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# Prevalence of Glucose-6-Phosphate Dehydrogenase Deficiency among the Ogoni and Etche Ethnic Groups in Rivers State, Nigeria

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***Authors' contributions***

*This work was carried out in collaboration between all authors. All authors read and approved the final manuscript.*

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## ABSTRACT

**Aims:** This study was aimed at determining the prevalence of Glucose-6-phosphate dehydrogenase deficiency (G6PD) among Ogoni and Etche ethnic groups in Rivers State, Nigeria,
**Study Design:** A total of 200 randomly selected subjects comprising of 100 Ogonis (49 males and 51 females) and 100 Etches (62 males and 38 females) participated in this study.
**Place and Duration of Study:** The study was conducted among members of Ogoni and Etche ethnic groups. Analysis was carried out at the Haematology Laboratory, Department of Medical Laboratory Science, Rivers State University, Port Harcourt, Nigeria, between May and August, 2018.
**Methodology:** Three milliliters of ethylene diamine tetra acetic acid (EDTA) anticoagulated blood was collected from each subject, The G6PD status of the subjects were determined by spectrophotometric method using semi-automated analyzer.
**Results:** The result obtained showed that the prevalence of G6PD deficiency in Ogoni was 9%

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while that of Etche was 13%. There was no significant difference (P=0.19) in the level of deficiency in both the Ogoni and Etche subjects with the value of 6.21±4.34 and 5.92±0.54 respectively.
**Conclusion:** G6PD is more common among Etches than the Ogonis, although with no significant difference. Therefore, G6PD screening test should be included in routine test before treatment with antimalarial drugs like quinine and premaquinine or other drugs that can predispose G6PD deficient individuals to haemolytic episode.

*Keywords: Prevalence; Glucose-6-phosphate dehydrogenase deficiency; Ogoni and Etche Ethnic Groups; Rivers State.*

## 1. INTRODUCTION

Red blood cell metabolism provides the cell with energy in the form of adenosine triphosphate (ATP) to pump ions against electrochemical gradients, maintain its shape, keep iron from haemoglobin in the reduced form and maintain enzyme and haemoglobin sulfhydryl groups. The main source of metabolic enzyme comes from glucose. Glucose is metabolized through the Embden-Meyerhof glycolytic pathway through the Hexose Monophosphate shunt (HMP) to produce adenosine triphosphate (ATP) and nicotinamide adenine dinucleotide (NADH), 2,3,-bisphosphoglycerate, an important regulator of the oxygen affinity of haemoglobin [1].

During the process in the glycolytic pathway, glucose-6-phosphate dehydrogenase is an important enzyme that catalyzes the oxidation of glucose, that is, the oxygen ultilisation of red blood cells (RBCs) and help in maintaining the normal life span of red blood cells [1]. To achieve this, the hexose monophosphate shunt (HMS) oxidize glucose-6-phosphate in the presence of the enzyme (G6PD), thereby generating nicotinamide adenine dinucleoside phosphate (NADPH) which serves the red cells to maintain high concentration of reduced glutathione (GSH) and the red cells are protected from oxidants, with 5-10% of the glucose metabolized [2].

Glucose-6-phosphate dehydrogenase (G6PD) is an X-linked chromosomal inheritance [2]. Thus, mutation of the gene in the X-chromosome, or any factor that can lead to the defects of the gene can cause deficiency of the enzyme (G6PD). Glucose-6-phosphate dehydrogenase deficiency is an X-linked chromosomal disorder and a high polymorphic enzyme affecting about 400 million people worldwide. The enzyme gene involved is located on the long arm of the X-chromosome Xq28 [3]. The resultant effect of the deficiency is the destruction of the red blood cells (RBCs) thereby given rise to the episode of haemolytic anaemia, characterized by the physical signs and symptoms of jaundice with haemolysis of red cells seen in susceptible individuals [2].

