

and 2.25 ± 0.33 in haemoglobin genotype AS and AC respectively ($F = 10.48$; $P=0.00$). The mean \pm SD of absolute monocyte count 0.27 ± 0.20 in haemoglobin genotype AA was significantly ($p<0.05$) higher compared to 0.20 ± 0.10 and 0.16 ± 0.08 in haemoglobin genotype AS and AC respectively ($F = 5.66$; $P = 0.00$). Multiple comparison between haemoglobin genotype AA and AS show that mean \pm SD of MPC, in haemoglobin genotype AA was significantly ($p<0.05$) higher compared to haemoglobin genotype AS; mean \pm SD of absolute platelet count and MPV in Hb genotype AA were higher compared to Hb genotype AS also mean \pm SD of PDW in haemoglobin genotype AA was lower compared to Hb genotype AS although the p-value show no statistical significant ($p>0.05$) difference. However, mean \pm SD of TWBC and relative neutrophil count were significantly ($p<0.05$) higher compared to Hb genotype AS while mean \pm SD of relative lymphocyte count in, haemoglobin genotype AA was significantly ($p<0.05$) lower compared to Hb genotype AS. Also, mean \pm SD of relative monocyte and relative eosinophil in Hb genotype AA were lower compared to Hb genotype AS; although the p-value show no statistical significant ($p>0.05$). Moreover, mean \pm SD of absolute neutrophil count, absolute lymphocyte and absolute monocyte in Hb genotype AA were significantly ($p<0.05$) higher compared to Hb genotype AS. However, multiple comparison between Hb genotype AA and AC show that mean \pm SD of MPC in Hb genotype AA was significantly ($p<0.05$) higher compared to Hb genotype AC, mean \pm SD of absolute platelet count and MPV in Hb genotype AA were higher compared to Hb genotype AC also, mean \pm SD of PDW in Hb genotype AA was lower compared to Hb genotype AC, although the P- value show no statistical significant ($p>0.05$). However, mean \pm SD of total WBC and relative neutrophil count were significantly ($p<0.05$) higher compared to Hb genotype AC while mean \pm SD of relative lymphocyte in haemoglobin AA was significantly ($p<0.05$) lower compared to Hb genotype AC. Also, mean \pm SD of relative monocyte and relative eosinophil in Hb genotype AA were lower compared to Hb genotype AC, P- value show no statistical significant ($p>0.05$). Moreover, mean \pm SD of absolute neutrophil count, absolute lymphocyte and absolute monocyte in Hb genotype AA were significantly ($p<0.05$) higher compared to Hb genotype AC. Multiple comparison between haemoglobin genotype AS and AC show that mean \pm SD of MPC, absolute platelet count and PDW in haemoglobin genotype AS were lower compared to Hb genotype AC, the P-value show no statistical significant ($p>0.05$). Mean \pm SD of mean platelet volume, in Hb genotype AS was higher compared to Hb genotype AC. Mean \pm SD total WBC, relative neutrophil count in Hb genotype AS were lower compared to Hb genotype AC. However, mean \pm SD of relative lymphocyte, relative monocyte, relative eosinophil and absolute monocyte in Hb genotype AS were higher compared to Hb genotype AC; mean \pm SD of absolute neutrophil and absolute lymphocyte in Hb genotype AS were lower compared to Hb genotype AC, the comparisons show no statistical significant ($p>0.05$).

Table 2 show the comparison of mean \pm SD in blood cell parameters on haemoglobin genotype variants in post anti-malaria drug treatment. The mean \pm SD of MPC 2477.30 \pm

