THE EFFECTS OF HAEMOGLOBINOPATHIES AND G6PD DEFICIENCY ON MALARIA AMONG CHILDREN OF THE KINTAMPO NORTH MUNICIPALITY OF GHANA

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INTRODUCTION AND BACKGROUND

- Malaria is the leading cause of childhood mortality, with 90% of this mortality occurring in Sub-Saharan Africa (Mc Combie, 2002).
- It causes between 300-600 million clinical malaria episodes and mortality in one to three million children under the age of five each year (WHO, 2000).
- Host genetic factors that confer resistance to disease are widespread in regions endemic for *Plasmodium falciparum* malaria.
- G6PD deficiency and haemoglobinopathies have been recorded in some studies to have protective effects on clinical malaria. (Aidoo *et al.,* 2002)

JUSTIFICATION

- Many people in the study area generally perceive every fever to be malaria induced and the holo-endemic nature of the disease could be the main reason for this perception.

- Therefore it is important to know the prevalence of these factors in any population and especially how they influence malaria pathogenesis particularly in areas where malaria interventions such as drug or vaccine trials are ongoing as pertains in the Kintampo North Municipality of Ghana

- This will help to avoid possible confounding of clinical trial research outcome.

OBJECTIVES

Broad Objective

- To determine the host genetic factors (haemoglobinopathies and G6PD deficiency) that predispose or protect children less than five years against clinical malaria in the Kintampo north municipality.

Specific Objectives

- To determine the prevalence of haemoglobinopathies and G6PD deficiency among children under-5 years who present to the Kintampo North Municipal Hospital with fever

- To determine the proportion of *P. falciparum* malaria infection among children with the different haemoglobinopathies and those with or without G6PD deficiency.

- To determine the association between *P. falciparum* malaria infection and haemoglobinopathies and G6PD deficiency among the children.

METHODOLOGY

- Cross sectional survey conducted at the Kintampo North Municipal Hospital in which blood samples of 341 children, aged 6-59 months were collected and analysed after informed consent have been given by caretakers of the children.

- Using the Kintampo Health and Demographic Surveillance System (KHDSS), it was possible to know all children who were true residents of the Kintampo North Municipal**ity** (as inclusion criteria) by means of their KHDSS identification cards and to trace them to their compounds for children with genetic abnormalities such as sickle cell disease and G6PD deficient for counselling on how to manage their conditions.

METHODOLOGY CONT'D

- Blood slide smears were prepared for each child, stained with 3% Giemsa and read by expert microscopist (a slide was declared negative after examining 200 high power fields).

- Malaria parasite density was estimated from parasite counted per 200 white blood cells(wbcs) and extrapolated to parasite per microlitre of blood using the subjects own absolute leucocytes

- G6PD deficiency status was determined using quantitative in vitro method (Randox Laboratory UK) and the haemoglobin variants were also determined by haemoglobin electrophoresis kit (Helena BioSciences, Beaumont, USA).

METHODOLOGY CONT'D

STATISTICAL ANALYSIS

- Statistical analysis was performed using Stata software version 9. for window (©college station, texas 77845 USA).

- Proportional data were compared using $\chi 2$-test and means between two groups were compared using t-test.

- Statistical significance was set at $p \leq 0.05$.

