

INCIDENCE RATE AND CONTRIBUTORY FACTORS OF MALARIA IN THE AMENFI WEST DISTRICT, GHANA

ABSTRACT

Aims: To determine the incidence rate and some contributory factors of malaria in the Amenfi West District of Ghana. This study investigated the incidence and determinants in predicting malaria status in the Amenfi West District.

Place and Duration of Study: Asankrangwa Catholic Hospital in the Amenfi West District of the Western Region, Ghana between May 2016 and October 2016.

Methodology and design of study: Primary data collection involved laboratory work, where blood samples were taken from patients to prepare blood films for microscopy, blood group determination and hematological analysis.

Purposive sampling was adopted to select 240 patients from Asankrangwa Catholic Hospital. Demographic data such as age, sex and the type of malaria control method(s) used were retrieved from patients. Data was organized into an excel sheet and analyzed using the Chi-square, Mann-Whitney, Kruskal-Wallis as well as Binary Logistic Regression.

Results: The results revealed that parasite density ($P = 0.048$) and incidence ($P = 0.000$) differed significantly based on age with younger persons recording higher values. Difference in prevalence rate was also found among the blood group types ($P = 0.041$) but no statistical difference was recorded in relation to parasite density ($P = 0.329$). Moreover, the logistic regression analysis showed that blood group ($P = 0.029$), type of malaria control used ($P = 0.019$), hemoglobin level ($P = 0.002$) and age ($P = 0.002$) are statistically significant in determining the malaria status (positive/negative) of an individual; for instance, use of treated nets or being in an older age group decreased a person's odds of experiencing malaria.

Conclusion: It is therefore suggested that, much emphasis on the use of ITNs should be focused on younger children since they are more vulnerable to malaria infection.

Keywords: Malaria, Parasitemia, Seasonality, Binary Logistic Regression, Ghana.

1. INTRODUCTION

Malaria remains one of the major killers of humans worldwide, threatening the lives of more than one-third of the world's population [1]. It is a vector borne disease mainly transmitted by the bite of female anopheles mosquitoes. Four species of Plasmodium protozoa namely; *Plasmodium falciparum*, *P. vivax*, *P. malariae* and *P. ovale* cause malaria in humans [2]. Of these species, *P. falciparum* is the most prevalent pathogenic species, responsible for most deaths from malaria in Africa and Ghana is of no exception [3]. An estimated 300 to 500 million cases of malaria are reported annually resulting in 1.5 to 2.7 million deaths, with 90% occurring in children [4]. According to Craig et al. [5] and Gaudart et al. [6], the distribution of malaria has always been geographically specific and biased towards sub-Saharan Africa [7]. Currently, there are 104 malaria endemic countries [8] and despite the fact that numbers of malaria cases as well as mortality in some endemic regions have been minimized, people of all ages remain susceptible [9] and still suffer from the burden of malaria mortality [10].

In Africa, malaria continues to be endemic in countries such as Ethiopia [11], Niger [4], Mali [6], Kenya [12] and Nigeria [7]. However, its eradication has been successful in other African countries like Algeria, Egypt, Libya, Morocco and Tunisia [13]. In Ghana, malaria is a principal public health problem that plagues all segments of the society [14]. Its effect on people is quite immense particularly with high risk occurring in pregnant women and children because they have less immunity [15]. Major factors that account for prevalence of malaria in this country include poor environmental conditions and lack of preventive services [16]. Aside these factors, transmission has also been influenced by urban agriculture where well irrigated sites often serve as breeding grounds for malaria vectors [17,18,19].

Human resistance or susceptibility to infection by malarial parasites are influenced also by several factors including a wide range of genetic polymorphism [20, 21]. It is known that, susceptibility to several infectious diseases are related to the patients' blood group and malaria is of no exception [22, 23]. For example, blood group O erythrocytes have reduced rosetting (a parasite virulence factor thought to contribute to pathogenesis of severe malaria by obstructing microvascular blood flow) compared with the non-O blood groups (A, B and AB) in *P. falciparum*. In addition, studies have suggested that blood group A may predispose African children to severe malaria [24, 25]. Contrarily, Joshi et al. [26] and Barragan et al. [27] reported that, A erythrocytes rather inhibits rosette formation.

Furthermore, several studies have documented age [28, 4] and gender [29] as drivers of malaria infection. Though Loha and Lindtjörn [30] revealed that malaria prevalence was higher in males than females, Dalatu et al. [31] indicated otherwise. In that study, the latter authors suggested that both sex and gender were not good determinants of malaria incidence. Other important factors that may contribute to malaria infection include body temperature [31], hemoglobin level [32] and type of malaria control [30]. Therefore, this study seeks to unravel the incidence rate and some contributory factors of malaria in the Amenfi West District of Ghana for better and improved management decisions locally and globally.

