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acta SATECH **3**(1): 122 - 131 (2009)

Malaria and genetic polymorphism of haemoglobin genotypes and ABO Blood Groups

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ABSTRACT

Malaria places a huge burden on human life, and has been reported to be a key health problem affecting developing countries. This study aimed at determining the distribution of Hemoglobingenotypes, ABO blood groups and severity of malaria infection among various hemoglobin genotypes.Seven hundred and sixty-five patients, who expressed symptoms of malaria, were clinically examined for malariasymptoms. Two hundred were clinically diagnosed for malaria infection. Blood films were prepared from these and examined for malaria parasites. Their Haemoglobin (Hb) genotypes and ABO blood groups were determined. The patterns of malaria infection were determined among Hb genotypes and ABO blood groups. One hundred and eighty had Plasmodium falciparum infection. Age group 1-5years had the highest frequency of malaria infection, 50 (27.77%) with a mean parasite count of 9,600/UL. Age group 36 – 40 was least infected, with a mean parasite count of 1,000/UL. The differences within the mean parasite counts, among the age groups were significant ($p<0.05$). Males 98 (54.4%) were more infected than the females 82 (45.6%). The mean parasite load of the males was also significantly higher than that of the females, ($P<0.05$). Hb genotype AA patients (60%) were more infected than the Hb genotype AS.

Key Words: *Malaria, Haemoglobin genotypes, Infection, ABO Blood group.*

INTRODUCTION

Malaria is a disease caused by parasites of the genus *Plasmodium* and transmitted by the female *Anopheles* mosquitoes. Malaria places a huge burden on human life. Malaria is one of the key health issues affecting developing countries, particularly in sub-Saharan Africa. It is also a major cause of disease for inhabitants of tropical and sub-tropical areas. Despite intensive control efforts during the twentieth century, approximately forty percent of the world population still remains at the risk of the infection.. It has been identified that children and pregnant women are most at risk with *P. falciparum* infection, as reported by (WHO, 1999) that most morbidity and mortality were caused by *P. falciparum* and the greatest disease is in African children under five years of age. Nearly twenty-five percent of all childhood mortality in Africa is caused by malaria (WHO, 2000), which has been identified as one of the causes of delay in economic development United Nations Children Fund (UNICEF) reported that malaria is a major cause of poverty and it was noted that poverty exacerbates the malaria situation (UNICEF, 2001). Haemoglobin transports oxygen in human red blood cells. In normal adult haemoglobin (HbA) chains are designated alpha (α) and beta (β). The four chains are folded and fitted together to form a rough globular molecule with a molecular weight of about sixty-four thousand, five hundred (Thompson and Thompson, 1980). The authors explained further, that the "formula" for the composition

is $\alpha_2^A\beta_2^A$, which translate to two α chains, typical of those in HbA, plus "two β chains, typical of those in HbA". The formula is often written as $\alpha_2\beta_2$ omitting the superscript. The two kinds of chains are almost equal in length, the α chain having 141 amino acids and the β chain 146.

Ingram (1956) showed that the difference was due to a single amino acid and since then the entire amino acid sequence of HbA and HbS has been determined. Taylor *et al.* (1997) reported that fault occurs at the sixth amino acid, in the β-chain. In HbS however, it is replaced by Val., as shown below:

HbA	= *Val*	– *His*	– *Leu*	– *Thr*	– *Pro*	– *Glu*	– *Glu*	– *Lys* –
HbS	= *Val*	– *Vis*	– *Leu*	– *Thru*	– *Pro*	– *Val*	– *Glu*	– *Lys* –
Amino Acid =	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*

The substitute of a valine, for glutamine acid in each of the two β-chains of the haemoglobin molecule, is the only biochemical difference between HbA and HbS. This difference, which results in an altered electric charge, explains the difference in electrophoretic mobility of the two haemoglobin Taylor *et al*. (1997) also noted that the presence of valine makes de-oxygenated HbS less soluble. Therefore when HbS loses its oxygen the molecules comes out of solution and crystallise into rigid, rod-like fibres. These change the shape of the red cell, which is normally a flat circular disc, to a sickle shape under low oxygen tension.

