

RISK DETERMINANTS TO CONGENITAL MALARIA IN ILORIN, NIGERIA

O.M. KOLAWOLE ^{*1a}, A.S. BABATUNDE ^b, A.A.G. JIMOH ^c,
O.R. BALOGUN ^c AND I.G. KANU ^a

^a Department of Microbiology, Faculty of Science, P.M.B. 1515,
University of Ilorin, Ilorin, Nigeria.

¹ Malaria I., Department of Molecular Parasitology, Bernhard Nocht
Institute for Tropical Medicine, Hamburg, Germany.

^b Department of Haematology, P.M.B. 1515, University of Ilorin, Ilorin, Nigeria.

^a Department of Obstetrics and Gynaecology, P.M.B. 1515, University of Ilorin, Ilorin, Nigeria.

Key words : Risk determinants, Malaria, Congenital

Abstract - A Study on risk determinants to congenital malaria was carried out for seven months between March and September 2006 at Ilorin using three different hospitals; Eytayo, Surulere Medical hospital and Centre Gboro specialist hospital. Malaria parasite detection was performed on a set of 130 maternal, fetal, placental and cord blood samples using thick film method. Blood group and haemoglobin genotype was also determined using tile technique and haematocrit method respectively. The prevalence of maternal, fetal, placental, and cord parasitaemia were 37(28.46%), 29(22.31%), 33(25.38%) and 30(23.08%) respectively. Fetal weight, sex, Blood group, maternal height, Gestation age, placental weight, Diabetes, Socio-economic status, maternal educational background, husbands occupation and husbands educational background did not show positive correlation with maternal, fetal, placental and cord parasitaemia ($p > 0.05$). However, Haemoglobin genotype and type of accommodation showed positive correlation with maternal, fetal, placental and cord parasitaemia ($p < 0.05$) while maternal weight showed positive correlation with maternal parasitaemia only ($p < 0.05$).

INTRODUCTION

Malaria is a protozoa disease transmitted by female Anopheles mosquito caused by minute parasitic protozoa of the genus *Plasmodium*, which infect human and insect host alternatively (Conell, 2002). Malaria and pregnancy are two conditions, which have an impact on one another.

Globally malaria infection during pregnancy is a major health problem (Ikeh *et al.* 2005). It has profound effect on pregnancy outcome and neonatal life (Jimoh, 2004). The hormonal and immunological changes brought about by pregnancy aggravate malaria. The increased nutritional needs and the immunodepressant action of certain hormones (mainly cortisol) lead to a state of immunodepression, which may affect the functions of T-lymphocytes (Ikeh *et al.* 2005).

Consequently materno-fetal complications such

as cerebral malaria, still birth, abortions, anaemia hypoglycemia, prematurity, intrauterine growth retardation, maternal mortality (Jimoh, 2004) and congenital malaria (Ikeh *et al.* 2005) has been reported in pregnant women with malaria.

This study was designed to find out risk determinants to congenital malaria at Ilorin, Kwara State using three hospital, Eytayo, Surulere Medical hospital and Centre Gboro Specialist hospital.

MATERIAL AND METHODS

STUDY AREA

The study was carried out in Ilorin at three different Hospitals, Eytayo Hospital, Surulere Medical Hospital and Centre Gboro Children Specialist; the first two representing privates and the last a public hospital respectively.

* Author to whom correspondence be addressed - Dr. O.M.A. Kolawole
Email: tomak74@yahoo.com; oml@unilorin.edu.ng

Subjects

A hundred and thirty mothers and their babies delivered at the labour wards of these Hospitals were used after due parental, consultant and Institutional consent. Pregnant mothers presenting at the labour ward with history of fever were excluded from the study.

Parameters

Questionnaires were applied to mothers and appropriate information filled such as Socio-economic status, types of accommodation, husband's occupation, educational background, gestational age, Diabetic etc. Moreso clinical examinations such as maternal height and weight were done. Laboratory test such as maternal haemoglobin genotype and blood group were carried out. Malaria parasite test was also carried out both on maternal and fetal blood samples. The weight of the placenta and fetal weight were taken on standard weighing scale and recorded.

Blood Collection

Aseptic procedures were adopted in the collection of the blood samples. The placenta was washed with clean water immediately after delivery before the blood was collected. The cord was cleaned with 70 percent alcohol to avoid maternal blood contamination before it was dispensed into EDTA bottles. Fetal blood was obtained from a peripheral vein on the dorsum of the hand using a 23G needle. Venous maternal blood was obtained using 5mL syringe and dispensed into EDTA bottles.

