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MODELING THE SPREAD OF MPOX VIRAL DISEASE IN AFRICAN COUNTRIES USING A BAYESIAN HIERARCHICAL MODEL

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Abstract. Mpox, a zoonotic disease similar to smallpox, has garnered increasing attention due to its sporadic outbreaks across different regions. This study employs a comprehensive statistical approach, combining Poisson regression, Generalized Linear Mixed Models (GLMM), and Bayesian Hierarchical Models (BHM) to analyze the spread of Mpox. The analysis accounts for regional variations in transmission dynamics and provides probabilistic estimates of key epidemiological parameters. Our findings reveal significant variability in the impact of covariates such as population density, healthcare capacity, and mobility on Mpox transmission. The Bayesian Hierarchical Model, in particular, offers a robust framework for understanding the complex transmission dynamics of the disease across diverse regions. These insights underscore the necessity of region-specific public health strategies to effectively control and prevent Monkeypox outbreaks.

Keywords: Mpox; Bayesian hierarchical model; Poisson regression; GLMM; epidemiology; regional variability.

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1. INTRODUCTION

Monkeypox, a zoonotic disease caused by the Monkeypox virus, has emerged as a significant public health concern due to its increasing prevalence and the potential for widespread outbreaks beyond endemic regions, [1, 2, 3, 4]. The virus is closely related to the variola virus, which causes smallpox, and shares many clinical features such as fever, rash, and lymphadenopathy, [5, 6]. However, unlike smallpox, monkeypox continues to pose a threat even in the post-smallpox eradication era, particularly in areas with limited healthcare infrastructure, [7, 8, 9].

Historically confined to certain regions of Central and West Africa, Mpox has recently been reported in various non-endemic countries, triggering global concerns about its potential to cause widespread outbreaks, [10]. The disease is primarily transmitted through direct contact with the blood, bodily fluids, or skin lesions of infected animals or humans, [11]. Secondary human-to-human transmission occurs through respiratory droplets and contaminated objects, making it a highly contagious disease, [12].

Understanding the transmission dynamics of Mpox is crucial for developing effective public health interventions, [13]. Traditional epidemiological models, such as the SIR (Susceptible-Infectious-Recovered) model, have been widely used to estimate key parameters like the basic reproduction number (R0) and to predict the course of infectious disease outbreaks, [14, 15]. However, these models often assume homogeneous mixing within populations, which may not accurately reflect the complex and heterogeneous nature of real-world populations. Factors such as regional variations in population density, healthcare access, and social behavior can significantly influence disease transmission dynamics.

To address these limitations, this study employs a Bayesian Hierarchical Model (BHM) to analyze the spread of Mpox across different regions. The BHM framework allows for the incorporation of regional heterogeneity and provides a probabilistic approach to estimating key epidemiological parameters. By accounting for regional variations, this model offers a more complex and detailed understanding of Mpox transmission dynamics and provides a robust method for forecasting the potential spread of the disease. The findings of this study underscore the importance of region-specific public health strategies to effectively control and prevent monkeypox outbreaks.

The study of infectious disease dynamics has a rich history, with numerous models developed to understand and predict the spread of diseases, [16, 17, 18]. The SIR (Susceptible-Infectious-Recovered) model is one of the most foundational approaches in epidemiology, providing a framework for estimating the basic reproduction number (R0) and understanding the progression of infectious diseases within a population, [19, 20]. However, the SIR model's assumption of homogeneous mixing has been critiqued for oversimplifying the complex social and environmental factors that influence disease transmission, [21, 22].

Recent advances in epidemiological modeling have sought to address these limitations by incorporating more realistic assumptions about population structure and behavior [23, 24, 25]. Bayesian Hierarchical Models (BHMs) have emerged as a powerful tool for modeling infectious disease spread, particularly in contexts where regional heterogeneity plays a significant role, [26]. Unlike traditional compartmental models, BHMs allow for the incorporation of multiple levels of data hierarchy, enabling researchers to account for variations in disease transmission across different regions or subpopulations, [27].

The application of BHMs in epidemiology has been well-documented, with successful implementations in modeling the spread of diseases such as Ebola, COVID-19, and Zika virus, [28, 29]. These models have provided valuable insights into the factors driving regional differences in disease transmission and have highlighted the importance of tailoring public health interventions to specific local contexts.

In the context of Mpox, the use of BHMs is particularly relevant given the disease's sporadic outbreaks and the significant variability in transmission dynamics across different regions. Studies have shown that factors such as population density, healthcare infrastructure, and mobility patterns can greatly influence the spread of monkeypox, making it essential to adopt modeling approaches that can capture this complexity. This study builds on the existing literature by applying a BHM to model the spread of monkeypox to provide more accurate estimates of transmission dynamics and inform targeted public health responses.