Normally there are two-functional β-globin genes, one inherited from each parent (father and mother). Heterozygotes for HbS have one normal and one defective β gene (they are said to have sickle cell trait and are designated AS). Their red cells which contain a mixture of HbA and HbS function fairly normally in most cases. By contrast, red cells of homozygotes, designated SS, contain mainly HbS, as both β-globin genes are of the abnormal type (Marsh, 1993). Their cells form an abnormal shape (sickle) under conditions of low oxygen tension, which leads to both increased in red cells lysis and obstruction of vascular flow as the abnormal cells lodge in small blood vessels (Marsh, 1993). Except under circumstance with a high availability of medical care, the homozygous condition (sickle cell disease) is almost uniformly fatal in childhood, indicating that there must be a very high degree of advantage to heterozygote, to maintain the gene at high frequencies (Marsh, 1993).

It has been observed that sickle trait provides a survival advantage over people with normal haemoglobin, in regions where malaria is endemic (Luzzatto, 1979). Population, field and clinical studies have provided convincing evidence that where *Plasmodium falciparum* malaria is endemic, non-immune subjects who have sickle cell trait [haemoglobin (Hb) AS] have a survival advantage over subjects who have only normal adult haemoglobin [Hb-AA] (Luzzatto, 1979). Children who are carriers of haemoglobin S have ninety percent protection against severe malaria, as reported by (Hill, et al., 1991).

Luzzatto (1979) explained sickle cell anaemia and natural selection, as related to malaria, compared to AS heterozygotes, people with the AA genotype (normal haemoglobin), have a greater risk of dying of malaria. Homozygotes who die young may not pass on their genes to the next generation. Individuals with the AS genotype do not develop sickle cell anaemia and also have less chance of getting malaria. They are able to survive and reproduce in malaria-infected regions.

It was also noted by Allison, (1954); Edington and Watson-Williams, (1965) and Livingstone, (1971) that there is conclusive evidence that the gene responsible for the production of haemoglobin HbS is maintained at high frequency in tropical Africa because of the biological advantages it confers on heterozygotes (HbAS or Sickle trait individual), through partial protection from *Plasmodium falciparum*. Salimonu (2003) also reported that patients who are heterozygous for the sickle gene (HbAS) are usually more resistant to *Plasmodium (P.) falciparum* infection, than those who are homozygous (HbSS, HbAA). Thus whilst HbAS patients, with a given load of *P. falciparum* infection do not show clinical signs and symptoms of malaria, HbAA or HbSS individuals with the same load of parasite would have been down with malaria fever, (Allison 1954 and 1956) Haemoglobin C is formed by substitution of a glutamate for lysine at position six in the β-chain (Marsh, 1993). The sequence is HbC: Val – His – Leu –Thru – Pro – Lys – Gly – Lys (Thompson and Thompson, 1980). Haemoglobin C has a close relationship in many ways, with HbS. It is present at high frequencies in a localized part of West Africa, which is Burkina-Fasso and Ghana (Marsh, 1993).

It was reported that haemoglobin C confers protection against malaria. In a study by Modiano *et al*. (2001) found that haemoglobin C heterozygotes had significantly fewer episodes of *P. falciparum* malaria than did controls with only haemoglobin A. The risk of malaria was lower still in subjects who were homozygous for haemoglobin C. It was suggested by Marsh (1993) that cells from the homozygote CC was refractory to parasite growth in culture, possibly due to their resistance to bursting and releasing Merozoites rather than any deficit in the intracellular environment.

An individual with antigen A in the red cell membranes is classified as having blood group A. If only B antigens are present the blood group is B. If both antigens are present the blood group will be AB and if no antigens are present the blood group is O as shown in Table 1 below (Taylor *et al*., 1997).

The relationship between blood group and susceptibility to malaria has been studied by several workers, the results have been contradictory (Livingstone, 1971). For

example, it has been suggested that blood group B confers a selective advantage in relation to malaria infection, and high B frequency in population groups and endemicity of malaria might be related (Osisanya, 1983). According to Bold (1983) some people were more prone to mosquito bites than others. Wood (1981) found that under laboratory conditions *Anopheles gambiense* seemed to recognize blood groups and to feed preferentially on group O. The basis for this recognition was not known although it may be related to the occurrence of ABO substances on skin cells and in sweat secretions (Bold, 1983)

In areas of intensive malaria transmission especially in Africa, epidemiological evidence suggested influence of genetic polymorphism on *Plasmodium sp*. Infection, with the notion that variations in host response to infection (malaria infection) might have a genetic basis is not new. Conditions affecting the structure of the beta globulin chain of haemoglobin (HbS, HbC, HbE) rates of synthesis of globin chains, (alpha-thalassaemia and beta-thalassaemia), the level of a key red cell emzyme (glucose-6-phosphate dehydrogenase, G6PD) and the condition affecting the red cell membrane and cytoskeleton (Duffy blood group negatively and hereditary ovalo-cytosis) have been identified by Marsh (1993) as the red cell polymorphisms for which there is now reasonable evidence to support a 'malaria' hypothesis.