2. MATERIAL AND METHODS

2.1 Study area

The study was carried out in the Amenfi West District with study participants selected from the Asankrangwa Catholic Hospital in the Amenfi West District of the Western Region, Ghana (Figure 1). It is the largest health facility (district hospital) serving as both the first consultation point and as a referral centre for about 13 health care facilities in the District. This hospital was chosen since most of the 13 health care facilities are community clinics and lack laboratory services. The district's geographical coordinates are 5°48' 18"N and 2°26'25" and is characterized by a bimodal rainfall regime, with the major season occurring from March to July and the minor season from September to mid-December. A dry spell is experienced between late December and February [33].

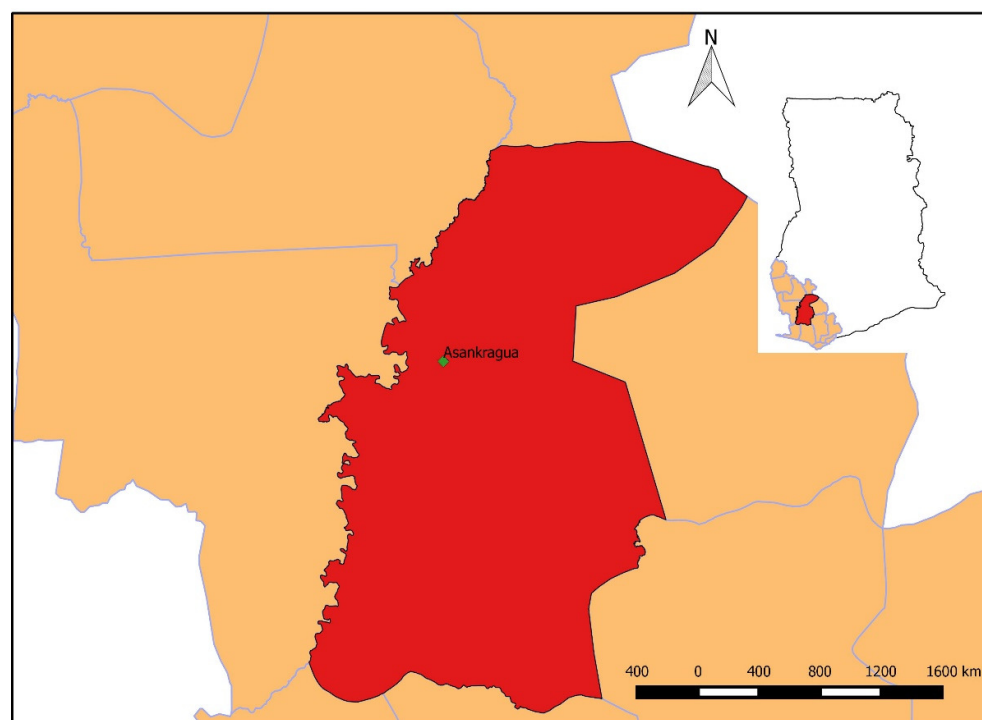


Figure 1: Map of Ghana showing study area in Amenfi West district.

2.2 Study population

Though hundreds of patients attend the health center for routine treatment daily, not all were sampled (i.e. purposive sampling), only patients diagnosed with clinical malaria according to standard national procedures for case management and sent to the laboratory were considered. Only patients present at the laboratory to do the malaria test were included while patients already on admission and have had their samples already taken was excluded.

2.3 Data collection

Primary data collection involved laboratory work, where blood samples was taken from patients to prepare blood films for microscopy, blood grouping and hematological analysis. Demographic data such as age, sex and the type of malaria control use were retrieved from patients. The duration dedicated to laboratory research was six months (3 months for each season) at the health facility. Blood samples were taken from patients who agreed to participate in this study by signing a consent form. A total of 240 samples were analyzed at the end of the study.

Three milliliters of venous blood was collected into EDTA tube from each patient, ABO typing for blood grouping was carried out using three drops of blood while some was used for hematological analysis (Full Blood Count). A drop of sample was used to prepare thick smear for microscopic detection of malaria parasites after standard Giemsa staining, by analyzing the slides under a 100X objective. Samples with no visible parasites after scoring 100 fields were considered negative for this test. Parasitemia (parasite/ μL) was determined by counting the number of parasites present per 200 white blood cells (WBC) in the thick smear and it was multiplied by 40, to obtain an approximate parasite count per ml of blood. This calculation was based on the assumption of a mean WBC count of 8,000/ μL of blood, considered as a representative standard value allowing calculation of the parasite count [34].

2.4 Data analysis

Descriptive statistics such as mean, median and coefficient of variation among others were estimated for the various datasets. Secondly, Chi-square test was performed to determine the effect of sex, age, season and blood group on malaria prevalence in the study area. For parasite density, factor effects

89 were examined using Mann-Whitney and Kruskal-Wallis tests where appropriate to investigate
90 statistical differences.