Preparation and Examination of Thick Blood Film

The procedure was carried out according to the method described by (Cheesbrough, 2000). Slides were examined microscopically by the X100 (oil immersion) objective.

Procedure for Haemoglobin Genotype and Blood Group

The procedure for the determination of haemoglobin genotype and blood group of the subjects were carried out according to the method described by Cheesbrough, (1987).

Statistical Analysis

The data generated in this study were analyzed using student's t-test for equality of means while Chi-square test was used to test for independence among the categorical variables at α level of 0.05 (level of significance).

RESULTS

The prevalence rates of maternal, fetal, placental and cord parasitaemia were 37(28.46%), 29 (22.31%), 33 (25.38%) and 30 (23.08%) respectively (Table 1). The numbers of male babies with parasitaemia were 18 (24.7%) and female babies with parasitaemia were 11 (19.3%), male babies were more parasitized compared to female babies (Table 2).

Parasitaemia prevalence was higher in babies that weighed 0.6-2.9kg compared to babies that weighed 3.0-4.0kg (Table 2). Fetal sex and weight did not show positive correlation with maternal, fetal, placental and cord parasitaemia ($p > 0.05$).

It was discovered genotypically that parasitaemia was higher in individual with haemoglobin genotype AA. This was followed by individual with haemoglobin genotype AS. Only one patient had haemoglobin genotype AC but showed negative parasitaemia except for the placenta, which showed positive parasitaemia. (Table 3).

With reference to blood group in Table 4, it was revealed that AB+ blood groups were most frequently parasitized. This was followed by A+ blood group and B+ blood group respectively. The lowest prevalence occurred in O+ and A+ blood group. Genotype showed positive correlation with maternal, fetal, placental and cord parasitaemia ($p < 0.05$) while blood group did not show positive correlation with maternal, fetal, placental and cord parasitaemia ($p > 0.05$).

According to Table 5 those that weighed 41-50kg had the highest prevalence (50.0%) followed by 51-60kg (36%). The lowest prevalence was seen in those that weighed 81-90kg (10.0%). Maternal weight showed positive correlation with maternal parasitaemia ($p < 0.05$).

With regard to maternal height in Table 6, parasitaemia prevalence was seen in mothers whose height were 1.90-2.00m (40.0%) while the least was seen in those whose height were 1.49-1.59m (25.0%). Maternal height did not show positive correlation with maternal, fetal, placental and cord parasitaemia.

Women with small placenta (0.3-0.5kg) had the highest placental parasitization compared to women with larger placenta (0.8-1.0kg) and average sized placenta (0.6-0.7kg) (Table 7).

With reference to gestation age in Table 7 the highest prevalence occurred in mother and babies

with gestation age 37-40 weeks while the least occurred in mothers and babies with 32-36 weeks gestation age. Only one sample was collected within the gestation age of 27-31 weeks and it showed positive parasitaemia. Placental weight and gestation age did not show positive correlation with maternal, fetal, placental and cord parasitaemia ($p>0.05$).

Table 8 depicts relationship between type of accommodation and malaria parasitaemia. A high prevalence rate of 35.6%, 28.8%, 52.0% and 30.1% maternal, fetal, placental and cord parasitaemia respectively was observed among those living in face to face type of accommodation while those living in self contained had low prevalence of 19.3%, 14.0%, 14.0% and 14.0% maternal, fetal, placental and cord parasitaemia respectively. Type of accommodation showed strong correlation with maternal, fetal, placental and cord parasitaemia ($p<0.05$).

Only one woman was diabetic and showed positive parasitaemia. Other women were not diabetic (Table 8). Diabetes did not show positive correlation with maternal, fetal placental and cord parasitaemia ($p>0.05$).

The socio economic status of the women was determined with reference to their occupation and their educational background. It was observed that middle class had the highest prevalence (30.6%) in maternal blood which was closely followed by higher class (28.3%) & lower class (25.0%) (Table 9).

With respect to maternal educational background, parasitaemia was highest among those with no formal education (50.0%), closely followed by Secondary school leavers (32.1%) and primary school leavers (26.1%). The lowest prevalence was seen among the graduates (22.9%) (Table 10). Maternal occupation and educational background did not show positive correlation with maternal, fetal, placental and cord parasitaemia ($p>0.05$).

