

**International Journal of TROPICAL DISEASE
& Health**

23(4): 1-11, 2017; Article no.IJTDH.33700
ISSN: 2278-1005, NLM ID: 101632866

Prevalence Rate and Contributory Factors of Malaria in the Amenfi West District, Ghana

Esther Love Darkoh^{1*} and John Aseidu Larbi¹

¹Department of Theoretical and Applied Biology, College of Science, Kwame Nkrumah University of Science and Technology, Kumasi, Ghana.

Authors' contributions

This work was carried out in collaboration between both authors. Both authors read and approved the final manuscript.

Article Information

DOI: 10.9734/IJTDH/2017/33700

Editor(s):

(1) Zhiheng Zhou, Thyroid Cancer Research Laboratory, Massachusetts General Hospital, Harvard Medical School, Boston, USA.

Reviewers:

(1) Dawit Getnet Ayele, The Johns Hopkins University, USA.
(2) Salakchit Chutipongvivate, Ministry of Public Health, Thailand.
(3) Aina Oluwagbemiga Olanrewaju Nigerian Institute of Medical Research, Yaba, Nigeria.
Complete Peer review History: <http://www.sciencedomain.org/review-history/19440>

Original Research Article

Received 26th April 2017
Accepted 10th May 2017
Published 9th June 2017

ABSTRACT

Aims: To determine the prevalence rate and some contributory factors of malaria in the Amenfi West District of Ghana. This study investigated the prevalence 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 March 2016 and November 2016.

Methodology: A purposive sampling technique was used to select 240 patients of both sexes aged 0- 81 years old at Asankrangwa District Hospital. Venous blood was collected and presence of malaria parasites was observed microscopically on thick smears. Demographic data such as age, sex and the type of malaria control method(s) used were retrieved from patients. All data was recorded and analyzed using SPSS (version 23) statistical software. Categorical data was compared using Pearson's Chi- Square test set at a significant level of 5%. For parasite density, factor effects were examined using Mann-Whitney and Kruskal-Wallis tests where appropriate to investigate statistical differences at 95% confidence interval. Regression analysis was also employed to model the presence (positive) or absence (negative) of malaria in a patient.

*Corresponding author: Email: amamaamekyedarkoh@gmail.com;

Results: Overall malaria prevalence was 27.9% (67/240). Parasite density ($P = 0.048$) and prevalence ($P = 0.000$) 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 infected female anopheles mosquitoes. Five species of Plasmodium protozoa namely; *Plasmodium falciparum*, *P. vivax*, *P. malariae*, *P. ovale* and *P. knowlesi* 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]. However, between 2010 and 2015, global incidence rate of malaria fell by 29% with decreased mortality rates of about 31% in the African region [5]. According to Craig et al. [6] and Gaudart et al. [7], the distribution of malaria has always been geographically specific and biased towards sub-Saharan Africa [8]. Currently, there are 104 malaria endemic countries [9] 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 [10] and still suffer from the burden of malaria mortality [11].

In Africa, malaria continues to be endemic in countries such as Ethiopia [12], Niger [4], Mali [7], Kenya [13] and Nigeria [8]. A study conducted by Ayele et al. [14] on risk factors of malaria revealed that malaria infection was positively correlated with poor socio-economic conditions. In addition, risk of malaria infection has been associated with poor housing conditions and sanitation as well as inaccessibility of insecticides treated nets [15]. Nevertheless, its eradication has been successful in other African countries like Algeria,

Egypt, Libya, Morocco and Tunisia [16]. In Ghana, malaria is a principal public health problem that plagues all segments of the society [17]. Its effect on people is quite immense particularly with high risk occurring in pregnant women and children because they have less immunity [18]. Major factors that account for prevalence of malaria in this country include poor environmental conditions and lack of preventive services [19]. Aside these factors, transmission has also been influenced by urban agriculture where well irrigated sites often serve as breeding grounds for malaria vectors [20-22].

Human resistance or susceptibility to infection by malarial parasites are influenced also by several factors including a wide range of genetic polymorphism [23,24]. It is known that, susceptibility to several infectious diseases are related to the patients' blood group and malaria is of no exception [25,26]. For example, blood group O erythrocytes have reduced resetting (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 [27,28]. Contrarily, Joshi et al. [29] and Barragan et al. [30] reported that, A erythrocytes rather inhibits rosette formation.