594.81 in haemoglobin genotype AA was significantly higher ($p<0.05$) compared to 1983.50 161.26 and 2026 \pm 165.03 in Hb genotype AS and AC respectively ($F=22.71$; $P=0.00$). The mean \pm SD of absolute platelet count 184.65 ± 59.58 in haemoglobin AA was higher compared to 170.26 ± 52.85 and 173.11 ± 53.03 in Hb genotype AS and AC respectively, comparisons show no statistical significant difference ($p>0.05$) ($F=1.33$; $P=0.27$). The mean \pm SD of platelet distribution width and mean platelet volume 13.59 ± 2.51 and 9.54 ± 0.67 respectively in Hb genotype AA were lower compared to mean \pm SD of PDW and MPV 13.93 ± 2.25 and 9.71 ± 0.63 in Hb genotype AS and 14.18 ± 2.70 and 9.79 ± 0.57 in Hb genotype AC with ($F = 0.67$, $P=0.51$) and ($F = 2.19$; $P= 0.12$) respectively; the comparisons show no statistical significant difference ($p>0.05$). The mean \pm SD of WBC and relative neutrophil 6.75 ± 1.79 and 55.04 ± 8.24 respectively in Hb genotype AA were significantly higher ($P<0.05$) compared to mean \pm SD of WBC and relative neutrophil 4.15 ± 0.93 and 48.41 ± 8.39 in Hb genotype AS and 4.43 ± 0.88 and 50.94 ± 5.42 in Hb genotype AC with ($F = 62.73$, $P=0.00$) and ($F= 13.44$, $P=0.00$) respectively. The mean \pm SD of relative lymphocyte count 42.78 ± 7.78 in Hb genotype AA was significantly ($P<0.05$) lower compared to mean \pm SD relative lymphocyte 48.80 ± 7.49 and 47.39 ± 5.45 in Hb genotype AS and AC respectively ($F=13.31$, $P=0.00$). The mean \pm SD of relative monocyte count 1.33 ± 0.97 in Hb genotype AC was lower compared to mean \pm SD of relative monocyte 1.79 ± 1.54 and 2.02 ± 1.57 in Hb genotype AA and AS respectively; comparison show no statistical significant ($p>0.05$) ($F=1.42$, $P=0.25$). The mean \pm SD of relative eosinophil 0.33 ± 0.49 in Hb genotype AC was significantly ($P<0.05$) lower compared to 0.38 ± 0.68 and 0.80 ± 1.29 in Hb genotype AA and AS respectively ($F = 4.57$, $P=0.01$). The mean \pm SD of absolute neutrophil, absolute lymphocyte and absolute monocyte 3.73 ± 1.22 , 2.87 ± 0.87 and 0.13 ± 0.14 respectively in Hb genotype AA were significantly ($P<0.05$) higher compared to absolute neutrophil, absolute lymphocyte and absolute monocyte 2.02 ± 0.61 , 2.02 ± 0.51 and 0.08 ± 0.07 respectively in Hb genotype AS and 2.29 ± 0.64 , 2.07 ± 0.33 and 0.06 ± 0.04 respectively in Hb genotype AC with ($F= 57.68$ $P = 0.00$) ($F = 28.99$; $P = 0.00$) and ($F = 3.96$; $P=0.02$) in absolute neutrophil, lymphocyte and monocyte respectively. Multiple comparison between Hb genotype AA and AS show that, mean \pm SD of MPC in haemoglobin genotype AA was significantly ($P<0.05$) higher compared to AS, mean \pm SD of platelet count in Hb genotype AA was higher compared to AS; it show no significant difference ($p>0.05$), the mean \pm SD of PDW and MPV in Hb genotype AA were lower compared to Hb genotype AS, there is no significant difference ($p > 0.05$). However, the mean \pm SD of WBC, relative neutrophil, in Hb genotype AA were significantly ($P< 0.05$) higher compared to Hb genotype AS, mean \pm SD of relative lymphocyte in Hb genotype AA was significantly ($P<0.05$) lower compared to Hb genotype AS. Furthermore, mean SD relative monocyte and relative eosinophil in Hb genotype AA were lower compared to Hb genotype AS. There is significant difference ($P > 0.05$). Moreover, mean \pm SD of absolute neutrophil, absolute lymphocyte and absolute monocyte in Hb genotype AA were significantly ($P < 0.05$) higher compared to Hb genotype AS. Multiple comparisons between Hb genotype AA and AC show that mean \pm SD of MPC in Hb genotype AA was significantly ($P < 0.05$) higher

