PREVALENCE OF CONGENITAL MALARIA IN JOS, PLATEAU STATE, NIGERIA

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Abstract

A cross-sectional study on the prevalence of congenital malaria was carried out at three hospitals within Jos Metropolis from September, 2007 to October, 2008. A total of 310 subjects, comprising of 210 pregnant women (35-40 weeks gestation) and 100 non-pregnant women attending antenatal and post natal clinics respectively were enrolled. Peripheral blood of women and neonates, placental and cord blood were examined for malaria parasites by microscopy using Giemsa stain technique. 137 (65.2%) pregnant women and 40 (40.0%) non-pregnant women were positive for malaria parasitaemia. The difference was significant ($p<0.05$). *Plasmodium falciparum* and *Plasmodium malariae* were found in the blood of the two groups of women. Malaria parasitaemia in the pregnant women was 76/93 (81.7%) in primigravidae and 61/117 (52.1%) in multigravidae ($p<0.05$). The prevalence of malaria parasites in peripheral blood of neonates, placenta and cord blood were 10/137 (7.3%), 74/137 (54.0%) and 46/137 (33.6%) respectively. Light 5/7 (71.4%) and moderate 2/7 (28.6%) infections were observed in the peripheral blood of neonates of primigravidae. All 3/3 (100.00%) of the neonates of multigravidae women had light infection, heavy infection was not observed in the neonates of these women. The average birthweights of healthy babies delivered of non-malarious mothers was higher 3.29 kg than 2.42 kg delivered of malarious mothers. Congenital malaria is associated with low birthweight. The study has revealed that there is congenital malaria in the study area and malaria parasitaemia is associated with low birthweight in newborns. Therefore, interventions for the treatment of malaria during pregnancy should be prompt and effective.

Keywords: congenital malaria, pregnant women, birthweight, Jos, Nigeria.

Accepted: 22 August, 2014.

Introduction

Malaria is a preventable and treatable mosquito-borne disease. Worldwide, an estimated 3.4 billion people are at risk of malaria, of which 1.2 billion are at high risk. In high-risk areas, more than one malaria case occurs per 1,000 population illness. In 2012, an estimated 482,000 children under the age of 5 died from malaria. That is, 1,300 children every day or one child almost every minute (WHO, 2013). Malaria is currently endemic in 99 countries worldwide (WHO, 2011). It is a disease found in poor and under-developed areas of the world. It remains the most complex and overwhelming health problem in the tropical and sub-tropical regions of the world with 300 to 500 million cases and 2 to 3 million deaths per year. About 90% of all malaria deaths in the world occur in sub-Saharan Africa because the main burden of malaria parasitaemia is caused by *Plasmodium falciparum*, the most

© *The Zoologist, 12:* 31-39 (2014), ISSN 1596 972X.
Zoological Society of Nigeria.

dangerous of the four human malaria parasites (*P. falciparum, P. ovale, P. vivax and P. malariae*) accounting for an estimated 1.4-2.6 million deaths per year in this region (WHO, 2000; WHO, 2003).

Malaria infection during pregnancy is a major public health problem in tropical and sub-tropical regions throughout the world. In most endemic areas of Africa, pregnant women are the main adult risk group for malaria. *P. falciparum* is responsible for the majority of malaria infections that occur in pregnancy. It is estimated that each year at least 30 million women become pregnant in malarious areas of Africa, with most living in areas of relatively stable malaria transmission (WHO, 2003). Malaria is the most important preventable cause of low birthweight (LBW) in malaria endemic areas in sub-Saharan Africa, which in turn is associated with increased susceptibility to illness and infant mortality. Malaria during pregnancy therefore is a serious problem in sub-Saharan Africa, affecting an estimated 24 million pregnant women. Each year, between 75,000 and 200,000 infant deaths are attributed to malaria infection in pregnancy globally (Steketee *et al,* 2001; WHO, 2003; Desai *et al,* 2007). In these areas, at least one in four pregnant women (all parities) has evidence of malaria infection at delivery detected as either peripheral malaria or placental malaria (Steketee *et al,* 2001; Guyatt and Snow, 2004). Poor outcome for both mother and foetus is associated with malaria in pregnancy and results in premature delivery, perinatal mortality, anaemia, abortion, death of the mother and LBW (birthweight <2.5 kg). Infant mortality for LBW babies is more than four times that for normal birth weight babies (McCormik, 1985; Bloland *et al,* 1996). Malaria exposure during pregnancy has a delayed effect on birthweight outcomes, but a more acute effect on still-birth risk. The highest rate of still-birth occurs in developing countries, especially in sub-Saharan Africa, and averages 20-40/1000 births (Van Geertruyden *et al,* 2004; Wort *et al,* 2006).

