



## The Prevalence of Glucose-6-Phosphate Dehydrogenase (G6PD) Deficiency in Students of Sultan Abdurrahman School of Health Technology Gwadabawa, Sokoto, North-Western Nigeria

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### **Authors' contributions**

*This work was carried out in collaboration between all authors. Authors MLJ and KKI designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Authors GA, AAN and JB managed the analyses of the study. Authors UIA and CN, managed the literature searches. All authors read and approved the final manuscript.*

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### **ABSTRACT**

**Background:** The aim of this study was to determine the prevalence of the deficiency of Glucose-6-phosphate 10 dehydrogenase (G6PD) activity among students of Sultan Abdurrahman School of Health Technology Gwadabawa, Sokoto State.

**Methodology:** A total of 164 subjects comprised of 82 (50%) males and 82 (50%) females were recruited for this study. Randox G6PD qualitative *in vitro* test screening was used for the diagnosis of G6PD deficiency.

**Results:** Of the 164 subjects tested, 144 (87.8%) were normal while 20(12.2%) were G6PD-deficient. Out of the 82 male subjects studied, 64 (78%) were normal while 18 (22%) were G6PD deficient compared to 80 (97.6%) and 2 (2.4%) females subjects who were normal and deficient for G6PD respectively. There was a significant difference between gender ( $p < 0.01$ ) using chi-square.

**Conclusions:** The Prevalence of G6PD deficiency was concentrated predominantly among male subjects (22%). There is a need for the routine screening of subjects on a much wider scale for G6PD deficiency in our environment, especially in malaria endemic areas, where quinine and its derivatives be used. This would allow for evidence-based management of subjects with G6PD deficiency and also educate them so as to avoid food, drugs, and other agents that can potentially predispose them to hemolytic crisis or oxidative stress.

*Keywords: Prevalence; G6PD; students; Gwadabawa; Sokoto; Nigeria.*

## 1. INTRODUCTION

Glucose-6-phosphatase dehydrogenase (G-6-PD) deficiency is one of the most common enzyme defects in humans affecting close to 400 million people worldwide. G6PD catalyzes the first reaction of the pentose phosphate pathway (PPP): in this reaction, NADPH which protects red blood cells from oxidative damage is produced [1].

This deficiency principally affects men of African and Mediterranean ethnic origins, with a prevalence of approximately 10% [2]. It is an X-linked disorder and has many genotypes (homozygous and heterozygous). The gene that code for the enzyme is located on the X-chromosome [1]. Males are mostly affected than female but occasionally homozygous females are affected. Deficiency in heterozygous females may provide protection against severe malaria where *P. falciparum* is endemic [3]. G6PD deficiency can also be inherited from females who are heterozygous i.e carry one copy of the causative gene on one of their X chromosomes. Males who inherit the defective gene from the mother develop G6PD deficiency, while females who receive the defective gene end up as carriers (carrier females generally do not show any characteristic symptoms) [4].

G6PD deficiency has many different variants and Mediterranean mutation is the most common variant of enzyme deficiency and often associated with favism [1]. The commonest in Africa is the variant A-G6PD [3].

According to the World Health Organization (WHO), 7.5-10% of the world population bears

one or two genes variants for G6PD deficiency and about 2.9% out of the said number are G6PD deficient. [5]. The prevalence of this deficiency varies around the globe, with frequencies ranging from 2% to 20% in Greece, Turkey, and Italy; but increased as much as 70% in groups of Kurdish Jews. Recently, a Bayesian geo-statistical model was used to estimate the prevalence of G6PD deficiency worldwide. This map presented the allelic frequency of the phenotypic deficiency, which is the prevalence of the disease in men. The results of this study showed that in Latin America (LA), the prevalence of G6PD deficiency is lower compared with other regions such as sub-Saharan Africa or Asia [2]. In Asia, the deficiency prevalence ranges from 6.0% to 15.8%. In India, it is 10.5%, while in the Middle East the prevalence varies from 3% to 29% [6,7]. In Brazil, a few studies have found a prevalence between 1.7% and 6.0% [8].

