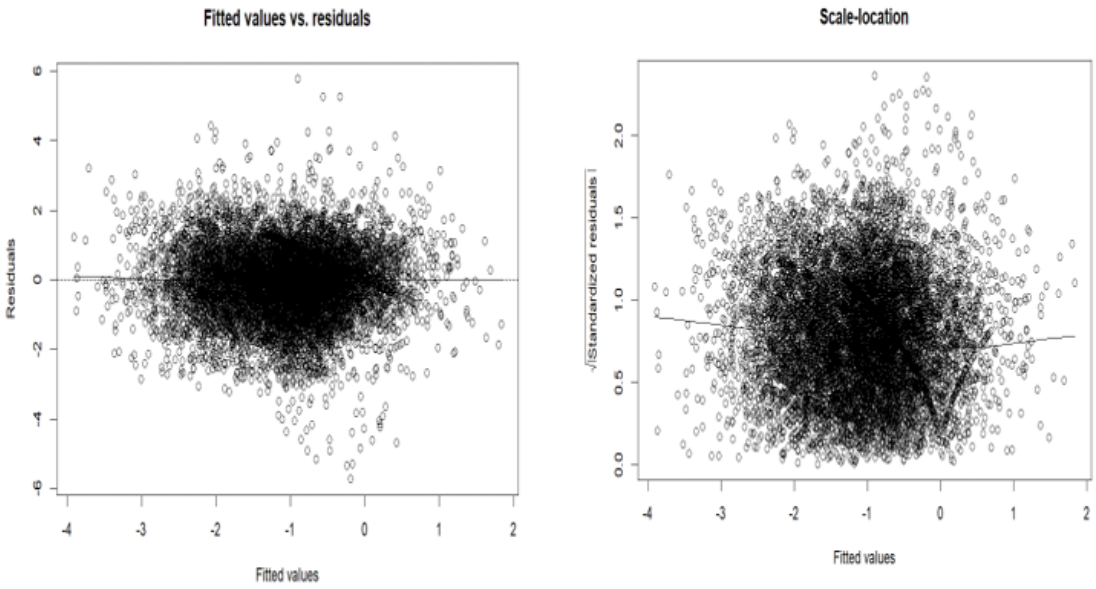

Figure 3.10: The residual and the scale-location plots

3.3.2 Discussion

According to our results, urban children are less likely than their rural counterparts to be underweight, and these results are credible. Better quality of the healthy environment and sanitation is present in urban regions. However, living in rural locations was thought to have numerous problems, such as poor health, a lack of access to clean water, a lack of charcoal as a fuel, a lack of milk intake, and poor personal cleanness or cleanliness. According to the findings of the study, the place of living has a major influence on being underweight. This result contradicts with [2], finding that where a mother resides (urban or rural), there is no statistical relevance for a child's weight-for-height; however, it is concise with [47, 78] findings that urban regions have a statistical significance for a child's underweight in Tanzania and Malawi. Similarly, female children were less likely than male children to be underweight. This outcome validates the findings from the earlier research [47, 53, 82]. Gibson, however, noted that there were no considerable gender discrepancies in underweight in Papua New Guinea [38].

Maternal education is a basic stimulus for child-care knowledge and behaviors. In our studies, mothers' educational attainment had a substantial influence on a child's underweight, and it lowered the risk of children being malnourished. This study favors the idea that a mother who has received an education is more responsible for delivering a sick child to medical treatment. In addition, the amount of time mothers spend discussing their children's sickness with a doctor is proportionate to their level of education. Uneducated women with their ill children benefit less from going to the doctor than educated women. Our findings suggest that maternal education has a significant impact on children's underweight which is consistent with studies conducted in underdeveloped countries [12, 17]. likewise, past research carried out in developing nations has demonstrated that several

African governments prohibit females from going back to school after giving children. Hence, a girl who abandons her studies would feed her kid poorly and perpetuate the cycle of poverty [74, 27, 31]

The BMI of a woman influences her capacity to effectively carry, give birth to, and care for her children. Malnutrition occurs when a non-pregnant woman's BMI falls below the recommended cutoff point (approximately 18.5 kg/m2). Women who are malnourished may deliver an underweight child. It implies that there is a link between the mother's BMI and her child's nutrition. According to our result, the relation between the BMI of a mother's and her weight-for-age z-score appears to be an inverted U shape for BMI between 12 and 50. Higher and lower maternal BMIs seem to have a significant impact on high underweights. This result contradicts [52], with all metrics exhibiting roughly linear trends with positive slopes.

The effects of maternal age on underweight are quite slight. It shows the weight-for-age of the Z-scores is high for mothers aged between 15 and 35 years. After the age of 35 years, the Z-score of weight-for-age decreases (underweight increases). It shows that their children are worth more in terms of nutrition status as compared with children whose mothers are in the younger age group. This is because as women age gets an increase, they are more likely to have chronic health conditions such as diabetes, hypertension, or heart diseases, which can affect the health of the developing fetus and increase the risk of being underweight [52, 71], and older mothers may be more likely to have unhealthy lifestyle habits, such as smoking, drinking alcohol, or poor nutrition, which can increase the risk of being underweight in babies [54, 92]. Therefore, our result contradicts the study conducted by [52], which found that mothers under the age of 20 have a greater effect on their underweight children.

A child's age also has a nonlinear trend towards underweight. Particularly, the child's underweight gradually worsens in an almost linear pattern at an age of less than 10 months. However, after 10 months, the tendency changes and then stabilizes at a moderate level in between the ages of 30 months. This means that, while the danger of malnutrition remains, it does not grow as fast as it did previously. This might indicate that younger children are more sensitive to underweight malnutrition owing to a lack of access to proper food and healthcare, while older children are more likely to be influenced by social and environmental variables such as poverty and food insecurity [84]. Hence, the results are consistent with other researchers' findings that child age affects underweight malnutrition non-linearly [47, 50, 67]. The study also found that children living in western, central, and eastern Ethiopia, as well as some other regions in the north, have underweight problems.

Furthermore, latent factors such as genetic predispositions, latent socioeconomic status, or environmental exposures can play crucial roles for child's underweight. Future research may include latent variable modelling for this the Bayesian geoadditive modeling approach which may capture unobserved heterogeneity and underlying factors, that

may not be directly measurable but significantly influence the children's underweight. It may allow for a more nuanced understanding of spatial dependencies and interactions between observed and latent variables, and it may also lead to more accurate and robust predictions and inferences about the factors affecting children's underweight. Our analysis also did not consider variables like household income, household size, and a child's birth weight, which might also significantly influence children's underweight. Therefore, in addition to the metrical covariates that we used, future researchers will try to check the existence of non linear relationship between the household income, household size, and child's birth weight metrical covariates and the underweight in Ethiopia.

In conclusion, this study addresses underweight in under-five children using a Bayesian geoadditive model. According to this analysis, factors such as the mother's education, the current mother's and child's residence the child's diarrhea, and anemia status, the sex of the child, and the availability of electricity were found to be significant based on the EDHS 2016 survey. However, the effects of sex on household heads are negligible. Our analysis also supports the flexible modeling of metrical factors (the mother's age, BMI, and child's age), and attention should also be given to unmeasured factors on childhood underweight at the community level, especially in central and eastern Ethiopia, which have indicated hotspot spatial impacts. Socio-demographic and community-based program development should be considered compressively in Ethiopian policy to combat childhood malnutrition.

3.4 Limitaions of the study

The current study has limitations despite its use of an innovative statistical method. First, it adopts a cross-sectional design, which means we cannot control for major confounders or make causal inferences, despite the analysis's robustness. Second, the study focuses solely on pertinent variables from our dataset, overlooking significant factors like breastfeeding practices, healthcare access, and breastfeeding practices.

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Chapter 4

The causality of infant mortality in Ethiopia: The application of Structural equation Modelling

Abstract

Infant mortality rate (IMR) serves as a proxy measure of population health. Previous studies have primarily focused on IMR in Ethiopia, considering only measured variables and one-directional effects. However, little attention has been given to simultaneously testing several causal paths. Data for the study were extracted from the World Bank Health Nutrition and Population Statistics between 2000 and 2019. We used structural equation modeling (SEM) to better understand the direct, indirect, and total effects relationships among causal variables in a single model. Path analysis was part of an algorithm that provided equations relating the variances and covariances of the indicators. GDP per capita (GDP) and out-of-pocket expenditure on health as a percentage of GDP (OOP) are exogenous variables, while immunization BCG (BCGI), maternal mortality ratio (MMR), fertility rate (FR), infant mortality rate (IMR), and domestic health expenditure as a share of GDP (GHE) are endogenous variables. The directed effects of OOP on MMR ($\beta = -0.071$, $p = 0.003$) and on BCGI ($\beta = 0.327$, $p = 0.024$), as well as the directed effects of GDP on FR ($\beta = -0.959$, $p < 0.001$), GHE ($\beta = -0.683$, $\Sigma = -0.69$, $p < 0.001$), and IMR ($\beta = -0.941$, $p < 0.001$), were significant. MMR significantly mediated the influence of OOP on IMR ($\beta = -0.012$, $p = 0.034$), and FR significantly mediated the influence of GDP on IMR ($\beta = 1.168$, $p < 0.001$). The discrepancy between the sample and the implied covariance matrix obtained from the five structural equation models was minimal. In conclusion, this study revealed that although IMR was declining, health and population variables remained the root cause of IMR in Ethiopia. MMR and FR were identified as mediating indicators, with FR having the highest standardized coefficients for increasing IMR. We recommend strengthening existing programs and interventions to reduce FR.