This investigation was therefore undertaken to determine thefollowing:

(1) Malaria status of the studied group
(2) Haemoglobin genotype of malaria patients
(3) Relationship between haemoglobin genotype and malaria infection
(4) The severity of malaria infection among various haemoglobin genotypes
(5) The distribution of ABO blood groups among malaria infected patients
(6) The relationship between ABO blood group and malaria
(7) Relate onset of infection to severity of infection among various haemoglobin genotype
(8) Relate onset of infection to severity among ABO blood groups.

MATERIALS AND METHODS

Study Population

Subjects used in the study were patients who were clinically diagnosed to have malaria infection, by a physician, in the military hospital. A verbal consent was obtained from the patients, prior to the study. A total of seven-hundred and sixty five subjects were examined, out of which two-hundred subjects were clinically diagnosed to have malaria fever and sent for malaria parasite test. Out of the two hundred subjects examined for malaria parasite, one hundred and eighty had patent parasitaemia.

Determination Of Parasite Burden

Blood film Preparation

Thick and thin blood films were prepared and stained with Field's staining technique. Thin blood films were used to identify the species of *Plasmodium*. The part of the slides where parasites were well stained and white blood cells were evenly distributed, in the middle and towards the tail, were selected, using the oil immersion objective, parasites (asexual) were counted, against two-hundred white blood cells and recorded as parasites per micro-litre (μl) of blood i.e. parasites/μl of blood.

$$= \frac{\text{Parasite Count} \times \text{WBC Count}}{200}$$

Where there was no parasite 'No parasite count was recorded'. Thick blood films were examined microscopically.

Determination Of Haemoglobin Genotype

Equal volumes of water (saponin could also be used) and blood samples were dropped into a test-tube and mixed very well to haemolyse the red blood cells. The haemolysed samples and the control samples (known Hb genotypes AS and AC) were applied on to the cellulose acetate paper for electrophoresis at 220V and left for thirty minutes. Electrophoretic movements of haemoglobin were observed, separations were also observed and movement of haemoglobin was recorded, after comparison with the control samples.

Determination Of Abo Blood Groups

Five drops each, of blood samples were dropped on glass slides and grouping sera, Anti A, Anti B, anti A+B and Anti D were added, mixed and observed for reactions, in form of agglutination. Observed reactions were recorded.

Analysis Of Data

Data collected from the study was analysed, using the following descriptive statistics of mean, frequency distribution, percentages, standard deviation, standard error, minimum and maximum limit. The Inferential Statistics of Analysis of Variance (ANOVA) was used with the alpha value set at 0.05. Correlation coefficient was also used.

RESULTS

Malaria Status Of The Studied Group

A total of seven hundred and sixty-five (765) subjects were examined for malaria parasites, out of which 200 (26.14%) patients were clinically diagnosed to have malaria parasites. Among the 200 patients, 180 (90%), had patent parasitaemia; while the remaining

20 (10%) had no malaria parasites.

Malaria Distribution Among Various Age Groups
Among the patients with parasites, 50 (27.77%) were within the ages of 1-5 years, 26 (14.44%) fell within the age group of 6-10 years, 14 (7.77%) were within ages 11 - 15 years, 24 (13.33%) fell in the age group 16-20 years. While 28(15.55%), 30 (16.16%), 6 (3.33%), 2 (1.11%) were within the ages of 21 - 25, 26-30, 31.35 and 36-40 years respectively as shown in table 2 below.
Age group 31-35 years had the highest mean parasite load of 25,333.3/ul of blood with minimum and maximum parasite counts of 18,000.00 and 40,000.00/ul of blood respectively followed by ages 21-25 years with mean parasite of 59206/ul of blood with minimum and maximum number parasite of 2,000.00 and 20,500.00/ul of blood respectively. While ages 6-10 years had the least mean parasite load of 5884.6/ul of blood, minimum and maximum number of parasite of 1,000.00 and 16,500.00/ul of blood respectively. The difference in the mean parasite count within the age groups: 1-5, 6-10, 11-15, 16-20, 21-25, 26-30, 31-35 and 36-40 in this study was significant ($p<0.05$) using Analysis of Variance (ANOVA).

