Frontiers in Bioscience Scholar

Front. Biosci. (Schol Ed) **2023**; 15(3): 11
https://doi.org/10.31083/j.fbs1503011

Original Research

Molecular Characterization of Erythrocyte Glucose-6-Phosphate Dehydrogenase Deficiency in Different Ethnic Groups of Blood Donors in Mauritania

Mariem Taleb[1,2], Meryem Bakour[1,3,†], Aminetou Taleb Brahim[2,4,†], Sidi Mohamed Ghaber[2,5], Seyid Abdellahi Ebnou Abdem[6], Aminetou Mohamed[2,4], Badiaa Lyoussi[1,*]

[1]Laboratory of Natural Substances, Pharmacology, Environment, Modeling, Health, and Quality of Life (SNAMOPEQ), Department of Biology, Faculty of Sciences Dhar El Mahraz, University Sidi Mohamed Ben Abdallah, 30000 Fez, Morocco
[2]Medical Analysis Laboratory (Maurilab), 2434 Nouakchott, Mauritania
[3]The Higher Institute of Nursing Professions and Health Techniques, 30000 Fez, Morocco
[4]Faculty of Sciences and Techniques, Department of Biology, Nouakchott University, 2373 Nouakchott, Mauritania
[5]Faculty of Medecine, Department of Basic Sciences, Nouakchott University, 2373 Nouakchott, Mauritania
[6]Center of Urban Systems (CUS), Mohamed VI Polytechnic University (UM6P), Lot 660, Hay Moulay Rachid, Ben Guerir 43150, Morocco
*Correspondence: lyoussi@gmail.com (Badiaa Lyoussi)
[†]These authors contributed equally.

Academic Editor: William Konigsberg
Submitted: 6 June 2023 Revised: 18 July 2023 Accepted: 17 August 2023 Published: 25 September 2023

Abstract

Background: Glucose-6-phosphate-dehydrogenase (G6PD) deficiency is the most frequent enzymopathy worldwide; it is a genetic disorder that affects red blood cells and causes hemolysis. Here, we conducted a study on G6PD-deficient subjects in Mauritania to evaluate the molecular characteristics associated with a deficiency in this enzyme and the frequency of nucleotide polymorphisms in the glucose-6-phosphate dehydrogenase gene. **Method and Materials**: A total of 943 blood samples were collected from blood donors (803 males and 140 females; 364 white Moors; 439 black Moors; 112 Pulaar; 18 Wolof; 10 Soninke). All blood samples were analyzed using a rapid screening test. G6PD status was analyzed quantitatively by the Randox G6PD test. Samples deficient in G6PD were extracted from the whole blood samples and subjected to DNA genotyping. The most frequent G6PD variants were determined by two molecular techniques: restriction fragment length polymorphism (RFLP) and multiplex PCR using the GENESPARK G6PD African kit. A total of six single nucleotide polymorphisms (SNPs) (*G202A*, *A376G*, *A542T*, *G680T*, *C563T*, and *T968C*) were identified. **Results**: The prevalence of G6PD deficiency in this population sample was 8.1%. The most common mutation was *A376G/202A* and was characterized by the G6PD A-phenotype, which is more common in the G6PD-deficient black Moors population. The wilaya in Nouakchott was the most affected among the 13 wilayas studied. **Conclusions**: This study shows, for the first time, the presence of the *G680T* mutation.

Keywords: G6PD; Mauritania; mutation; multiplex PCR; PCR-RFLP

1. Introduction

Enzymopathies are autosomal recessive or sex-linked genetic abnormalities [1]. Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most commonly known enzymopathy in humans and affects 400 million people worldwide. It is an X-linked recessive enzyme deficiency that can promote hemolysis in acute illnesses or after absorption of oxidizing drugs, including salicylates and sulfonamides [2].