KEYWORDS

infant mortality rate; Ethiopia; path analysis; structural equation model; standardize estimate

4.1 Introduction

The infant mortality rate (IMR), is defined as the number of deaths in children under a year of age one per 1000 live births in the same year [36], and it is an important indicator of the health of a nation, regarded as a highly sensitive measure of population health [7, 19, 52, 18, 45]. Infant mortality rate (IMR) is more than a marker of maternal and child health; it is a symbolic benchmark of a society's overall health, and recent studies highlight the health inequities experienced by this population and subsequent effects on infant morbidity and mortality [4].

The health of children improved dramatically over the twentieth century in the world [5, 9]. The infant mortality rate has declined across countries occupying very different positions in the world system, However, considerable cross-national variation in infant mortality remains at the beginning of the twenty-first century and Ethiopia's commitment to significantly reduce child mortality rates by two-thirds by 2015 (Goal 4) under the Millennium Development Goals (MDGs) ultimately fell short of this target [2]. This highlights the challenges faced in achieving ambitious development objectives. UN member states, instead of Millennium Development Goals, set out Sustainable Development Goals (SDGs) in 2015 as part of the 2030 agenda to end preventable deaths of new-borns and children under 5 years of age, with most of the countries directing to reduce neonatal mortality to at least as low as 12 per 1,000 live births and under-5 mortality to at least as low as 25 per 1,000 live births (SDG 3.2). Ethiopia's National Health Care Quality Strategy for 2016-2020 placed Maternal, Newborn and Child Health as a priority with the ambitious goals of reducing the maternal mortality ratio (MMR) from 412 to 199 per 100,000 live births by 2020; to reduce the neonatal mortality rate (NMR) from 28 to 10 per 1,000 live births by 2020 and reduce stillbirth rate from 18 to 10 per 1000 births by 2020 (WHO, 2019). Despite that, overall action to meet the goals is not yet advancing at the speed or scale required [2].

According to a 2021 report by the Institute for Health metrics and Evaluation (IHME). An estimated 5.2 million children under the age of 5 died globally, mostly from preventable d causes and 1.5 million of these deaths occurred within the first month [46] . Despite the burden of those death decreasing globally, Sub – Sahara Africa and southern Asia account for the maximum proportion of child deaths [46, 40]. Four out of every five deaths of children under age five occur in these regions, compared to children in high-income nations, children in sub-Saharan Africa face a staggering 15 times higher chance of dying before the age of five [40]. Afghanistan has the highest infant mortality rate of 110.6 and Monaco has the lowest infant mortality rate of 1.8 [10]. In Ethiopia, the infant mortality rate was 56.9 in the year 2009 and it was 36.5 in 2019 per 1,000 live births [66]. The country's IMR declined from 97 per 1000 live births in the year 2000, to 59 in the year 2011, and neonatal deaths per 1,000 live births showed a decline over time from 54 in the year 1990 to 37 in the year 2011, but it is unlikely that the MDG target of 31 per

1,000 live births in the year 2015 [13].

In countries where infant mortality is high, several factors are attributed to necessitating these deaths. Among these are poverty, malaria, malnutrition, undeveloped infrastructure, and poor health facilities conditions [10]. High infant mortality signifies demographic and socioeconomic, exposures and morbidity during pregnancy [49, 6]. Scholars confirm that there were different predictors of infant mortality rate. The study conducted by [41] in the UK, revealed that fertility rate domestic general government health expenditure (%GDP), and GDP per capita, significantly affect infant mortality rate. Furthermore, fertility and GDP per capita were the most influential variables in the infant mortality rate from all explanatory variables used in the analysis. Real GDP has a negative relationship with fertility, and in return, fertility is positively correlated with infant mortality rate [56].

The variables like coverage of the Bolsa Família program (BFP), per capita income, and fertility rate are associated with infant deaths [54, 43, 48]. A woman in a high fertility setting has a higher risk of maternal death than a low fertility setting, and the maternal mortality ratio was strongly associated with infant mortality [58]. Analogous to the maternal mortality ratio, the risk of maternal death varies largely across countries. woman in Sub-Saharan Africa has the highest risk of maternal death (1 in 38), followed by South Asia, 1 in 240 [39].

Out-of-pocket (OOP) health expenditure significantly drops maternal health as it leads to a decline in skilled birth attendance and enlarges the maternal mortality ratio [35, 16]. Population in low-income countries are often exposed to out-of-pocket (OOP) and related indirect costs for their illness for health care, and this infers that household's health expenditure reduces infant and maternal mortality across low-income countries to reach a goal of ensuring health lives and people's well-being [16].

The Calmette-Gue´rin bacillus (BCG)- vaccine is given soon after birth to infants to decrease the incidence of TB disease and TB-associated mortality [34, 63], and lack of BCG- vaccination in the first week of life of new birth was highly associated with infant mortality rate [24]. WHO currently suggests about Calmette-Gue´rin bacillus (BCG)-vaccination at birth for developing countries except for preterm infants who should be vaccinated when they reach the age of 40 weeks [10]. The infant mortality rate was lower for Calmette-Gue´rin bacillus (BCG) -vaccinated than for unvaccinated [47].

Accordingly, Infant mortality in Ethiopia could be attributed to many different factors [28, 55, 60, 61], and previous studies have mostly employed only variables that are measured and one directional effect to discover relationships through the data set in a difference-in-differences (Diff in Diff) analysis, spatial patterns of infant mortality, multiple linear regression and/or correlation analyses, multiple logistic analyses and other multivariate statistical models to explore the factors associated with Infant mortality rate. Even though, research conducted on infant mortality rate [1, 38] by using structural

equation modelling based on economic indicators, and these studies pass over the most influential variables mediating variables, model identification and validation which are the basic determinant for structural equation modelling.

In this paper, we examine the causality of IMR in Ethiopia between 2000 and 2019 based on the World Bank health nutrition and population statistics variables. We use Structural equation modelling (SEM), and multivariate statistical methods, for a better understanding of both direct, indirect, and total effects of the given variables. This approach improves the understanding of mechanisms of the relationships among various factors and allows to testing of the research hypotheses in a single process by modelling complex relationships among many observed and latent variables [37, 64]. Structural equation modelling or analysis of covariance structure is a confirmatory approach, more suitable for testing the hypothesis than other multivariate statistical methods, most of the statistical methods other than structural equation modelling try to discover relationships through the data set [26, 53].

While traditional statistical methods can identify relationships between these factors and infant mortality, Structural Equation Modelling (SEM) offers a more robust approach. SEM is a confirmatory technique that allows researchers to test pre-defined hypotheses about the complex interplay between these variables. This approach goes beyond simply identifying correlations. It allows researchers to estimate the direct and indirect effects of each factor on infant mortality while accounting for potential measurement errors and interrelationships between variables [59, 68].

Given that, in a recent commentary, scholars expressed concern about the scarcity of SEM models in epidemiological research even if there is the availability of user-friendly software (e.g., SPSS AMOS, EQS, Mplus) and urged epidemiologists to use SEM models more frequently [22, 62]. In our study, we thoroughly examine the diverse factors influencing infant mortality in Ethiopia through a methodological lens that critiques conventional approaches. The existing researchers focused on straightforward statistical analyses, neglecting pivotal mediating factors and forgoing rigorous model validation. To address these limitations, we used of Structural Equation Modelling (SEM) as a leverage alternative. SEM offers a confirmatory approach that empowers us to systematically test intricate hypotheses regarding the complex interactions influencing infant mortality in Ethiopia. Unlike conventional methods, SEM enables us to discern both direct and indirect effects while accommodating measurement errors and interdependencies among variables. This methodological advancement goes beyond mere correlation, allowing us to establish causal relationships with greater precision by estimating the parameters in the interest of obtaining minimal residual covariance from World Bank health nutrition and population statistics between 2000 and 2019. Furthermore, analyzing the entire system simultaneously, SEM provides a more comprehensive understanding of the underlying structure driving high infant mortality rates, and it is expected that findings from our study will

improve planning and intervention to measure infant mortality in Ethiopia.

