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*Research article*

# Bayesian joint spatial modelling of anemia and malaria in Guinea

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**Abstract:** In sub-Saharan Africa, anemia and malaria are the leading causes of morbidity and mortality among children under the age of five years. Guinea is one of the countries where the two diseases have devastating effects. Both of these diseases have been studied separately, but the two diseases exhibit inherent dependence between them, therefore, modelling them in isolation negates practical reality. This study aims at jointly estimating the spatial linear correlation between anemia and malaria, as well as to investigate the differences in contextual, socioeconomic and demographic factors affecting morbidity among children under five years in Guinea. Statistical approaches are used to handle modelling of binary outcomes with allowance for spatial components and joint responses. In particular, a latent model approach is proposed in the methodology to investigate the linear correlation between anemia and malaria allowing for spatial and non-spatial effects. All the parameters are estimated using Bayesian approach based on Markov chain Monte Carlo (MCMC) technique. According to the findings, 76.15% of children under the age of five years in Guinea were anemic, and 14.31% had malaria. Furthermore, the results showed that the child's malaria status is significantly associated with the place of residence, his/her age and ownership of television as an indicator of well being. In terms of anemia in children, there was a significant association with age, mother's education level and ethnicity group of the household head. The Nzerekore region, had both high malaria and anemia prevalences in children under five years. The latent model results showed that there was weak positive correlation between anemia and malaria in Nzerekore and Boke regions. Based on the shared component model, there was a significant unobserved risk factor that both diseases share.

**Keywords:** Bayesian inference; spatial modelling; correlation; latent model; binary outcome
**Mathematics Subject Classification:** 62F15, 62H20

## 1. Introduction

Anemia and malaria are still common in sub-Saharan Africa. According to recent reports, the number of malaria cases is on the rise, with an estimated 627,000 deaths, mostly in sub-Saharan Africa [1]. Furthermore, anemia affects roughly one-third of the world's population, with sub-Saharan Africa accounting for 67% of the cases [2,3]. Young children and women are the most severely affected by the impacts of both diseases [4, 5]. Guinea is a West African country with a high prevalence of anemia and malaria among children. According to Guinea Demographic and Health Survey (DHS) report [6], anemia and malaria prevalences in children under the age of five years were 75% and 17%, respectively. Regarding anemia, the prevalence in children has not changed significantly since 2012, falling from 77% in 2012 to 75% in 2018.

In the medical manifest, it has become increasingly obvious that human illnesses are not secluded from one another [7]. Malaria is a leading cause of anemia, among so many other factors. According to Sumbele et al. [8], the proportion of anemia attributed to malaria is usually high (79.5%). Malaria infects the blood, causing an anomalous reduction in the number of red blood cells, impeding quick recovery from anemia (a blood hemoglobin level of less than 110 g/L ) [9]. In malaria-endemic areas, almost all infants and younger children have low level of hemoglobin [10].

In the presence of malaria, anemia presents a health risk to children. It endangers children's quality of life by causing a variety of negative consequences such as delayed growth and development, liver disorder, heart murmur, spleen enlargement, and bone disease [11, 12].

In terms of public health policy, joint diseases modelling is very important. This type of analysis is more interesting than the univariate response models. It allows for the investigation of temporal patterns of disease outcomes and shared underlying spatial factors, as well as the distinction of different shared spatial factors. Specifically, joint diseases modelling aids in providing important information on the similarity of risk factors.

Spatial data analysis methods help in the search for scientific explanations of geographical patterns and distribution of diseases. Several authors have developed a univariate model, as well as a joint spatial model of diseases. Dicko et al. [13] used a multivariate backward logistic regression model to analyse the risk factors for malaria or anemia disease in pregnant women, as well as to investigate the relationship between malaria and anemia. Kateera et al. [14] assessed the prevalence of malaria parasitaemia, anemia, and malnutrition in preschool children in a rural Rwandan setting, as well as their interactions and risk factors. Ngesa et al. [15] developed a model with HIV and HSV-2 applications to capture the covariance spatial random effects. The findings of this study also shows that there is a significant positive relationship between HIV and HSV-2. Using a structured additive distributional biprobit model, Gayawan et al. [16] studied the spatial variability and correlation between fever and cough in various West African countries. Their results show that regions with huge burden of fever are also having burden of cough among children. Adebayo et al. [17] developed a geoadditive latent model for ordinal/binary indicators to investigate the effect of different types of variables on morbidity among children in Nigeria. The findings revealed a substantial geographical variations were found in likelihood of malaria in children under five years in Nigeria.

Some of the above mentioned approaches focused on the use of spatial random and non-spatial random effects. These models have also been developed to capture spatial random effects of covariance. Furthermore, most of the authors based their modelling processes on univariate separate regression, which links each illness to covariates separately. Univariate approaches assume that health outcomes are independent, which is unlikely to be true in practice since response variables are typically collected

from the same children at the same time. The authors also presented the biprobit model for joint estimation of correlated binary outcomes. The latent variables are used to consider a child to be ill if and only if the underlying latent variable has a positive value. The available covariates, which are a vector of different types of covariates, are then used to condition the latent variables.

This study explores statistical approaches to handle modelling of binary outcomes with allowance for spatial components and joint responses. In particular, a latent model approach is proposed in the methodology to investigate the linear correlation between anemia and malaria while allowing for both spatial and non-spatial effects. Univariate models and Multivariate Conditional Autoregressive (MCAR) model, as well as shared component model were explored under Bayesian framework through Markov chain Monte Carlo (MCMC) techniques.

The aim of this article is to determine the spatial linear correlation between anemia and malaria within regions, and investigate the differences in contextual, socioeconomic and demographic factors affecting morbidity among children under the age of five years in Guinea.

## 2. Methods

### *2.1. Study data*

The data used for this study were from the Guinea Multiple Indicator Cluster Survey (MICS5), which was conducted in all the eight (8) regions, including the capital city, Conakry. The survey data was collected in 2016 as part of round five of the MICS global survey program by the national institute of statistics and partners. Anemia and malaria data were gathered from 8081 households. The study was conducted for men and women aged 15 to 49, as well as children under the age of five years. The ethical considerations and survey design are explained in Guinea MICS report [18].

### *2.2. Variables of interest*

The study's dependent variables are malaria and anemia status in children under the age of five years. Both diseases were detected through a laboratory test. The variables have two levels: Positive (if the child has the illness) and negative (otherwise). Contextual, demographic, and socioeconomic variables are the independent variables considered for the analysis. The contextual covariates were administrative region and place of residence. The demographic and socioeconomic variables are: Age of the child, education level of the mother, ethnicity of the household head, wealth index, religion of the household head, access to electricity and ownership of television as an indicator of well being.