The molecular defect in this pathology mostly results from an exon point mutation, which causes an amino acid substitution [3]. Until now, more than 200 various mutations in the *G6PD* gene have been identified, and more than 400 variants of glucose-6-phosphate dehydrogenase have been distinguished previously [4,5]. These are distributed among five classes, based on activity and clinical manifestation [6]. Deficient subjects are usually asymptomatic and may have crises when exposed to situations that cause oxidative stress.

The clinical phenotype of G6PD deficiency is associated with the relationship between genetic and environmental factors and the molecular properties of the patients' enzymes [7]. G6PD deficiency is frequently observed in Africa, the Middle East, the Mediterranean, and Southeast Asia, although it can be encountered across all geographical locations due to population movements [8].

In recent years, the rate of transfusion in Mauritania has increased. In addition, most of the patients receiving blood products are on medication, meaning the transfusion of a unit of blood from a blood donor deficient in G6PD can be ineffective. Therefore, the present study was designed to determine the frequency of G6PD deficiency in Mauritanian blood donors, to identify the different mutations existing in this population, to establish their prevalence, and to discern which Mauritanian wilaya G6PD is the most widespread.

Table 1. Prevalence of G6PD deficiency in the five ethnic groups in Mauritania.

Ethnic group	Gender		G6PD deficiency			Frequency of deficit (%)
	Male	Female	Male	Female	Total	
White Moors	284	80	14	3	364	4.67
Black Moors	397	42	48	0	439	10.93
Pulaar	97	15	9	1	112	10.30
Wolof	16	2	2	0	18	11.11
Soninke	9	1	0	0	10	0
Total	803	140	73	4	943	8.1

G6PD, glucose-6-phosphate-dehydrogenase.

Table 2. Prevalence of G6PD deficiency in both sexes.

G6PD	Females, n (%)	Males, n (%)	Total, N (%)
Hemi and homozygous	2 (1.43)	73 (9.1)	75 (8)
Heterozygous	2 (1.43)	0 (0.0)	2 (0.2)
Normal	136 (97.14)	730 (90.9)	866 (91.8)
Total	140 (100)	803 (100)	943 (100)

2. Materials and Methods

2.1 Studied Population

Between June 16 and September 30, 2019, blood samples were collected from 943 blood donors (803 male and 140 female); (364 white Moors, 439 black Moors, 112 Pulaar, 18 Wolof, and 10 Soninke). The participants were voluntary and occasional blood donors at the National Blood Transfusion Center in Nouakchott, Mauritania. Blood samples were collected, irrespective of gender or ethnicity, into ethylenediaminetetraacetic acid (EDTA) tubes, and stored at 2–6 °C, until processed, which was within 24 hours of collection.

All samples were first screened for transfusion-transmitted diseases, such as HIV, syphilis, and hepatitis B and C viruses. Data were collected from the participants through a questionnaire (age, sex, ethnicity, origin, transfusion, consanguinity of the parents, and degree of consanguinity of the parents). The participants were all of Mauritanian origin, phenotypically normal, and had never been transfused. Anyone not of Mauritanian origin was excluded from the study. All blood donors who participated in this study were adults. Informed consent was obtained from each participant after explaining the purpose of the study.

2.2 Methodology

A venous blood sampling was performed in an EDTA tube for all the samples collected. The blood samples were subjected, in the hours following the collection, to a rapid screening test, using the G6PD screening kit (REF PD 2616, RANDOX Laboratories Ltd, Kearneysville, WV, USA). Positive subjects were confirmed spectrophotometrically using the RX Monza kit (REF PD 410, RANDOX Laboratories Ltd). Samples with G6PD deficiency were subjected to DNA genotyping after their extraction from whole blood using the QIAGEN kit (REF 69506).