Glucose-6-phosphate dehydrogenase deficiency is the most common enzymopathy in humans. It is triggered by predisposing biological agent, such as bacteria and virus infections and also with drugs used for the treatment of malaria infection (e.g. quinine) and exposure to these drugs, bacteria and virus in association with the deficiency of the enzyme increases the breakdown of RBCs in vulnerable individuals, thus leading to haemolysis [3].

Hereditary deficiency of red blood cell glucose-6-phosphate dehydrogenase (G6PD) is associated with clinical manifestations like drug-induced haemolytic anaemia, neonatal jaundice, favism, non-spherocytic congenital haemolytic anaemia [2];[4]. The drug-induced haemolytic anemia associated with G6PD deficiency has an increased frequency of occurrence in some population due to high prevalence of malaria in those areas e.g. Asia, Mediterranean and Africa, non-spherocytic congenital haemolytic anaemia occur mainly by involving the Embden-Meyerhof or hexose monophosphate pathway[ 5].

Reactive oxygen species (R0S) such as super oxide, hydrogen peroxide, hydroxyl radical, cause oxidative stress and damage of cells membrane [6,7]. Increase production of ROS or decrease antioxidative defense enzymes play a major role in oxidative injuries in different organs, tissues, and cells including brain, heart, vascular cells [6], and causes brain disease like Alzheimer and Parkinson disease. They are considered to contribute to the aging process [8,7]. Glucose-6-phosphate dehydrogenase, superoxide dismutases, catalase, glutathione peroxidases and glutathione reductase are antioxidant defense enzymes. In the human body, antioxidant defense system G6PD is an essential modulator enzyme with significant role in cells (red blood cells) [6].

The G6PD functions in maintenance of redox potential in cell producing NADPH in pentose phosphate pathway [6], and also in controlling of cell death.

## 2. MATERIAL AND METHODS

### 2.1 Study Design

Cross sectional non-randomized study was conducted to determine the prevalence of G6PD deficiency in Ogoni and Etche ethnic groups in Rivers State.

### 2.2 Study Area

The study was conducted within the Ogonis located at the south-East Nigeria, coast of the Gulf of Guinea, east of the city of Port Harcourt, River State. It extends across the local government area (LGA) of Khana, Gokana, Eleme, and Tai with a total population of over 500,000, living in a 404-square-mile (1,050 kilometres). They are surrounded by related ethnic groups such as Ibibio, Igbo, Ikwere, Ijaw, Efik, Ejagham and Annang; and the Etches located at the east region of the Rivers State South-South Nigeria with a total population of 351,200 living in area 805 kilometres density 436.3/km coordinates $4.991^0$N $7.05^0$E; they inhabits two local government area namely Etche and Omuma (LGA) surrounded by related ethnic groups such as Ikwere, Eleme, Abia State and Imo State.

### 2.3 Study Population

The G6PD deficiency individuals tested were people from Ogoni and Etche ethnic group, with study population of two hundred (200) subjects, One hundred (100) subjects from Ogoni and one hundred (100) subjects from Etche region. The research was conducted between March and August 2018.

### 2.4 Eligibility Criteria

The criteria for eligibility are that all the people from Ogoni and Etche are eligible, irrespective of age and gender. Non-Ogoni and Etche indigenes are totally excluded. Informed consent was obtained from subjects who were apparently healthy prior to enrolment. Ethical clearance was given by the Ethics Committee of the Department of Medical Laboratory Science, Rivers State University.

### 2.5 Sample Collection

Three (3) milliliters of blood were collected from each subject using a standard technique described for sample collection through venipuncture from the anticubital vein and dispensed into a labeled ethylenediaminetetracetic (EDTA) container capable of containing 10mls of blood with a concentration of 1.5mls of EDTA. The sample obtained was transported under heat-insulated envelopes and refrigerated stable transport container within 48 hours of collection to the Haematology Laboratory of Department of Medical Laboratory Science River State University and analyzed.

### 2.6 Sample Analysis

The method used for determination of glucose-6-phosphate dehydrogenase was by spectrophotometry at wavelength 340 nm was employed as described by RANDOX Laboratory Ltd.