### *2.3. Statistical analyses*

For each variable in the study, a summary statistic was produced. The first part of the analysis starts with the independent univariate models ($M1$ and $M2$). The second part contains the results of multivariate analysis. In this last part, we used the multivariate CAR model analysis (MCAR), as well as the shared component model developed by Knorr-Held and Best [19], respectively $M3$ and $M4$. However, in the full model, latent variables are used to explore the linear relationship between anemia and malaria (latent model, $M5$). WinBUGS software version 1.4 (MRC Biostatistics Unit, Cambridge, UK) was used for all of the analyses, and the findings were presented as posterior means and Adjusted Odds Ratios (AOR). All the interpretations of the models were done at 95% credible intervals (CI).

### 2.4. Models specification

In this section, different models are described:

#### 2.4.1. Univariate models

Let $Y_{ijk}$ be the diseases status of child $i$ for disease $k$ in region $j$. Especially, $k = 1$ for anemia and $k = 2$ for malaria, $i = 1, 2, \ldots, n_j$, $n_j$ is the number of children in region $j$ and $j = 1, 2, \ldots, 8$. We have binary responses, $Y_{ijk} = 1$ if child $i$ in region $j$ is positive for disease $k$ and zero otherwise.

The dependent variable $Y_{ijk}$ is assumed to be Bernoulli distributed, i.e $Y_{ijk} \mid p_{ijk} \sim$ Bernoulli $\left(p_{ijk}\right)$ with an unknown mean $\mathrm{E}\left(Y_{ijk} \mid x_{ij}, u_{kj}, v_{kj}\right) = p_{ijk}$, being related to the independent variables $X_{ij}^T$ as Eq (2.1) below,

$$\begin{aligned} g\left(E\left(Y_{ijk} \mid x_{ij}, u_{kj}, v_{kj}\right)\right) &= \log\left(\frac{P\left(Y_{ijk} = 1 \mid x_{ij}, u_{kj}, v_{kj}\right)}{1 - P\left(Y_{ijk} = 1 \mid x_{ij}, u_{kj}, v_{kj}\right)}\right) \\ &= X_{ij}^T\beta_k + u_{kj} + v_{kj}, \quad i = 1, \ldots, n_j, \\ j = 1, \ldots, 8 \text{ and } p_{ijk} &= P\left(Y_{ijk} = 1 \mid x_{ij}, u_{kj}, v_{kj}\right). \end{aligned} \tag{2.1}$$

In the two separate analyses, the independent variables and random effects are introduced as follows Eqs (2.2) and (2.3) below, which are known as convolution models, i.e., Besag, York and Mollie (BMY) models [23].

$$\bullet \text{ Model } M1: \quad g\left(E\left(Y_{ij1} \mid x_{ij}, u_{1j}, v_{1j}\right)\right) = X_{ij}^T\beta_1 + u_{1j} + v_{1j}, \tag{2.2}$$

$$\bullet \text{ Model } M2: \quad g\left(E\left(Y_{ij2} \mid x_{ij}, u_{2j}, v_{2j}\right)\right) = X_{ij}^T\beta_2 + u_{2j} + v_{2j}. \tag{2.3}$$

$M1$ and $M2$ are anemia and malaria models, respectively. $X_{ij}^T$ represent a $d$-dimensional vector of covariates which is the same for the two separate models, with $\beta_k$ the corresponding vector of regression estimated coefficients, $u_{kj}$ and $v_{kj}$ representing spatial and non-spatial random effects, respectively.

#### 2.4.2. Multivariate conditional autoregressive model (MCAR model)

Carlin et al. [25] proposed multivariate conditional autoregressive (MCAR) model. In the multivariate setting, the covariates and spatial random effects $u_j = \left(u_{1j}, u_{2j}\right)^T$ are introduced as follows:

$$\bullet \; Model\ M3: \begin{cases} \text{logit}\left(p_{ij1}\right) = \alpha_1 + X_{ij}^T\beta_1 + u_{1j} \\ \text{logit}\left(p_{ij2}\right) = \alpha_2 + X_{ij}^T\beta_2 + u_{2j} \end{cases} \tag{2.4}$$

$u_j = \left(u_{1j}, u_{2j}\right)^T$ are assigned a multivariate conditional autoregressive prior,

$$u_j \sim MCAR\left(1, \Sigma_u\right),$$

where $\Sigma_u$ is the variance-covariance matrix inducing the correlation.

2.4.3. Shared component model

Knorr-Held and Best [19] introduced a new joint modelling paradigm known as the shared component model. For the case of two diseases, the relative risk of each disease depends on an unobserved spatial component shared by both diseases, and a disease-specific latent component. The relative risk for each disease is modelled as in Eq (2.5) below,

$$\bullet\ Model\ M4: \begin{cases} \text{logit}\left(p_{ij1}\right) = \alpha_1 + X_{ij}^T\beta_1 + u_{1j}\delta + u_{2j} \\ \text{logit}\left(p_{ij2}\right) = \alpha_2 + X_{ij}^T\beta_2 + \dfrac{u_{1j}}{\delta}, \end{cases} \tag{2.5}$$

where $u_{1j}$ is the shared component, common to both diseases while $u_{2j}$ is the component specific to the first disease only. These two components of random effects are modelled using conditional autoregressive priors. The unknown parameter ($\delta > 0$) is introduced to allow for differential gradient of the shared component for the two diseases. The ratio of the scaling parameters $\delta$ to $1/\delta$ compares the weight of disease 1 relative to disease 2 associated with the shared component.

2.4.4. Latent model

For full explicit model, the correlation between and within region, latent variables are used such that,

$$Y_{ijk} = \begin{cases} 0, & W_{ijk} \leq 0 \\ 1, & W_{ijk} > 0. \end{cases} \tag{2.6}$$

A child is identified with anemia or malaria if and only if the underlying latent variable has a positive value. $W_{ijk}$ is a continuous variable that is considered to be normally distributed. The approach adopted offers the opportunity of unravelling the linear relationship between two diseases.