In this study, two molecular techniques were used: PCR-restriction fragment length polymorphism (RFLP) and multiplex PCR. Firstly, we performed PCR-RFLP, which is a reference method for identifying the two A variants (*A376G*) and A-(*A376G/202A*) in exons 4 and 5, respectively. Then, multiplex PCR was performed to confirm the PCR-RFLP results and identify further mutations in samples within the conclusive results. PCR-RFLP was performed on exons 4 and 5 to identify the A-variant, which is the most common in Sub-Saharan Africa, using two different primers: G6PD*A (*A376G*), 5′-CTGTCTGTGTCTGTCTGTC -3′ (forward) and 5′-GAGGGCAACGGCAAGCCTT -3′ (reverse), then, all samples positive for G6PD*A (*A376G*) were subjected to PCR amplification using primers for G6PD*A- (*G202A*): 5′-TACAGCTGTGCCCTGCCCT -3′ (forward) and 5′-CCGAAGCTGGCCATGCTGG -3′ (reverse) [9]. Verification was conducted using primer3 software for amplification conditions via https://www.primer3plus.com/.

PCR analysis was carried out for exons 4 and 5 to identify the A (*376G*) and A-(*G202A/376G*) variants. The PCR product was digested with the restriction enzymes FOK I and NLaIII, successively, using the protocol developed by Carter *et al.* [10]. The digested fragments, were subjected to electrophoresed on a 2.0% agarose gel (REF 4.658455). The rest of the samples were genotyped by multiplex PCR using the GENESPARK G6PD African kit (Immunospark, Rome, Italy) to detect the six single nucleotide polymorphisms (SNPs) (*G202A*, *A376G*, *A542T*, *G680T*, *T968C*, and *C563T*), which are the most common in Africa. PCR was performed using a reaction volume of 25 µL composed of 12.5 µL of PCR mix (2X), 2 µL of primer mix (G6PD African), 8.5 µL of sterile distilled water, and 2 µL of the fragment of DNA (50–100 ng) of each sample.

Multiplex PCR was carried out according to the manufacturer's recommendations, without enzymatic digestion.

Table 3. Prevalence of G6PD genotypes in both sexes.

	Females, n (%)	Males, n (%)	Total, N (%)
A (*376G*)	1 (25.0)	6 (8.2)	7 (9.1)
A-(*A376G/202A*)	1 (25.0)	48 (65.8)	49 (63.6)
Santamaria (*A376G/A542T*)	1 (25.0)	3 (4.1)	4 (5.2)
Betica-Selma (*A376G/T968C*)	0 (0.0)	5 (6.8)	5 (6.5)
G202A	0 (0.0)	5 (6.8)	5 (6.5)
G680T	0 (0.0)	1 (1.4)	1 (1.3)
MED (*C563T*)	1 (25.0)	5 (6.8)	6 (7.8)
Total	4 (100.0)	73 (100.0)	77 (100.0)

Table 4. G6PD genotypes in the five ethnic groups in Mauritania.

Ethnic groups	G6PD genotypes							
	A (*376G*)	A-(A*376G/202A*)	Santamaria (*A376G/A542T*)	Betica-Selma (*A376G/T968C*)	*G202A*	*G680T*	Med (*C563T*)	TOTAL, N = 77 (%)
White Moors	1 (1.3)	9 (11.7)	0 (0.0)	1 (1.3)	2 (2.6)	0 (0.0)	4 (5.2)	17 (22.1)
Black Moors	4 (5.2)	35 (45.5)	2 (2.6)	1 (1.3)	3 (3.9)	1 (1.3)	2 (2.6)	48 (62.3)
Pulaar	1 (1.3)	5 (6.5)	2 (2.6)	2 (2.6)	0 (0.0)	0 (0.0)	0 (0.0)	10 (13.0)
Wolof	1 (1.3)	0 (0.0)	0 (0.0)	1 (1.3)	0 (0.0)	0 (0.0)	0 (0.0)	2 (2.6)
Soninke	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
Total	7 (9.1)	49 (63.6)	4 (5.2)	5 (6.5)	5 (6.5)	1 (1.3)	6 (7.8)	77 (100.0)

The PCR products were separated on a 2.5% agarose gel by electrophoresis, and the results of the bands obtained after gel migration were interpreted using a table provided by the GENESPARK G6PD African kit (Immunospark, Rome, Italy).