2. Data and Methodology

2.1. Data Collection. Data for this study was gathered from multiple authoritative sources, including the World Health Organization (WHO) and various national health agencies. The

dataset comprises detailed records of confirmed monkeypox cases across different regions over a defined period(From 1 st January 2024 to 23 Aug 2024, a total cases of Mpox have been recorded from 13 African Union (AU) Member States (MS): Burundi, Cameroon, Central Africa Republic, Congo, Côte d'Ivoire, Democratic Republic of Congo, Gabon, Liberia, Kenya, Nigeria, Rwanda, South Africa and Uganda.). In addition to epidemiological data, we incorporated demographic and socio-economic variables such as population density, healthcare capacity, and human mobility patterns. These covariates are critical for understanding the factors driving regional variations in monkeypox transmission. A summary of the key variables utilized in the analysis is provided in Table 1.

TABLE 1. Summary of Data variable description

Variable	**Description**	**Source**
Number of Cases	Confirmed monkeypox cases by region and time	WHO
Population Density	Number of people per square kilometer	National Census
Healthcare Capacity	Number of hospital beds per 1000 people	National Health Agency
Mobility Data	Movement patterns within and between regions	Mobile Data Provider

3. METHODOLOGY

3.1. Poisson Regression Model. The Poisson regression model is used for count data, assuming that the response variable follows a Poisson distribution.

3.1.1. *Model Specification.* Let Y_i be the count of monkeypox cases for observation i. The Poisson regression model is given by:

$$Y_i \sim \text{Poisson}(\lambda_i)$$

where λ_i is the rate parameter for observation i, linked to the covariates via a log link function:

$$\log(\lambda_i) = \beta_0 + \beta_1 X_{i1} + \beta_2 X_{i2} + \cdots + \beta_p X_{ip}$$

Where, $X_{i1}, X_{i2}, \ldots, X_{ip}$ are the covariates (population density, healthcare capacity, mobility index), and $\beta_0, \beta_1, \ldots, \beta_p$ are the coefficients to be estimated.

3.1.2. *Likelihood.* The likelihood function for the Poisson model is:

$$L(\beta;Y) = \prod_{i=1}^{n} \frac{\lambda_i^{Y_i} e^{-\lambda_i}}{Y_i!}$$

Taking the log-likelihood:

$$\log L(\beta;Y) = \sum_{i=1}^{n} (Y_i \log(\lambda_i) - \lambda_i - \log(Y_i!))$$

Substituting $\lambda_i = e^{\beta_0 + \sum_{j=1}^{p} \beta_j X_{ij}}$:

$$\log L(\beta;Y) = \sum_{i=1}^{n} \left(Y_i \left(\beta_0 + \sum_{j=1}^{p} \beta_j X_{ij} \right) - e^{\beta_0 + \sum_{j=1}^{p} \beta_j X_{ij}} - \log(Y_i!) \right)$$

Maximizing this log-likelihood gives the maximum likelihood estimates (MLE) of the coefficients β.

3.2. Generalized Linear Mixed Model (GLMM). The GLMM extends the Poisson regression by incorporating random effects, accounting for variability across groups (e.g., different countries).

3.2.1. *Model Specification.* Let Y_{ij} be the count of monkeypox cases for observation i in group j. The GLMM can be expressed as:

$$Y_{ij} \sim \text{Poisson}(\lambda_{ij})$$

The rate λ_{ij} is linked to the covariates and random effects via:

$$\log(\lambda_{ij}) = \beta_0 + \beta_1 X_{ij1} + \cdots + \beta_p X_{ijp} + u_j$$

Where, u_j is the random effect for group j, assumed to follow a normal distribution:

$$u_j \sim \mathcal{N}(0, \sigma_u^2)$$

3.2.2. *Likelihood.* The likelihood for GLMM involves integrating out the random effects:

$$L(\beta, \sigma_u^2; Y) = \prod_{j=1}^{m} \int \left(\prod_{i=1}^{n_j} \frac{e^{-\lambda_{ij}} \lambda_{ij}^{Y_{ij}}}{Y_{ij}!} \right) \frac{1}{\sqrt{2\pi\sigma_u^2}} e^{-\frac{u_j^2}{2\sigma_u^2}} du_j$$

Given the complexity of this integral, numerical methods or approximation techniques (e.g., Laplace approximation, Gauss-Hermite quadrature) are typically used to maximize the likelihood and estimate the parameters β and σ_u^2.