Congenital malaria is defined as the diagnosis of malaria parasites in newborn babies within seven days of birth or later if there is possibility of post partum infection by either mosquito bite or blood transfusion; while neonatal malaria is described as symptoms attributed to the malaria parasites in the erythrocytes of an infant within the first twenty days of life (Laosombat, and Dharmasakti, 1981). Studies conducted from 1986 to 2006 in sub-Saharan Africa showed that congenital malaria is a rare occurrence with prevalence ranging from 0% to 0.7%, although maternal malaria rates of between 24.8% and 54.4% were reported (Djibo and Cenac, 2000; Sule-Odu *et al,* 2002). Other studies noted that congenital malaria was not uncommon with prevalence reaching up to 37% (Okoko *et al* 2002; Akum *et al,* 2005). These studies also reported a high frequency of neonatal peripheral parasitaemia ranging between 4.0 and 46.7% (Uneke, 2007). Postulated mechanisms for congenital transmission of malaria parasite include maternal transfusion into the foetal circulation either at the time of delivery or during pregnancy, and direct penetration through the premature separation of the placenta (De Silva *et al,* 1982). The remarkable capacity of the foetus to resist infection has been documented by Miller and Telford (1996). This resistance may reflect the physical barrier of the placenta to infected cells, the passive transfer of maternal antibodies, and fast elimination from the foetal circulation (Shear *et al,* 1998; Riley *et al,* 2001).

Despite all efforts to control malaria, it still remains one of the major causes of morbidity and mortality in Nigeria, with diverse consequences and implications (Okiro *et al,* 2007). Since malaria during pregnancy can result in LBW of neonates, an important risk factor in infant mortality, this study was carried out to determine the association between maternal peripheral malaria and placental malaria, and between placental parasitaemia and birthweight of newborn babies in Jos.

Materials and methods

The study was conducted in three hospitals within Jos Metropolis; Plateau State Specialist Hospital, Solat Hospital and New Crescent Hospital. Approval for the study was obtained from the hospitals' Ethical Committees. Before the enrollment of women, the aim and objectives of the study were explained to them. They were also informed that their participation was voluntary. Those who did not understand English language were told what the study was all about in a language (Hausa, Yoruba and Igbo) that they understood. Thereafter, a consent form was given to each volunteer. Volunteers were also told that all information emanating from the research will be treated with utmost confidentiality.

A total of 310 women were enrolled for the study including 210 pregnant women and 100 non-pregnant women who attended antenatal and post natal clinics respectively. The non-pregnant women were classified as control Group 1.

Collection of blood samples

Blood (2 ml) was collected from each of the pregnant and non-pregnant women by venepuncture. Information about age, parity and use of malaria chemoprophylaxis was obtained from each subject. Midwives assisted in collecting blood from placentas, cords and newborns. Blood was collected from the maternal surface of each placenta. The placenta was incised with a sterile scalpel and some amount of blood was aspirated from the intervillous with a sterile syringe. Blood (2 ml) was also collected from the cord close to its point of attachment to the placenta. Each blood sample was transferred into a labeled EDTA bottle. The peripheral blood of each neonate was collected onto a glass slide by heel pricking, using a sterile lancet. All the infants examined during this study were less than 24 hours old. The blood of each neonate was assigned a coded number traceable to that of the mother. The sex and weight of each new born was recorded immediately after delivery, a neonate whose weight was less than 2.5 kg was considered as having a LBW (Stekeetee *et al,* 1996a). 193 women allowed access to their placentas, while 200 women allowed access to the cord blood of their neonates. Only the corresponding placental and cord blood of mothers with peripheral malaria and those of their newborns were further used for statistical analyses. The non-parasitaemic pregnant women were classified as control Group 2.

Parasitological examination

Giemsa-stained thin and thick blood films of each blood sample was prepared (Cheesbrough, 2000) and the blood films were examined for malaria parasite by experienced microscopists. A slide was considered positive if it contained any of the asexual stages of malaria parasite.