2.3 Statistical Analysis

The data analysis and modeling were performed using the Python programming language version 3.7.3. (https://www.python.org/downloads/release/python-373/) with the following Python libraries: Numpy: for numeric calculations, mathematical operations, and matrix products; Pandas: to export, collect, and manipulate data; Matplotlib: for data visualization; Scipy: for statistical modeling more precisely inferential Statistics. The data is representative of samples from 943 individuals (people) and 7 statistical characters (variables). All these variables are nominal qualitative variables. The protocol used in this study can be separated into two steps:

Step 1: Univariate analysis: in this step, each variable was studied separately, and a descriptive statistic and a representation were acquired for this variable.

Step 2: Bivariate analysis: in this step, the dependencies between our variables were studied according to the object of the study.

The statistical analysis was performed using the Chi-Squared test of independence, and $p < 0.05$ was considered significant.

3. Results and Discussion

3.1 Sociodemographic Data and Prevalence of G6PD Deficiency in Mauritania

Due to its structure, the gene carrying the G6PD deficiency presents numerous genotypic variants [11]. The genotyping of six SNPs, which are the most common in Africa, and are involved in this hereditary disease, allowed us to evaluate the frequency of the different variants existing in blood donors in Mauritania [12].

It has been estimated that the prevalence of G6PD deficiency in Africa is 8.5% [13]. The results of our study determined that the prevalence of G6PD in the studied population was 8.1% (77/943) (Table 1). However, this result was lower than in two previously published Mauritanian studies, in 2018 and 2019, which found prevalence rates of 11.09% (58/523) and 11.03% (50/443), respectively [14,15]. This observed difference may be due to the choice of the population studied since in the first study, carried out in 2018, the researchers chose to define the epidemiological profile of G6PD deficiency in newborns in the city of Nouakchott [15]. Whereas, in the second study, carried out in 2019, the median age of the population was 30 (18–61; 12) years, and the ethnic groups presented in the study were white Moors, black Moors, black Africans, and mixed ethnic origins [14]. In addition, a comparison of our results with those found internationally and more specifically in African countries revealed that the prevalence of G6PD deficiency was less than in Burkina Faso (31%) [16], Congo (22.5%) [17], Mali (15.7%) [18], and Nigeria (ranged from 15.3% to 20%) [19–21]. The difference between our results and the results of other studies can be explained by the hypothesis that in the Mauritanian community, blood donation is not

Table 5. The percentage of mutations in each wilaya.

Wilayas	Type of mutations						
	A	A-	*A376G/A542T*	*A376G/T968C*	*G202A*	*G680T*	*MED*
Adrar	NF	6.25%	NF	NF	NF	NF	NF
Assaba	1.79%	10.71%	NF	NF	1.79%	1.79%	NF
Brakna	NF	5.36%	1.79%	0.89%	0.89%	NF	NF
Dakhlet Nouadhibou	NF	8.33%	NF	NF	NF	NF	NF
El HodhEcEcharghi	NF	2.13%	NF	NF	NF	NF	NF
El Hodh El Egharbi	NF	8.16%	NF	NF	NF	NF	NF
Gorgol	1.19%	4.76%	NF	1.19%	NF	NF	NF
Guidimagha	NF	3.03%	3.03%	NF	NF	NF	NF
Inchiri	NF	8.33%	NF	NF	NF	NF	NF
Nouakchott	1.21%	4.55%	0.30%	0.61%	0.61%	NF	1.52%
Tagant	NF	5.41%	NF	NF	NF	NF	NF
Trarza	0.76%	5.34%	NF	0.76%	0.76%	NF	NF
TirisZemmour	NF	8.33%	NF	NF	NF	NF	NF

NF, not found.

a common habit, especially among people who suffer from anemia, which explains why subjects with a deficit and who are symptomatic (anemic) will not donate blood [22].