Hypothesis Development

Developing hypotheses is important in SEM. Constructing a theoretical framework goes beyond data investigation. Researchers suggest that factors can have an impact on an outcome both directly and indirectly (e.g., infant mortality). These hypotheses then inform the construction of the SEM model and enable statistical testing of accepted theories [22]. In SEM, the development of hypotheses is guided by a robust theoretical framework that relies on previous research [20, 21]. The direction and strength of the direct and indirect effects among the factors that eventually affect the result (such as infant mortality rate) are specified by these hypotheses. Path analysis, an essential part of SEM, then visually depicts these suggested relationships by using arrows to show the direction and thickness to show the strength of the effect. The theoretical framework simply lays forth testable hypotheses that determine the modeled relationships; route analysis provides visual assistance to illustrate these ideas.

Based on these scholarly results and literature, we have developed the following hypotheses, and the hypothesized value of each path is included in the following directed diagram (see Figure 4.1). The hypotheses of this study are stated as:

H1: There is a direct effect of Out-of-pocket expenditure on health(% Gross domestic per capita (GDP) on maternal mortality ratio and Immunization Calmette-Guérin bacillus (BCG)

H2: Both BCG Immunization and Maternal mortality ratio mediate the influence of out-of-pocket expenditure on health (% GDP) on infant mortality rate.

H3: A higher level of fertility rate is associated with a higher level of maternal mortality ratio.

H4: Government health expenditure has a direct effect on fertility rate, BCG immunization, maternal mortality ratio, and infant mortality rate.

H5: GDP per capita has a direct effect on government health expenditure (percentage of GDP), Immunization (BCG), fertility rate, and Infant mortality rate

H6: Government health expenditure, fertility rate, and Immunization BCG mediate the influence of GDP on Infant mortality rate.

4.2 Materials and Methods

4.2.1 Data sources, and covariates

The study used pooled panel data from 2000 to 2019 in Ethiopia. The source of data for this study was the World Bank Development Indicators (World Bank Health Nutrition

and Population Statistics). We used the infant mortality rate as an outcome variable. The infant mortality rate is measured as the death of a child less than 1 year old per 1000 live births. The analyses were performed using SPSS AMOS and STATA 14. The dataset used is freely available at http://data.WorldBank.org. The variables in a structural equation model (SEM) are categorized as either endogenous or exogenous variables [29]. Moreover, endogenous and exogenous variables can be distinguished through the arrows that connect them within the model [11, 14]. Specifically, GDP per capita and out-of-pocket expenditure on health (as a percentage of GDP) are considered exogenous variables, while the infant mortality rate, immunization vaccination BCG, maternal mortality ratio, fertility rate, and domestic health expenditure (as a share of GDP) are endogenous variables and are explained within the model (see Table 4.1).

Table 4.1: List of endogenous and exogenous variables and their abbreviation

Variables	**World Bank Definition**	**Abbreviation**
Infant mortality rate	Number of deaths among infants (¡1 year of age) per 1000 live births in a given year.	IMR (y5)
Immunization, BCG (% of one-year-old children)	Child immunization rate, BCG is the percentage of children ages 12-23 months who received vaccinations before 12 months or at any time before the survey for BCG. A child is considered adequately immunized after one dose.	BCG (y3)
Out-of-pocket expenditure on health	Share of out-of-pocket payments of total current health expenditures. Out-of-pocket payments are spending on health directly out-of-pocket by households.	OOP (x2)
Maternal Mortality Ratio	Maternal mortality ratio is the number of women who die from pregnancy-related causes while pregnant or within 42 days of pregnancy termination per 100,000 live births.	MMR (y4)
Domestic general government health expenditure (% of GDP)	Public expenditure on health from domestic sources as a share of the economy as measured by GDP.	GHE (y1)
The fertility rate	represents the number of children that would be born to a woman if she were to live to the end of her childbearing years and bear children in accordance with age-specific fertility rates of the specified year.	FR (y2)
GDP per capita	Per capita GDP is typically expressed in local current currency, local constant currency, or a standard unit of currency in international markets, such as the U.S. dollar (USD).	GDP (x1)

4.2.2 Statistical Analysis

Path diagram/ Causal graphs

Path analysis represents a methodological improvement regarding multivariate techniques used in modelling indicators and it allows the investigation of more complex models [65]. Furthermore, the path analysis rule involves tracing paths in the graph as part of an algorithm giving equations relating the variances and covariances of the indicators, and it is represented by a diagram called a directed graph or path diagram [67]. In directed graphs, the vertices represent continuous variables, the edges some notion of correlation and causation, and the relations in the diagram are the parameters of the equations to be estimated, called path coefficients, which present the responses of endogenous variables to other endogenous or exogenous variables, while other variables in the model were held constant [17, 67].

Each node in path analysis was defined by the variables y_1 to y_n, and there was a directed edge from y_i to y_j if the coefficient of y_i in the equation for y_j was distinct from zero [8]. Besides, there is mediation where one variable (exogenous) causes variation in another variable (endogenous), and the mediator hypothesis is supported [3, 22].

From Figure 4.1, all indicators are represented by rectangles, and it indicates that no latent variable in the model, and all arrows flow one way, with no feedback looping (recursive model). The measurement errors for the endogenous variables are uncorrelated. Our directed graph, set out all the causal linkages between variables to evaluate the possible hypothesis and β_{ij} and γ_{ij} are the coefficients. Thus, the following figure (Figure 4.1) is the path diagram for based on our setting that shows the cause and effect relationship between based on the theoretical framework.

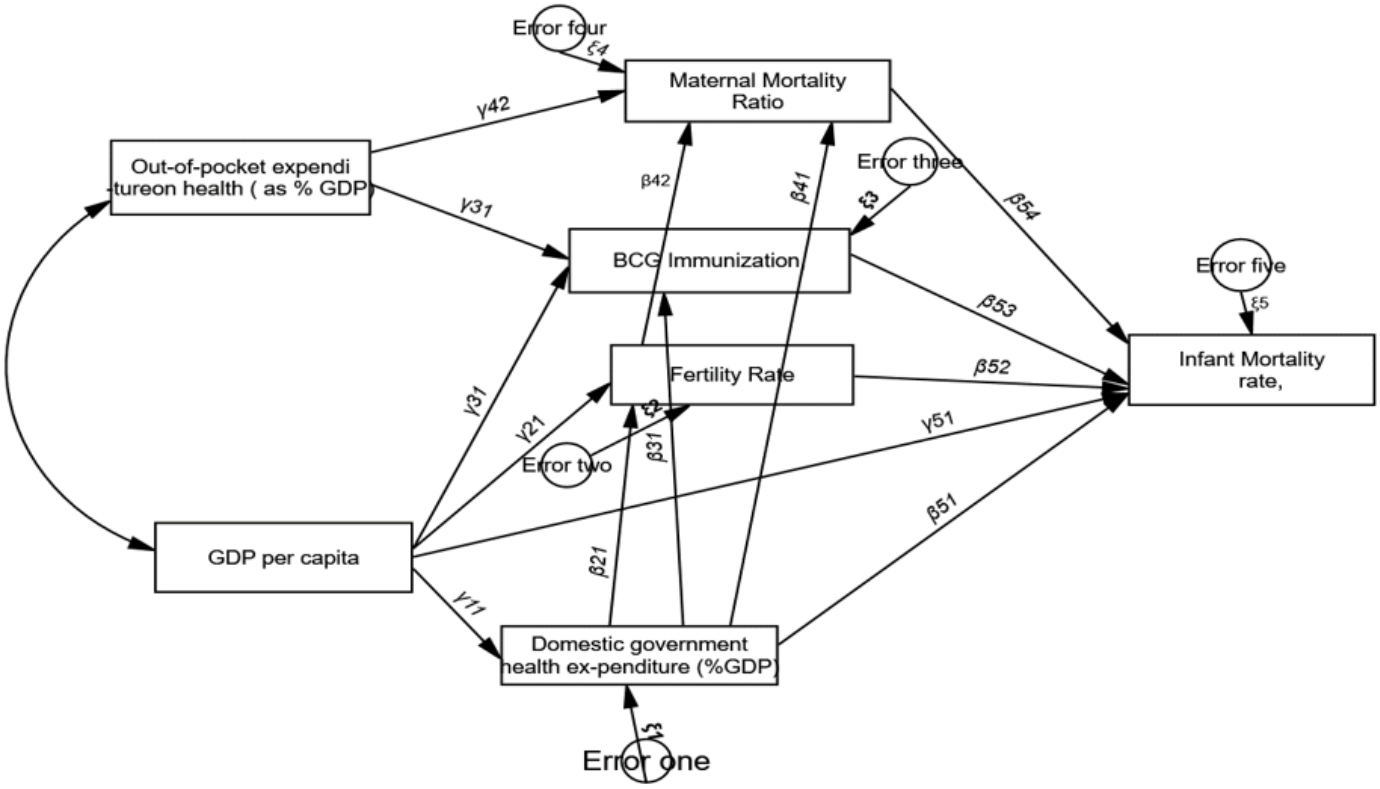

Figure 4.1: The directed cyclic graph or the path diagram of the theoretical model

Model Identification

Model identification also is critical in structural equation modelling path analysis, and no reliable quantitative conclusion can be derived from non-identified models (Carlos Brito and Judea Pearl, (2002). The process of model identification in structural equation modeling (SEM) establishes whether there is sufficient data to estimate the parameters of the model in a unique way. A just-identified model has all variables interconnected ($Df = 0$). Under-identified models ($Df < 0$) lack sufficient information, while over-identified models ($Df > 0$) have too many restrictions, potentially causing convergence issues [42].