Then, the bivariate augmented binary regression model is specified as,

$$\bullet\ Model\ M5: \begin{bmatrix} W_{ij1} \mid \theta_{ij1} \\ W_{ij2} \mid \theta_{ij2} \end{bmatrix} \sim N\left(\begin{bmatrix} \theta_{ij1} \\ \theta_{ij2} \end{bmatrix}, \Sigma\right) \text{ and } \Sigma = \begin{bmatrix} \sigma_{11}^2 & \rho_w\sigma_1\sigma_2 \\ \rho_w\sigma_1\sigma_2 & \sigma_{22}^2 \end{bmatrix} \tag{2.7}$$

with

$$\begin{bmatrix} \boldsymbol{\theta_{ij1}} \\ \boldsymbol{\theta_{ij2}} \end{bmatrix} \sim N\left(\begin{bmatrix} \boldsymbol{\gamma_{j1}} \\ \boldsymbol{\gamma_{j2}} \end{bmatrix}, \boldsymbol{\Lambda}\right) \text{ and } \boldsymbol{\Lambda} = \begin{bmatrix} \lambda_{11}^2 & \rho_b\lambda_1\lambda_2 \\ \rho_b\lambda_1\lambda_2 & \lambda_{22}^2 \end{bmatrix}, \tag{2.8}$$

where $\Sigma$ and $\Lambda$ are the within-region and between-region covariance matrices, respectively. The inclusion of within-region correlations (*i.e.*, $\rho_w$) and between-region correlations (*i.e.*, $\rho_b$) distinguishes bivariate random effects from two independent univariate random effects. We note that for two independent univariate random effects, $\rho_w = \rho_b = 0$, i.e., there is zero correlation.

According to Knorr-Held and Best [19], many diseases share common risk factors, which can provide more compelling evidence of true clustering in the underlying risk surface if similar patterns of geographical variation of related diseases are eventually realised. We consider $z_j$ unobserved covariate common to both diseases. The log relative risk $\gamma_{j1}, \gamma_{j2}$ for disease 1 and 2, respectively are given by,

$$\gamma_{j1} = \alpha_1 + \zeta_1 z_j \tag{2.9}$$

$$\gamma_{j2} = \alpha_2 + \zeta_2 z_j \tag{2.10}$$

$\alpha_p, p = 1, 2$ in Eqs (2.9) and (2.10) represents individual specific disease intercept and $\zeta_1$ and $\zeta_2$ are the distinct risk gradients related with the covariate for both the two diseases.

Now suppose we specify the following model for the log relative risk

$$\gamma_{j1} = \log\left(\eta_j\right)\delta \quad , \quad \gamma_{j2} = \log\left(\eta_j\right)/\delta,$$

where $\eta_j$ is the shared component.

In a general case, the models are expressed as,

$$\gamma_{j1} = \alpha_1 + \zeta_1 z_j + \varphi_1 q_j, \tag{2.11}$$

$$\gamma_{j2} = \alpha_2 + \zeta_2 z_j + \varphi_2 s_j. \tag{2.12}$$

The contribution of the shared component to overall relative risk is scaled by the $\delta$ scaling parameter. $q_j$ and $s_j$ are disease specific risk factors that are relevant to one or other of diseases only. The shared component $\eta_j$ will capture the spatial distribution of the $z_j$. Whereas, the cluster model for specific components $\omega_1$ and $\omega_2$ will account for the underlying distributions of the $q_j$ and $s_j$ respectively. $\varphi_1$ and $\varphi_2$ are the different risk gradients associated with the specific risk factors for the two diseases.

### *2.5. Parameters estimation*

All parameters in the Bayesian framework are assigned appropriate prior values. The posterior distributions of the parameters of interest are obtained by updating the priors with the observed data. The process model, which links the observed data to the explanatory variables and the spatial component, is the first stage. The spatial process model for spatial random effects ($u_j$) and non-spatial random effects ($v_j$) are used in the second stage. Prior distributions are then assigned to the parameters ($\beta$, $\varphi$, $\alpha$, and $\zeta$). The inverse variances of the non-spatial and spatial random effects, $\frac{1}{\sigma_v^2}$ and $\frac{1}{\tau_u^2}$ denote the hyperprior distributions, respectively.

The entire model, as well as its hierarchical structure, is illustrated below,

$$\text{level 1} \begin{cases} Y_{ijk} \mid p_{ijk} & \sim Bernoulli\left(p_{ijk}\right) \\ \text{Logit}(p_{ijk}) & = X_{ij}^T \beta_k + u_{kj} + v_{kj}, \end{cases}$$

$$i = 1, \ldots, n_j, \quad j = 1, \ldots, 8 \text{ and } \quad k = 1, 2.$$

$$\text{level 2} \begin{cases} u_j \mid u_{-j} \sim N\left(\frac{1}{n_j} \sum_{r \sim j} u_r, \frac{\tau_u^2}{n_j}\right) \\ v \sim N\left(0, \sigma_v^2\right) \end{cases}$$

$$\text{Priors} \begin{cases} \beta \sim N\left(\mu, \Sigma\right) \\ \varphi \sim N\left(\mu_\varphi, \sigma_\varphi^2\right) \\ \alpha \sim N\left(\mu_\alpha, \sigma_\alpha^2\right) \\ \zeta \sim N\left(\mu_\zeta, \sigma_\zeta^2\right) \end{cases}$$

$$\text{hyperpriors} \begin{cases} \frac{1}{\tau_u^2} \sim G\left(\alpha_\tau, \beta_\tau\right), \\ \frac{1}{\sigma_v^2} \sim G\left(\alpha_\sigma, \beta_\sigma\right). \end{cases}$$

The three components $z_j, q_j$ and $s_j$ are assumed to be independent. The $v$ capture region-wide heterogeneity via an exchangeable normal prior. Then, $u_j$ are the parameters that make this a truly spatial model by capturing regional clustering. They assumed that the random and fixed effects, and hyperpriors were mutually independent, also $u_j$ have a pairwise difference prior with joint probability density function,

$$p(\boldsymbol{u}) \propto \frac{1}{\kappa^{n/2}} \times e^{-\frac{1}{2\kappa}\sum_{j\neq r}(u_j-u_r)^2}.$$

Model estimation was carried by using a full Bayesian approach, with appropriate prior distributions assigned to all model parameters. For the intercept and coefficients, we used non-informative priors (normal prior with mean=0 and precision, inverse of variance=$1 \times 10^{-3}$). In the separate models, non-informative conditions were imposed on the inverse variances of both non-spatial and spatial random effects (gamma distributions with delimiting values of $1 \times 10^{-3}$ and $1 \times 10^{-3}$).

In the case of a joint model, the covariances matrix were assigned an inverse Wishart prior, denoted as $\Sigma \sim IW(\Omega_1, c_1)$ and $\Lambda \sim IW(\Omega_2, c_2)$ where $\Omega_1, \Omega_2$ are $p \times p$ matrices and $c_1, c_2$ are shape parameters. Since we have no prior knowledge regarding the nature or extent of dependence, we choose $\Omega_1$ and $\Omega_2$ diagonal.