Estimation of parasite density

The parasite densities of neonates, placentae and cords were estimated, with reference to WHO (1991) as follows:

Light infection: 1-10 parasites per 100 thick film fields (+ or 4-40 parasites per mm^3).
Moderate infection: 11-100 parasites per 100 thick film fields (+ + or 41-400 parasites per mm^3).
Heavy infection: 1-10 parasites per single thick film field (+ + + or 41-400 parasites per mm^3).

Statistical analysis

Data were analysed using excel spread sheet and SPSS 2002 16.0 software packages. One sample Kolmogrorov-Smirnov test was used to determine data distribution. Student *t*-test was carried out to compare the means of birthweights between infants of primigravidae and multigravidae for both parasitaemic and non-parasitaemic mothers. The frequency of types of birth in malarious women and non-malarious women was also analysed using *t*-test. *Chi*-square was used to determine correlation between variables. Difference was considered to be significant at $p \leq 0.05$.

Results

The overall prevalence of malaria parasites in the peripheral blood of pregnant and non-pregnant women are shown in Table 1. The prevalence of peripheral malaria parasitaemia in pregnant women (65.2%) was higher than in non-pregnant women (40.0%). The difference was significant x^2 = 19.77, $p<0.05$). The least (36.8%) infection rate in pregnant women was observed among women aged 31-40 years and the highest (88.2%) among those aged 20 years or less. Primigravidae had higher (81.7%) malaria prevalence compared to multigravidae (52.1%) among pregnant women and the difference was significant (x^2 = 6.64, $p<0.05$). The same trend was observed among their non-pregnant counterparts, the infection rates for primigravidae and multigravidae were (52.4% vs 31.0%). The difference was not significant (x^2 = 2.55, $p>0.05$) (Table 2).

Table 1. Prevalence of malaria parasite in peripheral blood of pregnant and non-pregnant subjects.

		Parasite type		
		Plasmodium falciparum	*Plasmodiu m malariae*	
	Number examined	Positive (%)	Positive (%)	Total positive (%)
Pregnant women	210	116 (55.2)	21 (10.0)	137 (65.2)
Non-pregnant women	100	32 (32.0)	8 (8.0)	40 (40.0)
Total	**310**	**148 (47.7)**	**29 (9.4)**	**77 (57.1)**

There was a decrease in malaria parasitaemia of peripheral blood of newborns, placenta and cord blood with ascending parity. However, there were significant associations between peripheral maternal malaria and placental malaria (x^2 = 18.80, $p<0.05$) and between peripheral maternal malaria and cord malaria (x^2 = 45.26, $p<0.05$). There were also significant associations between maternal peripheral malaria in newborns and placental parasitaemia (x^2 = 48.60, $p<0.05$) and between peripheral malaria of neonates and cord parasitaemia (x^2 = 23.14, $p<0.05$) (Table 3).

Light (71.4%) and moderate (28.6%) infections were observed in the peripheral blood of neonates of

Table 2. Prevalence of *Plasmodium* infection in pregnant and non-pregnant women according to age.

	Pregnant women				Non-pregnant women			
	Primigravidae		Multigravidae		Primigravidae		Multigravidae	
Age	No. Examined	+ve (%)	No. Examined	+ve (%)	N.o Examined	+ve (%)	No. Examined	+ve (%)
≤ 20	34	30 (88.2)	26	16 (61.5)	13	8(61.5)	10	5 (50.0)
21-30	48	39 (81.3)	44	27 (61.4)	24	13(54.2)	26	7 (26.9)
31-40	9	6 (66.7)	38	14 (36.8)	5	1(20.0)	18	4 (22.2)
≥41	2	1 (50.0)	9	4 (44.4)	0	0(0.0)	4	2 (50.0)
Total	**93**	**76 (81.7)**	**117**	**61 (52.1)**	**42**	**22 (52.4)**	**58**	**18 (31.0)**

+ve: Positive

Table 3. Distribution of malaria parasites in pregnant women, newborns, placentae and cord blood.

	Primigravidae			Multigravidae			Primigravidae and Multigravidae		
	No. Examined	No. Positive	Infection Rate %	No. Examined	No Positive	Infection Rate %	No. Examined	No. Positive	Infection Rate %
Pregnant Women	93	76	81.7	117	61	52.1	210	137	65.2
Newborn	53	7	13.2	84	3	3.6	137	10	7.3
Placenta	53	41	77.4	84	33	39.3	137	74	54.0
Cord	53	26	49.1	84	20	23.8	137	46	33.6

Table 4. Prevalence of light, moderate and heavy infections among primigravidae and multigravidae.