The highest parasitaemia prevalence rate was seen among the mothers whose husbands were secondary school leavers (35.4%). This was followed by those without formal education (25.0%). The lowest prevalence was seen among the graduates (21.3%) (Table 11).

According to husband occupation in Table 12, mothers whose husbands were businessmen showed highest parasitaemia (33.3%) while the lowest prevalence was seen among working class (22.2%). Husband educational background and

occupation did not show positive correlation with maternal, fetal, placental and cord parasitaemia ($p>0.05$).

DISCUSSION

The prevalence rates of maternal, fetal, placental and cord parasitaemia recorded in this study were 28.4%, 22.31%, 25.38% and 23.08% respectively (Table 1). Higher values of congenital malaria which were 55% and 46.7% have been reported by Reinhardt *et al.* (1978) and Obiajunwa *et al.* (2005) respectively.

Malaria parasitaemia did not seem to have affected the fetal weight of the babies in this study (Table 2). This could be due to the fact that malaria parasite present in the fetal blood were few as a result of barrier offered by placental, Ezechukwu *et al.* (2004) and Sule-Odun *et al.* (2002) made similar observation but this contradicts the work done by (Larkin and Thuma, 1991) who reported that parasitized babies had lower birth weight.

Male babies were more parasitized than female babies (Table 2). This could be as a result of their genetic make up. This is in agreement with the finding of Brabin, (1983). Fetal sex did not show positive correlation with fetal and maternal parasitaemia ($p>0.05$).

Women with haemoglobin genotype AA showed high prevalence of parasitaemia with about 41.6% as compared with 5% recorded in haemoglobin genotype AS women (Table 3). This is in accordance with the findings of Cheesbrough, (2000). Genotype showed strong correlation with maternal, fetal placental and cord parasitaemia ($p<0.05$).

It was observed that blood group A+ had the highest prevalence followed by AB+ while A- recorded the lowest prevalence, B- blood group did not show any positive parasitaemia (Table 4). The work of Macleod and Rhode, (1998) discovered high prevalence in A+ women. Blood group did not show positive correlation with maternal, fetal placental and cord parasitaemia ($p>0.05$).

Maternal weight in Table 5 showed that those with lower weight of 41-50kg had the highest parasitaemia while the least was found among those that weighed 81-90kg. This could be as a result of loss of appetite. Another reason may be due to fatty degeneration and muscle atrophication due to high rate of tissue metabolism as a result of fever. This is in consonance with the report of Francis and Warrel, (1993). Maternal

Table 1. Prevalence rates of malaria parasitaemia in maternal, fetal, placental and cord blood.

Group	Positive Parasitaemia (%)	Negative Parasitaemia (%)	Total (%)
Maternal	37 (28.46%)	93 (71.54%)	130(100%)
Fetal	29 (22.31%)	101(77.69%)	130(100%)
Placental	33 (25.38%)	97 (74.62%)	130(100%)
Cord blood	30 (23.08%)	100 (76.92%)	130(100%)

Table 2. Fetal Sex and Weight

Fetal sex	Maternal blood		Fetal blood		Placental blood		Cord blood	
	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)
Male	21(28.8%)	52 (71.2%)	18 (24.7%)	55(75.3%)	20(27.4%)	53(72.6%)	19(26.0%)	54(74.0%)
Female	16(28.1%)	41(71.9%)	11(19.3%)	46(80.7%)	13(22.8%)	44(77.2%)	11(19.3%)	46(80.7%)
p-value	P>0.05		P>0.05		P>0.05		P>0.05	
Fetal weight (kg)								
0.6-2.9	17(34.0%)	33(66.0%)	13(26.0%)	37(74.0%)	15(30.0%)	35(70.0%)	12(24.0%)	38(76.0%)
3.0-4.0	20(25.0%)	60(75.0%)	16(20.0%)	64(80.0%)	18(22.5%)	62(77.5%)	18(22.5%)	62(77.5%)
P-value	P>0.05		P>0.05		P>0.05		P>0.05	

P>0.05 showed no positive correlation

Table 3. Haemoglobin Genotype in relation to maternal, fetal, placental and cord parasitaemia.