3.3. Bayesian Hierarchical Model. The Bayesian Hierarchical Model is similar to GLMM but uses a fully Bayesian approach to estimate parameters, incorporating prior distributions on both the fixed and random effects.

3.3.1. *Model Specification.* Let Y_{ij} be the count of monkeypox cases for observation i in group j. The model is:

$$Y_{ij} \sim \text{Poisson}(\lambda_{ij})$$

With:

$$\log(\lambda_{ij}) = \beta_0 + \beta_1 X_{ij1} + \cdots + \beta_p X_{ijp} + u_j$$

u_j follows a normal distribution:

$$u_j \sim \mathcal{N}(0, \sigma_u^2)$$

But now, we assign prior distributions to the parameters:

$$\beta_0, \beta_1, \ldots, \beta_p \sim \text{Normal}(0, \sigma_\beta^2)$$

$$\sigma_u^2 \sim \text{Inverse-Gamma}(a, b)$$

Where σ_β^2 is the variance of the prior for the fixed effects, and a, b are hyperparameters for the inverse-gamma prior on the variance of the random effects.

3.3.2. *Posterior Distribution.* The goal is to compute the posterior distribution of the parameters given the data:

$$p(\beta, u, \sigma_u^2 \mid Y) \propto p(Y \mid \beta, u) \cdot p(\beta) \cdot p(u \mid \sigma_u^2) \cdot p(\sigma_u^2)$$

This posterior distribution is typically computed using Markov Chain Monte Carlo (MCMC) methods like Gibbs sampling or Hamiltonian Monte Carlo (as implemented in MCMCglmm).

3.3.3. *Inference.* The posterior samples obtained from MCMC can be used to estimate the posterior means, credible intervals, and make probabilistic statements about the parameters (e.g., the probability that a parameter is positive).

3.4. Parameter Estimation and Model Diagnostics.

3.4.1. *Parameter Estimation.* In the context of the models described, parameter estimation differs between the Poisson Regression, GLMM, and Bayesian Hierarchical Model.

3.4.2. *Poisson Regression.* For the Poisson regression model, parameters $\beta_0, \beta_1, \ldots, \beta_p$ are estimated using Maximum Likelihood Estimation (MLE). The MLE estimates are obtained by maximizing the log-likelihood function:

$$\hat{\beta} = \arg\max_{\beta} \log L(\beta; Y)$$

where

$$\log L(\beta; Y) = \sum_{i=1}^{n} \left(Y_i \left(\beta_0 + \sum_{j=1}^{p} \beta_j X_{ij} \right) - e^{\beta_0 + \Sigma_{j=1}^{p} \beta_j X_{ij}} - \log(Y_i!) \right)$$

3.4.3. *Generalized Linear Mixed Model (GLMM).* In GLMM, parameter estimation involves both fixed effects (β) and random effects (u_j). The estimation typically uses Restricted Maximum Likelihood (REML) or Maximum Likelihood (ML) methods, which maximize the likelihood function by integrating out the random effects:

$$L(\beta, \sigma_u^2; Y) = \prod_{j=1}^{m} \int \left(\prod_{i=1}^{n_j} \frac{e^{-\lambda_{ij}} \lambda_{ij}^{Y_{ij}}}{Y_{ij}!} \right) \frac{1}{\sqrt{2\pi\sigma_u^2}} e^{-\frac{u_j^2}{2\sigma_u^2}} du_j$$

Numerical techniques such as Laplace approximation or Gauss-Hermite quadrature are used to approximate this integral.

3.4.4. *Bayesian Hierarchical Model.* In the Bayesian Hierarchical Model[30], parameter estimation involves deriving the posterior distribution of parameters given the observed data and prior distributions:

$$p(\beta, u, \sigma_u^2 \mid Y) \propto p(Y \mid \beta, u) \cdot p(\beta) \cdot p(u \mid \sigma_u^2) \cdot p(\sigma_u^2)$$

Markov Chain Monte Carlo (MCMC) methods such as Gibbs sampling or Hamiltonian Monte Carlo are used to draw samples from the posterior distribution. The posterior means, credible intervals, and other summary statistics are then computed from these samples.

3.5. Model Diagnostics. Model diagnostics are crucial for assessing the fit and validity of the models. Different diagnostics apply depending on whether the model is a Poisson regression, GLMM, or Bayesian Hierarchical Model.