The principle is based on the evaluation of absorbance at 340 nm given by NADH formation, the quantitative evaluation is made by adding a precise amount of haemolysate to an assay mixture containing the substrate G6P and its cofactor NADP, the rate of NADP generation is spectrophotometrically measured at wavelength of 340 nm and G6PD activity is expressed as G6PD 1Unit/RBCs and G6PD Unit/g haemoglobin ratio when measured at $37^0$C.

The procedure is as follow: 1 ml of reagent R1 was pipetted into test tube. Then 0.03ml of reagent R2 was added. Thereafter, 0.015ml of haemolysate (test) was added into the tube. It was mixed by inversion and incubated at $37^0$C for 5minutes. And 0.015ml of reagent R3 (enzyme substrate) was added to the test tube. It was mixed by inversion and allowed for 5minutes and finally read spectrophotometrically at wavelength 340 nm.

## 3. RESULTS

This study was carried out to determine the prevalence of Glucose-6-phosphate dehydrogenase (G6PD) deficiency among the Ogoni and Etche ethnic groups in Rivers State. A total number of 200 subjects were used in the study out of which 100 subjects were from Ogoni and 100 subjects were those from Etche. Out of the 100 subjects from Ogoni 49 were male and

51 were female while out of the 100 subjects from Etche 62 were male and 38 were female as shown in Table 1.

When G6PD level deficiency among Ogoni subject and Etche were compared the result obtained shows the Ogoni subjects had 6.21±0.34Unit/gHb and that of Etche subjects had 5.92±0.54unit/gHb with both having percentage deficiency of 9% and 13% respectively and the non-deficiency subject at the rate of 91% and 87% respectively with (P = 0.19, t = 1.35) which shows non-significant difference at (P = 0.05). The Prevalence Mean ± SD of G6PD deficiency in percentage base on location of Ogoni and Etche is shown in Table 2.

When glucose-6-phosphere dehydrogenase level of the test subjects from Ogoni were compared with that of Etche, the result obtained showed that the test of Ogoni had 9.092 ± 1.51 Unit/gHb and test of Etche had 9.365 ± 2.25 Unit/gHb (P = 0.317, t = 1.004), which shows no significant difference at (P<0.05). Table 3 Shows the Prevalence of G6PD Deficiency and Non-deficiency in Ogoni and Etche Ethnic Groups.

When G6PD level of the test subjects from Ogoni were compared with that Etche subjects, the result obtained shows that the Ogoni male had 6.21±0.32 Unit/gHb and test of Etche male had 6.0±0.54 Unit/gHb (P = 0.41; t = 0.85) which indicates no significant difference at (P< 0.05). Table 4 shows the comparison Mean±SD of G6PD Deficiency among Ogoni and Etche Male Subjects.

When G6PD level of the test subjects from Ogoni were compared with that of Etche subjects, the result obtained shows that the Ogoni female had 6.2±0.4 Unit/gHb and test of Etche female had 5.8±0.51 Unit/gHb (P = 0.44; t = 0.84) which indicates no significant decrease at (P< 0.05). Table 5 Shows the Comparison of Mean±SD of G6DP Deficiency among Ogoni and Etche Female Subjects.

**Table 1. Demographic detail of participants**

| Subject | Total No of subjects | No. of male | No. of female |
|---|---|---|---|
| Ogoni | 100 | 49 | 51 |
| Etche | 100 | 62 | 38 |
| Total | 200 | 111 | 89 |

**Table 2. Percentage distribution of G6PD deficiency among Ogoni and Etche ethnic groups**

| Subject | Non deficient | Deficient | Mean ± SD | % Deficiency |
|---|---|---|---|---|
| Ogoni | 91 | 9 | 6.21±0.34 | 9 |
| Etche | 87 | 13 | 5.92±0.54 | 13 |
| P-Value | | 0.190 | | |
| T-value | | 0.35 | | |