Genotype	Maternal blood		Fetal blood		Placental blood		Cord blood	
	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)
AA	32(41.6%)	45(58.4 %)	25(32.5%)	52(.67.5%)	27(35.1%)	50(64.9%)	27(35.1%)	50(64.9%)
AS	5(9.6%)	47(90.4 %)	4(7.7)	48(92.3%)	47(9.6%)	47(90.4%)	3(5.8%)	49(94.2%)
AC	-	1(100%)	-	1(100%)	1(100%)	-	-	1(100%)
P-value	P<0.05		P<0.05		P<0.05		P<0.05	

P<0.05 showed positive correlation

weight showed positive correlation with maternal parasitaemia only ($p<0.05$).

Maternal height showed highest prevalence in those whose height were 1.90-2.00m (40.0%) while the least was seen in those whose height were 1.49-1.59 (25.0%) (Table 6). Maternal height did not show positive correlation with maternal, fetal, placental and cord parasitaemia ($p>0.05$).

It was observed that women with small placenta had the highest placental parasitization compared to those with larger and average sized placental (Table 7). Reason could be that malaria parasites affected their growth and development. This contradicts the work of Jimoh, (2003) who reported that women with average sized placental were more parasitized compared to women with small placenta.

The rate of parasitaemia was highest in babies with gestation age of 32-36 weeks (Table 7). This

could probably be due to the fact that most mothers were not infected at the early stage of pregnancy. Secondly it could possibly be that malaria parasite does not cross from the maternal blood through the placenta to the foetus immediately as a result of barrier offered by the placenta. This report contradicts the work of Goruip, (1989).

However this study confirmed that type of accommodation showed strong correlation with malaria parasitaemia ($p<0.05$) (Table 8). Women living in face to face type of accommodation had highest prevalence. The high prevalence rates in those living in face to face type of accommodation may be due to the fact that gutters with water which provide suitable breeding places for mosquito, surround the houses and also the houses are left without the doors and windows being covered with mosquito net. The prevalence of

Table 4. Blood group in relation to maternal, fetal, placental and cord parasitaemia.

Blood group	Maternal blood		Fetal blood		Placental blood		Cord blood	
	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)
A+	12(31.6%)	26(68.4 %)	9(23.7%)	29(76.3%)	12(31.6%)	26(68.4%)	8(21.1%)	30(78.9%)
A-	1(14.3%)	6(85.7%)	1(14.3%)	6(85.7%)	1(14.3%)	6(85.7%)	1(14.3%)	6(85.7%)
B+	8(30.8%)	18(69.2%)	5(19.2%)	21(80.8%)	5(19.2%)	21(80.8%)	6(23.1%)	20(76.9%)
B-	-	4(100%)	-	4(100%)	-	4(100%)	-	4(100%)
AB+	8(61.5%)	5(38.5%)	7(53.8%)	6(46.2%)	7(53.8%)	6(46.2%)	7(53.8%)	6(46.2%)
O+	7(19.4%)	29(80.6%)	6(16.7%)	30(83.3%)	7(19.4%)	29(80.6%)	7(19.4%)	29(80.6%)
O-	-	-	-	-	-	-	-	-
P-value	P>0.05		P>0.05		P>0.05		P>0.05	

P>0.05 showed no positive correlation.

Table 5. Maternal weights in relation to maternal, fetal, placental and cord parasitaemia.

Maternal weight (kg)	Maternal blood		Fetal blood		Placental blood		Cord blood	
	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)
41-50	5(50%)	5(50.0%)	3(30.0%)	7(70.0%)	3(30.0%)	7(70.0%)	3(30.0%)	7(70.0%)
51-60	24(36.4%)	42(63.6%)	21(31.8%)	45(68.1%)	18(27.3%)	48(72.7%)	18(27.3%)	48(72.7%)
61-70	6(19.4%)	25(80.6%)	6(19.4%)	25(80.6%)	6(19.4%)	25(80.6%)	7(22.6%)	24(77.4%)
71-80	1(7.7%)	12(92.3%)	1(7.7%)	12(92.3%)	1(7.7%)	12(92.3%)	1(7.7%)	12(92.3%)
81-90	1(100%)	9(80.0%)	2(20.0%)	8(80.0%)	1(10.0%)	9(90.0%)	1(10.0%)	9(90.0%)
P-value	P<0.05		P>0.05		P>0.05		P>0.05	

P<0.05 showed positive correlation ; P>0.05 showed no positive correlation.