According to [32]for the path analysis model, let P be the total number of exogenous and endogenous variables in the model, and let t be the number of free parameters.

Then, the t-rule is $\frac{p(P+1)}{2} \geq t$. The difference gives the number of degrees of freedom for the model:

$$Df = \frac{P(P+1)}{2} - t \tag{4.1}$$

From the observed covariance matrix of the given model, we have five endogenous and two exogenous variables, that is, seven observed variables (seven rectangles from the path diagram depicted above).

$$\sum 7 \times 7 = \frac{7(7+1)}{2} = 28\, variances, and, covariances.$$

Thus, we have 22 free parameters (8 non-zero from β; 6 non-zero from γ, 3 variances-covariances in Φ, and five residual variances in the diagonal of ψ). Therefore, the model $Df = 28 - 22 = 6$ is overidentified. This implies that the model has more constraints (equations) than unknowns (parameters). This excess constraint can potentially lead to convergence issues during estimation as the model searches for a solution that satisfies all the conditions simultaneously.

4.2.3 Structural equation Models

Structural equation model consists of a set of multivariate techniques that are confirmatory rather than exploratory in testing models that fit data [11]. It is used to examine linear causal relationships among variables; each equation describes the dependence of one variable in terms of the others. SEM incorporates stochastic error terms(residuals) into its equations to account for the influence of unobserved variables and measurement errors, providing a more nuanced understanding of the relationship between variables [11, 12].

SEM has three major advantages over traditional multivariate techniques: First, explicit assessment of measurement error; second, estimation of latent (unobserved) variables via observed variables; and third, model testing, where a structure can be imposed and assessed as to the fit of the data. SEMs allow for a joint analysis of multiple exposures with several outcome variables, a series of endogenous variables are related to each other as well as to a series of exogenous variables [51, 29].

The process of estimating SEM parameters determines the ideal values to represent relationships inside the model by taking observed data into account. Among these are route coefficients, factor loadings, and error term variances. OLS is not suitable for estimating path coefficients, factor loadings, and error term variance of SEM, despite its seeming simplicity, as it cannot handle latent variables, error terms, and multiple associations [29]. Thus, Maximum Likelihood (ML) is a prominent approach that effectively addresses these shortcomings in comparison to Ordinary Least Squares (OLS) [44], each indicator should follow Multivariate normality for each value of each other indicator.

Moreover, to examine the causal relationships between different determinants and infant mortality in Ethiopia (2000–2019), the model is constructed using the structural equation modeling (SEM) framework, and the specification of the model is as follows:

Let: $\mathbf{y}$ be a $p \times 1$ vector of endogenous variables (Infant mortality rate, Immunization, BCG, Maternal Mortality Ratio, Fertility rate, and Domestic health expenditure as a share of GDP), $\mathbf{x}$ is a $q \times 1$ vector of exogenous variables, $\boldsymbol{\beta}$ is a $p \times p$ matrix giving the regression coefficients of endogenous variables ($\mathbf{y}$) on other endogenous variables (i.e., the matrix of beta regression path coefficients between endogenous to endogenous), $\boldsymbol{\gamma}$ is a $p \times q$ matrix giving the regression coefficients of the exogenous variables ($\mathbf{x}$) on endogenous variables ($\mathbf{y}$), where the i-th row indicates the endogenous variable and the

j-th column indicates the exogenous variable, and $\boldsymbol{\epsilon}$ is a $p \times 1$ vector of errors in the equations (i.e., regression residuals), representing the model errors associated with each endogenous variable.

The variances and covariances of the endogenous variables are being modeled as a function of the exogenous variables. Then, the general form of a SEM path analysis model is expressed in the matrix equation

$$\begin{aligned} y &= \beta y + \gamma x + \varsigma \\ y &= (I-\beta)^{-1}\gamma x + (I-\beta)^{-1}\varsigma \end{aligned} \tag{4.2}$$

Then the variance of the endogenous variables (y variables) is looks like:

$$V(y), = E(yy'), = E\left[\left((I-\beta)^{-1}\gamma x + (I-\beta)^{-1}\varsigma\right)\left((I-\beta)^{-1}\gamma x + (I-\beta)^{-1}\varsigma\right)^{T}\right]$$

$$V(y), = \left((I-\beta)^{-1}\right)\left[\gamma\Phi\gamma' + \psi\right]\left((I-\beta')^{-1}\right) \tag{4.3}$$

Provided that the variances of exogenous variable, x variables are defined as
$V(x), = E(xx') = \Phi$, $\mathrm{V}(\varsigma) = E(\varsigma\varsigma') = \psi$. Similarly, the covariance between exogenous variable, x variables and endogenous variables (y variables) (covariance between x and y) is:
Cov(x,y) = E(xy') = $\mathrm{E}\left[x\left((I-\beta)^{-1}\gamma x + (I-\beta)^{-1}\varsigma\right)'\right.$

$$\Sigma = \Phi\gamma'(I-\beta')^{-1} \tag{4.4}$$

Assumptions

- (ς) is uncorrelated with (x), i.e., ($\mathrm{cov}\varsigma, x) = 0$
- $(|I-\beta| \neq 0) and is invertible (i.e., (\ \mathrm{I} \neq \beta))$
- $E(\varsigma) = 0)$
- $(E(x) = E(y) = 0)$

Therefore, Putting all the variance - covariance together,

$$\sum = \begin{bmatrix} \sum_{yy} & - \\ \sum_{xy} & \sum_{xx} \end{bmatrix}$$

Here, x, y, and ς are Gaussian random vectors:$x \sim N(\mu_x, \Sigma_x)$ and $y \sim N(\mu_y, \Sigma_y)$.

The stochastic error has a multivariate Gaussian distribution with a mean of the zero vector and a covariance matrix that is a diagonal matrix: $Cov(\varsigma) = \Psi = \mathrm{diag}(\psi_{11}, \psi_{22}, \psi_{33}, \psi_{44}, \psi_{55})$. The model acknowledges the inherent covariation of exogenous vari-

ables, determined outside the modeled system, through a variance-covariance system. This enhances the robustness of our causal inference by mitigating potential biases arising from untreated correlations among these external factors influencing infant mortality rate (IMR) in Ethiopia (2000-2019).

The causality of infant mortality based on the given indicators of exogenous variables, GDP per capita and Out-of-pocket expenditure on health (as percentage of GDP); and for endogenous variables, Domestic health on expenditure (as a share of GDP), Fertility rate, Immunization (BCG), Maternal Mortality Ratio and Infant mortality rate, can be expressed as a single matrix as:

$$\begin{bmatrix} \text{GHE} \\ \text{FR} \\ \text{BCG} \\ \text{MMR} \\ \text{IMR} \end{bmatrix} = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 \\ \beta_{21} & 0 & 0 & 0 & 0 \\ \beta_{31} & 0 & 0 & 0 & 0 \\ \beta_{41} & \beta_{42} & 0 & 0 & 0 \\ \beta_{51} & \beta_{51} & \beta_{51} & \beta_{51} & 0 \end{bmatrix} \begin{bmatrix} \text{GHE} \\ \text{FR} \\ \text{BCG} \\ \text{MMR} \\ \text{IMR} \end{bmatrix} + \begin{bmatrix} \gamma_{11} & 0 \\ \gamma_{21} & 0 \\ \gamma_{31} & \gamma_{32} \\ \gamma_{42} & 0 \\ \gamma_{51} & 0 \end{bmatrix} \begin{bmatrix} GDP \\ OOP \end{bmatrix} + \begin{bmatrix} \varsigma_1 \\ \varsigma_2 \\ \varsigma_3 \\ \varsigma_4 \\ \varsigma_5 \end{bmatrix}$$

This implies that some of the elements of β and γ re fixed to zero by hypothesis and the zeros on the diagonal of β implies that a variable cannot cause itself. So, from the above directed cyclic graph.