In addition, a prior of $log\delta \sim N(0, 0.17)$ was assigned to an extra parameter $\delta$ for the shared component models. This prior assumes that both $\delta$ and $1/\delta$ are positive, which is supported by the positive correlations between anemia and malaria, and that the ratio of the two scaling parameters (i.e., $\delta/(1/\delta)$) is between 0.2 and 5 with 95% probability [19].

The models' parameters were estimated using the Markov chain Monte Carlo (MCMC) technique. The number of MCMC chains were 100000, with a burn in period of 5000 iterations.

To examine the convergence of the simulated sequences in the models, we used the convergence diagnostic $\hat{R}$ [20], which was close to 1 for all correlation parameters. In Figure 3, the trace plots show that no convergence issues were detected. Furthermore, the total sampling path times were calculated, which are 573 seconds, 577 seconds, 578 seconds, and 301 seconds for univariate models, MCAR Model, shared component model, and latent model, respectively.

### *2.6. Diagnostics of the models*

On the basis of the proposed model fit criterion known as the deviance information criterion (DIC) [21], the results of the joint analyses were compared to those of separate analyses. According to [21, 22], comparing the models reveals that a difference in DIC of 3 or less between two models cannot be distinguished, whereas a difference between 3 and 7 can be weakly differentiated.

The DIC formula is described as follows,

$$DIC = \bar{D} + pD = \hat{D} + 2pD. \tag{2.13}$$

$\bar{D}$ is the posterior mean of the deviance, which is a measure of goodness of fit statistic for a statistical model, $\hat{D}$ is the point estimate of the deviance, and $pD$=$\bar{D}$-$\hat{D}$ is the effective number of parameters.

## 3. Results

The study population is described in Table 1. The current analysis included 2609 children under the age of five years who had full covariate information. According to the analysis, 77.00% of the children have anemia and about 15.14% have malaria. Children are distributed by place of residence, with 71.18% living in rural areas and only 28.82% in urban areas. In terms of administrative region, the region of Boke has the highest percentage of children (17.90%), followed by Nzerekore (16.67%), and Mamou (8.78% ). The higher percentage of children (29.86%) were between the ages of 48 and 59 months, while the lower (6.52%) were between the ages of 0 and 11 months. According to the wealth index, 24.68% of households were poor and 12.04% were rich. The ethnic distribution of household heads reveals that there were more Peuls (36.80%). Furthermore, 74.74% of mothers have not attended

a formal education. The muslim religion is represented by 84.44% of the household heads. Electricity was available to 27.06% of households. The proportion of household heads who owned a television were 25.53%.

**Table 1.** Respondents' socioeconomic and demographic characteristics.

| Variables | n | Percentage (%) |
|---|---|---|
| Status of anemia | | |
| Negative | 600 | 23.00 |
| Positive | 2009 | 77.00 |
| Status of malaria | | |
| Negative | 2214 | 84.86 |
| Positive | 395 | 15.14 |
| Place of residence | | |
| Urban | 752 | 28.82 |
| Rural | 1857 | 71.18 |
| Administrative Region | | |
| Boke | 467 | 17.90 |
| Conakry | 268 | 10.27 |
| Faranah | 369 | 14.14 |
| Kankan | 308 | 11.81 |
| Kindia | 288 | 11.04 |
| Labe | 245 | 9.39 |
| Mamou | 229 | 8.78 |
| NZerekore | 435 | 16.67 |
| Age of Child (months) | | |
| 0 – 11 | 170 | 6.52 |
| 12-23 | 427 | 16.37 |
| 24 – 35 | 523 | 20.05 |
| 36 – 47 | 710 | 27.21 |
| 48 – 59 | 779 | 29.86 |
| Education level of mother | | |
| None | 1950 | 74.74 |
| Primary | 329 | 12.61 |
| Secondary school or above | 330 | 12.65 |
| Ethnicity of household head | | |
| Soussou | 435 | 16.67 |
| Peul | 960 | 36.80 |
| Malinke | 665 | 25.49 |
| Kissi | 156 | 5.98 |
| Toma | 62 | 2.38 |
| Guerze/Kono/Mano | 187 | 7.17 |
| Other | 144 | 5.52 |

*Continued on next page*

| Variables | n | Percentage (%) |
|---|---|---|
| Religion of household head | | |
| Muslim | 2203 | 84.44 |
| Christian | 353 | 13.53 |
| Others(Animist, no religion) | 53 | 2.03 |
| Wealth index | | |
| Poorest | 644 | 24.68 |
| Second | 671 | 25.72 |
| Middle | 522 | 20.01 |
| Fourth | 458 | 17.55 |
| Richest | 314 | 12.04 |
| Access to electricity | | |
| Yes | 706 | 27.06 |
| No | 1903 | 72.94 |
| Ownership of television | | |
| Yes | 666 | 25.53 |
| No | 1943 | 74.47 |

Table 2 shows estimates of anemia and malaria prevalence among children in the country and by administrative region. In Guinea, the prevalences in children under the age of five years, for anemia and malaria were 76.15% and 14.31%, respectively. According to the finding by region, malaria infection and anemia were most prevalent in children in Nzerekore region with 29.48% and 83.60%, respectively. The Conakry region, on the other hand, had the lowest prevalences of malaria infection (1.36%) and anemia (70.32%) in children. In Figure 1, the different regions are grouped according to the levels of the prevalences.

**Table 2.** Prevalence estimates and corresponding 95% credible interval based on univariate models (anemia and malaria).

| | Prevalence of anemia (95% CI) | Prevalence of malaria (95% CI) |
|---|---|---|
| **Guinea** | 0.76 [0.74 0.78] | 0.14 [0.12 0.15] |
| Boke | 0.77 [0.75 0.81] | 0.07 [0.05 0.10] |
| Conakry | 0.70 [0.66 0.74] | 0.01 [0.00 0.03] |
| Faranah | 0.77 [0.74 0.80] | 0.21 [0.17 0.25] |
| Kankan | 0.77 [0.73 0.81] | 0.21 [0.16 0.25] |
| Kindia | 0.79 [0.75 0.82] | 0.09 [0.06 0.12] |
| Labe | 0.72 [0.67 0.76] | 0.09 [0.05 0.13] |
| Mamou | 0.71 [0.66 0.75] | 0.15 [0.11 0.20] |
| Nzerekore | 0.83 [0.80 0.86] | 0.29 [0.25 0.33] |

### 3.1. Comparison between models

Table 3 presents the fitness measure using Deviance Information Criteria (DIC) from the five models $M1$, $M2$, $M3$, $M4$ and $M5$. $M1$ and $M2$ are separate univariate anemia and malaria models, respectively. $M3$ represents the MCAR model, $M4$ the shared component model and $M5$ the latent model. The best model preferred has the lowest DIC. By making a comparison of the DICs, the latent model (M5) was found to be the best overall because it generated the smallest DIC value (-1014.75) [21, 22].