RESULTS

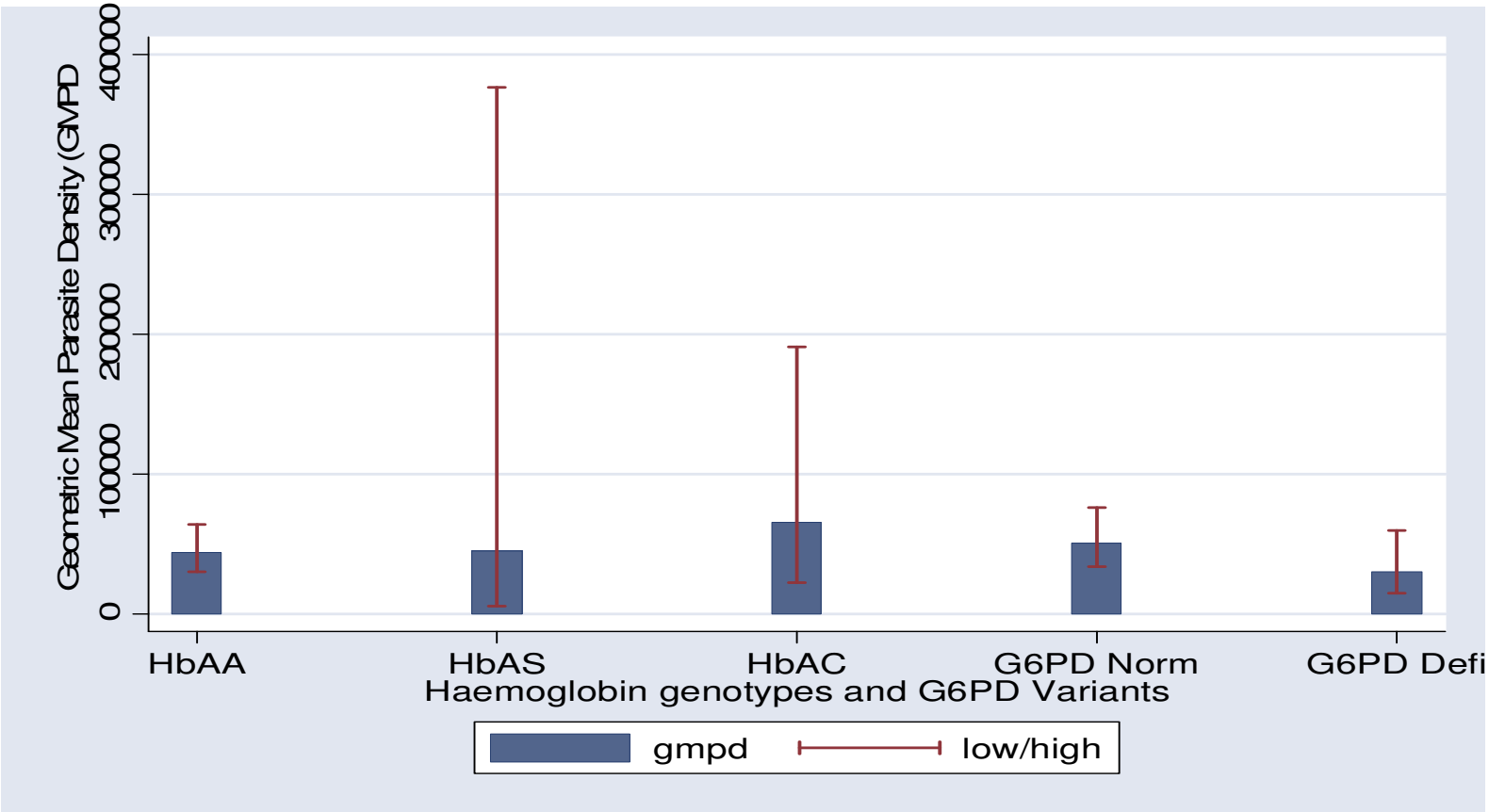
Table1: Prevalence of Haemoglobinopathies and G6PD Variants

Genetic factors	Prevalence n,(%)
G6PD normal	276 (80.9)
G6PD deficient	65 (19.0)
HbAA	255 (75.9)
HbAS	31 (9.2)
HbAC	50 (14.9)
HbSC	1 (0.3)
HbCC	1 (0.3)
HbSS	3 (0.9)

Prevalence of G6PD deficiency was 19.0 % (65/341) and for haemoglobinopathies were 75.9% (225/341),14.9% (50/341) and 9.2% (31/341) for HbAA, HbAC and HbAS respectively.