Malaria Distribution among sexes
Male 98 (54.4%) were more infected with malaria than the female 82 (45.6%). The mean parasite load of the male was 12428.57/ul of blood and that of female was 6963.4/ul of blood as shown in table 3. The difference in the mean parasite of the male and female was significant ($p<0.05$).

Hb Genotypes Among Patients with Malaria
108 (60%) had Hb genotype A while 72 (40%) had Hb genotype AS among the 180 patients with parasitaemia in the study as shown in table 4. None of the malaria patients was Hb genotype SS, SC, CC nor AC.

Parasite Count And Hb Genotypes
Patients with the genotype A haboured more malaria parasite with mean parasite of 11481.5/ul of blood with minimum and maximum number of parasite of 1,000.00 and 21,000.00/ul of blood respectively than the Hb genotype AS patients with mean parasite of 7,625.00/ul of blood with minimum and maximum number of parasite of 1,000.00 and 40,000.00/ul of blood respectively as shown in table 4. The difference in the mean parasite of the Hb genotype A and Hb genotype AS was significant ($p<0.05$).

Relationship between Hb Genotypes and Malaria infection
There was a positive correlation between Hb genotypes and malaria infection among the studied group ($r = .273$, $df = 179$, $p<0.01$.

ABO Blood Groups among Patients with Malaria
Patients who were blood group A were 59 (32.8%); blood group B was 19 (10.6%); blood group O were 99 (55.0%) while blood group AB were 3 (1.7%).

Malaria Parasite Density Among ABO Blood Groups Patients

The Mean parasite of blood group A was 8923.7/ul of blood with minimum and maximum number of parasite of 1,000.00 and 20,000.00/ul of blood respectively, while the mean parasite of blood group O was 10919.19/ul of blood with minimum and maximum number of parasites of 1,000.00 and 40,000.00/ul of blood respectively and then the mean parasite count of blood group AB patients was 7666.7/ul of blood with minimum and a maximum number of parasite of 3,000.00 and 10,000.00/ul of blood respectively. The mean parasite count among blood group B patients was 8342.1/ul of blood with minimum and maximum number of parasite of 1,000.00 and maximum number parasite of 1,000.00 and 18,000.00/ul of blood respectively as shown in table 5.

Onset of Infection to Severity of Malaria Parasite Among Hb A Genotype Patients
Twenty (16.53%) of the genotype A patients had an onset of infection of 2 days with mean parasite count of 11,500.00/ul (2,000.00 minimum and 20,000.00/ul maximum number of parasites). Those with three days of onset of infection was 32 (26.45%) with mean parasite count of 10875.00/ul of blood (4,000.00 minimum and 20,000.00 maximum number of parasite). Patients with 4 days and 5 days of onset of infection were 47 (238.84%) and 22 (18.18%) respectively with mean parasite count of 8617.00/ul of blood (2,000.00 minimum and 21,000/ul maximum number of parasite0 and 11681.82/ul of blood mean parasite count (3,000 minimum and 20,500.00/ul maximum number of parasite0 respectively as shown in table 6.
The difference in the mean parasite count of the onset of infection of two, three four and five days of onset of infection was significant ($p<0.05$) using Analysis of Variance OANOVA). There was a positive correlation between onset of infection and intensity of parasite among subjects with the Hb genotype A patients ($r = .077$, $df = 107$, $p<0.05$).

Onset of infection to Severity of malaria parasite among Hb Genotype AS
In Hb genotype AS patients with onset of infection of two, three, four, five and six days of infection were 18

(23.68%), 20 (26.4%), 20 (26.4%), 16 (13.2%) and 8 (10.56%) respectively with mean parasite count of 5555.56/ul (3,000.00minimum and 17,000/ul maximum number of parasite); 5200.00/ul (minimum of 1,000.00 and maximum of 2100.00/ul number of parasite); 5,950.00/ul (2000.00 minimum and 21,000/ul maximum number of parasite); 8700.00/ul (3,000.00 minimum and 20,500.00/ul maximum number of parasite0 and 14,875.00/ul (4,000.00 minimum and 40,000.00/ul maximum number of parasite) respectively as shown in table 7. The difference in the mean parasite count of the onset of infection within days two, three, four, five and six was significant ($p<0.05$).