91 In addition, binary logistic regression was employed to model the presence (positive) or absence
92 (negative) of malaria in a patient. The independent variables used to build the model included Age,
93 Sex, Blood group, Weight, HB, WBC, RBC, Season, and Type of malaria control used by patients.
94 This approach was preferred to other methods (eg. multiple regression) because it requires fewer
95 assumptions and can incorporate a mixture of numerical as well as categorical independent variables
96 [35]. With respect to age groups, 0-14 and 15-24 years were considered in the regression model. This
97 is because children and youth are considered highly susceptible to malaria infection than adults [14].
98 Thus, according to Uduakosu [36], WHO and UNICEF defines children and youth as people between
99 ages of 0-14 as well as 15-24 years respectively.

100 3. RESULTS

101 3.1 Determinants of Malaria Incidence

102 This study comprised of 65.4% females and 34.6% males with their ages ranging from 0 to 81 years
103 at the end of the laboratory investigation. Also, total positive prevalence rate of malaria in the study
104 area was approximately 27.9% (Table 1).
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106 **Table 1: Prevalence rates of malaria in the study area**

Variable	Number [Percentage]	Number Infected	Prevalence (%)	Chi-Square Value (χ^2)	P – Value
Sex					
Female	157 [65.4%]	37	55.200	4.268	0.039
Male	83 [34.6%]	30	44.800		
Age					
≥ 15	121 [54.4%]	8	11.900	57.263	0.000
10-14	10 [4.2%]	3	4.500		
5-9	22 [9.2%]	12	17.900		
0-4	87 [36.2%]	44	65.700		
Season					
Rainy	122 [50.8%]	31	46.300	0.775	0.379
Dry	118 [49.2%]	36	53.700		
Blood Group					
A	42 [17.5%]	13	19.400	8.277	0.041
B	69 [28.7%]	27	40.300		
AB	10 [4.2%]	1	1.500		
O	119 [49.6%]	26	38.800		
Total	240 [100%]	67	27.900		

107 From the table, significant differences in malaria prevalence was observed for both gender ($\chi^2 =$
108 4.268; $P = 0.039$) and age groups ($\chi^2 = 57.263$; $P = 0.000$). However, no statistical difference in
109 prevalence was found between the rainy and dry season ($\chi^2 = 0.775$; $P = 0.379$) though some
110 differences were observed among the different blood groups ($\chi^2 = 8.277$; $P = 0.041$) which had
111 positive rh factor D in all.

112 In order to investigate statistical differences in parasite density between seasons as well as age and
113 blood groups, Mann-Whitney (U) and Kruskal-Wallis (KW) tests were respectively performed due to
114 non-normality of the dataset (Table 2).
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Table 2: Test of normality on parasite density

Test	Statistic	P-value
KS	0.247	0.000
SW	0.667	0.000

KS = Kolmogorov-Smirnov; SH = Shapiro-Wilk

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Table 3: Descriptive statistics on parasite density

Variable		Mean	Median	Min.	Max.
Season	Dry	62951.06	47767.50	482.00	209000.00
	Rainy	89705.42	19500.00	551.00	610937.00
Age Group	0-4	86298.75	43843.00	551.00	610937.00
	5-9	74051.67	72868.00	2882.00	182384.00
	10-14	31419.67	27667.00	5342.00	61250.00
	≥15	33385.25	1571.50	482.00	230421.00
Blood Group	A	70600.38	61250.00	572.00	230421.00
	B	103280.70	22529.00	482.00	610937.00
	AB	2352.00	2352.00	2352.00	2352.00
	O	51475.77	22946.00	551.00	209000.00

Min. = Minimum value, Max. = Maximum value

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Descriptive statistics for parasite density in the study area are given in Table 3. Though dry season had the highest median parasite density (47767.50) than rainy season (19500.00), this difference was not statistically significant ($P = 0.930$). Parasite density also differed significantly according to age groups based on a KW test with younger individuals (0-4years = 43843.00; 5-9years = 72868.00) recording higher median values than older individuals (10-14years = 27667.00; ≥15years = 1571.50). Nonetheless, there was no statistical difference between the blood groups based on parasite density ($P = 0.329$).

Table 4: Parasite density comparative tests

Variable	Test Statistic	P-value
Season	551.000	0.930
Age	7.913	0.048
Blood Group	3.433	0.329

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Subsequently, a binary logistic regression (BLR) analyses was performed to model the probability of an individual testing positive or negative for malaria disease for persons between 0 – 24 years (later regrouped as: 0-14 and 15-24). The proposed model for the analysis included the following independent variables; Season, Sex, Type of Malaria Control, Blood Group, Age Group (0-14 and 15-24), Body Weight, HB, WBC and RBC. The final model with the variables; Age Group ($P = 0.0021$), HB ($P = 0.0021$), Type of Malaria Control ($P = 0.0191$), and Blood Group ($P = 0.0288$) were jointly found to be statistically significant in determining the Malaria status (positive/negative) of an individual in the study area. This significance was based on the likelihood ratio test ($\chi^2 = 51.7064$; $P < 0.0001$) and score test ($\chi^2 = 43.0286$; $P < 0.0001$) analyzed at a significance level of 5%.