RESULTS CON'T

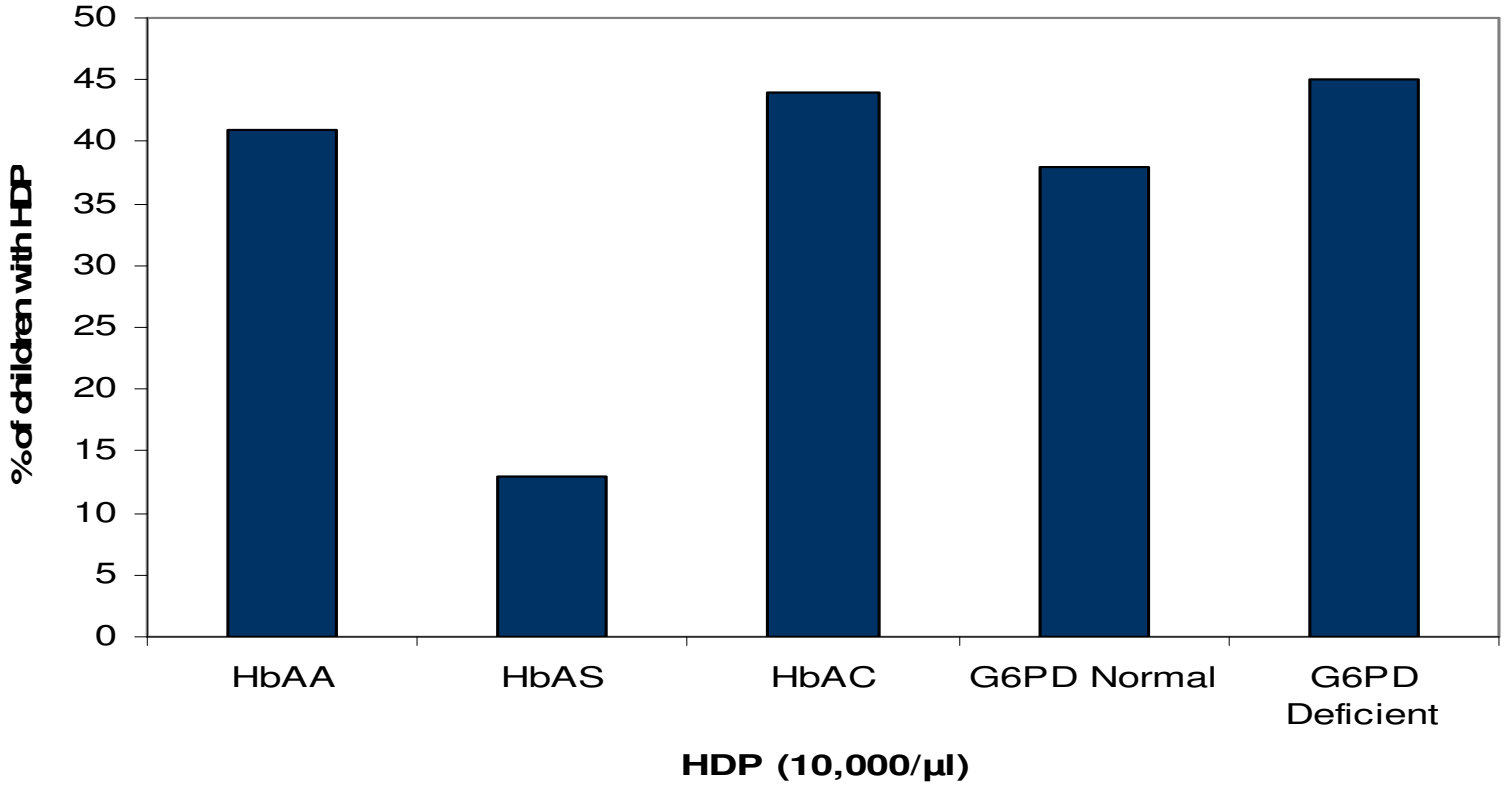
Figure-1:The Geometric Mean Parasite Density of the Different Genetic Variants



Results show that the malaria prevalence among the children was 48%.

RESULTS CONT'D

Figure 3: Proportion of High Density Parasitaemia (HDP) in the Haemoglobinopathies and G6PD Variants



Children with the sickle cell trait had reduced parasite density relative to the other genetic variants.

RESULTS CONT'D

Table 2: Association between Haemoglobinopathies and G6PD variants and Clinical malaria

Genetic Factors		No of children diagnosed with clinical malaria n (%)	No of children not diagnosed with clinical malaria n (%)	Univariate (unadjusted) Odds ratio (95% CI)	p-value	Multivariate (adjusted) Odds ratio (95% CI)	p-value
G6PD variants	Normal	182 (65.9)	94 (34.1)	1		-	-
	Deficient	37 (56.9)	28 (43.1)	0.7 (0.39 – 1.29)	0.257	-	-
Haemoglobinopathies	HbAA	167 (65.5)	88 (34.5)	1		1	
	HbAC	37 (74)	13 (26)	1.0 (0.47 – 1.94)	0.902	0.9 (0.45 – 1.94)	0.89
	HbAS	10 (32.3)	21 (67.7)	0.5 (0.21 – 0.98)	0.041	0.4 (0.20 – 0.96)	0.40

Univariate logistic model shows a negative association of malaria with the individuals with the sickle cell traits (HbAS) (OR 0.5, 95%CI (0.21 – 0.98), p=0.04)

CONCLUSION AND DISCUSSION

- Previous studies showed that the HbAS genotype had significantly reduced parasite density, and high density parasitaemia (HDP) compared with the HbAA genotype (Aidoo *et al.* 2002)

- This study showed significantly reduced parasite density among HbAS genotype and higher HDP among HbAC group

- Findings also showed that sickle cell trait HbAS was protective against clinical malaria but not G6PD deficiency in children less than 5 years in the Kintampo North Municipality of Ghana.

LIMITATION

- The study was hospital based, time for the conduct of the study was short, hence the sample size was small.

RECOMMENDATIONS

- The survey sample should consist of a larger population of age-appropriate subjects in order to detect large numbers of other erythrocytic polymorphisms which are believed to be in the population.

- In future studies, samples from community members should also be included.

- Molecular characterization of the G6PD and haemoglobinopathies should be performed (PCR).

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