In Africa, the prevalence of G6PD deficiency has been reported as high as 28.1% in south-west Nigeria, 22.5% in Congo (Brazzaville), 15.7% in Mali (Bamako), 13.0% in Uganda and 9.0–15.5% in Gabon [9]. Previous reports in Nigeria showed that the prevalence of G6PD deficiency ranged from 4% to 26%. However, a study by I.Z. Isaac et al (2016) [10] in Sokoto, Nigeria among 118 children visiting the Emergency Pediatrics unit of Usmanu Danfodiyo University Teaching Hospital for pediatrics related care indicated G6PD deficiency of 14.4%. Also a study by Bello et al (2016) [4] in Katsina among children aged 0 – 5 years reported an overall prevalence of 16.2% [4,11,12]. It is most frequent among individuals of African descent, with a frequency ranging from 3.6% to 28.0% [6,7].

Most people with the deficiency do not present any symptoms until exposed to oxidative drugs, some infections or ingestion of Fava beans. Majority of deficient patient develop mild to severe chronic hemolysis only when oxidative hemolysis occurs [3]. The degree of hemolysis changes according to the degree of enzyme deficiency and the oxidant agent exposure [13]. Avoiding the oxidative agents that induces hemolysis is the most important step in the treatment of the enzyme deficiency. Screening of Neonates and proper health education can reduce the rate of G6PD deficiency [1].

## **2. MATERIALS AND METHODS**

### **2.1 The Study Area**

The study area is Sultan Abdurrahman School of Health Technology (SASHT) Gwadabawa. Gwadabawa is one of the local government areas in Sokoto State, North Western Nigeria. The local government has a land mass of 991km<sup>2</sup> and a population of 231,569 in 2006 census, and a projected population of 269,050 by 2011 [14]. The inhabitants are predominantly Hausa-Fulani by tribe, and most of the students attending the school are from the town and nearby cities which shares the same ethnicity.

### **2.2 Study Subjects**

A total of 164 apparently healthy individuals comprised of 82 (50%) males and 82 (50%) females attending Sultan Abdurrahman School of Health Technology (SASHT), Gwadabawa and its environment were recruited for this study.

### **2.3 Study Design**

This was a cross sectional study designed to determine the prevalence of G6PD deficiency among the subjects attending SASHT, Gwadabawa and its environment. The patients were conveniently enrolled into the study. Qualitative data was elicited using interviewer self-administered questionnaire, while quantitative test data was obtained through screening for G6PD status of the subjects.

#### **2.3.1 Inclusion criteria**

Apparently healthy male and female subjects, without prior knowledge of their G6PD status.

#### **2.3.2 Exclusion criteria**

Subjects with known cases of infection or use of therapeutic drugs.

### **2.4 Ethical Considerations**

Ethical approval was obtained from the school authority, and Informed consent was obtained from the patients prior to the commencement of the study as directed by the management of SASHT, Gwadabawa, Sokoto, North-Western Nigeria.

### **2.5 Sampling Techniques**

Apparently healthy individuals were enrolled for the study, structured interviewer self-administered questionnaires were used to obtain the patient's data, such as the demographic data including age, gender, occupation, etc. and other relevant data were obtained from the them.

#### **2.5.1 Specimen collection and processing**

Two (2 milliliters of whole blood was collected from each study participants and the blood emptied into S-Monovette, ethylene-diamine-tetra-acetic acid (K<sub>2</sub>EDTA) anti coagulated vacutainer tubes. The anti-coagulated blood was stored in a refrigerator till required for use in the screening of subjects for G6PD deficiency (qualitative test). All sample collected were analysed within 48 hours.

### **2.6 Test Principles and Procedures**

#### **2.6.1 Methaemoglobin method of screening for G6PD deficiency**

##### *2.6.1.1 Principle*

Sodium nitrite converts hemoglobin (Hb) to methaemoglobin (Hi) when no methylene blue is added. Methaemoglobin persists but incubation of the samples with methylene blue allows stimulation of the pentose phosphate pathway in subject with normal G6PD levels. The Hemoglobin level (Hb) is reduced during the incubation period. In G6PD deficient subjects, blockage in the pentose phosphate pathway prevent this reduction.