The results of the molecular study showed heterogeneity in variants in blood donors. In this study, the A-variant was the highest in blood donors (Table 2). This incidence was consistent with the results of another study conducted in Mauritania that presented a percentage of 3.6% (36/996) for the A-variant [23], which was higher than the results obtained in a Senegalese study [24].

Mauritania is made up of two main ethnolinguistic groups: The first group is black Africans, which is composed of Pulaar, Soninke, and Wolof, each of which has its own dialect; the second group is the Moors, which are composed of white and black Moors, who speak "Hassaniya", a mixed language between Arabic and Berber [14]. The results summarized in Table 1 illustrate the presence of the five ethnic groups in the studied population: black Moors, represented by a percentage of 46.5%; white Moors (38.6%); Pulaar (11.87%); Wolof (1.90%); Soninke (1.06%). The prevalence of G6PD deficiency in the five ethnic groups was 11.11% in Wolof, 10.93% in black Moors, 10.30% in Pulaar, and 4.67% in white Moors. Concerning gender, 85.1% are men and 14% are women. The median age in the studied population was 30 years, while it ranged between 18 and 66 years.

The results presented in Table 2 show that G6PD deficiency is significantly higher in men than women (7.74%; 73/943) *vs.* (0.42%; 4/943) ($p = 0.020$), which is in agreement with previous results [25–27]. This is a consequence of the *G6PD* gene being X-linked. Moreover, hemizygous G6PD-deficient males are much more common than homozygous G6PD-deficient females [28].

The PCR-RFLP analysis detected the *A376G* and *A376G/202A* variants in 54 samples, prior to the rest being genotyped by multiplex PCR. The visualization of the PCR product via agarose gel electrophoresis identified six studied nucleotides with different percentages. The A-variant was the most common among them with a rate of 63.6% (49/77), followed by the A variant at 9.1% (7/77), the Med variant at 7.8% (6/77), the *G202A* mutation at 6.5% (5/77), the *A376G/T968C* (Betica-Selma) variant at 6.5% (5/77), the Santamaria variant (*A376G/A542T*) at 5.2% (4/77), and the rate of the G680T mutation was 1.3% (1/77), as shown in Table 3. This implies that the A-variant is the most dominant of the six SNPs found in our population, with a percentage of 45.5% (35/77) in black Moors and 11.7% in white Moors (9/77), as shown in Table 4. This mutation was the most common deficient variant in Sub-Saharan Africa [29]. The A-variant has been grouped by the World Health Organization as a "mild deficit" (Class III) [30] and variant A as a normal (Class IV phenotype) [31].

There is a significant correlation between the ethnic groups and the variant type ($p = 0.028$), which was also found in other studies [23,32]. G6PD deficiency was absent in the Soninke group; no mutation was found in this ethnic group, while the six SNPs were detected in black Moors.

3.2 Percentage of Mutations in Each Wilaya

According to the data obtained concerning the origins of the parents of the donors, the deficit in G6PD was observed in all the wilayas in Mauritania, with the highest frequency observed in the wilayas in Nouakchott, the capital of Mauritania. G6PD deficiency was found to have a very high prevalence in subjects whose parents were from the wilaya in Nouakchott (37.6%, 29/77) followed by 12.9% (10/77) in both Trarza and Brakna wilayas (12.9%, 10/77). Table 5 shows the prevalence of each mutation in the 13 wilayas, with the A-variant being the dominant mutation in Nouakchott. There was no correlation found between the type of mutation and the origin of the parents ($p = 0.97$).

Table 6. Frequency of consanguinity of parents in each ethnic group.