	Primigravidae			Multigravidae		
Parasite density	**Newborn peripheral blood No. infected (%)**	**Placenta No. infected (%)**	**Cord blood No. infected (%)**	**Newborn peripheral blood No. infected (%)**	**Placenta No. infected (%)**	**Cord blood No. infected (%)**
Light infection	5 (71.4)	21 (51.2)	17 (65.4)	3 (100.0)	26 (78.8)	17 (85.0)
Moderate infection	2 (28.6)	15 (36.6)	8 (30.8)	0 (0.0)	7 (21.2)	3 (15.0)
Heavy infection	0 (0.0)	5 (12.2)	1 (3.8)	0 (0.0)	0 (0.0)	0 (0.0)
Total	**7**	**41**	**26**	**3**	**33**	**20**

Table 5. Frequency of birth types in malarious and non-malarious mothers.

		Malarious Mothers			Non-Malarious Mothers		
Types of deliveries	No. of delivery (%)	Male infants (%)	Female infants (%)	Total No. (%)	Male infants (%)	Female infants (%)	Total No. (%)
Normal delivery	199 (94.8)	59 (29.6)	67 (33.7)	126 (63.3)	32 (16.1)	41 (22.1)	73 (36.7)
Stillbirth	1 (0.5)	0 (0.0)	1 (100.0)	1 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)
Premature birth	10 (4.8)	6 (60.0)	4 (40.0)	10 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)
Total	**210**	**65 (31.0)**	**72 (34.3)**	**137 (65.2)**	**32 (15.2)**	**41 (19.5)**	**73 (34.8)**

Table 6. Mean birthweight (kg) of newborns of non-parasitaemic and parasitaemic mothers.

	Non-parasitaemic mothers						Parasitaemic Mothers					
	PM			MG			PM			MG		
Type of delivery	Male new-born (*n*=18)	Female newborn (*n*=22)	Overall mean	Male new-born (*n*=14)	Fe-male new-born (*n*=19)	Overall mean	Male new-born (*n*=30)	Female new-born (*n*=34)	Overall mean	Male new-born (*n*=35)	Female new-born (*n*=38)	Overall mean
Normal delivery	3.27	3.29	3.28±1.89	3.35	3.23	3.29±1.89	2.38	2.28	2.33±1.28	2.49	2.35	2.42±1.31
Stillbirth	0.00	0.00	0.00	0.00	0.00	0.00	0.00	1.79	0.00	0.00	0.00	0.00
Premature birth	0.00	0.00	0.00	0.00	0.00	0.00	2.03	1.89	1.96±0.09	2.10	1.99	2.05±0.07

PG: Primigravidae **MG:** Multigravidae.

primigravidae. However, all (100.0%) neonates of multigravidae had light infections. The placental and cord blood of primigravidae had light to heavy infections, while the placentae and cords of multigravidae had light to moderate infections (Table 4).

There were 199 (94.8%) normal deliveries, 1 (0.5%) stillbirth and 10(4.8%) premature births. There was significant association between the different types of deliveries $p < 0.05$) (Table 5).

The mean birthweight was lower among babies from malaria infected mothers than those from uninfected mothers. The overall mean birthweight of newborns from non-parasitaemic and parasitaemic mothers were 3.29±1.89 kg and 2.42±1.31 kg respectively. However, the difference was not statistically significant ($p > 0.05$). There was also no significant difference between the overall mean birthweights of the primigravidae and multigravidae of non-parasitaemic mothers ($p > 0.05$) and between the overall mean birthweights of primigravidae and multigravidae of parasitaemic mothers ($p > 0.05$). There was significant association between birthweight and placental malaria ($x^2 = 19.32$, $p<0.05$) (Table 6).