Parameter estimates of the final model that are useful determinants in predicting Malaria status are given in Table 5. From the table, the odds of testing positive for malaria is 1.2740 higher given only Mosquito Repellant (MRO) use compared to combined use of Repellant and Insecticide Treated Nets (MR-ITN). However, persons who use only ITNs are 0.2340 times as likely as those who use MR-ITNs to test positive for malaria (i.e. ITN use decreases odds of experiencing the disease). Though individuals with type 'A' and 'B' Blood Groups have increased odds of testing positive for malaria, only the latter was marginally insignificant when compared to blood group 'O'. The result for HB indicates that a unit change in its level reduces the odds of an individual testing positive for malaria. Also, being an older person (i.e. 15-24 years) decreases one's odds of experiencing the disease or testing positive for it compared to younger persons or children (i.e. 0-14 years).

Table 5: Parameter estimates of final BLR model

Parameter	Estimate	Std. Error	Wald χ^2	P-value	Odds Ratio
Intercept	2.2295	1.2951	2.9635	0.0852	NA
NCON	-0.3531	0.3842	0.8445	0.3581	0.4170
ITN	-0.9323	0.4101	5.1684	0.0230	0.2340
MRO	0.7639	0.3225	5.6105	0.0179	1.2740
BG-A	0.7732	0.5449	2.0133	0.1559	2.8750
BG-B	0.8706	0.4490	3.7589	0.0525	3.1700
BG-AB	-1.3607	0.9801	1.9274	0.1650	0.3400
15-24yrs	-1.2260	0.3989	9.4464	0.0021	0.0860
HB	-0.3777	0.1229	9.4509	0.0021	0.6850

NCON=No Malaria Control; ITN=Insecticide Treated Net;
MRO=Mosquito Repellant (Coil & Spray); BG=Blood Group

Finally, a diagnostic check with the Hosmer and Lemeshow test, which gave a test statistic of 6.6062 with a P-value of 0.5797, indicates that the final (i.e. selected) model is adequate. In totality, approximately 72% of the observations were correctly classified by the final model and based on the area under the ROC curve (0.8340), the model could be used to discriminate between the two Malaria Statuses 83.4000% of the time (Figure 2).

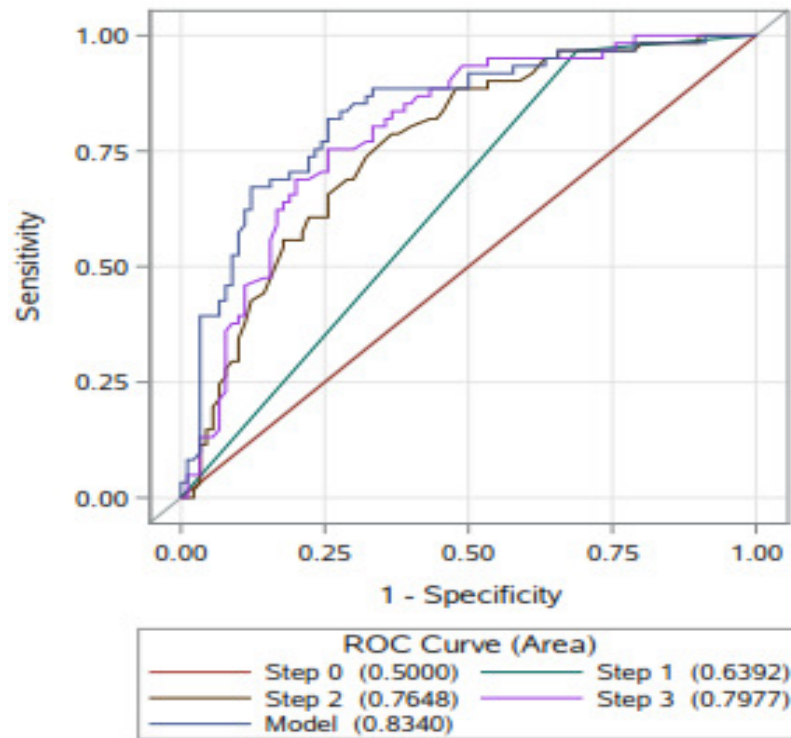


Figure 2: ROC curve for all model building steps – BLR

4. DISCUSSION

According to Abdul-Aziz et al. [28] and Muntaka and Opoku-Okrah [37], age is an important determinant of malaria occurrence. Similarly, the result of this work indicated a significant difference in malaria prevalence between age groups. Also, children ≤ 4 as well as 5 – 9 years contracted and had more parasite density than people above 15 years. This affirms the findings of [4] who reported higher parasite and prevalence rate among children below 10 years. These differences may arise because some age groups either lack acquired or have inadequate immunity [30] and as such harbor more parasite at a given time [38].

Furthermore, incidence percentage based on our result was significantly higher in females than males. This corroborates the findings of Vlassoff and Bonilla [39] as well as Otajevwo [40] who reported a higher infection rate in females than males. In contrast, Bonilla and Rodriguez [41] stated a higher infection rate in males than females. This higher rate of infection among females than males in our study could be an indication that more females were highly exposed to mosquitoes that transmit the malaria parasite. Also, it may be due to the fact that more females were present at the study site (i.e. health facility) to test for malaria.