##### *2.6.1.2 Reagents*

Sodium nitrite (180 mmol/L), Dextrose (280 mmol/L) 5 g of AR dextrose and 1.25 g of NaNO<sub>2</sub> were dissolve in 100 ml of water.

Methylene blue (0.4 mmol/L);.Dissolve 150 mg of Methylene blue chloride, (Sigma, country) was dissolve in 1 liter of water.

2.6.1.3 Procedure

Two milliliters (2 mL) of anticoagulated (EDTA) blood was added to the tube containing 0.1 ml of the freshly prepared Sodium Nitrite Glucose reagent and 0.1 ml Methylene blue and mixed gently by inverting the tube marked (Test).

The Control tube was prepared by adding 2 ml of blood without the Sodium Nitrite Glucose reagent and Methylene blue marked (normal reference tube).

Two milliliters (2 ml) of blood was also added to another tube containing 0.1 ml of Sodium Nitrite – Glucose reagent without methylene blue and mixed gently marked (deficient reference tube) [3].

All the tubes were incubated at 37°C for 90 minutes.

2.6.1.4 Interpretation

Normal blood will produce a colour similar to that in the normal reference tube (clear red). Blood from a deficient subject give a brown colour similar to that in the deficient reference tube.

Table of Protocol

Tube	Test	Normal reference	Deficient reference
Sodium-nitrite-glucose reagent	0.1 ml	-	0.1 ml
Methylene-blue reagent	0.1 ml	-	-
Sample	2 ml	2 ml	2 ml

2.7 Statistical Analysis

The data collected were entered into the data editor of statistical package for social sciences (SPSS Version 22) software. Analysis was based on simple percentages, or proportions. A Chi-square test at a 95% confidence level was also used to test for association of G6PD deficiency between male and female subjects. A p-value of <0.05 was considered significant in all statistical analysis.

3. RESULTS

Table 1 shows the percentages and number of G6PD normal and deficient subjects in the study. From the Table it can be seen that the number of deficient males is higher than that of the females.

Table 1. The percentages of G6PD status in both male and female

G6PD status	Male	Female	Percentage (%)
Normal	64 (78.0)	80(97.6)	144(87.8%)
Deficient	18 (22.0)	2(2.4)	20 (12.2%)
Total	82 (100)	82(100)	164 (100%)

p = 0.000

4. DISCUSSION

In this study, 87.8% of the students were found to be normal, while 12.2% are deficient, which was consistent to the study carried out by Akanni et al. [15] in Osogbo, South- west Nigeria, that reported a prevalence of 80.5% normal and 19.5% deficient in male subjects. The male subjects had a higher prevalence (22%) of G6PD deficiency compared to their female's counterparts with a prevalence of 2%. This further supports the history of G6PD deficiency as an X-linked recessive genetic disorder carried by genes on X chromosome; and the fact that male hemizygotes and female homozygotes are most frequently affected, Amiwero C. E. and Olatunji P. O. [16] hence this deficiency will largely affect males, since the deficiency is inherited in a sex linked fashion and the fact that the abnormal genes are mostly found in Africans and Mediterranean's than others.

This study also agrees with the work of Ademowo and Falusi [17] that reported a G6PD deficiency prevalence of 4.6% in females and 23.9% in males, and Isaac et al. (2013) that reported a prevalence of 22.1% in male children in their study carried out among children in Sokoto, North-west Nigeria. Another study by Ouattara et al. [18] found a higher prevalence in males than Females, even though their overall prevalence was 9.5% in a rural community in Burkina Faso. This difference in the overall prevalence might be due to ethnic differences between their study group and ours, another factor may be that they are having a different variant of the enzyme than the variant found in North-western Nigeria, even though the A(-) variant (with 10 – 20% activity of the B variant) and a frequency of up to 15% is common in sub Saharan Africa.