compared to Hb genotype AC; mean \pm SD of absolute platelet count in Hb genotype AA was higher compared to Hb genotype AC. There is no significant difference ($P > 0.05$) in the comparison, mean \pm SD of PDW and MPV in Hb genotype AA were lower compared to Hb genotype AC, it show no significant difference ($P > 0.05$). However, mean \pm SD WBC and relative neutrophil in Hb genotype AC; mean \pm SD of relative lymphocyte in Hb genotype AA was significantly ($P > 0.05$) lower compared to Hb AC. Moreover, mean \pm SD of relative monocyte and eosinophil in Hb genotype AA were higher compared to Hb genotype AC. There is no significant difference in the comparison ($P > 0.05$). However, mean \pm SD of absolute neutrophil, absolute lymphocyte and absolute monocyte in Hb genotype AA were significantly ($P < 0.05$) higher compared to Hb genotype AC. Multiple comparison between Hb genotype AS and AC show that mean \pm SD of MPC, absolute platelet count, PDW, MPV, total WBC and relative neutrophil count in Hb AS were lower compared to Hb genotype AC; there is no significant difference ($P > 0.05$) in the comparison. However, mean \pm SD of relative lymphocyte, relative monocyte and relative eosinophil in Hb genotype AS were higher compared to Hb genotype AC there is no significant difference ($P > 0.05$). Moreover, mean \pm SD of absolute neutrophil, absolute lymphocyte and absolute monocyte in Hb genotype AS were lower compared to Hb genotype AC. There is no significant difference ($P > 0.05$).

Table 3: show comparison of mean \pm SD in blood cell parameters on haemoglobin genotype variants in control subjects. The mean \pm SD of absolute platelet 271.00 ± 25.46 in Hb genotype AC was lower compared to 284.53 ± 46.27 and 291.24 ± 55.68 in Hb genotype AA and AS respectively ($F = 0.26$; $P = 0.78$). The mean \pm SD of PDW 11.99 ± 1.61 in Hb genotype AA was lower compared to 12.01 ± 2.26 and 12.60 ± 1.69 in Hb genotype AS and AC respectively ($F=0.12$; $P=0.89$). The mean \pm SD of MPV 9.25 ± 0.49 in Hb genotype AC was lower compared to 9.52 ± 0.22 and 9.52 ± 0.31 in Hb genotype AA and AS respectively ($F = 1.71$; $P=0.31$). The mean \pm SD of total WBC 4.43 ± 0.32 in Hb genotype AA was higher compared to 4.41 ± 0.39 and 4.5 ± 0.35 in Hb genotype AS and AC respectively ($F = 0.08$; $P=0.92$). The mean \pm SD of relative neutrophil count 57.19 ± 4.48 in Hb genotype AA was lower compared to 57.57 ± 3.19 and 58.00 ± 7.07 in Hb genotype AS and AC respectively ($F = 0.08$, $P= 0.93$). The mean \pm SD of relative lymphocyte 42.00 ± 7.07 in Hb genotype AC was higher compared to 41.62 ± 4.76 and 41.29 ± 2.90 in Hb genotype AA and AS respectively, the comparison show no statistical difference ($p > 0.05$). ($F = 0.06$, $P = 0.95$). The mean \pm SD of relative monocyte and eosinophil 0.97 ± 1.07 and 0.33 ± 0.71 respectively in Hb genotype AA were lower compared to mean \pm SD of relative monocyte and eosinophil 0.86 ± 0.96 and 0.29 ± 0.56 respectively in Hb genotype AS with respect to ($F=0.91$, $P = 0.41$) and (0.25 , $P= 0.78$) in relative monocyte and eosinophil respectively; the comparison show no significant difference ($p > 0.05$). The mean \pm SD of absolute neutrophil 2.53 ± 0.51 in Hb genotype AC was lower compared to 2.54 ± 0.31 and 2.54 ± 0.22 in Hb genotype AA and AS respectively ($F = 0.00$; $P= 0.99$). The mean \pm SD of absolute lymphocyte 1.84 ± 0.23 in Hb genotype AA was higher compared to 1.82 ± 0.23 and 1.82 ± 0.16 in Hb genotype AS and AC respectively ($F = 0.06$,