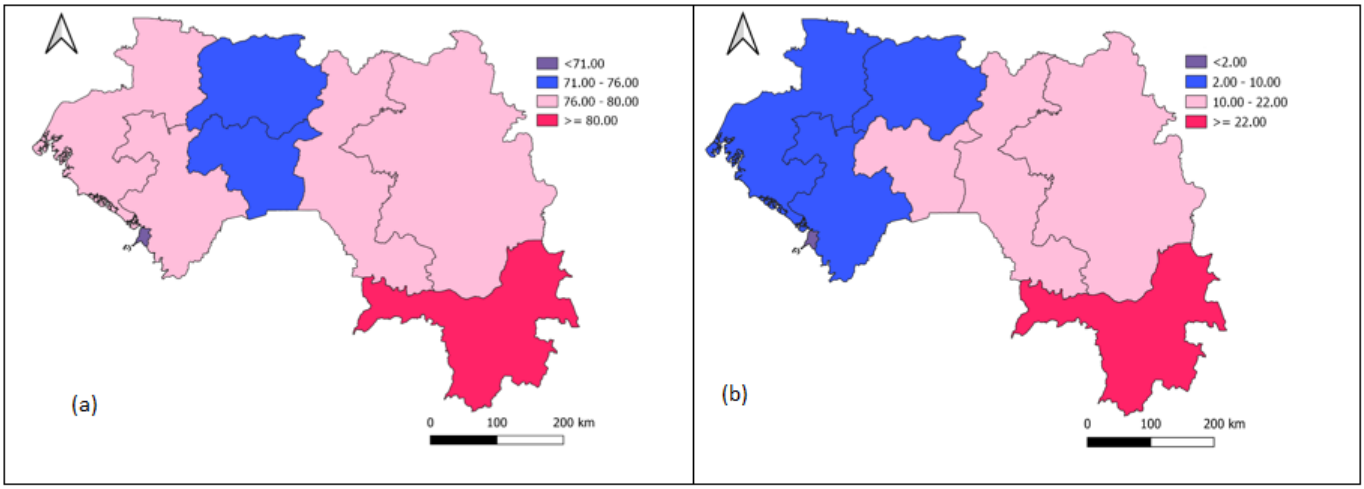


**Figure 1.** (a) Prevalence estimates of anemia; (b) Prevalence estimates of malaria.

**Table 3.** Models comparison statistics.

| | $\bar{D}$ | $\widehat{D}$ | $pD$ | $DIC$ |
|---|---|---|---|---|
| **Separate models** | | | | |
| $M1$ | 2694.93 | 2667.97 | 26.96 | 2721.89 |
| $M2$ | 1932.97 | 1903.31 | 29.66 | 1962.62 |
| **Multivariate CAR models,** $M3$ | | | | |
| Anemia | 2694.12 | 2668.02 | 26.10 | 2720.22 |
| Malaria | 1930.29 | 1902.48 | 27.81 | 1958.10 |
| Total | 4624.41 | 4570.50 | 53.91 | 4678.32 |
| **Shared Component models,** $M4$ | | | | |
| Anemia | 2695.26 | 2670.42 | 24.84 | 2720.10 |
| Malaria | 1932.58 | 1905.11 | 27.47 | 1960.05 |
| Total | 4627.84 | 4575.53 | 52.31 | 4680.15 |
| **Latent model** $M5$ | -1015.80 | -1016.85 | 1.05 | -1014.75 |

Notes: $\bar{D}$ is the posterior mean of the deviance, $\hat{D}$ is the point estimate of the deviance, and pD=$\bar{D}$-$\hat{D}$ is the effective number of parameters.

### 3.2. Results from the separate models

The results presented in the Table 4 include the estimation of parameters from models $M1$ and $M2$ via independent univariate models. In anemia model ($M1$), the covariates such as child's age, mother's education level and ethnicity of household head were found to be significant. Indeed, children whose mothers attained secondary school or above were less likely to have anemia (AOR: 0.67, CI [0.49 0.90]) than children of mothers with no formal education (no educational attainment). The children from 48–59 months age group were less likely to be anemic (AOR: 0.47, CI [0.29 0.70]) than those from 0–11 months age group. Furthermore, children who were under the responsibility of household heads from Kissi, Peul ethnic group are less likely to have anemia (AOR: 0.48 [0.23 0.92], AOR: 0.58 [0.42 0.79], respectively) compared to their counterparts whose household heads are from Soussou ethnic group. With reference to malaria model ($M2$), place of residence and child's age were significantly associated with the status of malaria in children. When compared to children from rural to those from urban areas, children from rural areas were more likely to have malaria (AOR: 3.07, CI [1.64 5.43]). Furthermore, children aged 12–23 months and 24–35 months were less likely to have malaria than children aged 0–11 months, (AOR: 0.45, CI [0.22 0.80]), (AOR: 0.60, CI [0.39 0.86]) respectively.

### 3.3. Results from joint analyses (MCAR)

A Multivariate conditional autoregressive model (MCAR) was used to jointly model risk of anemia and malaria diseases ($M3$ in Table 5). Estimating both diseases simultaneously perform better than the results of the univariate analysis. The findings revealed that the child's malaria status was significantly associated with the place of residence, child's age and ownership of television. Indeed, children in rural areas were more likely to have malaria (AOR: 3.11, CI [1.77 5.25]) than those in urban areas. Children in households where the head did not own a television were more likely to have malaria (AOR: 2.26, CI [1.02 4.49]) than those in households where the head owned a television. Malaria is more likely to affect children aged 48–59 months (AOR: 2.62, CI [1.34 4.97]) than those aged 0–11 months.

The findings also showed that anemia in children is significantly related to the child's age, education level of the mother, and ethnicity group of the household head. As expected, children whose mothers attained secondary school or above were less likely to be anemic (AOR: 0.67, CI [0.49 0.90]) than children whose mothers had no formal education (no educational attainment). Children aged 48–59 months were more likely to be at risk of anemia (AOR: 1.35, CI [1.05 1.71]) than those aged 0–11 months. We also found that children with Peul household heads are less likely to have anemia (AOR: 0.63, CI [0.45 0.87]) than their counterparts whose leaders are Soussou.