Onset of Infection to Severity of Malaria Infection Among Blood Group A

In blood group A patients with onset of infection f two, three, four, five and six days were 15 (122.72); 15 (22.72%); 18 (27.36%); 14 (21.28%); 4 (4.08%) respectively and with mean parasite count of 9100.00/ul (2000.00 minimum and 20,000.00/ul maximum number of parasite); 8733.33/ul (1,500.00 minimum and 20,000/ul maximum number of parasite); 7833.33/ul (1,000.00 minimum and 18,000.00 maximum number of parasite); 7857.14/ul (1,500.00 mi9nimum and 19,000.00/ul maximum number of parasite; 2,000.00/ul (2,000.00 minimum and 2,000/ul maximum number of parasite) respectively (Table 8). The difference in the mean parasite count of the onset of infection of days two, three, four, five and six was significant ($p<0.05$). There was a negative correction between onset of infection compared to intensity of parasite infection ($r = -.093$, of =58 $p<0.05$).

Onset of Infection to Severity of Malaria infection among Blood Group B.

In blood group B patients with onset of infection of two days were 6 (31.55%) with mean parasite count of 3,000.00/ul (1,000.00 minimum and 5,000.00/ul maximum number of parasite); 3 days was 5 (26.3%) with mean parasite count of 13500.00/ul (9,500.00 minimum and 18,000.00/ul maximum number of parasite) and lastly patients with onset of infection of four days were 8 (42.8%) had mean parasite count of 9125.00/ul (1,500.00 minimum and 16,000.00/ul maximum number of parasite as shown in table 9.

The difference in the mean parasite count of the onset of infection of days two, three, and four was significant ($p<0.05$). There was positive correlation between onset of infection and intensity of infection in blood group B ($r = .383$, $df = 18$, $p<0.05$).

Onset of Infection to Severity of Malaria Infection Among Blood Group O

In blood group O, 16 (14.14%) had onset of infection of two days with mean parasite count of 10312.50/ul (3000.00 minimum and 17,000/ul maximum number of parasite); 30 (27.00) had onset of infection of three days with mean parasite count of 8133.33/ul (1000.00 minimum and 21,000.00/ul maximum number of parasite); 42 (37.8%) with onset of infection of four days and mean parasite count of 7857.14/ul (2000.00 minimum and 21,000.00/ul maximum number of parasite); 17 (15.3%) had onset of infection of five days with mean parasite count of 13588.24/ul (3000.00 minimum and 20,500.oo/ul maximum number of parasite and lastly 7 (5.4)\%) had onset of infection of six days with mean parasite count of 18,500.00 /ul (4,000.00 minimum and 40,000.00/ul maximum number of parasite as shown in table 10. The difference in the mean parasite count of the onset of infections of days two, three, four, five and six was significant ($p<0.05$). There was positive correlation between onset of infection and the intensity of parasite in blood group O ($r = .353$, $df = 98$, $p<0.05$).

Onset of Infection to Severity of Parasitic Infection Among Blood Group AB

In blood group AB, 1 (25%) had onset of infection of 2 days with mean parasite count of 10,500/ul (10,500.00/ul minimum and maximum number of parasite); 2 (50%) had onset of infection of 3 days with mean parasite count of 4750.00/ul (9500.00/ul minimum and maximum number parasite and lastly 1 (25%) of patients had onset of infection of five days with mean parasite count of 2750.00/ul (3000.00 minimum ad maximum number of parasite) (Table 11). The difference in the mean parasite count of the onset of infection of days two, three and five was not significant ($p<0.05$). There was a negative correlation between onset of infection and intensity of parasites ($4 =-.9978$; $df = 2$ $p<0.05$).

DISCUSSION AND CONCLUSION

Out of the subjects who were examined for malaria parasite, ages one to five years were most affected with malaria. This agreed with WHO (1999), which reported that most morbidity and mortality was caused by P. *falciparum* infection and the greatest disease was in African children under five years of age. The least affected was in the ages of thirty six to forty years. These categories of people (adult) would have developed immunity against malaria infection, which believed that adults in malaria endemic areas enjoyed substantial immunity which provided protection against malaria. The males were more infected than the females. Finding more malaria cases in males agreed with the findings of most researchers, as supported by Madhu and Rai Chowdhun (1980) in their study, that more males were infected with malaria than females. The males also harboured more of the malaria parasite than the females in the present study.