$P= 0.95$). The mean \pm SD of absolute monocyte 0.04 ± 0.05 and 0.04 ± 0.05 in Hb genotype AA and AS respectively is the same mean value; hence, the comparison show no significant difference ($p > 0.05$). ($F = 0.86$; $P=0.43$). However multiple comparisons between Hb genotype AA and AS show that, there is no significant difference ($P > 0.05$) in any of the parameters compared. The mean \pm SD of absolute platelets and PDW in Hb genotype AA were lower compared to Hb genotype AS; the mean \pm SD of MPV in Hb genotype AA and AS was the same; the mean \pm SD of WBC in Hb genotype AA was higher compared to Hb genotype AS. However, mean \pm SD of relative neutrophil in Hb genotype AA was lower compared to Hb genotype AS. The mean \pm SD of relative lymphocyte in Hb genotype AA was higher compared to Hb genotype AS. The mean \pm SD of relative monocyte and eosinophil in Hb genotype AA were higher compared to Hb genotype AS. The mean \pm SD of absolute neutrophil and absolute monocyte in Hb genotype AA and AS were of the same mean value; however mean \pm SD of absolute lymphocyte in Hb genotype AA was higher compared to Hb genotype AS. Multiple comparisons of Hb AA and AC show that, the mean \pm SD of absolute platelet count, in Hb AA was higher compared to Hb genotype AC. Comparisons show no significant difference ($P > 0.05$); the mean \pm SD of PDW in Hb AA was lower compared to Hb genotype AC, comparison show no significant difference ($P > 0.05$); the mean \pm SD of MPV and WBC in Hb genotype AA were higher compared to Hb genotype AC, comparisons show no significant difference ($P > 0.05$); however, mean \pm SD of relative neutrophil and relative lymphocyte in Hb genotype AA was lower compared to Hb genotype AC, the comparisons show no significant difference ($P > 0.05$). The mean \pm SD of relative monocyte, eosinophil and absolute monocyte in Hb genotype AA has nothing to compare with in Hb genotype AC; however mean \pm SD of absolute neutrophil and lymphocyte in Hb genotype AA were higher compared to Hb genotype AC. The comparison shows no significant difference ($P > 0.05$). Multiple comparison of Hb AS and AC show that the mean \pm SD of platelet in Hb genotype AS was higher compared to the genotype AC, the comparison show no significant difference ($P > 0.05$). The mean \pm SD of PDW in Hb genotype AS was lower compared to Hb genotype AC; the comparison show no significant difference ($P > 0.05$); the mean \pm SD of MPV and WBC in Hb genotype AS were higher compared to Hb AC; the comparison show no significant difference ($P > 0.05$). However, the mean \pm SD of relative neutrophil and relative lymphocyte in Hb genotype AS were lower compared to Hb genotype AC, the comparisons show no significant difference ($P > 0.05$); the mean \pm SD of relative monocyte, relative eosinophil and absolute monocyte in Hb genotype AS has no mean \pm SD value compared to Hb genotype AC. Hence, the mean \pm SD of absolute neutrophil in Hb genotype AS were higher compared to Hb genotype AC, comparison show no significant difference ($P > 0.05$); absolute lymphocyte in Hb genotype AS and AC has the same mean \pm SD value.

6. Discussion

Out of 202 *plasmodium falciparum* malaria patients in this study, 130 were haemoglobin genotype AA, 54 were haemoglobin genotype AS, and 18 were haemoglobin