### 3.4. Results from the latent model

To grasp disease explanations clearly, we need to understand any possible correlations that may exist between that disease and another. Table 6 shows the results of the correlation (model $M5$). The posterior correlation between anemia and malaria among the children associated with $\Sigma$, i.e., $\frac{\Sigma_{12}}{\sqrt{(\Sigma_{11}\Sigma_{22})}}$, has mean 0.13 with interval estimates [0.09 0.17]. This means that there is a weak positive correlation between anemia and malaria among the children under five years in Guinea. The administrative region findings also revealed a weak positive linear relationship between anemia and malaria in Boke and Nzerekore regions, 0.20 [0.11 0.29] and 0.23 [0.14 0.32], respectively. The interaction between these two diseases is that malaria parasites infect red blood cells after entering the bloodstream via an infective mosquito bite. Red blood cells rupture at the end of the infection cycle. This process reduces the number of red blood cells, which can lead to anemia in children.

**Table 4.** Posterior means (95%CI) estimates for anemia and malaria from the two separate models $M1$ and $M2$, respectively.

| | AOR (95% CI), M1 | AOR (95% CI), M2 |
|---|---|---|
| Place of residence | | |
| Urban | 1 | 1 |
| Rural | 1.32 [0.93 1.82] | 3.07 [1.64 5.43] |
| Age of Child (months) | | |
| 0-11 | 1 | 1 |
| 12-23 | 1.30 [0.77 2.04] | 0.45 [0.22 0.80] |
| 24-35 | 1.02 [0.62 1.56] | 0.60 [0.39 0.86] |
| 36-47 | 0.63 [0.39 0.93] | 0.99 [0.69 1.36] |
| 48-59 | 0.47 [0.29 0.70] | 1.06 [0.77 1.42] |
| Wealth index | | |
| Poorest | 1 | 1 |
| Second | 0.89 [0.66 1.17] | 1.01 [0.74 1.35] |
| Middle | 0.75 [0.55 1.00] | 0.82 [0.57 1.14] |
| Fourth | 0.71 [0.42 1.15] | 0.93 [0.38 1.93] |
| Richest | 0.62 [0.31 1.12] | 1.43 [0.30 4.46] |
| Education level of mother | | |
| None | 1 | 1 |
| Primary | 1.06 [0.78 1.43] | 0.84 [0.55 1.21] |
| Secondary school or above | 0.67 [0.49 0.90] | 0.78 [0.44 1.26] |
| Ethnicity of household head | | |
| Soussou | 1 | 1 |
| Peul | 0.58 [0.42 0.79] | 1.06 [0.59 1.77] |
| Malinke | 0.86 [0.59 1.21] | 1.48 [0.74 2.64] |
| Kissi | 0.48 [0.23 0.92] | 1.44 [0.49 3.26] |
| Toma | 1.84 [0.52 4.99] | 0.93 [0.23 2.52] |
| Guerze/Kono/Mano | 1.05 [0.44 2.16] | 2.16 [0.67 5.23] |
| Other | 0.89 [0.53 1.43] | 1.30 [0.58 2.49] |
| Religion of household head | | |
| Muslim | 1 | 1 |
| Christian | 1.79 [0.90 3.24] | 1.47 [0.61 2.97] |
| Others(Animist, no religion) | 0.93 [0.30 2.28] | 2.28 [0.73 5.40] |
| Ownership of television | | |
| Yes | 1 | 1 |
| No | 1.09 [0.71 1.61] | 2.19 [0.96 4.75] |
| Access to electricity | | |
| Yes | 1 | 1 |
| No | 0.91 [0.59 1.35] | 1.50 [0.73 2.79] |

**Table 5.** Posterior means (95%CI) estimates for anemia and malaria from the joint models, *M*3.

| | AOR (95% CI), anemia | AOR (95% CI), malaria |
|---|---|---|
| Place of residence | | |
| Urban | 1 | 1 |
| Rural | 1.35 [0.94 1.91] | 3.11 [1.77 5.25] |
| Age of Child (months) | | |
| 0-11 | 1 | 1 |
| 12-23 | 2.45 [1.54 3.79] | 1.53 [0.73 2.99] |
| 24-35 | 2.81 [2.09 3.83] | 2.53 [1.26 4.85] |
| 36-47 | 2.20 [1.63 2.90] | 2.73 [1.39 5.20] |
| 48-59 | 1.35 [1.05 1.71] | 2.62 [1.34 4.97] |
| Wealth index | | |
| Poorest | 1 | 1 |
| Second | 0.89 [0.65 1.18] | 1.01 [0.76 1.33] |
| Middle | 0.75 [0.54 1.00] | 0.82 [0.57 1.13] |
| Fourth | 0.70 [0.40 1.15] | 0.95 [0.41 1.85] |
| Richest | 0.61 [0.30 1.12] | 1.40 [0.32 3.96] |
| Education level of mother | | |
| None | 1 | 1 |
| Primary | 1.07 [0.78 1.44] | 0.84 [0.55 1.21] |
| Secondary school or above | 0.67 [0.49 0.91] | 0.78 [0.44 1.25] |
| Ethnicity of household head | | |
| Soussou | 1 | 1 |
| Peul | 0.63 [0.45 0.87] | 1.10 [0.64 1.79] |
| Malinke | 0.90 [0.60 1.31] | 1.60 [0.83 2.81] |
| Kissi | 0.48 [0.22 0.93] | 1.59 [0.53 3.73] |
| Toma | 1.77 [0.48 4.92] | 1.04 [0.26 2.85] |
| Guerze/Kono/Mano | 0.99 [0.40 2.11] | 2.41 [0.72 6.06] |
| Other | 0.91 [0.53 1.48] | 1.33 [0.60 2.52] |
| Religion of household head | | |
| Muslim | 1 | 1 |
| Christian | 1.73 [0.87 3.15] | 1.47 [0.60 3.04] |
| Others(Animist, no religion) | 0.87 [0.28 2.16] | 2.29 [0.72 5.53] |
| Ownership of television | | |
| Yes | 1 | 1 |
| No | 1.09 [0.69 1.64] | 2.26 [1.02 4.49] |
| Access to electricity | | |
| Yes | 1 | 1 |
| No | 0.90 [0.57 1.34] | 1.53 [0.79 2.75] |