4. RESULTS

Parameter	Estimate	Std. Error	z value	Pr($>\|z\|$)	2.5%	97.5%
(Intercept)	3.7015	0.1154	32.0664	1.295E-225	3.4758	3.9286
population_density	-0.0221	0.0007	-32.6921	2.023E-234	-0.0234	-0.0208
healthcare_capacity	-3.8782	0.0689	-56.3130	0.0000	-4.0187	-3.7484
mobility_index	3.9620	0.0614	64.5319	0.0000	3.8416	4.0824

TABLE 2. Summary of Poisson regression model parameter estimates

4.1. Poisson Regreesion Model Summary. Table 2 presents the results from the Poisson regression model, indicating the effects of covariates like population density, healthcare capacity, and mobility index on the number of confirmed monkeypox cases. The intercept and mobility index have positive effects, indicating that as mobility increases, the number of cases also increases. Population density has a negative effect, implying that higher population density might be associated with fewer cases. Healthcare capacity also has a significant negative effect, suggesting that better healthcare capacity might reduce the number of cases.

4.2. GLM Fixed Effects and Random Effects.

Effect	Covariate	post.mean	l-95% CI	u-95% CI	eff.samp
Fixed Effects	Intercept	3.573324	0.436228	7.012442	844.4457
	Population Density	-0.003545	-0.011929	0.003465	1000
	Healthcare Capacity	-2.770876	-5.181056	-0.729619	1000
	Mobility Index	2.379011	0.012969	4.717297	897.2286
Random Effects	Countries	0.019574	7.01E-17	1.29E-06	134.5301

TABLE 3. Summary of GLM MCMC results for Fixed and Random Effects

Table 3 provides estimates from the GLMM, showing both fixed effects (similar covariates as in the Poisson model) and random effects (variance attributed to different countries). The fixed effects indicate similar trends to the Poisson model, with significant effects from mobility index and healthcare capacity. The random effects demonstrate variability among different countries, which is captured by the model.

4.3. Bayesian Hierachical.

Effect	**Covariate**	**post.mean**	**l-95% CI**	**u-95% CI**	**eff.samp**	**pMCMC**
Fixed Effects	Intercept	3.561961	0.279791	6.98385	1000	0.036
	Population Density	-0.003736	-0.011177	0.004282	1000	0.302
	Healthcare Capacity	-2.718427	-5.141706	-0.687872	1000	0.022
	Mobility Index	2.362036	-0.032897	4.566143	1000	0.052
Random Effects	Countries (Mean)	2.183151				
	Countries (SD)	3.145294				
	Countries (2.5% CI)	0.001415				
	Countries (97.5% CI)	10.19093				
Variance Components	Units (Mean)	2.486032				
	Units (SD)	3.145251				
	Units (2.5% CI)	0.00154				
	Units (97.5% CI)	9.357558				

TABLE 4. Summary of Bayaesian Hierarchical MCMC results for Fixed Effects, Random Effects, and Variance Components

Table 4 presents the Bayesian Hierarchical Model results, highlighting the mean, credible intervals, and effective sample size for fixed effects, random effects, and variance components. The BHM results align with the GLMM, capturing the variability between countries while also providing a probabilistic interpretation of the parameter estimates.