genotype AC, while in control group, 79 were haemoglobin genotype AA, 21 were haemoglobin genotype AS and 02 were haemoglobin genotype AC. Prevalence of haemoglobin genotype AA and haemoglobin genotype AS in this study was similar to the report of Francis and Pete (14), stated that of the four hundred (400) subjects screened for haemoglobin genotype in malaria infected patients, two hundred and sixty-eight (268) (67.0%) were dominant homozygous (HbAA), one hundred and twenty-eight (128) (32.0%) were sickle heterozygous (HbAS), four (4) (1.0%) were recessive homozygous (HbSS), while none of the subjects had HbAC or HbSC genotype, similarly, Edith *et al.*, (15) reported the frequencies of Hb genotypes in *Plasmodium falciparum* infected patients as 73.2% AA; 15.0% AC; 8.2% AS; 2.2% CC; 1.1% CS and 0.2% SS. This study showed Hb AS had lower malaria parasite count compared to Hb AC and Hb AA in pre-treatment and post-treatment. Hb AA had higher malaria parasite count; result in this present study showed that Hb AS and AC had genetic resistance to malaria attack compared to Hb AA. This was supported by Francis and Pete (14), reported that heterozygote individuals (HbAS) were more resistant to *Plasmodium falciparum* malaria than normal dominant homozygous (HbAA) individuals. This present study support the facts that AS and AC genotype was associated with lower incidence of clinical malaria relative to AA genotype which has been widely reported. According to Rihet *et al.*; Williams *et al.*; Verra *et al.*, and Kreuels *et al.*, (16, 17a, 17b, 18 and 19) they reported that HbAS is widely known to confer significant protection from severe and uncomplicated malaria. Similar protection afforded by haemoglobin C (HbC) was more recently demonstrated although findings are less conclusive. Clinical studies performed in Nigeria and Mali has found no protection (20, 21), while other Malian study and Burkina study indicated an association between HbAC and clinical malaria (22, 23). However, there was significant decrease in malaria parasite count mean value of Hb AA, AS and AC in post treatment, Hb AS still had significantly lower mean malaria parasite count compared to Hb AC and AA. This was due to effect of anti-malaria drug used during treatment. In pre-treatment, absolute platelet count of Hb AS was lower compared to Hb AC while Hb AA had higher mean value. In post treatment, there was generally increase in absolute platelet count although no significant difference was observed, in both pre treatment and post treatment. Hb AA still had higher absolute platelet mean value compared with Hb AS and AC. low platelet in this present study support the facts that platelet could form 'clumps' with *Plasmodium*-infected erythrocytes, hence thrombocytopenia may be helpful as a sensitive but not specific marker of active infection. However, low amount of platelets may not only be a marker of parasite burden but may be protective against severe disease (24, 25). Control subject in this present study of haemoglobin variants Hb AA, AS and AC had absolute platelet count within the normal range although there is no significant difference. Platelet distribution width was lower in Hb AA compared to Hb AS and AC while mean platelet volume was higher in Hb AA compared to Hb AS and Hb AC in pre treatment. However, in post treatment, platelet distribution width and mean platelet volume were low in Hb AA compared to Hb AS and Hb AC although there is no significant difference in pre treatment and post treatment. There was general increase in immune response

to malaria attack in Hb AA compared to Hb AS and AC. Hb AA had slightly high total white blood cell count, relative and absolute neutrophil count, absolute lymphocyte and monocyte were slightly higher in Hb AA compared to Hb AS and Hb AC in pre treatment. Moreover, there was general decrease in total and differential count except slight lymphocytosis that was observed in post treatment compared to pre treatment, it was generally observed that, immune response started returned to normal while compared the mean value in post treatment with control subject. This decrease was due to the effect of anti-malaria drug used during treatments. There is significant difference in total and differential leukocyte count in pre treatment and post treatment. The findings in this present study was similar to the report of Francis and Pete (14), stated that white blood cell and granulocyte counts were higher in Hb AA subjects compared with other haemoglobin variants and on the contrary they reported that, lymphocyte count was higher in Hb AA compared with other haemoglobin variants during the progress of malaria infection which could be associated with severe or acute malaria. However, in Hb AS and Hb AC, there is genetic immune resistance to malaria infection which reflected in this present study as supported by Francis and Pete (14), who reported that heterozygote individuals (HbAS) were more resistant to *falciparum malaria* than normal dominant homozygous (HbAA) individuals and Hb AA subjects suffered malaria more frequently, had significantly higher parasite density than other haemoglobin variants which is similar to this present study. Multiple comparisons among Hb genotype AA, AS and AC showed significant difference in most of the comparison in pre treatment and post treatment, but there is no significant difference in most of the comparison in control.

7. Conclusion

Mean parasite density was markedly lower in HbAS relative to HbAA genotype normal haemoglobin, Parasite density was however higher among AC relative to AA genotype, suggesting potential mechanistic variation among protection afforded by abnormal genotypes. Indeed, abnormal haemoglobin may not allow for optimal development of *Plasmodium* in deep organs where oxygen pressure is reduced.

References

- [1] World Health Organization (WHO) Expert Committee on Malaria Twentieth report Geneva, world health organization, (WHO Technical report serie, No 892); (2000) 120pp
- [2] Breman JG, Egan A, Kensch GT. The intolerable burden of malaria: a new look at the numbers. *AM J Trop Med Hyg*; (2001) **64** 1-2.
- [3] Miller LH, Baruch D I, Marsh K, Doumbo OK. The pathogenic basis of malaria *Nature* ; (2002) **415**: 673-67
- [4] WHO/UNICEF. Rapport Sur le Paludisme en Afrique; (2003) p 36-59
- [5] Tiffert T, Lew VL, Ginsburg H, et al. The hydration state of human red blood cells and their susceptibility to invasion by *Plasmodium falciparum*. *Blood*; (2005); **105**:4853.