**Table 6.** Linear correlations between anemia and malaria by administrative region from latent model, $M5$.

| | Posterior means (95% CI) estimates |
|---|---|
| **Guinea** | 0.13 [0.09 0.17] |
| Boke | 0.20 [0.11 0.29] |
| Conakry | 0.10 [-0.02 0.22] |
| Faranah | 0.09 [-0.01 0.19] |
| Kankan | 0.11 [-0.01 0.21] |
| Kindia | 0.10 [-0.02 0.21] |
| Labe | 0.10 [-0.03 0.22] |
| Mamou | 0.10 [-0.03 0.23] |
| Nzerekore | 0.23 [0.14 0.32] |

### *3.5. Results from shared component model*

The values of the two disease-specific components $\omega_1$ and $\omega_2$ are shown in Table 7 (model $M4$). The specific component for malaria has a distinct spatial pattern, with higher values in the regions of Kankan (0.41) and Nzerekore (0.40) and lower value (-0.62) in the region of Conakry. On the other hand, the anemia specific component shows a different spatial pattern with less variation and higher value in the region of Conakry (0.03).

According to the shared component, the posterior mean value is significant in the region of Nzerekore 0.09 [0.01 0.58]. This suggests that there is unobserved risk factor that is shared by both diseases. In Figure 2, the different regions are grouped according to the anemia and malaria specific factors, as well as the shared unobserved factor.

Furthermore, in Table 8, the findings show that Nzerekore region has a higher overall odds ratio of anemia and malaria than the other regions, with 1.09 [0.19 1.54] and 1.67 [1.07 2.51], respectively.

**Table 7.** Estimates posterior means (95%CI) estimates of the different risk gradients associated with the specific risk factors and shared risk factors for anemia and malaria by administrative region, $M4$.

| Region | Specific risk factors for anemia | Specific risk factors for malaria | Shared risk factors |
|---|---|---|---|
| Boke | -0.01 [-1.08 0.30] | -0.40 [-0.83 0.03] | -0.02 [-0.41 0.17] |
| Conakry | 0.03 [-0.97 0.44] | -0.62 [-1.65 0.11] | -0.08 [-0.86 0.16] |
| Faranah | -0.17 [-2.72 0.12] | 0.37 [-0.01 0.75] | 0.03 [-0.13 0.39] |
| Kankan | -0.23 [-2.89 0.12] | 0.41 [-0.05 0.89] | 0.02 [-0.21 0.46] |
| Kindia | -0.04 [-1.49 0.26] | -0.14 [-0.53 0.25] | 0.01 [-0.20 0.21] |
| Labe | -0.15 [-1.64 0.11] | -0.28 [-0.74 0.09] | -0.08 [-0.44 0.06] |
| Mamou | -0.18 [-2.67 0.11] | 0.26 [-0.10 0.70] | 0.01 [-0.17 0.28] |
| Nzerekore | -0.08 [-2.39 0.35] | 0.40 [-0.08 0.89] | 0.09 [0.01 0.58] |

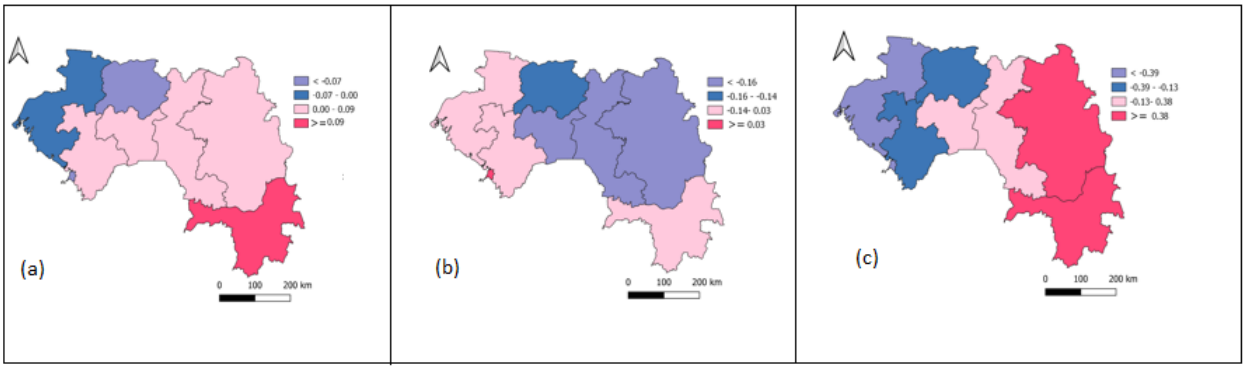


**Figure 2.** (a) Mean estimates of shared risk factors, (b) Mean estimates of specific risk factors of anemia, and (c) Mean estimates of specific risk factors of malaria by administrative region.

**Table 8.** Posterior means (95%CI) estimates of overall odds of anemia and malaria, *M*4.

| Region | Overall odds of anemia | Overall odds of malaria |
|---|---|---|
| Boke | 1.02 [0.16 1.32] | 0.67 [0.45 0.94] |
| Conakry | 0.97 [0.14 1.29] | 0.55 [0.18 1.06] |
| Faranah | 0.94 [0.13 1.18] | 1.52 [1.09 2.09] |
| Kankan | 0.89 [0.11 1.15] | 1.57 [1.03 2.35] |
| Kindia | 1.02 [0.16 1.36] | 0.90 [0.60 1.32] |
| Labe | 0.86 [0.10 1.07] | 0.71 [0.45 1.01] |
| Mamou | 0.91 [0.11 1.14] | 1.34 [0.91 1.97] |
| Nzerekore | 1.09 [0.19 1.54] | 1.67 [1.07 2.51] |