The hypothesized model is comprised of five linear regressions like:
$\text{GHE} = \gamma_{11}\text{GDP} + \varsigma_1$
$\text{FR} = \beta_{21}\text{GHE} + \gamma_{21}\text{GDP} + \varsigma_2$
$\text{BCG} = \beta_{31}\text{GHE} + \gamma_{31}\text{GDP} + \gamma_{32}\text{OOP} + \varsigma_3$
$\text{MMR} = \beta_{41}\text{GHE} + \beta_{42}\text{FR} + \gamma_{42}\text{OOP} + \varsigma_4$
$\text{IMR} = \beta_{51}\text{GHE} + \beta_{52}\text{FR} + \beta_{53}\text{BCG} + \beta_{54}\text{MMR} + \gamma_{51}\text{GDP} + \varsigma_5.$

And the parameters pertaining to variances and covariances of the exogenous variables GDP and OOP and the error terms $(\varsigma_1, \varsigma_2, \varsigma_3, \varsigma_4, and, \varsigma_5)$.

The variance-covariance matrix of the exogenous variables is given by:

$$\Phi = \begin{bmatrix} \text{var(GDP)} & \\ \text{Cov(GDP, OOP)} & \text{var(OOP)} \end{bmatrix}$$

Similarly, the variance-covariance matrix of the error terms $(\varsigma_1, \varsigma_2, \varsigma_3, \varsigma_4, and, \varsigma_5) is given by:$

$$\psi i = \begin{bmatrix} \psi 11 & 0 & 0 & 0 & 0 \\ 0 & \psi 22 & 0 & 0 & 0 \\ 0 & 0 & \psi 33 & 0 & 0 \\ 0 & 0 & 0 & \psi 44 & 0 \\ 0 & 0 & 0 & 0 & \psi 55 \end{bmatrix}$$

Typically, these variances and covariances of the exogenous variables GDP and OOP and the error terms the error variances are free parameters, but the covariances of error

variances are fixed to zero.

Model Fit Statistics

Model Fit Statistics measures how closely the (population) model-implied covariance matrix $\Sigma(\theta)$ matches the (population) observed covariance matrix Σ. Since SEM is also known as covariance structure analysis, the hypothesis of interest is regarding the covariance matrix. In SEM, relying solely on numerous fit indices can increase the risk of rejecting valid models. Using a combination of at least two fit indices is recommended for a more robust assessment of a model fit [15, 23]. Table 4.2 provides the information about goodness of fit indexes selected for this study and their cut-off values for model evaluation, based on the scholars [30].

Table 4.2: The Model Goodness of Fit Indices and Cut-Off Values

Indices	**Cut-Off**	**Scholars**
χ^2	≥ 0.5	Wan (2002); Schermelleh-Engel et al. (2003);
SRMR	≤ 0.05 (good) $0.05 <$ value ≤ 0.08 (acceptable)	Garson (2009); Wan (2002)
RMSEA	$0.05 <$ value ≤ 0.08	Browne and Cudeck (1993);
CFI	$0.90 \leq$ value < 0.95 (acceptable) ≥ 0.95 (good)	Hu and Bentler (1999); Schreiber, Stage,
TLI	$0.90 \leq$ value < 0.95 (acceptable) ≥ 0.95 (good)	Hoe (2003); Hu and Bentler (1999)

RMSEA = Root Mean Square Error of Approximation, TLI = Tucker-Lewis Index, CFI = Comparative Fit Index, SRMR = Standardized Root Mean Square Residual, χ^2 p= chi-square with p-value.

Assessment of multivariate normality

In SEM, each indicator should follow multivariate normality for each value of each other indicator and maximum likelihood estimation (MLE) is the dominant method for estimating structure (path) coefficients [29]. If we have a p x 1 random vector X that is distributed according to a multivariate normal distribution with population mean vector μ and population variance-covariance matrix, Σ, then this random vector X could have the joint density function in the form of:

$$\phi(x) = \frac{1}{(2\pi)^{\frac{p}{2}}|\Sigma|^{\frac{-1}{2}}} \exp\left\{-\frac{1}{2}(x-\mu)^T\Sigma^{-1}(x-\mu)\right\}, \quad X \sim N(\mu, \Sigma) \tag{4.5}$$

Where $|\Sigma|$ is the determinant of the variance-covariance matrix Σ and Σ^{-1} is the inverse of the variance covariance matrix Σ.

4.3 Results and discussions

4.3.1 Results

The main purpose of our study is to develop and test a hypothesized model that uses SEM for a better understanding of both direct and indirect effects of the given indicators on IMR by estimating the parameters so that the discrepancy between the sample covariance matrix and the implied covariance matrix is minimal from the data of world Bank: health nutrition and population statistics between 2000 and 2019. Accordingly, the analysis is carried out in SPSS AMOS and STATA 14.

Descriptive statistics were used to summarize the baseline characteristics of the population. As shown in the following table (Table 4.3), the mean infant mortality rate was 58.16 in the sample of 20 years for World Bank data from 2000 to 2019 in Ethiopia. In our settings, the maximum number of infants dying before reaching one year of age was recorded in the year 2000 with a value of 87.2 and the minimum value has been recorded in the year 2019 with a value of 36.6, per 1,000 live births each year. The maximum value of the fertility rate was 6.543 in the year 2000, and 1030 was the maximum maternal mortality ratio encountered in the year 2000. Thus, the fertility rate and maternal mortality ratio declined from the year 2000 to the year 2019. The mean number of public expenditures on health from domestic sources as a share of the economy as measured by GDP was 1.18 and the means of out-of-pocket expenditure, GDP per capita, and BCG immunization were given as 37.81, 67.8, and 395.23, respectively.

Checking for Multivariate normality

In the assessment of maximum likelihood estimation of loadings(parameters) for SEM, it is important to determine whether the data follows Gaussian normal distribution or not. From Table 4.3 of the assessment of normality column, the critical values of both skewness and Kurtosis of Observed, endogenous variables, and exogenous variables lie between -1.96 and +1.96 (all these p-values are ≥ 0.05) in the univariate case and the critical values of the multivariate normality of the model were - 0.191, we retain the null hypothesis and consider the sample as coming from a normal distribution.

Table 4.3: Descriptive statistics of the causality of infant mortality study in Ethiopia

Variables	**N**	**Min**	**Max**	**Mean**	**SD**	**Skew**	**CR**	**Kurt.**	**CR**
Fertility rate	20	4.15	6.54	5.26	0.75	0.180	0.328	-1.186	-1.082
Out-of-pocket expenditure	20	31.34	46.54	37.81	0.607	1.108	-0.028	-0.026	0.607
Maternal Mortality ratio	20	354.00	1030.00	663.35	231.94	0.281	0.513	-1.380	-1.259
Infant mortality ratio	20	36.60	87.20	58.16	16.09	0.347	0.634	-1.134	-1.035
Immunization (BCG)	20	56.00	80.00	67.80	6.79	-0.332	-0.606	-832	-0.759
Gov't expenditure on health	20	0.38	2.28	1.18	0.54	0.672	1.227	-0.606	-0.553
GDP per capita	20	111.93	855.76	395.23	251.41	0.447	0.815	-1.160	-1.059
Valid N (listwise)	20								
Multivariate							-0.960		-0.191

CR = Critical ratio, SD= standard deviation,Min= Minimum,Max = Maximum,skew= skewness and Kurt.= Kurtosis

Furthermore, World Bank data shows a clear downward trend in Ethiopia's IMR between 2000 and 2019 (see Figure 4.2). While Ethiopia has experienced a decline in IMR [39], the country still faces challenging in achieving and the country did not achieve the extent of the sustainable development goals(SDGs) related to infant health. According to the [39], the country's IMR remained high at 34.5 percent. This falls short of the SDGs targets within Goal 3: "Ensuring healthy lives and promoting the wellbeing of all" with specific targets to "end preventive coverage (UHC), through access to quality, safe, effective, affordable and essential health care services" and to "to end preventable deaths of new-born and child under five years of age" [2].