Though limited, a number of studies have reported the importance of blood group in malaria transmission and prediction models [22, 24] with the available literature showing varying results relating to the subject [42, 43]. For example, an examination conducted on blood groups in determining malaria incidence in Nigeria [44] showed that, “O” had higher prevalence of parasitaemia but lower prevalence of severe malaria when compared with non – “O” blood groups. Secondly, studies have suggested A blood group as more susceptible to malaria infection [24, 25]. Nevertheless, Joshi et al. [26] and Baragnan et al. [27] provided evidence that A group are rather protective against malaria. Deepa et al. [45] also researched on ABO blood groups and malaria using several parameters such as parasitic load, hemoglobin level, and platelets count among others. They concluded that A, B and AB groups were predisposed to severe malaria infection than O group. The findings herein which explicitly indicates statistical significance in blood group being a determinant of malaria incidence and or prevalence conforms to the aforementioned works. In this study, blood groups B and O recorded higher malaria incidence than A and AB. Again, blood group A and B had increased odds of testing positive for malaria when compared to O although the difference was not statistically significant. These differences in incidence/prevalence and non-significance of the

parameters (i.e. A and B compared to O) could be attributed to unequal sample sizes as well as inadequate sample sizes of blood groups present in the study (eg. only one person with blood group AB was infected). It is also believed that whereas O group may have the ability to impair rosetting and vascular cytoadherence [44, 23], it is equally (non- O groups) susceptible to malaria infection [46].

Malaria transmission is influenced by seasonality (wet and dry) and according to [47], the highest prevalence/ density rate occurs in the dry season. Similarly, we found increased percentage occurrence and median density of malaria parasite in the dry season than in the rainy season although the relationship was not statistically significant. This difference though not significant is presumably due to the fact that there were higher parous, survival and life expectancy rate of disease vectors during the dry season and as such accounted for greater malaria transmission [48]. It is also noted that heavy rainfall flushes out breeding sites of mosquitoes [49] and may account for the differences observed in the study area. Thus, all these factors given favor parasitic vectors [19] and may be a suggestive reason of increased parasitaemia recorded in the dry season. Our findings, which indicated that season did not have any statistical effect on transmission of malaria, could be attributed to the site's geographical location that influences other potential contributors (eg. Housing, behaviour, disaster, socio-economic factors) of malaria transmission other than season [50].

In addition, the logistic regression model revealed that the type of malaria control used influences malaria transmission in the study area whiles hemoglobin level was found to be associated with malaria parasitaemia. It is known that hematological changes (e.g. Hb, Platelets, Red blood cells etc.) are common complications and play significant roles in malaria pathology. For instance, Maina et al. [32] and Kotepui et al. [51] reported a lower Hemoglobin level (Hb) among malaria-infected people which is consistent with our results. The observation of decreased Hb based on our findings could be due to increased hemolysis or a decreased rate of erythrocytes production caused by malaria parasites [52, 53]. With reference to the type of malaria control, this study showed that the use of Insecticide Treated Nets (ITN) decreased the odds of experiencing the disease whiles use of Mosquito repellents only (MRO) increased an individual's odds of contracting it. Curtis and Mnzava [54] suggest that the combined use of ITNs and MROs are highly effective in combating malaria than the separate use of these methods. The reasons are that though mosquitoes have become resistant to both methods [55], bed nets may still intercept or reduce infected mosquitoes from biting during late nights. On the other hand, when indoors are sprayed at preferred time in the evening, anopheles may not rest there long enough to pick up a lethal dose of insecticide [54]. Additionally, some mosquito species may still survive after the efficacy of the spray has reduced and may bite at night thereby increasing risk of infection.

4. CONCLUSION

This study investigated the determinants of malaria in patients using binary logistic regression. A total positive malaria incidence rate of 27.9% was observed in the study area. Also, this study provides evidence that the type of malaria control used, blood group, gender and Hb are determinants of malaria infection in the district. Hence, it is recommended that various interventions such as sleeping in insecticide treated nets (ITNs), combined use of mosquito repellants and ITNs, proper drainage systems and sanitation practices should be encouraged to help curb the disease. In addition, since the results suggest that younger persons have increased odds of contracting the disease, much emphasis on the use of ITNs should be focused on them since they are more vulnerable. The limitations of this study and its likely impact on the results cannot be ignored since microscopy was the only method used in malaria parasite detection. It is believed that some malaria negative patients could have had malaria parasites if it were subjected to PCR and vice versa thus influencing the findings of this study. Nonetheless, it is expected that this level of variation will be minimal based on the necessary precautions taken. Moreover, future diagnoses of malaria should shift from RDTs and microscopy to PCR techniques to provide more accurate data that can be used to generate very reliable results for the creation of efficient intervention strategies.