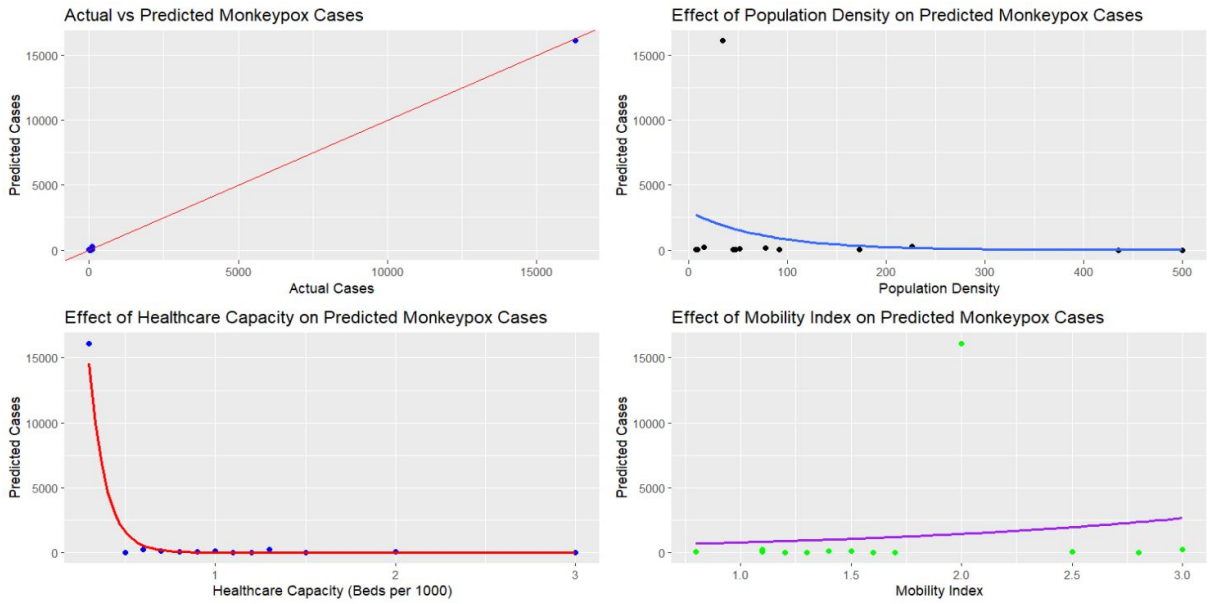

FIGURE 1. Effects of covariates on Predicted MonkeyPox cases.

4.4. Graphical Representation. Figure 1 visualizes the relationship between the covariates (population density, healthcare capacity, and mobility index) and the predicted number of monkeypox cases. The figure shows how each covariate individually influences the predicted number of cases, with the mobility index having a positive association, while population density and healthcare capacity have mixed effects.

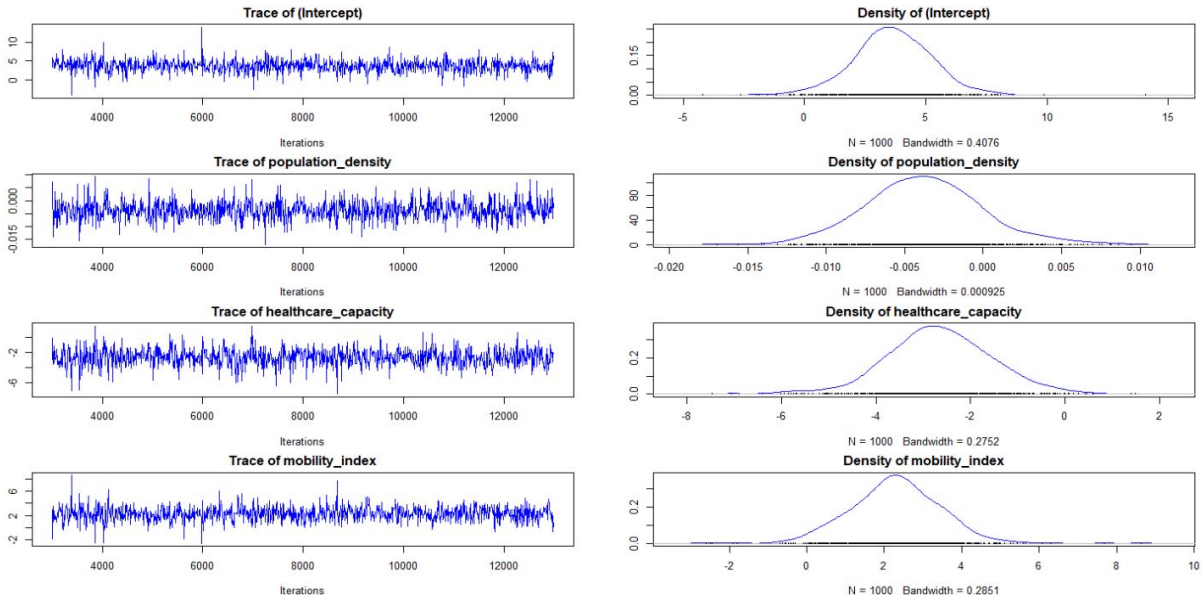

FIGURE 2. Trace and density plot for the covariates on MonkeyPox cases prediction

Figure 2 presents the trace and density plots from the MCMC sampling for the covariates, showing the stability and distribution of the estimates. It provides a diagnostic check on the MCMC convergence, ensuring that the sampling is sufficient to represent the posterior distribution.