Table 6. Maternal height in relation to maternal, fetal, placental and cord parasitaemia

Maternal height (m)	Maternal blood		Fetal blood		Placental blood		Cord blood	
	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)
1.49-1.59	2(25.0%)	6(75.0%)	1(12.5%)	7(87.5%)	1(12.5%)	7(87.5%)	1(12.5%)	7(87.5%)
1.60-1.69	6(28.6%)	15(71.4%)	6(28.6%)	15(71.4%)	5(23.8%)	16(76.2%)	6(28.6%)	15(71.4%)
1.70-1.79	13(28.0%)	37(74.0%)	9(18.0%)	41(82.0%)	11(22.0%)	39(78.0%)	10(20.0%)	40(80.0%)
1.80-1.89	10(27.8%)	26(72.2%)	9(25.0%)	27(75.0%)	9(25.0%)	27(75.0%)	9(25.0%)	27(75.0%)
1.90-2.00	6(40.0%)	9(60.0%)	4(26.7%)	11(73.3%)	7(46.7%)	8(53.3%)	4(26.7%)	11(73.3%)
P-value	P>0.05		P>0.05		P>0.05		P>0.05	

P>0.05 showed no positive correlation.

parasitaemia was low among those living in self contained type of accommodation because it is believed that the inhabitants could possibly be educated and fall in the higher class. They however ensure that all preventive measure to reduce mosquito bite are properly taken by using bed nets, bed net insecticides treatment services, insect repellants also draining of gutters and clear all receptacles that may collect water that can serve as breeding places for mosquito. This report is in agreement with the findings of Cattani *et al.* (1986)

and Goruip, (1989).

The entire group of patient did not have diabetes except one (Table 8). Absence of diabetes may be as a result of hypoglycaemia, which is due to quinine, or quinine induced hyperinsulinaemia, which results in reduced hepatic gluconeogenesis and increase uptake of glucose by peripheral tissues. Diabetes did not show positive correlation with malaria parasitaemia ($p>0.05$). This finding is in consonance with the work of Loorensan *et al.* (1995).

Table 7. Placental weight and Gestation age in relation to maternal, fetal, placental and cord parasitaemia.

Placenta Weight	Maternal blood		Fetal blood		Placental blood		Cord blood	
	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)
0.3-0.5 kg	17(41.5%)	24(58.5%)	13(31.7%)	28(68.3%)	15(36.6%)	2(63.4%)	14(34.1%)	27(65.9%)
0.6-0.7 kg	17(22.4%)	59(77.6%)	14(18.4%)	62(81.6%)	15(19.7%)	61(80.3%)	14(18.4%)	62(81.6%)
0.8-1.0 kg	3(23.1%)	10(76.9%)	2(15.4%)	11(84.6%)	3(23.1%)	10(76.9%)	2(15.4%)	11(84.6%)
P-value	P>0.05		P>0.05		P>0.05		P>0.05	
Gestation age								
27-31 weeks	1(100%)	-	1(100%)	-	1(100%)	-	1(100%)	-
32-36 weeks	29(26.9%)	79(73.1%)	23(21.3%)	85(78.7%)	27(25.0%)	81(75.0%)	25(23.1%)	83(76.9%)
37-40 weeks	7(33.3%)	14(66.7%)	5(23.8%)	16(76.2%)	5(23.8%)	16(76.2%)	4(19.0%)	17(81.0%)
P-value	P>0.05		P>0.05		P>0.05		P>0.05	

P>0.05 showed no positive correlation

Table 8. Type of accommodation and Diabetes in relation to maternal, fetal, placental and cord parasitaemia.

Type of accommodation	Maternal blood		Fetal blood		Placental blood		Cord blood	
	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)
Flat/ self contained	11(19.3%)	46(80.7%)	8(14.0%)	49(86.0%)	8(14.0%)	49(86.0%)	8(14.0%)	49(86.0%)
Compound/ face to face	26(35.6%)	47(64.4%)	21(28.8%)	52(71.2%)	52(52%)	48(48.0%)	22(30.1%)	51(69.9%)
P-value	P<0.05		P<0.05		P<0.05		P<0.05	
Diabetes								
Yes	1	-	1	-	1	-	1	-
No	36	93	29	100	32	97	30	99
P-value	P>0.05		P>0.05		P>0.05		P>0.05	

P < 0.05 showed positive correlation; P > 0.05 showed no positive correlation

Table 9. Maternal occupation in relation to maternal, fetal, placental and cord parasitaemia.