CONSENT

Informed consent was obtained from the participants (prior to sample collection) after the study objectives and procedures were clearly explained to them.

ETHICAL APPROVAL

The study was undertaken with permission of the Committee on Human Research Publication and Ethics (CHRPE/ AP/444/16) of the Kwame Nkrumah University of Science and Technology (KNUST), Ghana.

REFERENCES

1. World Health Organization (WHO). World Malaria Report: 2014; World Health Organization: Geneva, Switzerland; 2014.
2. Li J, Collins WE, Wirtz RA, Rathore D, Lal A, McCutchan TF. Geographic subdivision of the range of the malaria parasite *Plasmodium vivax*. *Emerg Infect Diseases*. 2001; 7:35–42.
3. Gnémé A, Guelbéogo WM, Riehle MM, Tiono AB, Diarra A, Kabré GB, Sagnon N, Vernick KD. *Plasmodium* species occurrence, temporal distribution and interaction in a child-aged population in rural Burkina Faso. *Malar J*. 2013; 12:67.doi: 10.1186/1475-2875-12-67.
4. Guillebaud J, Mahamadou A, Zamanka H, Katzelma M, Arzika I, Ibrahim ML, Eltahir EAb, Labbo R, Druilhe P, Duchemin JB, Fandeur T. Epidemiology of malaria in an area of seasonal transmission in Niger and implications for the design of a seasonal malaria chemoprevention strategy. *Malaria J*. 2013; 12: 379. doi: 10.1186/1475-2875-12-379. pmid:24172107.
5. Craig M.H., Snow R.W. and le Sueur D. (1999). A Climate-based Distribution Model of Malaria Transmission in Sub-Saharan Africa. *Parasitology Today*, 15(3):105- 111 PII: S0169-4758(99)01396-4.
6. Gaudart J, Touré O, Dessay N, Dicko AL, Ranque S, Forest L, Demongeot J, Doumbo OK. Modelling malaria incidence with environmental dependency in a locality of Sudanese savannah area, Mali. *Malar J*. 2009; 8:61. doi: 10.1186/1475-2875-8-61.
7. Okafor EE, Amzat J. Problems of Malaria Menace and Behavioral Intervention for its Management in Sub-Saharan Africa. *J Hum Ecol*. 2007; 21(2): 155-162.
8. Kiszewski A, Mellinger A, Spielman A, Malaney P, Sachs SE, Sachs J. A global index representing the stability of malaria transmission. *Am J Trop Med Hyg*. 2004; 70(5): 486–498.
9. Kiszewski AE, Teklehaimanot A. A review of the clinical and epidemiologic burdens of epidemic malaria. *Am J Trop Med Hyg*. 2004; 71(2 Suppl):128-35.
10. Sarpong N, Owusu-Dabo E, Kreuels B, Fobil JN, Segbaya S, Amoyaw F, Hahn A, Kruppa T, May J. Prevalence of malaria parasitaemia in school children from two districts of Ghana earmarked for indoor residual spraying: a cross-sectional study. *Malar J*. 2015; 14:260 doi: 10.1186/s12936-015-0772-6.
11. Teklehaimanot HD, Lipsitch M, Teklehaimanot A, Schwartz J. Weather-based prediction of *Plasmodium falciparum* malaria in epidemic-prone regions of Ethiopia I. Patterns of lagged weather effects reflect biological mechanisms. *Malar J*. 2004; 3:41 doi:10.1186/1475-2875-3-4.
12. Kristan M, Abeku TA, Beard J, Okia M, Rapuoda B, Sang J, Cox J. Variations in entomological indices in relation to weather patterns and malaria incidence in East African highlands: implications for epidemic prevention and control. *Malar J*. 2008; 7: 231. Doi: 10.1186/1475-2875-7-231.
13. World Health Organization (WHO). The African Malaria Report 2003. Geneva. WHO/CDS/MAL, World Health Organization: Geneva, Switzerland; 2003.
14. World Health Organization (WHO). Global burden of disease: Update. 2008. Geneva, Switzerland: World Health Organization; 2004.

- 298 15. Asante FA, Asenso-Okyere K. Economic burden of malaria in Ghana. World Health
299 Organization (WHO). 2003; 1-81; Accessed 13 February 2016.