[6] Ayi K, Turrini F, Piga A, Arese P. Enhanced phagocytosis of ring-parasitized mutant erythrocytes: a common mechanism that may explain protection against falciparum malaria in sickle trait and beta-thalassemia trait. *Blood*; (2004); **104**:3364-3371.

[7] Williams TN: How do hemoglobins S and C result in malaria protection? *J Infect Dis*, (2011); **204**:1651-1653.

[8] Kennedy JR: Malaria: a vaccine concept based on sickle haemoglobin's augmentation of an innate autoimmune process to band 3. *Int J Parasitol*, (2001), **31**:1275-1277.

[9] Roberts DJ, Williams TN: Haemoglobinopathies and resistance to malaria. *Redox Rep*, (2003); **8**:304-310.

[10] Ferreira A, Marguti I, Bechmann I, Jeney V, Chora A, Palha NR, Rebelo S, Henri A, Beuzard Y, Soares MP: Sickle hemoglobin confers tolerance to Plasmodium infection. *Cell*, (2011) **145**:398-409.

[11] Williams T. N. "Red blood cell defects and malaria," Molecular and Biochemical *Parasitology*, (2006) vol. **149**, no. 2, 121-127.

[12] Monica Cheesbrough. Discrete Laboratory Practice in Tropical Countries Part 1, Cambridge Second Editions. Published by Press Syndicate of the University of Cambridge, (2005) chp. 5, page 247 – 258

[13] Uzoegwu P.N and Onwurah A.E. Prevalence of hemoglobinopathy and malaria disease in the population of old Aguata Division, Anambra State, Nigeria. *Biokemistri*, (2003) **15**: 57-66

[14] Francis M. AWAH and Pete N. UZOEGWU. Influence of sickle heterozygous status and glucose-6-phosphate dehydrogenase deficiency on the clinico-haematological profile of Plasmodium falciparum-infected children *Biokemistri* (2006); **18**(2):89-97

[15] Edith C Bougouma, Alfred B Tiono, Alphonse Ouédraogo, Issiaka Soulama, Amidou Diarra, Jean-Baptiste Yaro, Espérance Ouédraogo, Souleymane Sanon, Amadou T Konaté, Issa Nébié. Haemoglobin variants and Plasmodium falciparum malaria in children under five years of age living in a high and seasonal malaria transmission area of Burkina Faso; *Malaria Journal* (2012); **11**: 154

[16] Rihet P, Flori L, Tall F, Traore AS, Fumoux F: Hemoglobin C is associated with reduced Plasmodium falciparum parasitemia and low risk of mild malaria attack. *Hum Mol Genet*, (2004); **13**:1-6.

[17] A. Williams TN, Mwangi TW, Wambua S, Alexander ND, Kortok M, Snow RW, Marsh K Sickle cell trait and the risk of Plasmodium falciparum malaria and other childhood diseases. *J Infect Dis*, (2005) **192**:178-186.

B. Williams TN, Mwangi TW, Roberts DJ, Alexander ND, Weatherall DJ, Wambua S, Kortok

[18] M, Snow RW, Marsh K : An immune basis for malaria protection by the sickle cell trait. *PLoS Med*, (2005)**2**:e128.

[19] Verra F, Bancone G, Avellino P, Blot I, Simpre J, Modiano D: Haemoglobin C and S in natural selection against Plasmodium falciparum malaria: a plethora or a single shared adaptive mechanism? *Parassitologia*, (2007); **49**:209-213.

[20] Kreuels B, Kreuzberg C, Kobbe R, Ayim-Akonor M, Apiah-Thompson P, Thompson B,

[21] Ehmen C, Adjei S, Langefeld I, Adjei O, May J: Differing effects of HbS and HbC traits on uncomplicated falciparum malaria, anemia, and child growth. *Blood*, (2010); **115**:4551-4558.