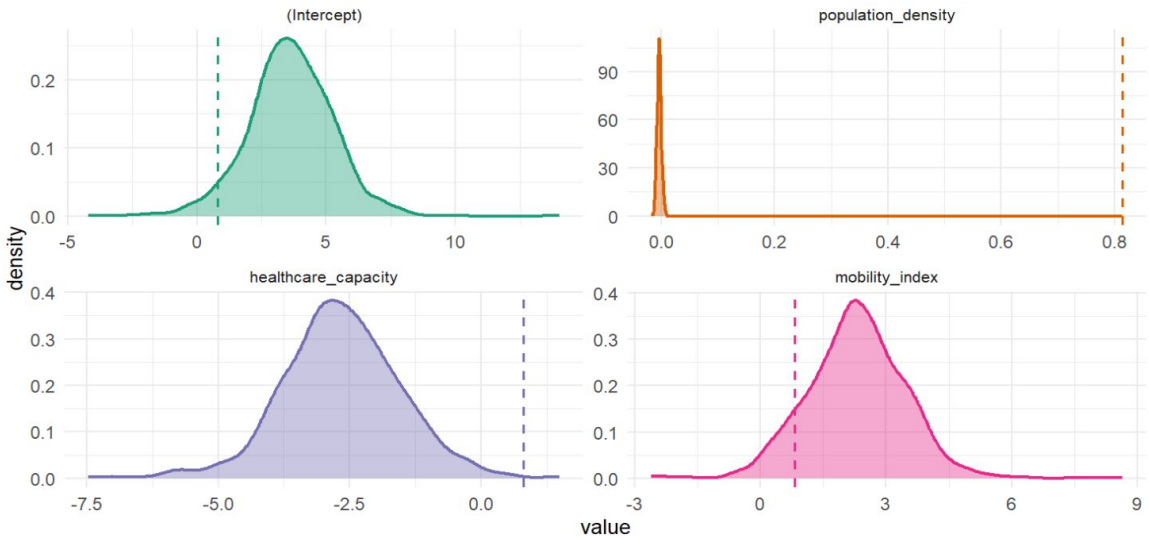

FIGURE 3. Posterior Distribution of Model parameters

Figure 3 shows the posterior distributions of the model parameters, emphasizing the uncertainty in the parameter estimates. It highlights the variability in the parameter estimates, especially for the random effects, and helps to assess the credibility of the model predictions, [31, 30].

5. DISCUSSION

The study utilized a combination of Poisson regression, GLMM, and Bayesian Hierarchical Modeling to analyze the spread of monkeypox across different regions. The Poisson regression provided initial insights into the relationship between key covariates and the number of confirmed cases. The GLMM added an additional layer by incorporating random effects, allowing for variability between countries to be considered. Finally, the Bayesian Hierarchical Model provided a comprehensive probabilistic framework, capturing both fixed and random effects while accounting for the uncertainty in the estimates.

The results indicate significant variability in monkeypox transmission across regions, influenced by factors such as mobility, healthcare capacity, and population density. The findings underscore the need for region-specific public health interventions, as different areas may respond differently to the same covariates. For instance, while increased mobility generally leads to more cases, the effect of population density and healthcare capacity is more complex and varies across regions.

The Bayesian Hierarchical Model proved particularly useful in this analysis, offering a nuanced understanding of the transmission dynamics and enabling more robust forecasts. The MCMC diagnostics confirmed the reliability of the model, with trace plots indicating good convergence and the effective sample size ensuring sufficient representation of the posterior distribution.

6. Conclusion

This study demonstrates the utility of advanced statistical modeling techniques in understanding the spread of infectious diseases like monkeypox. By combining Poisson regression, GLMM, and Bayesian Hierarchical Modeling, the analysis was able to capture both the fixed effects of key covariates and the random effects associated with different regions. The findings highlight the significant heterogeneity in monkeypox transmission, which has important implications for public health strategies.

The Bayesian Hierarchical Model, in particular, provided a robust framework for dealing with the complexity of disease transmission, offering insights that would not be apparent from simpler models. The study underscores the importance of tailoring public health interventions to specific regional contexts to effectively control and prevent monkeypox outbreaks.

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Conflict of Interests

The authors declare that there is no conflict of interests.