	White Moors	Black Moors	Pulaar	Soninke	Wolof
No consanguineous marriage	17.8%	27.1%	4.2%	0.5%	0.8%
Consanguineous marriage	20.8%	19.4%	7.6%	0.5%	1.1%

3.3 Frequency of Parental Inbreeding

The present study shows that 50.58% (477/943) of the parents in the studied population did not partake in a consanguineous marriage, while the rest were in a consanguineous marriage. The highest prevalence was 33.93% (320/943) among marriages of close first cousins (Fig. 1).

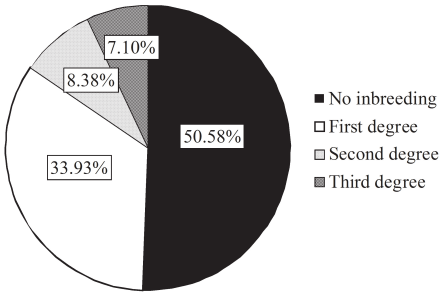

Fig. 1. Frequency and the degrees of parental inbreeding.

Consanguineous marriages were very high among white Moors, with a frequency of 20.8%; the lowest frequency was found among the Wolof ethnic group. A total of 50 people was found with a deficit among the 477 whose parents were not consanguineous and 27 people were shown to have a deficit among the 466 whose parents are cousins (Table 6). The correlation between the deficient in individuals and the consanguinity of the parents (p = 0.012) indicates that the two variables are dependent. In a study conducted by Hammami *et al*. [33], the results showed that consanguinity in Mauritania leads to infant and child mortality, especially in the Soninke and Pulaar ethnic groups.

4. Conclusion

This study confirmed heterogeneity in variants in the Mauritanian population, with the A-variant presenting as the dominant mutant. Moreover, it shows the presence of other mutations that also potentially exist in our population. G6PD deficiency is very common in donors of black Moorish ethnicity, whose parents originated from Nouakchott. These results are not surprising given that Mauritania is a mosaic of different populations due to its geographical location, which makes this country a crossing point for many civilizations.

Availability of Data and Materials

The data used to support the findings of this study are available from the corresponding author upon request.

Author Contributions

MT: data collection, blood analysis, writing original draft preparation, data analysis and interpretation. MB: writing original draft preparation, data analysis and interpretation. ATB: data collection and analysis. SMG: data analysis and interpretation. SAEA: statistical analysis. AM: supervision, designed the research study, writing—review and editing, data analysis and interpretation. BL: supervision, writing—review and editing, data analysis and interpretation, and submitted the manuscript. All authors read and approved the final manuscript.

Ethics Approval and Consent to Participate

All blood donors who participated in this study are adults. Informed consent was obtained by each participant after explaining the purpose of the study. The work was conducted in accordance with the Ethical Guidelines of the World Medical Association Declaration of Helsinki. The protocol was reviewed and approved by the institutional ethics committee of the Nouakchott University, Nouakchott, Mauritania (approval no. 00000062).

Acknowledgment

The research was performed as part of the employment of the authors within the MAURILAB laboratory..

Funding

Not applicable

Conflict of Interest

The authors declare no conflict of interest.

References

[1] Nadkarni A, Hariharan P. Molecular Techniques for Prenatal Diagnosis. Hematopathology: Advances in Understanding (pp. 469–482). Springer: Singapore. 2019.

[2] Anurogo D, Yuli Prasetyo Budi N, Thi Ngo MH, Huang YH, Pawitan JA. Cell and Gene Therapy for Anemia: Hematopoietic Stem Cells and Gene Editing. International Journal of Molecular Sciences. 2021; 22: 6275.

[3] Alakbaree M, Amran S, Shamsir M, Ahmed HH, Hamza M, Alonazi M, *et al*. Human G6PD variant structural studies: elucidating the molecular basis of human G6PD deficiency. Gene Reports. 2022; 27: 101634.