Maternal occupation	Maternal blood		Fetal blood		Placental blood		Cord blood	
	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)
Lower class	13(25.0%)	35(72.9%)	1(22.9%)	37(77.1%)	15(38.5%)	24(61.5%)	10(20.8%)	38(79.2%)
Middle class	11(30.6%)	25(69.4%)	8(22.2%)	28(77.8%)	8(22.2%)	28(77.8%)	9(25.8%)	27(75.0%)
Higher class	13(28.3%)	33(71.7%)	10(21.7%)	36(78.3%)	10(18.2%)	45(81.8%)	11(23.9%)	35(76.1%)
P-value	P>0.05		P>0.05		P>0.05		P>0.05	

P>0.05 showed no positive correlation

Furthermore according to Socio-economic status in Table 9, middle class had the highest parasitaemia. In maternal educational background, the highest prevalence was noted among people without formal education and secondary school leavers (Table 10). It was also discovered that in husband educational background, parasitaemia was highest in mothers whose husbands were

secondary school leavers (Table 11). Prevalence was very low among the graduates. The high prevalence of parasitaemia among people without formal education and secondary school leavers could possibly be that they are not enlightened on how to prevent malaria infection. This report contradicts the work of Okonofua *et al.* (1990).

Finally, women whose husbands were

Table 10. Maternal educational background in relation to maternal, fetal, placental and cord parasitaemia .

Educational background	Maternal blood		Fetal blood		Placental blood		Cord blood	
	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)
Primary school certificate	6(26.1%)	17(73.9%)	4(17.4%)	19(82.6%)	3(13.0%)	20(87.0%)	5(21.7%)	18(78.3%)
Secondary school certificate	17(32.1%)	36(67.9%)	11(20.8%)	42(79.2%)	15(28.3%)	38(71.7%)	10(18.9%)	43(81.1%)
Tertiary certificate	11(22.9%)	37(77.1%)	11(22.9%)	37(77.1%)	12(25.0%)	36(75.0%)	12(25.0%)	36(75.0%)
No formal education	3(50.0%)	3(50.0%)	3(50.0%)	3(50.0%)	3(50.0%)	3(50.0%)	3(50.0%)	3(50.0%)
P-value	P>0.05		P>0.05		P>0.05		P>0.05	

P>0.05 showed no positive correlation.

Table 11. Husband Educational Background in relation to Maternal, Fetal, Placental and Cord parasitaemia

Educational Background	Maternal blood		Fetal blood		Placental blood		Cord blood	
	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)
Secondary Education	23 (35.4%)	42 (64.6%)	18 (27.7%)	47 (72.3 %)	21 (32.3%)	44 (67.7%)	18 (27.7%)	47 (72.3%)
Higher Certificate	13 (21.3%)	48 (78.7%)	10 (16.4%)	51(83.6%)	12(19.7%)	49 (80.3%)	10 (16.4%)	51(83.6%)
No Formal Education	1(25.0%)	3 (75.0%)	1(25.0%)	3 (75.0%)	-	4 (100%)	2 (50%)	2 (50%)
P-value	P > 0.05		P > 0.05		P > 0.05		P > 0.05	

P > 0.05 showed no positive correlation.

Table 12. Husband occupation in relation to maternal, fetal, placental and cord parasitaemia.

Husband occupation	Maternal blood		Fetal blood		Placental blood		Cord blood	
	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)	Positive (%)	Negative (%)
Farmer	-	1(100%)	-	1(100%)	-	1(100%)	-	1(100%)
Business	25(33.3%)	50(66.7%)	20(26.7%)	55(73.3%)	21(28.0%)	54(72.0%)	22(29.3%)	53(70.7%)
Working class	12(22.2%)	42(77.8%)	9(16.7%)	45(83.3%)	12(22.2%)	42(77.8%)	8(14.8%)	46(85.2%)
P-value	P>0.05		P>0.05		P>0.05		P>0.05	

P>0.05 showed no positive correlation

businessmen had the highest malaria parasitaemia (33.3%) compared to those that their husbands were working classes (Table 12). This could be due to the fact that most of these businessmen are travelers and are exposed to mosquito bite in their new environment in the course of their business transactions. This finding is in accordance with the report of Myron, (1989).