Furthermore, several studies have documented age [31,4] and gender [32] as drivers of malaria infection. Though Loha and Lindtjörn [33] revealed that malaria prevalence was higher in males than females, Dalatu et al. [34] 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 [34],

hemoglobin level [35] and type of malaria control [32]. Nonetheless, little is known about how these factors influence malaria infection in the district. Therefore, this study sort to unravel the prevalence rate and some contributory factors of malaria infection in the Amenfi West District of Ghana for improved management decisions locally and globally.

2. MATERIALS 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 (Fig. 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 [36].

2.2 Study Population

The study population comprised of patients who presented at the Asankrangwa district hospital and were referred to the laboratory for malaria test by the consulting physicians. After obtaining an informed consent, demographic information such as age, sex and the type of malaria control use were retrieved from patients. A total of 240 patients with ages between 0-81 years were recruited in the study. Six months (3 months for each season) was dedicated to the laboratory research between March 2016 and November 2016.

2.3 Collection of Blood Samples

Three milliliters of venous blood was collected into EDTA tube from each patient. The blood was smeared immediately onto a clean grease-free slide for preparations of thick blood films, while some was used for blood group identification using ABO typing. The rest of the blood was used for hematological analysis (Full Blood Count).

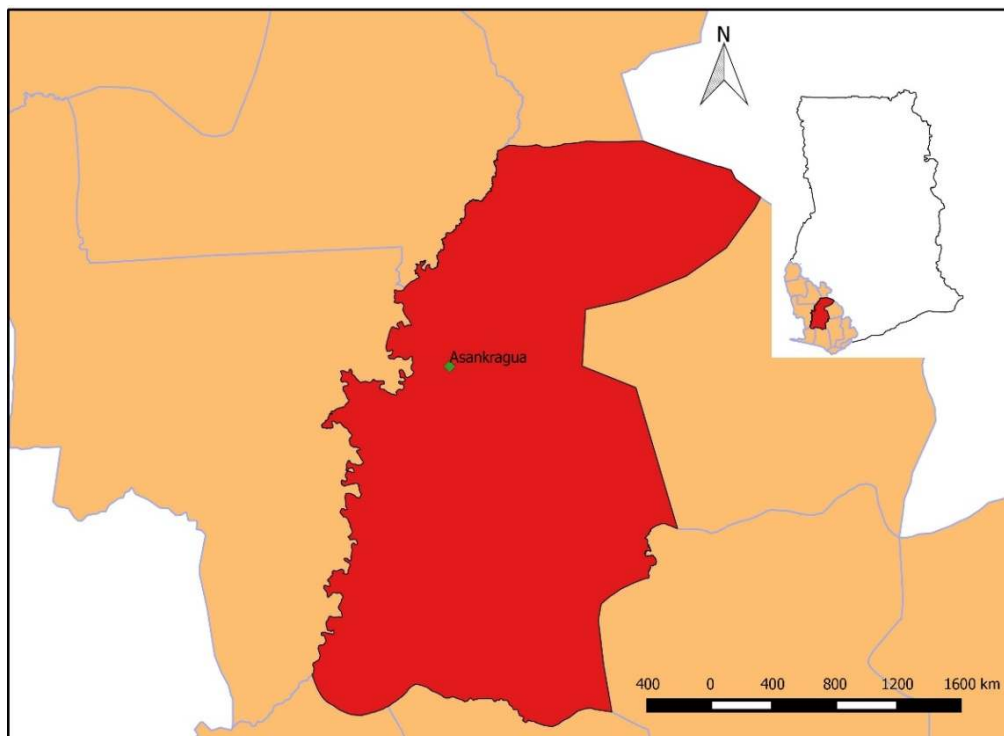


Fig. 1. Map of Ghana showing study area in Amenfi West district

2.4 Staining of Blood Film and Determination of Malaria Parasitaemia

Thick blood films were stained with 10% Giemsa for 10 minutes for the detection of *Plasmodium* parasites. The slides were then observed microscopically under the x 100 (oil immersion) objectives. Samples with no visible parasites after observing 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 [37].