APPENDIX

APPENDIX A. STATISTICAL ANALYSIS

The appendix includes the full R code used for the statistical analysis, covering data preparation, model fitting, and visualization. The code provides a detailed roadmap for reproducing the results and can be adapted for similar studies in other contexts. This transparency ensures that the findings are reproducible and that the methodology can be further refined by other researchers.

```
 Install necessary packages
install.packages("ggplot2")
install.packages("dplyr")
install.packages("lme4")
install.packages("MCMCglmm")
install.packages("brms")
install.packages("Matrix", dependencies = TRUE)
install.packages("lme4", dependencies = TRUE)
install.packages("devtools")
install_github("Matrix", repo = "cran/Matrix")

# Load necessary libraries
library(ggplot2)
library(dplyr)
library(lme4)
library(MCMCglmm)
library(brms)
library(Matrix)
library(devtools)

# Load the data
# Load necessary libraries
library(readr)  # For reading CSV files

# Load the data from a CSV file
```

```r
data <- read_csv("monkeypox_data.csv")

# View the first few rows of the data to verify
head(data)

# Fit a Poisson regression model
model <- glm(confirmed_cases ~ population_density + healthcare_capacity + mobility_index,
             data = data, family = poisson())

# Summary of the model
summary(model)

# Predicting new cases based on the model
data$predicted_cases <- predict(model, type = "response")

# Extract coefficients
coefficients <- coef(model)
coefficients

# Confidence intervals for coefficients
confint(model)

# Plot the actual vs predicted cases
ggplot(data, aes(x = confirmed_cases, y = predicted_cases)) +
  geom_point(color = "blue") +
  geom_abline(slope = 1, intercept = 0, color = "red") +
  labs(title = "Actual vs Predicted Monkeypox Cases",
       x = "Actual Cases", y = "Predicted Cases")

# Plot effect of population density
ggplot(data, aes(x = population_density, y = predicted_cases)) +
  geom_point() +
  geom_smooth(method = "glm", method.args = list(family = "poisson"), se = FALSE) +
```

```
  labs(title = "Effect of Population Density on Predicted Monkeypox Cases",
       x = "Population Density", y = "Predicted Cases")


#Plot effect of Healthcare Capacity on Predicted Cases
ggplot(data, aes(x = healthcare_capacity, y = predicted_cases)) +
  geom_point(color = "blue") +
  geom_smooth(method = "glm", method.args = list(family = "poisson"), se =
      FALSE, color = "red") +
  labs(title = "Effect of Healthcare Capacity on Predicted Monkeypox
      Cases",
       x = "Healthcare Capacity (Beds per 1000)", y = "Predicted Cases")

# Plot effect of mobility on predicted cases
ggplot(data, aes(x = mobility_index, y = predicted_cases)) +
  geom_point(color = "green") +
  geom_smooth(method = "glm", method.args = list(family = "poisson"), se =
      FALSE, color = "purple") +
  labs(title = "Effect of Mobility Index on Predicted Monkeypox Cases",
       x = "Mobility Index", y = "Predicted Cases")

# Arrange the plots into a 2x2 grid layout
grid.arrange(p1, p2, p3, p4, nrow = 2, ncol = 2)

# Fit a GLMM model
glmm_model <- MCMCglmm(confirmed_cases ~ population_density +
    healthcare_capacity + mobility_index,
                       random = ~ countries, family = "poisson", data =
                          data)

# Summary of the model
summary(glmm_model)
```

REFERENCES

[1] Y.A. Tajudeen, H.J. Oladipo, A.O. Muili, et al. Monkeypox: a review of a zoonotic disease of global public health concern, Health Promotion Perspect. 13 (2023), 1–9. https://doi.org/10.34172/hpp.2023.01.

[2] R.A. Farahat, R. Sah, A.A. El-Sakka, Human monkeypox disease (MPX), Infez. Med. 30 (2022), 372–391. https://doi.org/10.53854/liim-3003-6.

[3] M.G. Reynolds, J.B. Doty, A.M. McCollum, et al. Monkeypox re-emergence in africa: a call to expand the concept and practice of one health, Expert Rev. Anti-Infect. Therapy 17 (2019), 129–139. https://doi.org/10.1080/14787210.2019.1567330.

[4] M.S. Hakim, S.A. Widyaningsih, The recent re-emergence of human monkeypox: would it become endemic beyond Africa?, J. Infect. Public Health 16 (2023), 332–340. https://doi.org/10.1016/j.jiph.2023.01.011.

[5] S.D. Falcinelli, J. Ciric, J. Kindrachuk, Variola virus: clinical, molecular, and bioterrorism perspectives, in: S.K. Singh, J.H. Kuhn (Eds.), Defense Against Biological Attacks, Springer, Cham, 2019: pp. 55–102. https://doi.org/10.1007/978-3-030-03071-1_3.

[6] Z.S. Moore, J.F. Seward, J.M. Lane, Smallpox, The Lancet 367 (2006), 425–435. https://doi.org/10.1016/S0140-6736(06)68143-9.

[7] N. Hussain, M.H. Ashraf, R. Arooj, et al. An overview of monkeypox virus: molecular biology, epidemiology, pathogenesis, treatment and prevention strategies, preprint, (2024). https://doi.org/10.20944/preprints202402.1430.v1.

[8] M.M. Naseer, M. Afzal, T. Fatima, et al. Human monkeypox virus: a review on the globally emerging virus, Biomed. Lett. 10 (2024), 26–41.

[9] A. Rabaan, N. Alasiri, M. Aljeldah, et al. An updated review on monkeypox viral disease: emphasis on genomic diversity, Biomedicines 11 (2023), 1832. https://doi.org/10.3390/biomedicines11071832.