Our study was also consistent with the prevalence of 20% recorded by Egesie et al. [12], in subnormal G6PD activity in the male population in Jos, North Central, Nigeria. In another research carried out by Al-Riyami and

Ebrahim, [6] in the Sultanate of Oman reported a prevalence of 25% for males children which was consistent with our study and 10% for female children, which is at variance to the 2% reported by this study. G6PD deficiency is believed to be beneficial as it is reported that red cells that are G6PD deficient, are also resistant to *Plasmodium falciparum* invasion since the *Plasmodium* parasite requires the enzyme for its normal survival in the host. Even though this deficiency offers selective protection against *P. falciparum* malaria, there are reports that some *P. falciparum* parasite strains have been able to synthesize their own G6PD enzyme, and thereby evading the immunity conferred by the absence of the enzyme [12].

Nguetse et al. [19] also reported an overall G6PD prevalence of 13% in a study carried out on Gabonese, Ghanaian and Kenyan children, this result is similar to that obtained in this study.

## 5. LIMITATIONS

This study is limited by the fact that, though the methaemoglobin reduction screening test employed is sensitive and specific, there are conflicting reports from other studies about its sensitivity and specificity. However, Methaemoglobin reduction screening test remains a useful and reliable alternative for researches most especially in developing countries like Nigeria; where there are limited resources, no research grants and the cost of routine screening of G6PD is an issue. Other techniques regularly employed for the estimation of G6PD enzyme includes the NADPH fluorescent spot test (qualitative). This method detects the fluorescence of NADPH, which is proportional to G6PD activity, under long-wave UV light (365 nm), the spectrophotometric enzyme assay (quantitative) These tests are run in duplicates and incubated at 30°C in a water bath for 5 minutes, the Enzyme activity determine using a temperature regulated spectrophotometer, the lateral flow colorimetric test (qualitative) which must be performed between 18°C and 25°C, Cytochemical staining and flow cytometry. As well as the Methaemoglobin reduction test (qualitative) [20].

## 6. CONCLUSION

This study has shown a high prevalence (22%) of G6PD deficiency among Male subjects residing at SASHT, Gwadabawa Local government area of Sokoto State, north-western Nigeria. Our

findings indicated a male-sex bias in the prevalence of G6PD deficiency among the subjects presented in this study. There is need for the inclusion of G6PD screening as a routine test in the Laboratory, bearing in mind that Gwadabawa Local Government is one of the mosquito endemic areas and as such the possibility of hemolytic crisis for patients that are deficient in G6PD enzyme that may take quinine containing antimalarial drugs or any oxidative drug that can trigger the hemolysis. Creating awareness among health professionals is very essential in this respect.

## COMPETING INTERESTS

Authors have declared that no competing interests exist.

## REFERENCES

1. Farhud DD, Yazdanpanah L. Glucose-6-phosphate dehydrogenase (G6PD) deficiency. Iranian Journal of Public Health. 2008;37(4):1-18.
2. Gómez-ManzoSaúl, Marcial-Quino Jaime, Vanoye-Carlo America, Serrano-Posada Hugo, Ortega-Cuellar Daniel, González-Valdez Abigail, Castillo-Rodríguez Rosa Angélica, Hernández-Ochoa Beatriz, Sierra-Palacios Edgar, Rodríguez-Bustamante Eduardo, Arreguin-Espinosa Roberto. Glucose-6-Phosphate dehydrogenase: Update and analysis of new mutations around the World. International Journal of Molecular Science. 2016;17(12):2069. DOI: 10.3390/ijms17122069
3. Cheesbrough M. District laboratory practice in tropical countries part two Cambridge University Press India. 2010; 274,309.
4. Bello Ibrahim, Abdulsalami Mohammed Sani, Bulus Timothy. Prevalence of glucose-6-phosphate dehydrogenase deficiency among children aged 0-5 years infected with *Plasmodium falciparum* in Katsina State, Nigeria. Advances in Biochemistry. 2016;4(6):66-73.
5. Hussein UY, Yusoff NM. Prevalence of glucose 6phosphate dehydrogenase (G6PD) deficiency (Favism) in thamar province-republic of Yemen Biohealth Science Bulletin. 2011;3(1):12 –19.
6. Al-Riyami A, Ebrahim GJ. Genetic blood disorders survey in the sultanate of Oman. Journal of Tropical Pediatrics. 2003;49(1): i1– 20.