Discussion

In this study, the overall prevalence of malaria parasites in both pregnant and non-pregnant women was 57.1% thus, supporting earlier assertions that malaria is endemic in Nigeria (Okon *et al*, 1992; Eneanya, 1996; Uneke *et al*, 2008a; Akanbi *et al*, 2009; Omalu *et al*, 2012; George *et al*, 2013). The pregnant women were more susceptible to malaria infection than their non-pregnant counterparts and the difference was significant ($p<0.05$). Similar trend in malaria parasitaemia have been reported in Nigeria by Akanbi *et al* (2004) in Ibadan and Ekejindu *et al* (2011) in a semi-urban area of Anambra State. However, our results are at variance with that of Omalu *et al* (2012) who found no significant difference in prevalences between the two groups of women in Minna, Nigeria. Differences in malaria parasitaemia between the two groups of women have been attributed to poor nutrition, micronutrient imbalances, particularly Vitamin A and iron, which are known to exacerbate the impact of pregnancy associated malaria (Breman *et al*, 2004). Pregnancy also causes a number of physiological changes that affect the suppression of T-helper Cell 1 (Th l) thus, making the pregnant woman more reliant on humoral immunity (Th 2) for protection thereby, putting her at an increased risk of acquiring malaria infection (Samak, 2004). It is not clear why a higher proportion of the pregnant women were found with malaria parasites in this study compared with their counterparts without malaria (137 *vs* 73) in spite of the fact that the all claimed to be on malarial chemo-prophylaxis. Nevertheless, we attributed this to infection acquired late in pregnancy. *P. falciparum* and *P. malariae* were found in peripheral blood of pregnant women in this study. This finding is consistent with those of Okon *et al* (1992) in Cross River State, Nigeria and McGregor *et al* (1983) in The Gambia.

Factors which increase the risk of malaria include young maternal age and gravidity (Agomo *et al*, 2009). Our results showed that malaria parasitaemia occurred most frequently in women aged 20 years and less for both pregnant and non-pregnant subjects. These findings are comparable to those of Ekejindu *et al* (2011) but contrary to those of George *et al* (2013). Furthermore, primigravidae of both groups were more infected than multigravidae, because the latter gain immunity against malaria infection during successive pregnancies, particularly in areas of high malaria transmission (Fried *et al*, 1998).

Some studies have revealed that pregnancy results in foetal exposure to maternal malaria parasites (Okon *et al* 1992; Egwunyenga *et al*, 1995, 1996; Uneke *et al*, 2008a; Omalu *et al*, 2012). Our findings concur with this observation, malaria parasites were found in 54.0% and 33.6% of placental and cord blood respectively with significant association occurring between maternal peripheral parasitaemia and placenta malaria and between maternal peripheral parasitaemia and cord malaria ($p<0.05$). Significant association was also observed between placental and congenital malaria ($p<0.05$) (Table 3). These prevalences are higher than those reported by Redd *et al* (1996) but lower than those of Obiajunwa *et al* (2005). These may be due to factors such as perinatal clearance of occult parasitaemia, maternal immunity and co-existing infections (Miller and Telford, 1996).

Evidence from this study and others (Morgan, 1994; Steketee *et al*, 1996b; Mutabingwa *et al*, 2005) suggest that placental malaria especially in primigravidae may increase malaria risk for newborns. In this study, 7.3% of the newborns had *falciparum* peripheral parasitaemia. *Falciparum* malaria is an important cause of perinatal and neonatal morbidity in high transmission settings in sub-Saharan Africa (Valley *et al*, 2007). The frequency of peripheral blood malaria in neonates obtained in this study is higher than those reported by Egwunyenga *et al* (1995) and Omalu *et al* (2012). Some studies showed that congenital malaria was rare (Djibo and Cenac, 2000; Sule-Odu *et al*, 2002).

However, the present report and others (Obianjuwa *et al*, 2005; Runsewe-Abiodun *et al*, 2006; Omalu *et al*, 2012) show that malaria is not a rare occurrence. These recent reports show a new trend, since neonatal malaria was previously considered as an uncommon occurrence due to the protective effect of maternal immunity (IgG) after birth, the poor environment afforded by foetal erythrocytes for plasmodial replication and fast elimination of the parasites from the foetal circulation (Shear *et al*, 1998; Riley *et al*, 2001). Furthermore, maternal age, host genetics, gravidity, nutrition, use of prophylaxis, level of immunity as well as parasite genetics and transmission rates have been suggested to influence the prevalence of placental malaria in pregnant women (Tako *et al*, 2005).

In this study neonates with peripheral parasitaemia had low parasite density, which is in agreement with the report of Eweronu-Laryea *et al* (2013) in a study of congenital malaria among high risk Ghanian newborns. Light to heavy infections of the cord were observed among neonates of parasitaemic mothers in this study. Similar observations were made by Egwunyenga *et al* (1996). The only stillbirth recorded in this study was in a primigravidae (Table 6). This is consistent with the finding of McGregor *et al* (1983) who recorded stillbirths in primigravidae in urban and rural Gambia. A single still-birth was reported in this study which is lower than the number recorded in Tanzania by Wort *et al* (2006).