[4] Dore MP, Parodi G, Portoghese M, Pes GM. The Controversial Role of Glucose-6-Phosphate Dehydrogenase Deficiency

on Cardiovascular Disease: A Narrative Review. Oxidative Medicine and Cellular Longevity. 2021; 2021: 5529256.
[5] Luzzatto L, Nannelli C, Notaro R. Glucose-6-Phosphate Dehydrogenase Deficiency. Hematology/oncology Clinics of North America. 2016; 30: 373–393.
[6] Cappellini MD, Fiorelli G. Glucose-6-phosphate dehydrogenase deficiency. Lancet (London, England). 2008; 371: 64–74.
[7] Bancone G, Chu CS. G6PD Variants and Haemolytic Sensitivity to Primaquine and Other Drugs. Frontiers in Pharmacology. 2021; 12: 638885.
[8] Koromina M, Pandi MT, van der Spek PJ, Patrinos GP, Lauschke VM. The ethnogeographic variability of genetic factors underlying G6PD deficiency. Pharmacological Research. 2021; 173: 105904.
[9] Daoud BB, Mosbehi I, Préhu C, Chaouachi D, Hafsia R, Abbes S. Molecular characterization of erythrocyte glucose-6-phosphate dehydrogenase deficiency in Tunisia. Pathologie Biologie. 2008; 56: 260–267.
[10] Carter N, Pamba A, Duparc S, Waitumbi JN. Frequency of glucose-6-phosphate dehydrogenase deficiency in malaria patients from six African countries enrolled in two randomized anti-malarial clinical trials. Malaria Journal. 2011; 10: 241.
[11] Zheng Y, Wang J, Liang X, Huang H, Ma Y, Lin L, *et al*. Epidemiology, evolutionary origin, and malaria-induced positive selection effects of G6PD-deficient alleles in Chinese populations. Molecular Genetics & Genomic Medicine. 2020; 8: e1540.
[12] Ouattara AK, Yameogo P, Diarra B, Obiri-Yeboah D, Yonli A, Compaore TR, *et al*. Molecular Heterogeneity of Glucose-6-Phosphate Dehydrogenase Deficiency in Burkina Faso: G-6-PD Betica Selma and Santamaria in People with Symptomatic Malaria in Ouagadougou. Mediterranean Journal of Hematology and Infectious Diseases. 2016; 8: e2016029.
[13] Nkhoma ET, Poole C, Vannappagari V, Hall SA, Beutler E. The global prevalence of glucose-6-phosphate dehydrogenase deficiency: a systematic review and meta-analysis. Blood Cells, Molecules & Diseases. 2009; 42: 267–278.
[14] Djigo OKM, Bollahi MA, Hasni Ebou M, Ould Ahmedou Salem MS, Tahar R, Bogreau H, *et al*. Assessment of glucose-6-phosphate dehydrogenase activity using CareStart G6PD rapid diagnostic test and associated genetic variants in Plasmodium vivax malaria endemic setting in Mauritania. PLoS ONE. 2019; 14: e0220977.
[15] Mohamed GS, Lemine SM, Cheibetta S, Mohamed A. Neonatal screening for glucose-6-phosphate dehydrogenase (G6PD) deficiency in Mauritania. The Pan African Medical Journal. 2018; 30: 224.
[16] Meissner PE, Coulibaly B, Mandi G, Mansmann U, Witte S, Schiek W, *et al*. Diagnosis of red cell G6PD deficiency in rural Burkina Faso: comparison of a rapid fluorescent enzyme test on filter paper with polymerase chain reaction based genotyping. British Journal of Haematology. 2005; 131: 395–399.
[17] Bouanga JC, Mouélé R, Préhu C, Wajcman H, Feingold J, Galactéros F. Glucose-6-phosphate dehydrogenase deficiency and homozygous sickle cell disease in Congo. Human Heredity. 1998; 48: 192–197.
[18] Duflo B, Diallo A, Toure K, Soula G. Glucose-6-phosphate dehydrogenase deficiency in Mali. Epidemiology and pathological aspects. Bulletin de la Societe de Pathologie Exotique et de ses Filiales. 1979; 72: 258–264.