2.5 Data Analysis

The Scientific Program for Social Sciences (SPSS, version 23) was used to analyze the data collected. 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. The level of significance was set at $P < .05$. For parasite density, factor effects were examined using Mann-Whitney and Kruskal-

Wallis tests where appropriate to investigate statistical differences at 95% confidence interval.

In addition, binary logistic regression was employed to model the presence (positive) or absence (negative) of malaria in a patient. The independent variables used to build the model included Age, Sex, Blood group, Weight, HB, WBC, RBC, Season, and Type of malaria control used by patients. This approach was preferred to other methods (eg. multiple regression) because it requires fewer assumptions and can incorporate a mixture of numerical as well as categorical independent variables [38]. With respect to age groups, 0-14 and 15-24 years were considered in the regression model. This is because children and youth are considered highly susceptible to malaria infection than adults [17]. Thus, according to Uduakosu [39], WHO and UNICEF defines children and youth as people between ages of 0-14 as well as 15-24 years respectively.

3. RESULTS

3.1 Malaria Prevalence and Intensity

Out of 240 patients recruited in the study, 157 (65.4%) were females while 84 (34.6%) were males with their ages ranging from 0 to 81 years. Also, after examining the blood films, malaria parasites were detected in 27.9% (67/240) of the participants in the study area (Table 1).

Table 1. Prevalence 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				57.263	0.000
≥ 15	121 [54.4%]	8	11.900		
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				0.775	0.379
Rainy	122 [50.8%]	31	46.300		
Dry	118 [49.2%]	36	53.700		
Blood group				8.277	0.041
A	42 [17.5%]	13	19.400		
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		

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

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

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

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; 5-9 years = 72868.00) recording higher median values than older individuals (10-14years = 27667.00; ≥ 15 years = 1571.50). Nonetheless, there was no statistical difference between the blood groups based on parasite density ($P = 0.329$).

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 4. 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 3. Mean parasite density in relation to age, blood group and season

Variable		Mean	Median	Min.	Max.	Test statistic	P-value
Age Group	0-4	86298.75	43843.00	551.00	610937.00	7.913	0.048
	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	3.433	0.329
	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		
Season	Dry	62951.06	47767.50	482.00	209000.00	551.000	0.930
	Rainy	89705.42	19500.00	551.00	610937.00		

Min. = Minimum value, Max. = Maximum value

Table 4. 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 repellent (Coil & Spray); BG = Blood group

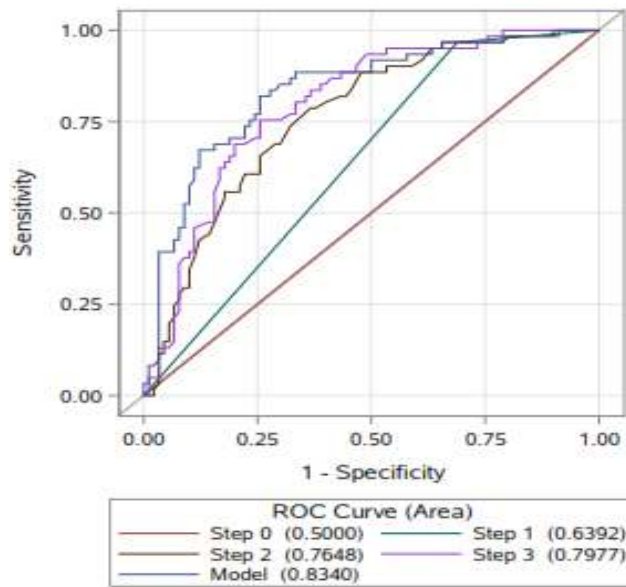


Fig. 2. ROC curve for all model building steps – BLR

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 (Fig. 2).

4. DISCUSSION

According to Abdul-Aziz et al. [31] and Muntaka and Opoku-Okrah [40], 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 [33] and as such harbor more parasite at a given time [41].