7. Usanga EA, Ameen R. Glucose-6-phosphate dehydrogenase deficiency in Kuwait, Syria, Egypt, Iran, Jordan and Lebanon. *Human Heredity*. 2000;50(3): 158–161.
8. Castro S, Weber R, Dadalt V, Tavares V, Giugliani R. Prevalence of G6PD deficiency in newborns in the south of Brazil. *Journal of Medial Screening*. 2006;13(2):85–86.
9. Beutler E, Duparc S. Glucose-6-phosphate dehydrogenase deficiency and antimalarial drug development. *American Journal of Tropical Medicine and Hygiene*. 2001; 77(4):779-789.
10. Isaac IZ, Mainasara AS, Osaro Erhabor, ST, Omojuyigbe MK, Dallatu LS, Bilbis TC. Adias. Glucose-6-phosphate dehydrogenase deficiency among children attending the Emergency Paediatric Unit of Usmanu Danfodiyo University Teaching Hospital, Sokoto, Nigeria. *International Journal of General Medicine*. 2013;6:557–562.
11. Luzzatto L, Gordon-Smith EC. Inherited hemolytic anemia. In: Hoffbrand AV, Lewis SM, Tuddenham EGD, editors. *Postgraduate Hematology*. 4th ed. London: Arnold. 2001;120–143.
12. Egesie D, Joseph DE, Isiguzoro I, Egesie UG. Glucose-6-phosphate dehydrogenase (G6PD) activity and deficiency in a population of Nigerian male residents in Jos. *Niger Journal of Physiological Science*. 2008;23(1–2):9–11.
13. Carl AB, Edward RA, David EB. *Fundamentals of clinical chemistry (TIET2)* Elsevier Inc India. 2012;335.
14. National Population Commission; 2006. Available:[www.citypopulation.de](http://www.citypopulation.de) 2015-01-31
15. Akanni EO, Oseni BSA, Agbona VO, Tijani BA, Tosan E, Fakunle EE, Mabayoje VO. Glucose-6-phosphate dehydrogenase deficiency in blood donors and jaundiced neonates in Osogbo, Nigeria. *Journal of Medical Laboratory and Diagnosis*. 2010; 1(1):1-4.
16. Amiwero CE, Olatunji PO. Prevalence of G6PD deficiency in children presenting with jaundice in Ilorin, Nigeria *International Journal of Biomedical and Health Sciences*. 2012;8(1):21-26.
17. Ademowo OG, Falusi AG. Molecular epidemiology and activity of erythrocyte G6PD variants in a homogeneous Nigerian population. *East African Medical Journal*. 2002;79(1):42–45.
18. Ouattara AbdoulKarim, Bisseye Cyrille, Valery Jean Bapio, Elvira Bazie Télesphore, Diarra Birama, Tegwindé Rebeca Compaore, Djigma Florencia, Pietra Virginio, Moret Remy, Jacques Simpure. Glucose-6-phosphate dehydrogenase (G6PD) deficiency is associated with asymptomatic malaria in a rural community in Burkina Faso. *Asian Pacific Journal of Tropical Biomedicine*. 2014;4(8):655–658.
19. Nguetse Christian N, Christian G. Meyer, Ayola Akim Adegnika, Tsiri Agbenyega, Bernhards R. Ogutu, Peter G. Kremsner and Thirumalaisamy P. Velavan. Glucose-6-phosphate dehydrogenase deficiency and reduced haemoglobin levels in African children with severe malaria. *Malaria Journal*. 2016;15:346. DOI: 10.1186/s12936-016-1396-1
20. LaRue, Nicole Kahn Maria, Murray Marjorie, Leader Brandon T, Bansil, Pooja, McGray Sarah, Kalnoky Michael, Zhang Hao, Huang Huiqiang, Jiang Hui, Domingo Gonzalo J. Comparison of quantitative and qualitative tests for glucose-6-phosphate dehydrogenase deficiency. *American Journal of Tropical Medicine and Hygiene*. 2014;91(4):854–861. DOI: 10.4269/ajtmh.14-0194

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