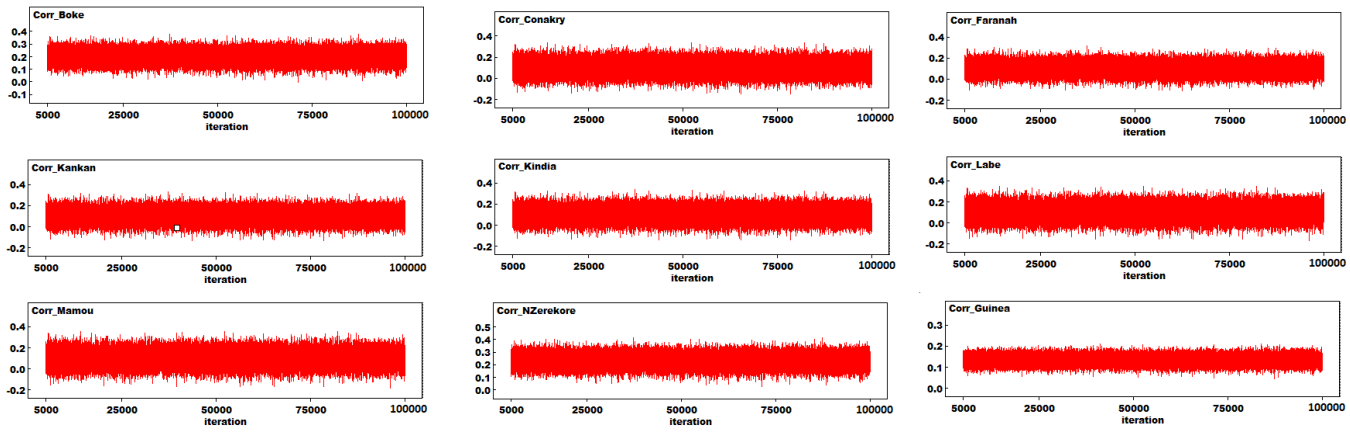


**Figure 3.** Trace plots of the correlation parameters based on latent model (*M*5).

## 4. Discussion

The study was designed to estimate spatial linear correlation between anemia and malaria, as well as to investigate the differences in socioeconomic and demographic factors affecting morbidity among children under the age of five years in Guinea.

In the statistical framework, we proposed to fit two separate models and joint models, and compared their performance. Joint modelling helps in the stabilization of parameter estimates, particularly in small area estimation where sample sizes at regions are small with respect to each disease. Furthermore, from an epidemiological standpoint, joint modelling aids in determining divergent and similar disease patterns, as well as understanding diseases associations.

The results of the MCAR model revealed some significant factors between contextual, socio-demographic factors and epidemiological issues. The malaria status of the child was significantly associated with the child's age, place of residence and ownership of television. Indeed, children in rural areas were more likely to have malaria than those in urban areas. Children from households where the head did not own a television were more likely to have malaria than those from households where the head did own a television. Then, as children grow older, malaria and anemia become more likely to affect them. We also found that anemia in children is significantly related to the education level of the mother, and the ethnicity of the household head. Previous studies has also shown the obtained results. Mother's education, which is known to have strong tie with child health, children whose mothers are educated are less likely to be ill [26]. A plausible explanation is that educational attainment has strong influence on women behaviour. Furthermore, since the majority of well-educated women are likely to be employed in formal jobs, they have the financial resources to care for their children. In terms of child age, the two health outcomes were significant in the positive direction, indicating that as a child's age increases, he/she is more likely to suffer from both illnesses. One would normally have considered it to be the other way round because age is known to have strong a relationship with children's health, and almost all infants and young children have low haemoglobin levels in malaria-endemic areas [10]. Among those who are affected by malaria, the blood is infected and the result is an abnormal drop in the number of red blood cells, which compromises the rapid recovery from anemia [27]. We also note that the results in the right direction were found with the age of the child in the separate models of these illnesses. As a child grows older, the risks of getting anemia and malaria decreases. This agrees with the findings of other morbidity investigations, such as the anemia study conducted by Osório et al. [28] in children aged 6–59 months.

Malaria was more prevalent in children from rural areas than those from urban areas. It is well known that most healthcare services in most African countries are concentrated in urban rather than rural areas. Hospitals in urban areas have a greater supply of medicines. Children in urban centers have better access to essential services, which could explain why they are less likely to get sick [29]. Whereas, for anemia the difference between children living in urban and rural settings was not significant. In agreement with previous studies [30], there were no significant differences in anemia prevalence among children living in a city, township and rural area.

Children whose household heads are from the Peul ethnic group are less likely to have anemia than their counterparts whose household heads are Soussou ethnic group. This could be explained by ethnic differences being viewed as differences in cultural attitudes and practices [31]. The lifestyle may pose a significant challenge to the health status of individuals [32]. The fact of having a television in the household is associated with reduction in the odds of malaria, but not for ownership television and anaemia. This component's involvement could be explained by the fact that having good

information and well-being advice from healthcare providers contribute in more prevention strategies being practiced [33]. The reasoning could also be to do with differing associated risk behaviours. for example, more time spent outside if a television is not available, or owning a television could reflect what the house itself looks like, where a household without a television might generally be less well built, therefore increasing possible exposure to vectors.

The findings also show that there is a relationship between epidemiological outcomes (anemia and malaria), particularly in the Boke and Nzerekore regions. These results could not be accounted for by univariate models. In Nzerekore region, the prevalences of anemia and malaria in children under the age of five years are high. These findings may explain the significance of the relationship of these two outcomes. In addition, we found that the posterior mean value of the shared component is significant in Nzerekore region. This means that there is an unobserved covariate common to both diseases.

In the limitations, our models make a number of assumptions. We assumed that the random and fixed effects, as well as the hyperpriors, were all independent of one another. Similarly, we assumed that a single latent variable for each disease may adequately capture the spatial structure of the disease-specific covariates in our shared component models. In addition, the models assumed that the shared and specific components were not dependent, ignoring the possibility of interaction between the observed covariates.

In the analysis, only contextual, demographic and socioeconomic factors were used. MICS data used was from a cross-sectional study, it was impossible to determine the temporality or causality of the observed relationships with anemia or malaria in children. Regional medical conditions and epidemic prevention conditions as variables to analyze the spatial linearity between anemia and malaria could be very useful, but no information from those variables was collected. Likewise, sample error could have an influence on the results obtained. The linear correlation between variable $Y_1$ at site $i$ and variable $Y_2$ at site $j$ is another potential limitation of the model. This has not been considered and may be thought of as spatial cross-correlation of binary outcomes.

It should be noted that the administrative region, which is made up of several smaller geographical units (prefectures, sub-prefectures), is the unit of analysis in the study for spatial correlation. As a result, variations in these small units are to be expected, as is the correlation between anemia and malaria. In the future, it would be useful to consider these smaller units.

## 5. Conclusions

In conclusion, the methods used in this study allows for the investigation of the relationship between anemia and malaria in children in Guinea. As a result, the study provides an inherent picture of the prevalences of anemia and malaria among children in Guinea's different regions. The findings suggest that effective policy interventions should focus on contextual, socioeconomic, and demographic factors in order to reduce anemia and malaria prevalences. Furthermore, prevent malaria in general across the country, but particularly in Boke and Nzerekore regions, since its could induce anemia in children.

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## Conflict of interest

The authors declare that there are no conflicts of interest.

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