Another study in The Gambia, Okoko *et al* (2002) observed a two-fold increased risk of still-birth among mothers with malaria infected placenta. However, (McGegor *et al*, 1983) reported that placental malaria infection was not found to be associated with early neonatal death or foetal death.

In this study, there were 4.8% premature births, the finding conform with the assertion of earlier workers that immature (premature) labour occur in malarious mothers due to the parasitisation of the placenta (Uneke *et al*, 2008a; Mwangoka *et al*, 2008). Furthermore, we found no significant difference in infection rates between male and female neonates ($p>0.05$) (Table 5). McGregor *et al* (1983) also made a similar observation in their study in The Gambia.

On the average, the birthweight of babies from parasitaemic mothers in this study was lower than that of babies from non-parasitaemic mothers. How maternal malaria influences birthweight is not clear, but two obvious possibilities have been suggested, first by inducing premature labour and second, by decreasing placental function (McGregor *et al,* 1983).

Our result showed that infants born to parasitaemic mothers had LBW less than 2.5 kg; similar observations were made by Brabin and piper (1997) in Papua New Guinea, Morgan (1994) in Sierra Leone and Matteelli *et al* (1997) in Tanzania. LBW is the greatest single risk factor for neonatal and infant mortality (McCormick, 1985). Steketee *et al* (1996b) in Malawi and Uneke *et al* (2008a) in Nigeria, also revealed that parasitized placenta is associated with low birthweight. Elsewhere it is associated with reduced early life weight development independent of LBW (Walther *et al,* 2010).

Our findings revealed that neonates who were prematurely born had *falciparum* peripheral malaria parasitaemia. *P. falciparum* infection has also been associated with preterm delivery and intrauterine growth retardation in a study in Papua New Guinea (Allen *et al,* 1998).

Limitations

In this study, conventional blood microscopy was used to diagnose malaria infection. This method of diagnosis still remains the gold-standard for the detection of *Plasmodium* species (Johnston *et al,* 2006) but may not be able to detect placental infection as parasites can be sequestered in the placenta (Anchang-Kimbi *et al,* 2009). Other methods of diagnosis, such as the rapid diagnostic tests (RDTs) and polymerase chain reaction (PCR) have been shown to detect malaria specific antigen(s) in the circulation, even when parasites are sequestered in the placenta and not visible by microscopy. These diagnostic methods are, however, more expensive to perform (Uneke, 2008b; Kyabayinze *et al,* 2011). Due to the lower cost of conventional blood microscopy compared with RDTs and PCR (Wongsrichanalai *et al,* 2007), microscopy was used to diagnose malaria infection in this study. Questionnaires were used to obtain information from the study participants, so, there is a possibility of bias in recalling past events and there was no way to tell how truthful the participants were. Thus, we could not ascertain whether the pregnant participants were on intermittent preventive treatment in pregnancy (IPTp) with sulfadoxine-pyrimethamine (IPTp-SP), to determine factors that contributed to the higher prevalence of malaria parasitaemia in them compared with their non-pregnant counterparts. The higher prevalence may have been as a result of late acquisition of malaria parasite in pregnancy, non-compliance with IPTp-SP or resistant strains of *P. falciparum* to IPTp-SP. However, all the participants in this study claimed to have been on regular routine anti-malarial chemo-prophylaxis. Some of the participants did not allow access to the examination of the placenta and cord blood of their neonates which may have been due to religious beliefs and socio-cultural factors. In some communities, the placenta is considered to be the companion of the newborn baby, has spiritual life and should therefore be protected and respected (Jenkins and Sugarman, 2005).

Conclusion

In conclusion, our data suggest that congenital malaria is associated with low birthweight in the area of study. Therefore, interventions for the prevention and treatment of malaria during pregnancy should be prompt and effective.

Acknowledgements

The authors thank the Managements of Plateau State Specialist Hospital, New Crescent Hospital and Solat Hospital for permission to carry out the study in the respective hospitals. We are also grateful to the midwives of the various hospitals for their assistance in collecting blood samples and to the mothers and babies who participated in the study.

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Ajayi, O. O., Ajayi, O. A., Turshak, L. G. and Dakyahas, J. Y.
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Zoological Society of Nigeria.