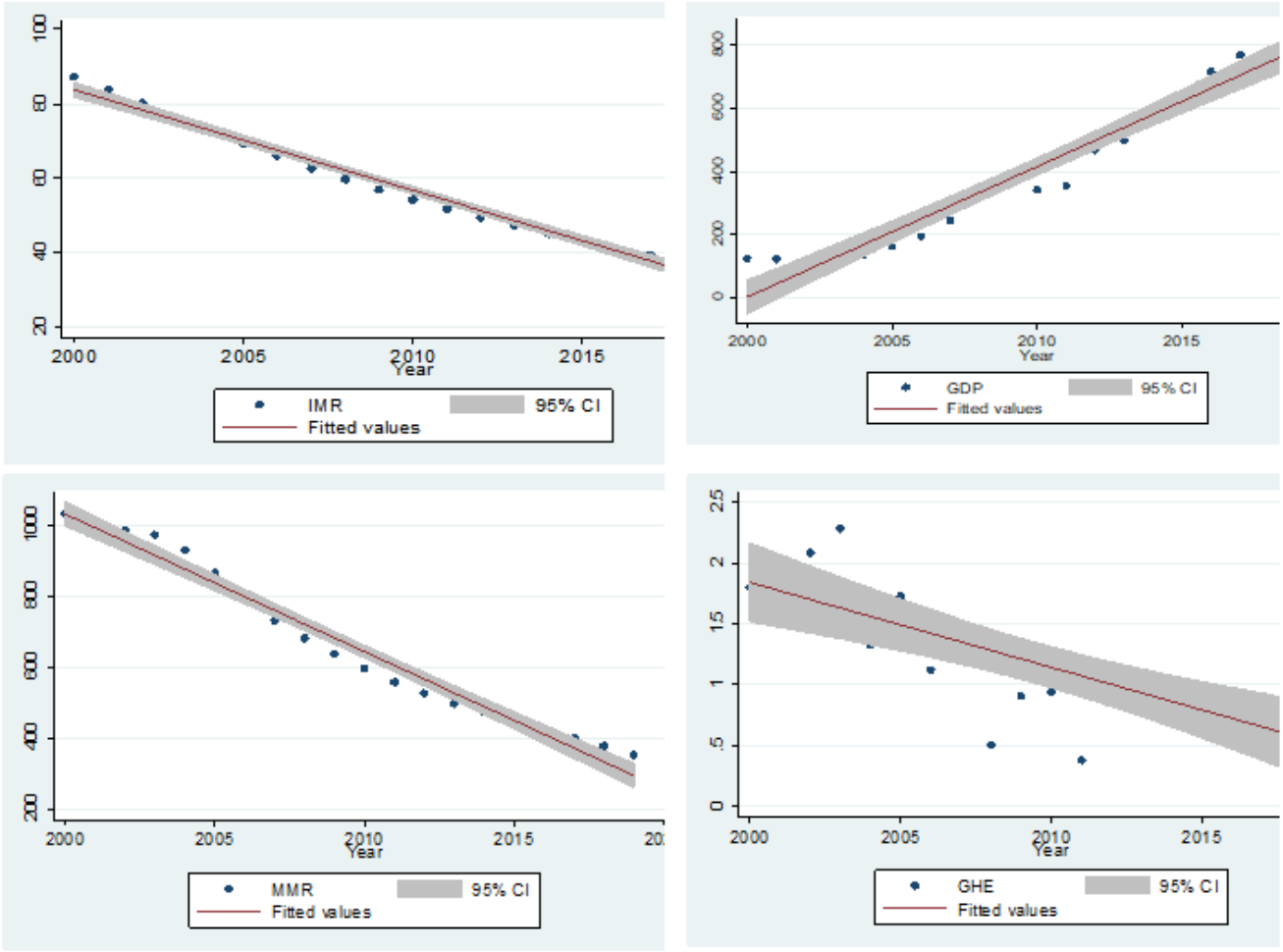

Figure 4.2: Trends of the different observed variables from 2000 to 2019

Structural equation model path analysis.

Path analysis

In Figure 4.3, the directed graph was displayed for each variable to test the hypothesis for IMR, and the diagram shows how one variable was associated with a subsequent variable in the causal chain. The direct effects were dedicated to the straight influence of one variable on another observed variable without any mediation and the effects of more distance variables were mediated indirectly through intervening. Moreover, the numbers written on the arrow are coefficients that show the influence of one variable on another variable. The path coefficients and errors displayed were standardized estimates and accordingly, the analysis is carried out in SPSS AMOS.

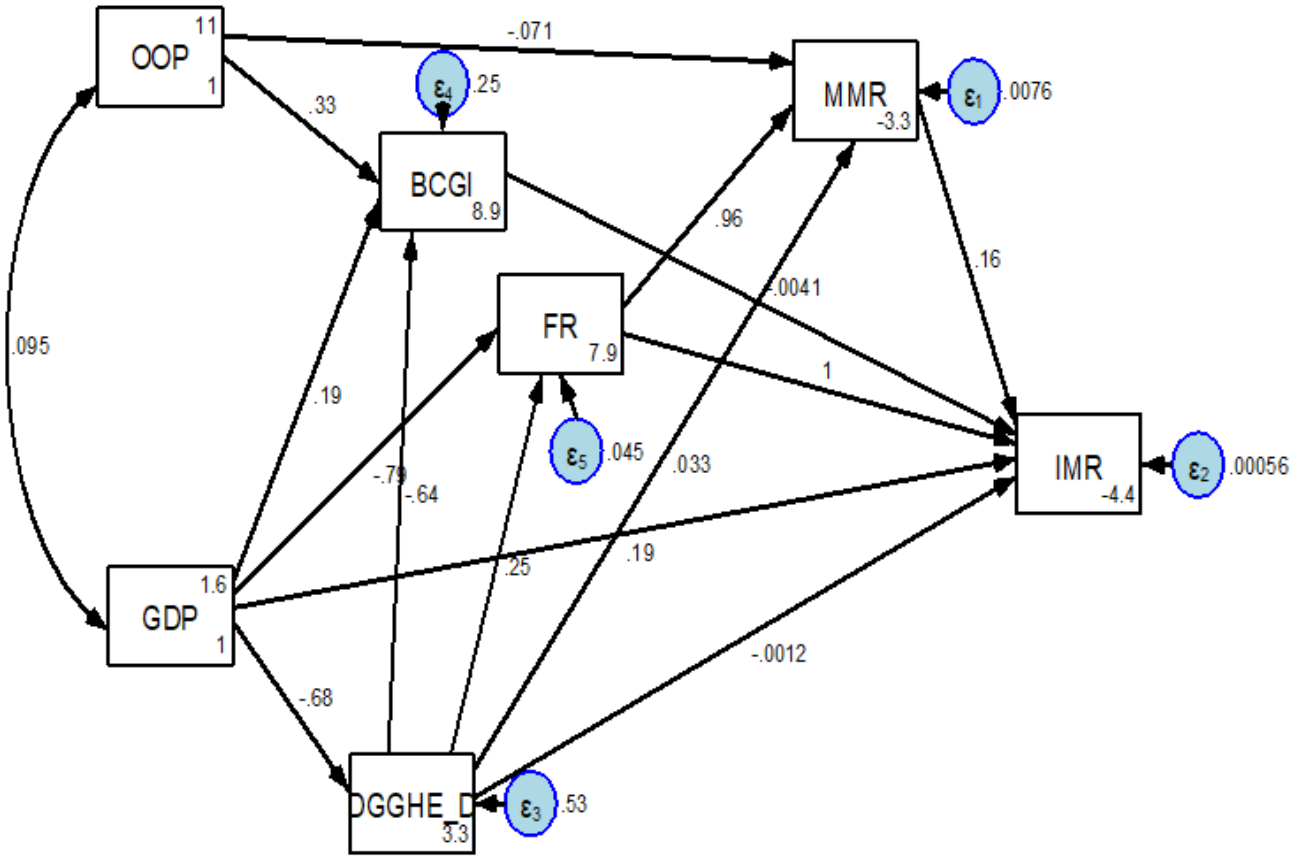

Figure 4. 3: Path diagram, path standardized coefficients of the risk factors on IMR.

Parameter estimation for Structural equation modeling

Table 4.3 shows the values of the standardized parameter estimate (direct, indirect, and total effects) of the structural equation model by employing maximum likelihood estimation which gathers the loadings for each variable on the model.

This study found evidence that out-of-pocket expenditure (OOP) has directed effects on maternal mortality ratio (MMR) ($\beta = -0.071$, $p = 0.003$) and BCG immunization ($\beta = 0.327$, $p = 0.024$), and as OOP increases by one unit, MMR decreases by 0.071 unit, and immunization (BCG) increases by 0.327 unit, while other variables were held constant. Besides, the coefficient for maternal mortality ratio (MMR) is a statistically significant predictor of infant mortality rate in Ethiopia with ($\beta = 0.141$, $p = 0.009$), while the coefficient of BCG immunization is insignificant for infant mortality rate with ($\beta = -0.0041$, $p = 0.774$). Based on loading and p-values (see in Table 4.3), the indirect path coefficient of OOP to IMR through MMR was negative and significant ($\beta = -0.012$, $p = 0.034$). Thus, MMR was significantly mediating the influence of OOP on IMR, and BCGI was not a mediator for OOP to IMR. In conclusion: H_1: There is a direct effect of out-of-pocket expenditure on health (percentage of GDP) on BCG immunization and maternal mortality ratio was fully supported and H_2: Both BCG Immunization and Maternal mortality ratio mediate the influence of out-of-pocket expenditure on health (percentage of GDP) on the infant mortality rate of the research hypothesis was partially supported.

Looking at the effects of GDP on endogenous variables, GDP has a significant total effect on fertility rate with ($\beta = -0.959$, $p < 0.001$), part of which ($\beta = -0.175$ and $p = 0.004$) was indirect through GGHE-D, and when GDP goes up by 1 unit, FR goes down by 0.175 unit due to the indirect (mediated) effect of GDP on FR in addition to any direct (unmediated) effect that GDP may have on FR. GDP was also a significant predictor of infant mortality rate ($\beta = -0.941$, $p < 0.001$) and government expenditure on health ($\beta = -0.683$, $p < 0.001$), respectively. The direct path coefficient from GDP to BCGI was insignificant ($\beta = 0.188$, $p = 0.260$). Moreover, as GDP increases by one unit, FR decreases by 0.959 units, and government expenditure on health decreases by 0.683 units, and IMR decreases by 0.941 units by 0.625, while other variables were held constant. The research hypothesis H_5: there is a direct effect of GDP on GGHE-D, BCGI, FR, and IMR is partially supported.