[10] M.O. Oduoye, T.O. Faloye, S.C. Ubechu, et al. Factors responsible for the re-emergence of monkeypox and implications for global health, Int. J. Surg.: Glob. Health 7 (2024), e0459. https://doi.org/10.1097/GH9.0000000000000459.

[11] P.L. Nicoletti, Relationship between animal and human disease, CRC Press, 2020.

[12] J.S. Kutter, M.I. Spronken, P.L. Fraaij, et al. Transmission routes of respiratory viruses among humans, Curr. Opin. Virol. 28 (2018), 142–151. https://doi.org/10.1016/j.coviro.2018.01.001.

[13] M.A. Nakhriry, C.J. Aarthy, Evaluating monkeypox transmission dynamics: implications for public health interventions, Eigenpub Rev. Sci. Technol. 7 (2023), 71–84.

[14] H. Ajmal, E. Hunter, J. Duggan, et al. Agent-based modelling of mpox infection dynamics: simulating disease transmission and control strategies, J. Artif. Soc. Soc. Simul. 27 (2024), 6. https://doi.org/10.18564/jasss.5380.

[15] V. Singh, S.A. Khan, S.K. Yadav, et al. Modeling global monkeypox infection spread data: a comparative study of time series regression and machine learning models, Curr. Microbiol. 81 (2024), 15. https://doi.org/10.1007/s00284-023-03531-6.

[16] H. Heesterbeek, R.M. Anderson, V. Andreasen, et al. Modeling infectious disease dynamics in the complex landscape of global health, Science 347 (2015), aaa4339. https://doi.org/10.1126/science.aaa4339.

[17] O. Diekmann, J.A.P. Heesterbeek, T. Britton, Mathematical tools for understanding infectious disease dynamics, Princeton University Press, Princeton, 2013. https://doi.org/10.1515/9781400845620.

[18] J.K. Mutinda, A.K. Langat, Modeling the impact of air pollution and meteorological variables on COVID-19 transmission in Western Cape, South Africa, Int. J. Math. Math. Sci. 2024 (2024), 1591016. https://doi.org/10.1155/2024/1591016.

[19] M.Y. Li, An introduction to mathematical modeling of infectious diseases, Springer, Cham, 2018. https://doi.org/10.1007/978-3-319-72122-4.

[20] C. Ding, X. Liu, S. Yang, The value of infectious disease modeling and trend assessment: a public health perspective, Expert Rev. Anti-Infect. Therapy 19 (2021), 1135–1145. https://doi.org/10.1080/14787210.2021.1882850.

[21] A.M.G. García, Epidemic spreading model, Thesis, Universitat Politècnica de Catalunya, 2021.

[22] J. Tolles, T. Luong, Modeling epidemics with compartmental models, JAMA 323 (2020), 2515–2516. https://doi.org/10.1001/jama.2020.8420.

[23] H. Caswell, A.M. John, From the individual to the population in demographic models, in: Individual-Based Models and Approaches In Ecology, Chapman and Hall/CRC, pp. 36–62, 2018.

[24] W.J. Sutherland, From individual behaviour to population ecology, Oxford University Press, 1996.

[25] A. Langat, G. Orwa, J. Koima, Cancer cases in Kenya; forecasting incidents using Box & Jenkins arima model, Biomed. Stat. Inf. 2 (2017), 37–48.

[26] A.B. Lawson, Using R for bayesian spatial and spatio-temporal health modeling, Chapman and Hall/CRC, 2021. https://doi.org/10.1201/9781003043997.

[27] S.L. Taylor, Access to mental health services among persons with HIV/AIDS and need: A multi-level analysis, Columbia University, 2002.

[28] A.J. Powell, I.K. Tezaur, W.L.D. Iv, et al. Predicting future disease burden in a rapidly changing climate, Technical Report, Sandia National Laboratories, 2020. https://doi.org/10.2172/1670999.

[29] Q. Li, T. Bedi, G. Xiao, Y. Xie, Evaluating short-term forecast among different epidemiological models under a bayesian framework, medRxiv 2020.10.11.20210971, (2020). https://doi.org/10.1101/2020.10.11.20210971.

[30] A.K. Langat, S.M. Mwalili, L.N. Kazembe, Hierarchical bayesian semi-parametric models for measurement error correction in determining optimal fertilizer application levels, Sci. Afr. 26 (2024), e02423. https://doi.org/10.1016/j.sciaf.2024.e02423.

[31] A.K. Langat, S.M. Mwalili, L.N. Kazembe, Mixed effects and semi-parametric Bayesian integration models for measurement error correction in the context of fertilizer application levels: a simulation study, Commun. Math. Biol. Neurosci., 2024 (2024), 105. https://doi.org/10.28919/cmbn/8744.