**Table 3. Prevalence of G6PD deficiency (DF) and non-deficiency (NDF) in Ogoni and Etche Ethnic groups**

| Subject | Total | No. of NDF | No. of DF | Mean ± SD |
|---|---|---|---|---|
| Ogoni | 100 | 91 | 9 | 9.092±1.51 |
| Etche | 100 | 87 | 13 | 9.365±2.25 |
| P-Value(0.05) | 0.317 | | | |
| T-value | 1.004 | | | |

**Table 4. Comparison of Mean ± SD of G6PD deficiency (DF) among Ogoni and Etche male subjects**

| Subject | Total male | DF male | Mean ± SD | % DF |
|---|---|---|---|---|
| Ogoni | 49 | 7 | 6.21±0.32 | 14.3 |
| Etche | 62 | 8 | 6.0±0.54 | 12.9 |
| P-Value | | 0.41 | | |
| T-value | | 0.85 | | |

**Table 5. Comparison of Mean ± SD of G6PD deficiency (DF) among Ogoni and Etche female subjects**

| Subject | Total female | No. of NDF | Mean ± SD | % DF |
|---|---|---|---|---|
| Ogoni | 51 | 2 | 6.2±0.4 | 3.9 |
| Etche | 38 | 5 | 5.8±0.51 | 13.2 |
| P-Value | | 0.44 | | |
| T-value | | 0.84 | | |

## 4. DISCUSSION

The present study was aimed at determining the prevalence of glucose-6-phosphate dehydrogenase deficiency among Ogoni and Etche ethnic groups in Rivers State. This study, out of the 100 Ogoni and 100 Etche subjects, 9% subjects recruited from Ogoni and 13% of subjects recruited from Etche were deficient of the enzyme with non-deficient rate of 91% and 87% respectively among the two groups.

In G6PD deficiency, the red blood cell breaks down when the body comes in contact with certain drugs and infection. This is as a result of lack of glucose-6-phosphate dehydrogenase, an enzyme that enables the physiological function of red blood cells [9]. Based on our finding in this study, individuals who are deficient of the G6PD enzyme may likely have issues with normal physiological function of their red cells if they are exposed to drugs and stressful conditions.

The study showed that out of the 49 male subjects and 51 female subjects from Ogoni, 42 males and 49 females are non-deficient while 7 males and 2 females are deficient with total of 9 subjects being deficient with P = 0.41 for male and P = 0.44 for female at (P = 0.05). In comparison with Etche subjects, the study shows that out of the 62 males subjects and 38 females subjects from Etche 54 males and 33 females are non-deficient while 8 males and 5 females making a total of 13 subjects being deficient, with P = 0.41 for male and P= 0.44 for female, which shows no significant difference at (P= 0.05).

This study was compared to the previous study carried out by [10] on Nigeria children. Where result obtained showed that the odd of G6PD deficiency were 3.6 times higher in males compared to females. This confirms that males were more affected than females as indicated by this study. The findings of this study indicates that G6PD enzyme defect is an X-linked chromosomal inheritance which affects more of male due to the inheritance of X chromosome as the only sex X chromosome while the female having two X-chromosome are less affected. The study also agreed with an earlier study [11] where males were found to have a high G6PD deficiency in the Niger Delta region of Nigeria.

Comparing our observation in this study to the meta-analysis carried out recently [12,13,14], G6PD deficient individual in this study may not have any advantageous protection against malaria as it was observed that subjects had in one time or the other suffered from malaria infection and some of them do take malaria medications that contains quinine even without prescription.

## 5. CONCLUSION

This study confirms that incidence of glucose-6-phosphate dehydrogenase (G6PD) deficiency is common among the Etche ethnic group than the Ogoni, although with no significant difference and occur more in male than in female of subjects from these two regions of the population study.

## CONSENT AND ETHICAL APPROVAL

Informed written consent was obtained from apparently healthy subjects prior to enrolment upon ethical clearance by the Ethics Committee of the Department of Medical Laboratory Science, Rivers State University.

## COMPETING INTERESTS

Authors have declared that no competing interests exist.

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