Further, when we consider the direct effects of government expenditure on health for other endogenous variables, the path coefficient was negative and significant for BCGI ($\beta = -0.640$, $p < 0.001$), and positive and significant for FR ($\beta = 0.256$, $p < 0.001$), and insignificant for MMR ($\beta = 0.246$, $p = 0.386$) respectively. The total effects of government expenditure on health (GGHE-D) on IMR was significant ($\beta = 0.306$, $p = 0.017$), part of which ($\beta = 0.308$, $p < 0.001$) was indirect through FR. There was also a significant effect of fertility rate on maternal mortality ratio ($\beta = 0.96$, $p < 0.001$). In conclusion, H_4: there is a direct effect of GHE on FR, BCGI, MMR, and the IMR was partially supported and H_3: a higher level of FR is associated with a higher level of MMR was supported.

Our model also revealed that there were direct positive effects between FR and IMR ($\beta = 1.168$, $p < 0.001$), and between MMR and IMR ($\beta = 0.156$, $p = 0.009$). The direct path coefficients from BCGI and GHE to IMR were insignificant with the standardized beta coefficient and p-values of ($\beta = -0.007$, $p = 0.774$) and ($\beta = -0.002$, $p = 0.915$) respectively. Based on the loadings or standardized coefficients, the FR has the highest standard coefficient ($\beta = 1.168$, $p < 0.001$) for increasing infant mortality rate (IMR), part of which was indirect through MMR ($\beta = 0.136$ and $p = 0.009$). As the fertility rate increased by one unit, the Infant mortality rate increased by 1.168, through which 0.136 unit was indirect through the maternal mortality ratio while all other variables held constant.

Table 4.4: Standardized paths for direct, indirect, and total effects of each factor of the causality of infant mortality

To ← From	**Direct**	**Indirect**	**Total**
MMR ← OOP	$-.071^{*}$	-	$-.071^{*}$
MMR ← FR	$.96^{**}$	-	$.96^{**}$
MMR ← GGHE-D	$.246(P = 0.386)$	$.032^{**}$	$.278^{**}$
MMR ← GDP	-	$-.861^{**}$	$-.861^{**}$
BCGI ← OOP	$.327^{*}$	-	$.327^{*}$
BCGI ← GDP	.188(0.260)	$.437^{*}$	$.625^{**}$
BCGI ← GGHE-D	$-.640^{**}$	-	$-.640^{**}$
FR ← GGHE-D	$.256^{**}$	-	$.256^{**}$
FR ← GDP	$-.784^{**}$	$-.175^{*}$	$-.959^{**}$
GGHE-D ← GDP	$-.683^{**}$	-	$-.683^{**}$
IMR ← MMR	$.141^{*}$	-	$.141^{*}$
IMR ← BCGI	−.007(0.774)	-	−.007(0.774)
IMR ← FR	1.032^{**}	$.136^{*}$	1.168^{*}
IMR ← GGHE-D	−.002(0.915)	$.308^{**}$	$.306^{*}$
IMR ← GDP	$.186^{**}$	-1.126^{**}	$-.941^{**}$
IMR ← OOP	-	$-.012^{*}$	$-.012^{**}$

**** Significant at 1% level of significance** ($p < 0.001$) **and * Significant at 5% level of significance** ($p < 0.05$).

In addition to the above-established relationships of the variables in the model, structural relationships between the set of variables taken into consideration, Table 4.5 represents the covariance of how much two variables move together. The relationship between MMR and IMR ($\Sigma = 0.99$), MMR and FR ($\Sigma = 0.99$), and MMR and GGHE-D ($\Sigma = 0.79$) was positive and increasing, while the relationship between MMR and BCGI ($\Sigma = -0.75$), MMR and OOP ($\Sigma = -0.17$), and MMR and GDP ($\Sigma = 0.96$) was negative and decreasing (see Table 4.5). The value of the covariance does not give any more information further than directionality [33].

Table 4.5: Fitted covariances of observed variables (standardized) for each factor of the causality of infant mortality in Ethiopia

	MMR	**BCGI**	**IMR**	**GGHE-D**	**FR**	**OOP**	**GDP**
MMR	1						
BCGI	-0.75	1					
IMR	0.99	-0.73	1				
GGHE-D	0.79	-0.79	0.81	1			
FR	0.99	-0.72	0.99	0.80	1		
OOP	-0.17	0.39	-0.11	-0.07	-0.09	1	
GDP	-0.96	0.66	-0.95	-0.69	-0.96	0.09	1

Assessment of the overall Goodness of fit

The model summary (see Table 4.6) provides the equation-by-equation goodness of fit statistics for the endogenous variable, which is displayed by equation-level variance decomposition along with the coefficient of determination (R^2), Bentler-Raykov squared multiple correlation coefficient (mc^2), and the correlation between them and their predictors (mc). The values of the coefficient of determination (R^2) and Bentler-Raykov squared multiple correlation coefficient (mc^2) are measures of goodness of fit statistics that are equivalent in recursive structure equation modeling [6].

According to the results in Table 4.6 below, the correlation between MMR and its predictors was 0.996, and the variance of MMR explained by its predictors is 0.993, or 99.3% of the variation explained by MMR in the equation for the endogenous variable MMR. Similarly, the correlation between FR and its predictors was 0.978, and 95.5% of the data fits the model for the endogenous variable FR. The model equation of the endogenous variable IMR has explained 99.5% of the total variation of implied causality.

Further, because the χ^2 goodness-of-fit criterion is very sensitive to sample size, often other descriptive measures of fit are used in addition to the absolute χ^2 test, and there should be a combination of at least two goodness-of-fit [42, 50]. The overall model fit for the structural equation model was adequate to good in terms of CFI (0.932) and TLI (0.961).

Table 4.6: Equation-level goodness of fit for the causality of infant mortality in Ethiopia

Dependent variables	Fitted variance	Predicted variance	Residual variance	R-squared	mc	mc2
MMR	49877.49	49500.85	376.65	0.993	0.996	0.993
BCGI	36.63	27.65	8.98	0.755	0.869	0.755
IMR	244.65	244.51	0.14	0.999	0.996	0.999
GGHE-D	0.276	0.13	0.15	0.466	0.682	0.466
FR	0.544	0.52	0.025	0.955	0.978	0.955
Overall				0.995		

mc = correlation between depvar and its prediction and mc^2 = Bentler-Raykov squared multiple correlation coefficient

Table 4.7 reveals residual covariances (i.e., the difference between the sample covariances based on the sample data and the covariances implied by the fitted model) provide a natural estimate of the fit of covariance structure models, and this covariance residual value was smaller (all values are less than 1.96 in absolute value). The model is supported as the implied covariance matrix did not differ significantly from the empirical covariance matrix. this smaller value indicates the best fit of the covariance structure model). The larger in absolute value of the residual covariance, the worse the fit [30].

Table 4.7: Covariance residuals for each factor of the causality of infant mortality

	MMR	BCGI	IMR	GGHE-D	FR	OOP	GDP
MMR	0.076						
BCGI	0.446	0.515					
IMR	0.047	-0.445	0.016				
GGHE-D	0.113	-0.472	0.020	0.000			
FR	0.037	-0.370	0.007	0.000	0.000		
OOP	0.731	1.034	-0.818	-1.841	-0.716	0.000	
GDP	0.022	0.000	-0.003	-0.000	-0.000	0.000	0.000

The final structural equation modeling path analysis

Results presented in Table 4.8, indicate the parameter estimation of coefficients of observed variables, the standard error, significant values, and the 95% confidence interval for the final Structural equation model for infant mortality in Ethiopia. The estimated coefficient for each observed variable represents the magnitude and direction of their influence in IMR. A positive coefficient indicates a positive relationship, while a negative coefficient suggests a negative association. Additionally, the s standard error quantifies the potential variability in the estimated coefficient due to sampling error. A p-value less than a chosen significance level (e.g., 0.05) suggests that the observed effect is unlikely to be due to chance alone. Finally, the 95% confidence intervals capture a range of plausible values within which the true population value of each coefficient is likely to fall within 95% certainty [22].