In conclusion, malaria parasite represents

additional risk in pregnancy. The disease is particularly severe in the pregnant women, and can as a result of transplacental transmission cause congenital malaria which is associated with neonatal mortality. It is therefore recommended that there should be regular screening of pregnant women in malaria endemic zone for malaria infection and also effective treatment to reduce the mortality of both women and their foetus.

REFERENCES

- Brabin, B.J 1983. Analysis of malaria in pregnancy in Africa. *Bull World Health Organization*. 61: 1005-16.
- Cattani, J.A Moir, J.S. Gibson F.D., Ginny M., Paino., Davidson, W. and Alpers, M.P. 1986. Small- area variation in epidemiology of malaria in Madang province Papua. *New Guinea Medical Journals*. 29 : 11-17.
- Cheesbrough, M. 1987. *Medical Laboratory Manual for Tropical Countries*. Butterworth Publisher, London. PP. 221-250
- Cheesbrough, M. 2000. *District Laboratory Practice in Tropical Countries Part 2*. Cambridge University Press, United Kingdom. pp.284-312.
- Conell, M. 2002. History of Malaria: <http://www.rph.wa.gov.au/abs/haem/malaria/history.html>.
- Ezechukwu, C.C Ekejindu, I.M., Ugocchukwu, E.F., Egbuonu, I., Oguatu, M. and Chuwuka, J.O. 2004. Congenitally Acquired Malaria in a hyperendemic Area : A cohort study. 8 (2): 44-47.
- Francis, N. and Warrel, D.A. 1993. Pathology and pathophysiology of human Malaria. In: Gilles H.M. Warrel, D.A. (Eds.), Bruce Chwatt's. *Essential Malariology*. 3rd Edition. Edward Arnold London. Pp. 50 - 59.
- Goruip, S. 1989. Practical and constrains on chemotherapy and practical consideration in the use of vaccines. *Transactions of the Royal Society of Tropical Medicine and Hygiene*. 83 : 81-83
- Ikeh, E.L., Akudo, S.N. and Uguru, V.E. 2005. Prevalence of Malaria parasitaemia in pregnant women attending antenatal clinic at Jos University Teaching Hospital. *Africa Journal of Clinical and Experimental Microbiology*. Unilorin Press. 6 (2): 91-92.
- Jimoh, A.A.G. 2003. Epidemiological study of malarial parasites in pregnant others, placenta and Newborns at Mongomo, Guinea Equatorial. *Nigeria Clinical Review*. Pp. 24-27.
- Jimoh, A.A.G. 2004. Materno -fetal Haematological. Relationship in malaria at Mongomo, Guinea Equatoria. *African Journal of clinical and Experimental Microbiology*. Unilorin Press. 5 (3) : 217-219.
- Larkin, G.L. and Thuma, P.E. 1991. Congenital malaria in a hyperendemic area. *Am. J. Trop. Med. Hyg.* 45 (5) : 587-592.
- Loorenswan, S., Philips, R.E., White, N.J. et al. 1995. Quinine and Severe faciparum malaria in late pregnancy. *Lancet* II: 4-8.
- Macleod, J. and Rhode, R. 1998. Retrospective follow - up of malaria deaths and their associated risk factors in rural district of Tanzania. *Tropical Medicine and International Health*. 3 : 130-137.
- Myron, G. and Schults E. 1989. Malaria in-migrants and travelers. *Transactions of the Royal society of Tropical Medicine and Hygiene*: 83 : 31-34. *Tropical Medicine and Public Health*. 29 : 356-360.
- Obiajunwa, J.A., Owa.O and Adeodu, O.O. 2005. Prevalence of congenital Malaria in Ile-Ife Nigeria. *Journals of Tropical Pediatrics Advance Access*. Oxford University Press. PP: 1-3
- Okonofua, F., Adeniran, M., Adetugbo, D. and Nganwuchu, A. 1990. Women attending antenatal clinic of the Obafemi Awolowo University Teaching Hospital, Ile-ife Nigeria. *Medicare*. 25 : 8 - 11.
- Reinhardt, M.C., Ambroise -Thomas, P., Cavallo -Serra, R., Meylan, C and Gautier, R. 1978. Malaria at Delivery in Abidjan. *Helvetica Paediatrica Acta*. 33 (suppl. 41): 65-84.
- Sule- Odun A.O., Ogunledun, A. and Olatunji, A.O. 2002. Impact of Asymptomatic malaria parasitaemia at parturition outcome. *J. Obstet. Gynaecol.* 22 (1):25-8