Furthermore, incidence percentage based on our result was significantly higher in females than males. This corroborates the findings of Vlassoff and Bonilla [42] as well as Otajewwo [43] who reported a higher infection rate in females than males. In contrast, Bonilla and Rodriguez [44] 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 [23,25] with the available literature showing varying results relating to the subject [45,46]. For example, an examination conducted on blood groups in determining malaria incidence in Nigeria [47] 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 [27,28]. Nevertheless, Joshi et al. [29] and Baragnan et al. [30] provided evidence that A group are rather protective against malaria. Deepa et al. [48] 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 [47,26], it is equally (non- O groups) susceptible to malaria infection [49].

Malaria transmission is influenced by seasonality (wet and dry) and according to [50], 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 [51]. It is also noted that heavy rainfall flushes out breeding sites of mosquitoes [52] and may account for the differences observed in the study area. Thus, all these factors given favor parasitic vectors [22] 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 [53].

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. [35] and Kotepui et al. [54] 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 [55,56]. 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 [57] 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 [58], 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 [57]. 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 especially for the younger ones, proper drainage systems and sanitation practices should be encouraged to help curb the disease.

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.

ACKNOWLEDGEMENTS

We express our appreciation to Mr. Eric Adjei Lawer at the University for Development Studies (UDS) for his critical reading/insightful comments of the manuscript.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. World Health Organization (WHO). World malaria report. 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. World Health Organization. World malaria report; 2016. (Accessed 4 May 2017) Available:<http://www.who.int/malaria/media/world-malaria-report-2016/en/>
6. Craig MH, Snow RW, Le Sueur D. A climate-based distribution model of malaria transmission in Sub-Saharan Africa. *Parasitology Today*. 1999;15(3):105-111. PII: S0169-4758(99)01396-4
7. 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
8. 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.
9. 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.
10. 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.
11. 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
12. 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
13. 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
 14. Ayele DG, Zewotir TT, Mwambi HG. Prevalence and risk factors of malaria in Ethiopia. *Malaria Journal.* 2012;11:195.
DOI: 10.1186/1475-2875-11-195
 15. Molla E, Ayele B. Prevalence of malaria and associated factors in Dilla Town and the surrounding rural areas, Gedeo Zone, Southern Ethiopia. *J Bacteriol Parasitol.* 2015;6:242.
DOI: 10.4172/2155-9597.1000242
 16. World Health Organization (WHO). The African Malaria Report 2003. Geneva. WHO/CDS/MAL, World Health Organization. Geneva, Switzerland; 2003.
 17. World Health Organization (WHO). Global burden of disease: Update 2008. Geneva, Switzerland: World Health Organization; 2004.
 18. Asante FA, Asenso-Okyere K. Economic burden of malaria in Ghana. *World Health Organization (WHO).* 2003;1-81.
(Accessed 13 February 2016)
Available:http://www.who.int/countries/gha/publications/Economic_Burden_of_Malaria_in_Ghana
 19. Martens WJM, Niessen LW, Rotmans J, Jetten TH, McMichael AJ. Potential impact of global climate change on Malaria risk. *Environ Health Perspect.* 1995;103(5): 458-464.
 20. Afrane YA, Klinkenberg E, Drechsel P, Owusu-Daaku K, Garms R, Kruppa T. Does irrigated urban agriculture influence the transmission of malaria in the city of Kumasi, Ghana. *Acta Trop.* 2004;89:125-134.
 21. Klinkenberg E, McCall PJ, Hastings IM, Wilson MD, Amerasinghe FP, Donnelly MJ. Malaria and irrigated crops, Accra, Ghana. *Emerg Infect Diseases.* 2005; 11(8):1290–1293.
 22. Stoler J, Weeks JR, Getis A, Hill AG. Distance threshold for the effect of urban agriculture on elevated self - reported Malaria Prevalence in Accra, Ghana. *Am J Trop Med Hyg.* 2009;80(4):547–554.
 23. Kwiatkowski DP. How malaria has affected the human genome and what human genetics can teach us about malaria. *Am J Hum Genet.* 2005;77(2):171–192.
 24. Williams TN. Human red blood cell polymorphisms and malaria. *Curr Opin Microbiol.* 2006;9(4):388-94.
DOI: 10.1016/j.mib.2006.06.009
 25. Singh IP, Walter H, Bhasin MK, Bhardwaj V, Sudhakar K. Genetic markers and malaria observations in Gujarat, India *Hum Hered.* 1986;36:31-6.
 26. Gayathri BN, Harendra KML, Gomathi N, Jeevan S, Reethesh RP. Relationship between ABO blood groups and malaria with clinical outcome in rural area of South India. *GJMEDPH.* 2013;2(2):1-7.
 27. Fischer PR, Boone P. Short report. Severe malaria associated with blood group. *Am J Trop Med Hyg.* 1998;58:122–123.
 28. Pathirana SL, Alles HK, Bandara S, Phone-Kyaw M, Perera MK, Wickremasinghe AR, Mendis KN, Handunnetti SM. ABO-blood-group types and protection against severe, *Plasmodium falciparum* malaria. *Ann Trop Med Parasitol.* 2005;99:119–124.
DOI: 10.1179/136485905X19946
 29. Joshi H, Raghavendra K, Subbarao SK, Sharma VP. Genetic markers in malaria patients in Delhi. *Indian J Malariol.* 1987; 24(1):33-8.
 30. Barragan A, Kreamsner PG, Wahlgren M. Blood group antigen is a co receptor in *Plasmodium falciparum* resetting. *Infect Immun.* 2000;68:2971-5.
 31. Abdul-Aziz AR, Harris E, Munyakazi L. Risk factors in malaria mortality among children in Northern Ghana: A case study at the tamale teaching hospital. *IJBSR.* 2012;2(5):35-45.
 32. Shayoa EH, Rumishaa SF, Mlozib MRS, Bwanac VM, Mayalaa BK, Malimac RC, Mlachaa T, Mboera LEG. Social determinants of malaria and health care seeking patterns among rice farming and pastoral communities in Kilosa District in central Tanzania. *Acta Trop.* 2015;144: 41–49.
 33. Loha E, Lindtjörn B. Predictors of *Plasmodium falciparum* Malaria incidence in Chano Mille, South Ethiopia: A Longitudinal Study. *Am J Trop Med Hyg.* 2012;87(3):450–459.
DOI: 10.4269/ajtmh.2012.12-0155
 34. Dalatu AB, Garba M, Oguejiofor NJ. An investigation of Malaria predictors using