[22] Guinet F, Diallo DA, Minta D, Dicko A, Sissoko MS, Keita MM, Wellem TE, Doumbo O : A comparison of the incidence of severe malaria in Malian children with normal and C-trait hemoglobin profiles. *Acta Trop*, (1997); **68**:175-182.

[23] Jeremiah ZA, Jeremiah TA, Emelike FO: Frequencies of some human genetic markers

[24] and their association with Plasmodium falciparum malaria in the Niger Delta, Nigeria. *J Vector Borne Dis*, (2010); **47**:11-16.

[25] Modiano D, Luoni G, Sirima BS, Simpre J, Verra F, Konate A, Rastrelli E, Olivieri A, Calissano C, Paganotti GM, D'Urbano L, Sanou I, Sawadogo A, Modiano G, Coluzzi M: Haemoglobin C protects against clinical Plasmodium falciparum malaria. *Nature*, (2001); **414**:305-308.

[26] Aggarwal R, Mishra A, Crochet P, Sirimanna P, Darzi A: Effect of caffeine and taurine on simulated laparoscopy performed following sleep deprivation. *Br J Surg*, (2011); **98**:1666-1672.

[27] Lou, J., Lucas, R. and Grau, G. E. Pathogenesis of Cerebral Malaria: Recent Experimental Data and Possible Applications for Humans *Clin. Microbiol Rev.* (2001) **14**:810-820.

[28] Pain, A., Ferguson, D. J. and Kai, O. Platelet-Mediated Clumping of Plasmodium falciparum-Infected Erythrocytes is a Common Adhesive Phenotype and is Associated with Severe Malaria. *Proc. Natl. Acad. Sci. USA*. (2001) **98**:1805-1810.

Table 1: Blood Cell Parameters on Haemoglobin Genotype Variant in Pre Anti-Malaria Drug Treatment Of Malaria Infected Subjects

Groups	MPC μ/L	PLT X10 ⁹ /L	PDW fl	MPV pL	WBC X10 ⁹ /L	NEU %	LYMP %	MONO %	EOSIN %	NUE X10 ⁹ /L	LYM X10 ⁹ /L	MONO X10 ⁹ /L
Hb AA (N=130)	2795.10 ±378.60	171.61 ±50.88	13.45 ±2.51	9.79 ±0.81	7.52± 1.37	59.81± 9.28	35.77± 8.79	3.65± 2.34	0.79± 0.99	4.53± 1.20	2.66± 0.72	0.27± 0.20
Hb AS (N=54)	2311.10 ±295.37	162.57 ±48.51	13.83 ±2.16	9.78 ±0.78	5.09± 1.01	51.44± 10.29	43.17± 9.31	3.96± 1.97	1.44± 1.99	2.63± 0.78	2.19± 0.64	0.20± 0.10
Hb AC (N=18)	2374.60 ±279.74	165.22 ±49.40	13.98 ±2.71	9.59 ±0.75	5.47± 0.87	54.89± 5.79	41.61± 6.30	2.94± 1.39	0.67± 0.97	3.03± 0.72	2.25± 0.33	0.16± 0.08
F (P-value)	41.56 (0.00*)	0.67 (0.52)	0.96 (0.50)	0.52 (0.59)	81.59 (0.00*)	15.93 (0.00*)	15.08 (0.00*)	1.49 (0.23)	5.04 (0.01*)	66.29 (0.00*)	10.48 (0.00*)	5.66 (0.00*)
Hb AA vs Hb AS p-value	0.00*	0.49	0.57	0.99	0.00*	0.00*	0.00*	0.62	0.06	0.00*	0.00*	0.01*

Hb AA vs Hb AC p-value	0.00*	0.87	0.72	0.54	0.00*	0.01*	0.01*	0.18	0.87	0.00*	0.00*	0.00*
Hb AS vs HbAC p-value	0.69	0.98	0.97	0.63	0.31	0.19	0.71	0.05*	0.08	0.13	0.89	0.16

P<0.05 significance, P>0.05 no Significant, F (P-value) = mean ± SD of parameters compared using ANOVA

Table 2: Blood Cell Parameters On Haemoglobin Genotype Variants In Post Anti Malaria Drug Treatment In Malaria Infected Subjects