Distribution of Hb genotypes among the malaria patients indicated that patients with Hb genotype A were more infected than the Hb genotype individuals, in the present study. This agrees with Salimonu (2003), which report

that patients who were heterozygous for sickle gene (Hb AS) were usually more resistant to *plasmodium falciparum* infection, than those who were homozygous (Hb A). Thus, whilst Hb AS patients, with a given load of Plasmodium falciparum infection do not show clinical signs and symptom of malaria; Hb A or Hb AS individuals with the same parasite load, would have been severely affected by malaria fever. Also in this study, the mean parasite count of patients with Hb genotype A was higher than the mean parasite count among Hb AS individuals. This agrees with Fleming et al (1984) report that population, field and clinical studies have provided evidence that, the presence of sickle haemoglobin (Hbs) in the red cell had been found to limit the multiplication of malaria parasite.

This study also revealed that there was relationship between malaria infection and Hb genotypes. This indicated that there were differences in susceptibility to malaria among Hb genotypes A and AS individuals. This study also indicated that blood group O individuals were more susceptible to malaria, than other blood groups A, AB and B, while blood group AB were least infected with malaria. The present study did not agree with Livingstone (1971), Osisanya (1983), that blood group B conferred a selective advantage, in relation to malaria infection, and high B frequency in population groups and endemicity of malaria might be related. In their study, it was found that there was no significant relationship between ABO blood groups and susceptibility to malaria infection. Hence, the more an individual was bitten by the malaria vector, the more chance of being infected with malaria parasites.

Blood group O patients harboured more malaria parasites than other blood groups A, AB and B; while blood group AB had the least mean malaria parasite count.In Hb genotypes A and AS, there was positive correlation between onset of infection and intensity of parasite infection. As days of symptom of malaria infection increased, parasite density also increased. There were differences in the mean parasite count of the onset of infection of two, three, four, five, six days in Hb genotypes A and AS. Among blood groups A, AB and O patients, there were significant differences ($p<0.05$), in the mean parasite count at the onset of infection,) while in the blood group AB patients, the differences in the mean parasite of the days of onset of infection, was not significant. In blood group A and AB patients, there was a negative correlation between onset of infection and malaria parasites intensity, i.e. as the days of onset of infection increased, the parasite decreased. In blood groups B and O patients, there was positive correlation between onset of infection and the intensity of parasite count i.e. as the onset of infection increased parasite density also increased.

Table 1: Human Blood Groups

Blood Group	A	B	AB	O
Antigen	A	B	A + B	–
Antibody	B	A	–	A + B

Table 2: Distribution of Malaria According to Age

Age Group	N	%	Mean parasite/ul of blood	Standard Deviation	Standard Error	Minimum No. of Parasite	Maximum No. of Parasite
1 –5	**50**	27.77	9600.00	6353.5	898.5	1000.00	20000.00
4– 10	**26**	14.44	5884.62	5130.9	1006.3	100.00	16500.00
11– 15	**14**	7.77	9928.6	3251.4	869.0	3500.00	13000.00
16 – 20	**24**	13.33	9916.7	6823.3	1392.8	1000.00	21000.00
21 – 25	**28**	15.55	13357.1	5920.6	1118.9	2000.00	20500.00
26 – 30	**30**	16.66	7700.0	5384.5	983.1	1500.00	16000.00
31 – 35	**6**	3.33	25333.3	11360.8	4638.00	18000.00	40000.00
36 – 40	**2**	1.11	11000.0	5000.0	-000	11000.00	11000.00
Total	**180**	**99.96**	**9938.9**	**6936.2**	**5170**	**1000.00**	**40000.00**

Table 3: Distribution of Malaria among Sexes

Sex	N	Percentage	Mean parasite/ul of blood	Standard Deviation
Male	98	54.4	12428.6	6973.4
Female	82	45.6	6963.4	5623.4

Table 4: Distribution of Hb Genotype Among Malaria Patients

Hb Genotype	N Freq.	%	Mean parasite/ul of blood	Standard Deviation	Standard Error	Minimum No. of Parasite	Maximum No. of Parasite
AA	108	60	11481.48	5692.23	547.73	7000.00	21000.00
AS	72	40	7625.00	7963.27	938.48	1000.00	40000.00
TOTAL	180	100	9938.89	6936.19	516.99	1000.00	40000.00