300 Available:
301 http://www.who.int/countries/gha/publications/Economic_Burden_of_Malaria_in_Ghana .
- 302 16. Martens WJM, Niessen LW, Rotmans J, Jetten TH, McMichael AJ. Potential Impact of Global
303 Climate Change on Malaria Risk. *Environ Health Perspect.* 1995; 103(5): 458-464.
- 304 17. Afrane YA, Klinkenberg E, Drechsel P, Owusu-Daaku K, Garms R, Kruppa T. Does
305 irrigated urban agriculture influence the transmission of malaria in the city of Kumasi, Ghana.
306 *Acta Trop.* 2004; 89:125-134.
- 307 18. Klinkenberg E, McCall PJ, Hastings IM, Wilson MD, Amerasinghe FP, Donnelly MJ. Malaria
308 and Irrigated Crops, Accra, Ghana. *Emerg Infect Diseases.* 2005;11(8):1290 – 1293.
- 309 19. Stoler J, Weeks JR, Getis A, Hill AG. Distance Threshold for the Effect of Urban Agriculture
310 on Elevated self - reported Malaria Prevalence in Accra, Ghana. *Am J Trop Med Hyg.* 2009:
311 80(4): 547–554.
- 312 20. Kwiatkowski DP. How Malaria Has Affected the Human Genome and What Human Genetics
313 Can Teach Us about Malaria. *Am J Hum Genet.* 2005: 77(2), 171–192.
- 314 21. Williams TN. (2006). Human red blood cell polymorphisms and malaria. *Curr Opin Microbiol.*
315 2006: 9(4):388-94. doi:10.1016/j.mib.2006.06.009
- 316 22. Singh IP, Walter H, Bhasin MK, Bhardwaj V, Sudhakar K. Genetic markers and malaria
317 observations in Gujarat, India *Hum Hered.* 1986: 36:31-6.
- 318 23. Gayathri BN, Harendra KML, Gomathi N, Jeevan S, Reethesh RP. Relationship between
319 ABO blood groups and malaria with clinical outcome in rural area of South India. *GJMEDPH.*
320 2013; 2(2):1-7.
- 321 24. Fischer PR, Boone P. Short report: severe malaria associated with blood group. *Am J Trop*
322 *Med Hyg.* 1998: 58:122–123.
- 323 25. Pathirana SL, Alles HK, Bandara S, Phone-Kyaw M, Perera MK, Wickremasinghe AR, Mendis
324 KN, Handunnetti SM. ABO-blood-group types and protection against severe, *Plasmodium*
325 *falciparum* malaria. *Ann Trop Med Parasitol.* 2005: 99:119–124 doi:
326 10.1179/136485905X19946.
- 327 26. Joshi H, Raghavendra K, Subbarao SK, Sharma VP. Genetic markers in malaria patients in
328 Delhi. *Indian J Malariol.* 1987: 24(1):33-8.
- 329 27. Barragan A, Kremsner PG, Wahlgren M. Blood group A antigen is a co receptor in
330 *Plasmodium falciparum* resetting. *Infect Immun.* 2000: 68:2971-5.
- 331 28. Abdul-Aziz AR, Harris E, Munyakazi L. Risk factors in malaria mortality among children in
332 Northern Ghana: a case study at the tamale teaching hospital. *IJBSR.* 2012: 2(5):35-45.
- 333 29. Shayoa EH, Rumishaa SF, Mlozib MRS, Bwanac VM, Mayalaa BK, Malimac RC, Mlachaa T,
334 Mboera LEG. Social determinants of malaria and health care seeking patterns among rice
335 farming and pastoral communities in Kilosa District in central Tanzania. *Acta Trop.* 2015: 144:
336 41–49.
- 337 30. Loha E, Lindtjørn B. Predictors of *Plasmodium falciparum* Malaria Incidence in Chano Mille,
338 South Ethiopia: A Longitudinal Study. *Am J Trop Med Hyg.* 2012: 87(3): 450–459
339 doi:10.4269/ajtmh.2012.12-0155.