Groups	MPC μ/L	PLT X10 ⁹ /L	PDW fl	MPV pL	WBC X10 ⁹ /L	NEU %	LYMP %	MONO %	EOSIN %	NUE X10 ⁹ /L	LYM X10 ⁹ /L	MONO X10 ⁹ /L
Hb AA (N=130)	2477.30±59 4.81	184.65± 59.58	13.59± 2.51	9.54± 0.67	6.75± 1.79	55.04± 8.24	42.78± 7.78	1.79± 1.54	0.38± 0.68	3.73± 1.22	2.87± 0.87	0.13± 0.14
Hb AS (N=54)	1983.50±16 1.26	170.26± 52.85	13.93± 2.28	9.71± 0.63	4.15± 0.93	48.41± 8.39	48.80± 7.49	2.02± 1.57	0.80± 1.29	2.02± 0.61	2.02± 0.51	0.08± 0.07
Hb AC (N=18)	2026± 165.03	173.11± 53.03	14.18± 2.70	9.79± 0.57	4.43± 0.88	50.94± 5.42	47.39± 5.45	1.33± 0.97	0.33± 0.49	2.29± 0.64	2.07± 0.33	0.06± 0.04
F (P-value)	22.71 (0.00*)	1.33 (0.27)	0.61 (0.51)	2.19 (0.12)	62.73 (0.00*)	13.44 (0.00*)	13.31 (0.00*)	1.42 (0.25)	4.57 (0.01*)	57.68 (0.00*)	28.99 (0.00*)	3.96 (0.02*)
Hb AA vs Hb AS p-value	0.00*	0.24	0.65	0.21	0.00*	0.00*	0.00*	0.65	0.08	0.00*	0.00*	0.02*
Hb AA vs Hb AC p-value	0.00*	0.68	0.67	0.22	0.00*	0.02*	0.01*	0.21	0.92	0.00*	0.00*	0.00*
Hb AS vs HbAC p-value	0.61	0.98	0.94	0.88	0.48	0.31	0.67	0.08	0.08	0.28	0.85	0.21

P<0.05 Significance, P>0.05 no Significant, F (P-value) = mean ± SD of parameters compared using ANOVA

Table 3: Mean ±Sd Of Blood Cells Parameters On Haemoglobin Genotype Variants In Non-Malaria Infected Subjects (Control)

Groups	MPC μ/L	Platelet X10 ⁹ /L	PDW fl	MPV pL	WBC X10 ⁹ /L	NEU %	LYMP %	MONO %	EOSIN %	NEU X10 ⁹ /L	LYM X10 ⁹ /L	MONO X10 ⁹ /L
Hb AA (N=79)	-	284.53± 46.27	11.99± 1.61	9.52± 0.22	4.43± 0.32	57.19± 4.98	41.62± 4.76	0.97± 1.07	0.33± 0.71	2.54± 0.31	1.84± 0.23	0.04± 0.05
Hb AS (N=21)	-	291.24± 55.68	12.01± 2.26	9.52± 0.31	4.41± 0.39	57.57± 3.19	41.29± 2.90	0.86± 0.96	0.29± 0.56	2.54± 0.22	1.82± 0.23	0.04± 0.05
Hb AC (N=02)	-	271.00± 25.46	12.60± 1.67	9.25± 0.49	4.35± 0.35	58.00± 7.07	42.00± 7.07	-	-	2.53± 0.51	1.82± 0.16	-
F (p-value)	-	0.26 (0.78)	0.12 (0.89)	1.17 (0.31)	0.08 (0.92)	0.08 (0.93)	0.06 (0.95)	0.91 (0.41)	0.25 (0.78)	0.00 (0.99)	0.06 (0.95)	0.86 (0.43)
Hb AA vs Hb AS p-value	-	0.87	1.00	1.00	0.97	0.91	0.91	0.88	0.95	1.00	0.95	0.93
Hb AA vs Hb AC p-value	-	0.79	0.89	0.78	0.95	0.99	0.99	0.00*	0.00*	1.00	0.98	0.00*
Hb AS vs HbAC p-value	-	0.68	0.90	0.78	0.93	1.00	0.98	0.00*	0.07	1.00	1.00	0.00*

P<0.05 Significance, P>0.05 no Significant, F (P-value) = mean ± SD of parameters compared using ANOVA