- logistic regression model. IJSRST. 2015; 1(5):153-157.
35. Maina RN, Walsh D, Gaddy C, Hongo G, Waitumbi J, Otieno L, Jones D, Ogutu BR. Impact of *Plasmodium falciparum* infection on haematological parameters in children living in Western Kenya. *Malar J.* 2010; 9(Suppl 3):S4.
DOI: 10.1186/1475-2875-9-S3-S4
 36. Ministry of local government and rural development. *Wassa Amenfi West*; 2006.
(Accessed 12 February 2015)
Available:http://www.ghanadistricts.com/districts/?rs&_id=140&sa=427
 37. Trape JF, Rogier C, Konate L, Diagne N, Bouganali H, Canque B, Legros F, Badji A, Ndiaye G, Ndiaye P, Brahimi K, Faye O, Druilhe P, Pereira Da Silva L. The Dielmo project. A longitudinal study of natural malaria infection and the mechanisms of protective immunity in a community living in a holoendemic area of Senegal. *Am J Trop Med Hyg.* 1994;51:123-137.
 38. Hosmer W, Lemeshow S. *Applied logistic regression.* 2nd Ed. David Wiley, New York. 2000;xii+373.
ISBN: 0-471-35632-8
(Accessed 15 April 2015)
Available:<https://www.ncbi.nlm.nih.gov/books/NBK2621/>
 39. Uduakosu G. Re-define and expand global youth age bracket from 15-24 years to 15-35 years to capture the youths de facto, and enhance effective representation in global youth issues; 2014.
(Accessed 12 June 2016)
Available:<https://gpy2015.crowdcity.com/post/47266>
 40. Muntaka S, Opoku-Okrah C. The prevalence of Malaria Parasitaemia and Predisposition of ABO Blood Groups to *Plasmodium falciparum* Malaria among blood donors at a Ghanaian Hospital. *AU J Tech.* 2013;16(4):255-260.
 41. Bottius E, Guanzirolli A, Trape JF, Rogier C, Konate L, Druilhe P. Malaria. Even more chronic in nature than previously thought; Evidence for subpatent parasitaemia detectable by the polymerase chain reaction. *Trans R Soc Trop Med Hyg.* 1996;90(1):15-9.
 42. Vlassoff C, Bonilla E. Gender-related differences in the impact of tropical diseases on women: What do we know? *J Biosoc Sci.* 1994;26(1):37-53.
 43. Otajevwo FD. Prevalence of malaria parasitaemia and its association with ABO blood grouping among students of Igbinedion University Okada, Nigeria. *Br J Med Med Res.* 2013;3(4):1164-1177.
 44. Bonilla E, Rodriguez A. Determining malaria effects in rural Columbia. *Soc Sci Med.* 1993;37:1109-1114.
 45. Panda AK, Panda SK, Sahu AN, Tripathy R, Ravindran B, Das BK. Association of ABO blood group with severe falciparum malaria in adults: Case control study and meta-analysis. *Malar J.* 2011;19:10-309.
DOI: 10.1186/1475-2875-10-309
 46. Tadesse H, Tadesse K. Assessing the association of severe malaria infection and ABO blood groups in Northwestern Ethiopia. *J Vector Borne Dis.* 2013;50: 292-296.
 47. Akhigbe RE, Ige SF, Adegunlola GJ, Adewumi MO, Azeez MO. Malaria, haemoglobin genotypes and ABO blood groups in Ogbomoso, Nigeria. *Int J Trop Med.* 2011;6(4):73-76.
DOI: 10.3923/ijtmed.2011.73.76
 48. Deepa Alwar VA, Rameshkumar K, Ross C. ABO blood groups and malaria related clinical outcome. *J Vector Borne Dis.* 2011;48:7-11.
 49. Singh N, Shukla MM, Uniyal VP, Sharma VP. ABO blood groups among malaria cases from district Mandla, Madhya Pradesh. *Indian J Malariol.* 1995;32(2):59-63.
 50. Kobayashi J, Somboon P, Keomanila H, Inthvongsa S, Nambanya S, Inthakone S, Sato Y, Miyagi I. Malaria prevalence and brief entomological survey in a village surrounded by rice fields in Khammouan province, Lao PDR. *Trop Med Int Health.* 2000;5:17-21.
 51. Vythilingam I, Phetsouvanh R, Keokenchanh K, Yengmala V, Vanisaveth V, Phompida S, Hakim SL. The prevalence of anopheles (*Diptera: Culicidae*) mosquitoes in Sekong Province, Lao PDR in relation to malaria transmission. *Trop Med Int Health.* 2003;8(6):525-535.
 52. Nkurunziza H, Gebhardt A, Pilz J. Bayesian modelling of the effect of climate on malaria in Burundi. *Malar J.* 2010; 9:114.
 53. Riedel N, Vounatsou P, Miller JM, Gosoni L, Chizema-Kawesha E, Mukonka V, Steketee RW. Geographical patterns and predictors of malaria risk in Zambia:

- Bayesian geostatistical modelling of the 2006 Zambia national malaria indicator survey (ZMIS). *Malar J.* 2010;9:37. DOI: 10.1186/1475-2875-9-37
54. Kotepui M, Phunphuech B, Phiwklam N, Chupeerach C, Duangmano S. Effect of malarial infection on haematological parameters in population near Thailand-Myanmar border. *Malar J.* 2014;13:218. Available:<http://www.malariajournal.com/content/13/1/218>
55. Weatherall D. The anaemia of malaria. McGregor I WW, editor. New York: Churchill Livingstone; 1988.
56. Phillips RE, Pasvol G. Anaemia of *Plasmodium falciparum* malaria. *Bail Cli Hae.* 1992;5:315–330. PMID: 1511178
57. Curtis CF, Mnzava AEP. Comparison of house spraying and insecticide treated nets for malaria control. *Bull World Health Organ.* 2000;78:1389–1400.
58. N'Guessan R, Corbel V, Akogbéto M, Rowland M. Reduced efficacy of insecticide-treated nets and indoor residual spraying for malaria control in pyrethroid resistance area, Benin. *Emerg Infect Diseases.* 2007;13:199–206.

© 2017 Darkoh and Larbi; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:

*The peer review history for this paper can be accessed here:
<http://sciencedomain.org/review-history/19440>*