[19] Luzzatto L, Allan NC. Relationship between the genes for glucose-6-phosphate dehydrogenase and for haemoglobin in a Nigerian population. Nature. 1968; 219: 1041–1042.
[20] Nnamezie Igwilo H, Salawu L, Adeoye Adedeji T. The impact of glucose-6-phosphate dehydrogenase deficiency on the frequency of vasoocclusive crisis in patients with sickle cell anemia. Plasmatology. 2021; 15: 26348535211040528.
[21] Egesie OJ, Joseph DE, Isiguzoro I, Egesie UG. Glucose-6-phosphate dehydrogenase (G6PD) activity and deficiency in a population of Nigerian males resident in Jos. Nigerian Journal of Physiological Sciences: Official Publication of the Physiological Society of Nigeria. 2008; 23: 9–11.
[22] Hmida S, Boukef K. Transfusion safety in the Maghreb region. Transfusion Clinique et Biologique: Journal De La Societe Francaise De Transfusion Sanguine. 2021; 28: 137–142.
[23] Djigo OKM, Ould Ahmedou Salem MS, Diallo SM, Bollahi MA, Boushab BM, Garre A, *et al*. Molecular Epidemiology of G6PD Genotypes in Different Ethnic Groups Residing in Saharan and Sahelian Zones of Mauritania. Pathogens (Basel, Switzerland). 2021; 10: 931.
[24] Doupa D, Boye O, Ndiaye M, DjitÃ M, Kandji PM, Seck SM, *et al*. Prevalence of glucose-6-phosphate dehydrogenase (G6PD) deficiency (A-376/202) in homozygous sickle cell patients in Senegal and its clinical impact. African Journal of Biochemistry Research. 2018; 12: 55–62.
[25] Moosazadeh M, Amiresmaili M, Aliramezany M. Prevalence of G6PD deficiency in Iran, a mata-analysis. Acta Medica Iranica. 2014; 52: 256–264.
[26] Ouattara AK, Bisseye C, Bazie BVJTE, Diarra B, Compaore TR, Djigma F, *et al*. 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: 655–658.
[27] Kamali M, Mehralizadeh A, Sarvtin MT. Prevalence of Glucose-6-phosphate Dehydrogenase (G6PD) Deficiency in Jiroft City in Southern Iran. International Journal of Medical Laboratory. 2022; 9: 81–84.
[28] Domingo GJ, Advani N, Satyagraha AW, Sibley CH, Rowley E, Kalnoky M, *et al*. Addressing the gender-knowledge gap in glucose-6-phosphate dehydrogenase deficiency: challenges and opportunities. International Health. 2019; 11: 7–14.
[29] Williams O, Gbadero D, Edowhorhu G, Brearley A, Slusher T, Lund TC. Glucose-6-phosphate dehydrogenase deficiency in Nigerian children. PLoS ONE. 2013; 8: e68800.
[30] Glucose-6-phosphate dehydrogenase deficiency. WHO Working Group. Bulletin of the World Health Organization. 1989; 67: 601–611.
[31] Mura M, Saidi R, Wolf A, Moalic JL, Oliver M. Congenital hemolytic anemia due to glucose-6-phosphate dehydrogenase deficiency. Medecine Tropicale: Revue du Corps de Sante Colonial. 2009; 69: 551–555.
[32] Moradkhani K, Mekki C, Bahuau M, Te VLT, Holder M, Pissard S, *et al*. Practical approach for characterization of glucose 6-phosphate dehydrogenase (G6PD) deficiency in countries with population ethnically heterogeneous: description of seven new G6PD mutants. American Journal of Hematology. 2012; 87: 208–210.
[33] Hammami A, Chalbi N, Ben Abdallah M, Elgazzeh M. Effects of consanguinity and social factors on mortality and fertility in Mauritania. La Tunisie Medicale. 2005; 83: 221–226.