- 340 31. Dalatu AB, Garba M, Oguejiofor NJ. An Investigation of Malaria Predictors Using Logistic
341 Regression Model. IJSRST. 2015: 1(5): 153-157.
- 342 32. Maina RN, Walsh D, Gaddy C, Hongo G, Waitumbi J, Otieno L, Jones D, Ogutu BR. Impact
343 of Plasmodium falciparum infection on haematological parameters in children living in
344 Western Kenya. Malar J. 2010: 9(Suppl 3):S4DOI: 10.1186/1475-2875-9-S3-S4.
345
- 346 33. Ministry of Local Government and Rural Development (2006). Wassa Amenfi West. Accessed
347 12 February 2015. Available: http://www.ghanadistricts.com/districts/?rs&_id=140&sa=427.
- 348 34. Trape JF, Rogier C, Konate L, Diagne N, Bouganali H, Canque B, Legros F, Badji A, Ndiaye
349 G, Ndiaye P, Brahimi K, Faye O, Druilhe P, Pereira da Silva L. The Dielmo project: A longitudinal
350 study of natural malaria infection and the mechanisms of protective immunity in a community living in
351 a holoendemic area of Senegal. Am J Trop Med Hyg. 1994; 51: 123- 137.
- 352 35. Hosmer W, Lemeshow S. Applied logistic regression. 2nd ed. David Wiley, New York, No. of
353 pages: xii+373. ISBN 0-471-35632-8; 2000. Accessed 15 April 2015. Available:
354 <https://www.ncbi.nlm.nih.gov/books/NBK2621/>.
- 355 36. Uduakosu G. Re-define and expand global youth age bracket from 15-24 years to 15-35
356 years to capture the youths de facto, and enhance effective representation in global youth issues.
357 2014. Accessed 12 June 2016. Available; <https://gpy2015.crowdcity.com/post/47266>.
- 358 37. Muntaka S, Opoku-Okrah C. The Prevalence of Malaria Parasitaemia and Predisposition of
359 ABO Blood Groups to Plasmodium falciparum Malaria among Blood Donors at a Ghanaian
360 Hospital. AU J Tech. 2013: 16(4): 255-260.
- 361 38. Bottius E, Guanzirolli A, Trape JF, Rogier C, Konate L, Druilhe P. Malaria: even more
362 chronic in nature than previously thought; evidence for subpatent parasitaemia detectable by the
363 polymerase chain reaction. Trans R Soc Trop Med Hyg. 1996; 90(1):15-9.
- 364 39. Vlassoff C, Bonilla E. Gender-related differences in the impact of tropical diseases on women:
365 what do we know? J Biosoc Sci. 1994; 26 (1): 37-53.
- 366 40. Otajevwo FD. Prevalence of malaria parasitaemia and its association with ABO blood
367 grouping among students of Igbinedion University Okada, Nigeria. Br J Med Med Res. 2013; 3(4):
368 1:164-77.
- 369 41. Bonilla E, Rodriguez A. Determining malaria effects in rural Columbia. Soc Sci Med. 1993; 37:
370 1109–1114.
- 371 42. Panda AK, Panda SK, Sahu AN, Tripathy R, Ravindran B, Das BK. Association of ABO blood
372 group with severe falciparum malaria in adults: case control study and meta-analysis. Malar J.
373 2011; 19:10-309. doi: 10.1186/1475- 2875-10-309.
- 374 43. Tadesse H, Tadesse K. Assessing the association of severe malaria infection and ABO blood
375 groups in northwestern Ethiopia. J Vector Borne Dis. 2013; 50: 292–296.
- 376 44. Akhigbe RE, Ige SF, Adegunlola GJ, Adewumi MO, Azeez MO. Malaria, Haemoglobin
377 Genotypes and ABO Blood Groups in Ogbomoso, Nigeria. Int J trop Med. 2011; 6(4): 73-76 DOI:
378 10.3923/ijtm.2011.73.76.
- 379 45. Deepa, Alwar VA, Rameshkumar K, Ross C. ABO blood groups and malaria related clinical
380 outcome. 2011: J Vector Borne Dis. 48: 7–11.
- 381 46. Singh N, Shukla MM, Uniyal VP, Sharma VP. ABO blood groups among malaria cases from
382 district Mandla, Madhya Pradesh. Indian J Malariol. 1995; 32(2): 59-63.

- 383 47. Kobayashi J, Somboon P, Keomanila H, Inthvongsa S, Nambanya S, Inthakone S, Sato Y,
384 Miyagi I. Malaria prevalence and brief entomological survey in a village surrounded by rice
385 fields in Khammouan province, Lao PDR. *Trop Med Int Health*. 2000; 5:17-21.
- 386 48. Vythilingam I, Phetsouvanh R, Keokenchanh K, Yengmala V, Vanisaveth V, Phompida S,
387 Hakim SL. The prevalence of *Anopheles* (Diptera: Culicidae) mosquitoes in Sekong Province, Lao
388 PDR in relation to malaria transmission. *Trop Med Int Health*. 2003; 8(6):525–535.
- 389 49. Nkurunziza H, Gebhardt A, Pilz J. Bayesian modelling of the effect of climate on malaria in
390 Burundi. *Malar J*. 2010; 9:114.
- 391 50. Riedel N, Vounatsou P, Miller JM, Gosoni L, Chizema-Kawesha E, Mukonka V, Steketee
392 RW. Geographical patterns and predictors of malaria risk in Zambia: Bayesian geostatistical
393 modelling of the 2006 Zambia national malaria indicator survey (ZMIS). *Malar J*. 2010; 9:37, DOI:
394 10.1186/1475-2875-9-37.
- 395 51. Kotepui M, Phunphuech B, Phiwklam N, Chupeerach C, Duangmano S. Effect of malarial
396 infection on haematological parameters in population near Thailand-Myanmar border. *Malar J*.
397 2014; 13:218 <http://www.malariajournal.com/content/13/1/218>.
- 398 52. Weatherall D. The anaemia of malaria. McGregor I WW, editor. New York: Churchill
399 Livingstone; 1988.
- 400 53. Phillips RE, Pasvol G. Anaemia of *Plasmodium falciparum* malaria. *Bail Clin Hae*. 1992; 5:315–
401 330. PMID: 1511178.
- 402 54. Curtis CF, Mnzava AEP. Comparison of house spraying and insecticide treated nets for
403 malaria control. *Bull World Health Organ*. 2000; 78: 1389–1400.
- 404 55. N'Guessan R, Corbel V, Akogbéto M, Rowland M. Reduced efficacy of insecticide-treated
405 nets and indoor residual spraying for malaria control in pyrethroid resistance area, Benin. *Emerg*
406 *Infect Diseases*. 2007; 13: 199– 206.