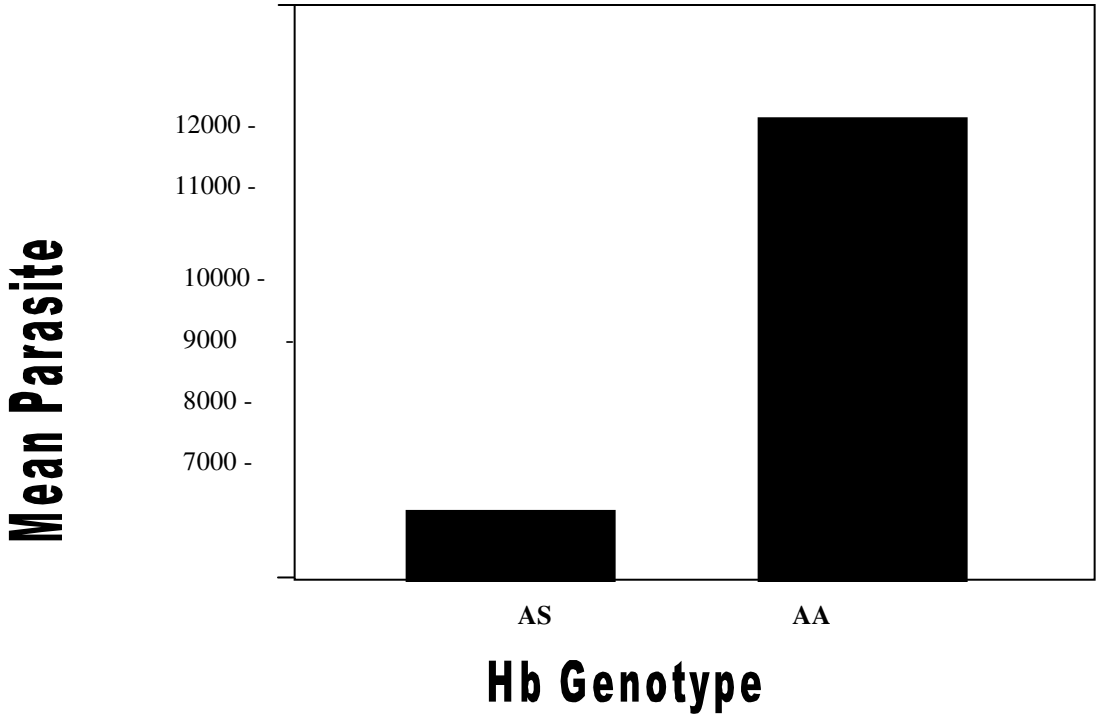

Figure 1:Hb genotype and mean parasite density

Table 5 below.Table 5: ABO Blood Groups and Malaria Infection

Blood group	N	%	Mean Parasite/Ul of Blood	Standard Deviation	Minimum No. of Parasite	Maximum No. of Parasite
A	59	32.9	8923.73	6911.9	1000.00	20000.00
B	19	10.6	8342.1	6353.2	1000.00	18000.00
O	99	55.0	10919.19	7043.1	1000.00	40000.00
AB	3	1.3	7666.7	4072.3	3000.00	10500.00
Total	180	100	9938.9	6936.2	1000.00	40000.00