Table 4.8: The finalized and accepted Structural equation model for infant mortality

	Coef.	**Std. Err.**	**z**	$P > \|z\|$	**[95% Conf.**	**Interval]**
GGHE ←						
GDP	-0.6819305	0.1196231	-5.70	0.000	-0.9163875	-0.4474736
cons	3.343989	0.4232345	7.90	0.000	2.514464	4.173513
FR ←						
GGHE	0.2547491	0.071664	3.55	0.000	0.1142903	0.3952079
GDP	-0.7855652	0.0623844	-12.59	0.000	-0.9078363	-0.663294
cons	7.893804	1.260267	6.26	0.000	5.423727	10.36388
BCGI ←						
GGHE	-0.6394371	0.1900197	-3.37	0.001	-1.011869	-0.2670054
OOP	0.3266416	0.1577665	2.07	0.038	0.0174249	0.6358584
GDP	0.1888563	0.1671475	1.13	0.259	-0.1387468	0.5164595
cons	8.858733	2.392873	3.70	0.000	4.168789	13.54868
MMR ←						
GGHE	0.0330499	0.0531217	0.62	0.534	-0.0710667	0.1371665
FR	0.9609476	0.0323262	29.73	0.000	0.8975893	1.024306
OOP	-0.0707044	0.0289071	-2.45	0.014	-0.1273614	-0.0140475
cons	-3.269208	0.5745308	-5.69	0.000	4.395268	-2.143148
IMR ←						
MMR	0.1554458	0.0593495	2.62	0.009	0.0391229	0.2717687
BCGI	-0.0041356	0.0144006	-0.29	0.774	-0.0323602	0.024089
GGHE	-0.0012406	0.0420536	-0.03	0.976	-0.0836643	0.081183
FR	1.025088	0.0543037	18.88	0.000	0.9186548	1.131521
GDP	0.1912811	0.0313778	6.10	0.000	0.1297818	0.2527805
cons	-4.382065	0.7871316	-5.57	0.000	-5.924815	-2.839315
Mean (OOP)	10.6367	1.696609	6.27	0.000	7.311404	13.96199
Mean (GDP)	1.612898	0.3391696	4.76	0.000	0.9481377	2.277658
Var (e.GGHE)	0.5349707	0.1631493			0.2942661	0.9725676
Var (e.FR)	0.0450516	0.0196886			0.01913	0.1060976
Var (e.BCGI)	0.2451441	0.0921224			0.1173679	0.5120277
Var (e.IMR)	0.0005578	0.0002494			0.0002322	0.0013399
Var (e.MMR)	0.0076	0.0033638				
Var (OOP)	1					
Var (GDP)	1					
Cov(OOP,GDP)	0.095317	0.2215753	0.43	0.667	−0.3389626	0.5295965

e = error for each observed variable, cons = constant for each observed variable.

Therefore, based on tables 4.8 and Figure 4.3, the final structural equation model was:

$$\textbf{GHE} = -0.683 \cdot \text{GDP} + 0.0450516, \quad R^2 = 46.6\%$$

$$\textbf{FR} = 0.256 \cdot \text{GHE} - 0.786 \cdot \text{GDP} + 0.0450516, \quad R^2 = 95.5\%$$

$$\textbf{BCG} = -0.639 \cdot \text{GHE} + 0.189 \cdot \text{GDP} + 0.327 \cdot \text{OOP} + 0.2451441, \quad R^2 = 75.5\%$$

$$\textbf{MMR} = 0.034 \cdot \text{GHE} + 0.961 \cdot \text{FR} - 0.071 \cdot \text{OOP} + 0.0075514, \quad R^2 = 99.3\%$$

$$\textbf{IMR} = -0.005 \cdot \text{GHE} + 1.03 \cdot \text{FR} - 0.005 \cdot \text{BCG} + 0.156 \cdot \text{MMR} + 0.192 \cdot \text{GDP} + 0.0005578,$$

$R^2 = 99.5\%$

4.3.2 Discussion

We use SEM to estimate the direct, indirect, and total effects of variables, to accredit the presence of connections between them, and to test the hypothesized model based on World Bank data on IMR. From the sample of 20 years of World Bank data, the occurrence of IMR was decreasing and that could be justified by the advancement of mother and childcare activity in Ethiopia. Although this represents an overall decline in infant mortality between the years 2000 to the year 2019, Ethiopia accounts for the highest infant mortality rate, it was reported at 35.4 % in 2020, and the country did not achieve the extent of the sustainable development goals (SDGs) of target focuses on "ensuring healthy lives and promoting the wellbeing of for all" [10].

From the study using path analysis (directed graph) and structural equation modeling, we found that variables MMR, FR, and GDP significantly affect the IMR directly. Besides, the indirect path coefficients from OOP and FR to IMR through MMR and indirect path coefficients GGHE-D and GDP to IMR through FR were significant. However, the variable BCGI was not influential for IMR. Consequently, the FR and MMR were the mediating variables on IMR, and among all variables that had an influence on IMR, FR had the highest standardized coefficient. Complementarily, OOP, and FR had an effect on MMR directly, and GDP and GGHE-D affect MMR indirectly through FR. Besides, GGHE-D affects FR directly while GDP affects FR directly and indirectly. In our analysis, residual covariances of this SEM were smaller (all values are less than 1.96 in absolute value). This smaller value indicates the best fit of the covariance structure model. The larger in absolute value of the residual covariance, the worse the fit [23].

There were significant direct effects of OOP on MMR and BCGI. Moreover, MMR was significantly mediating the influence of OOP on IMR, but no indirect effect of OOP on IMR through BCGI. Ultimately, H1: There is a direct effect of OOP on BCGI, and MMR was fully supported while H2: Both BCGI and MMR mediate the influence of OOP on IMR of the research hypothesis was partially supported. This finding is also in line with another previous study in Egypt [1]. Considering this result, BCGI was not significantly associated with IMR. Contrary to our results, authors Roth et al (2004), revealed that IMR was lower for BCGI vaccinated than unvaccinated. This variability could be better BCGI vaccination coverage in Ethiopia, and it was 56 percent in 2000 and 90.27 percent in 2019 [25, 57].

Looking at the direct effects of GDP on other endogenous variables, GDP has a significant and negative predictor of FR part of which, was indirect through GGHE-D, and this is in addition to any direct (unmediated) effect that GDP may have on FR. This study was in accordance with the study conducted in Pacific Island countries by [31], and the study from the developed world by [56]. Our results in Ethiopia were entirely consistent

with those from studies that observed GDP had a negative association with FR, and in return, IMR was positively correlated with fertility [41, 31, 56]. This is because, in the developing world, parents consider children as virility, they use their children for work and to bring in an income for the family, and Ethiopia has a total fertility rate of 4.6 children per woman [66]. Lastly, our research hypothesis H5 was partially supported.

There had also been a significant effect of FR on MMR and this result was in line with the study conducted in Nepal by [27]. In conclusion, H4: there is a direct effect of government health expenditure on fertility rate, BCG immunization, maternal mortality ratio, and infant mortality rate was partially supported, and H3: a higher level of fertility rate is associated with a higher level of maternal mortality ratio was supported.

Furthermore, our results emphasize how important maternal mortality is. However, the variables influencing maternal mortality in this study were not sufficiently highlighted. Several factors, like as diet, care, prenatal vaccinations, and birth settings, may be connected to the overall health of mothers. It is crucial to identify these variables and other childhood vaccines in order to evaluate how they can impact the model for future study in this field.

In conclusion, this structural equation model path analysis is used to examine the different connections between observed variables (both endogenous and exogenous) and recognize both direct, indirect, and total effects of IMR based on Health Nutrition and Population Statistics indicators. The study found that maternal mortality ratio, fertility rate, and GDP per capita do have a significant impact on the infant mortality rate in Ethiopia and the study showed that there was a reverse association between IMR and GDP. However, the model shows that both government expenditure on health and BCGI were insignificant to the IMR. As we observed in the present study, reduction in fertility rate, improve the general care of mothers, and increase the per capita GDP of the country is the most important factors to decrease IMR. From the given mediators of GDP to IMR and predictors MMR, FR has the highest standard coefficients for increasing infant mortality rate (IMR) directly and indirectly through MMR. Moreover, OOP and FR were significantly predicting the MMR, but GHE was insignificant for MMR. In line with this, both government and stockholders should design and implement programs to decrease the FR and MMR, and increase per capita GDP and OOP to decrease the rate of infant mortality. Therefore, from our research hypotheses, H1 and H3 are fully supported while the rest research hypotheses H2, H4: H5: and H6: were partially supported. From our model, the covariance residual value is smaller (all values are less than 1.96 in absolute value) and it shows a good estimate of the fit of covariance structure models.

4.3.3 Limitations of the study

The study was based on secondary pooled data. Although we attempted to examine the causal relationships of variables over an extended period, many variables had missing

or incomplete values. Additionally, various literature sources identify factors influencing the infant mortality rate in Ethiopia, such as sanitation facilities, maternal nutrition (the mother's nutritional status before and during pregnancy), health infrastructure, malaria incidence, and urbanization. However, data on these variables were not available in the World Bank Development Indicators (World Bank Health Nutrition and Population Statistics), so these variables were not included in this study.

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