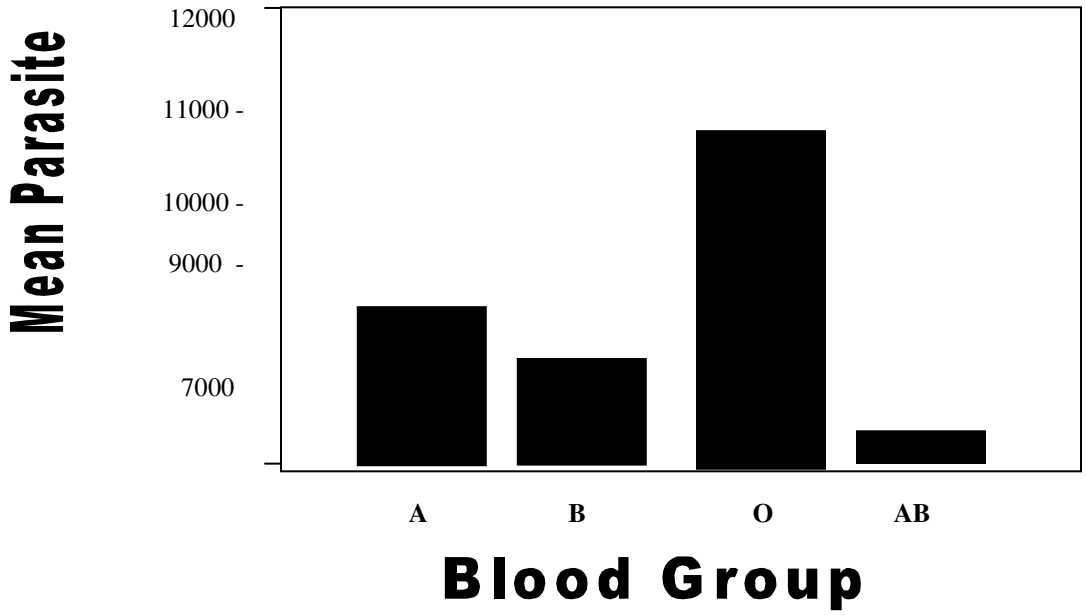

Figure 2 shows the ABO Blood Groups and mean parasite density

Table 6: Onset of infection Among Hb Genotype A Patients

Onset/day	N	%	Mean Parasite/ul of Blood	Minimum No. of Parasite	Maximum No. of Parasite	Standard Deviation
2	20	16.53	11500.00	2,000.00	20,000.00	5567.76
3	32	26.45	20875.02	4,000.00	20,000.00	6157.87
4	47	38.84	8617.02	2,000.00	21,000.00	6207.65
5	22	18.18	11681.82	3,000.00	20,500.00	7700.37

Table 7: Onset of Infection among HB Genotype AS patients

Onset/day	N	%	Mean Parasite/ul of Blood	Minimum No. of Parasite	Maximum No. of Parasite	Standard Deviation
2	18	23.68	5555.56	3,000.00	17,000.00	4901.65
3	20	26.4	5200.00	1,000.00	21,000.00	6135.32
4	20	26.4	6900.00	2,000.00	21,000.00	6006.36
5	10	13.2	8700.00	3,000.00	20,500.00	6350.10
6	8	10.56	14875.00	4,000.00	40,000.00	16589.08

Table 8: Onset of Infection among Blood Group A Patients

Onset/day	N	%	Mean Parasite/ul of Blood	Minimum No. of Parasite	Maximum No. of Parasite	Standard Deviation
2	15	22.72	9100.00	2,000.00	20,000.00	7091.75
3	15	22.72	8733.32	1,500.00	20,000.00	8364.18
4	18	27.36	7833.33	1,000.00	18,000.00	6087.00
5	14	21.28	7857.14	1,500.00	19,000.00	7769.34
6	4	6.08	2000.00	2,000.00	2,000.00	-000

Table 9: Onset of infection Among Blood Group B Patients

Onset/day	N	%	Mean Parasite/ul of Blood	Minimum No. of Parasite	Maximum No. of Parasite	Standard Deviation
2	6	31.58	3,000.00	1,000.00	5,000.00	1788.85
3	5	26.3	13,500.00	9,500.00	18,000.00	4153.31
4	8	42.08	6890.73	1,500.00	16,000.00	6890.73

Table 10: Onset of infection Among Blood Group O Patients

Onset/day	N	%	Mean Parasite/ul of Blood	Minimum No. of Parasite/ul	Maximum No. of Parasite/ul	Standard Deviation
1	16	14.41	10312.50	3,000.00	17,000.00	4966.13
2	30	27.00	8133.32	1,000.00	21,000.00	5981.19
3	42	37.8	7857.43	2,000.00	21,000.00	6223.76
4	17	15.3	13588.24	3,000.00	20,500.00	5974.52
5	6	5.4	18500.00	4,000.00	40,000.00	18038.55

Table 11: Onset of Infection Among Blood Group AB

Onset/day	N	%	Mean Parasite/ul of Blood	Minimum No. of Parasite/ul	Maximum No. of Parasite/ul	Standard Deviation
2	1	25.00	10,500.00	10,500.00	10,500.00	-
3	2	50.00	4,750.00	9,500.00	9,500.00	6717.51
5	1	25.00	3,000.00	3,000.00	3,000.00	-

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