HIV and Glucose-6-Phosphate Dehydrogenase (G-6-PD) Deficiency among University Athletes in Rivers State, Nigeria

I. O. Okonko1*, S. Adewuyi-Oseni1, T. I. Cookey1 and K. C. Anugweje2,3

1Virus Research Unit, Department of Microbiology, University of Port Harcourt, Port Harcourt, Rivers State, Nigeria.
2Centre for Advanced Research in Sports Science, Sports Medicine and Sports Technology, University of Port Harcourt Sports Institute, Nigeria.
3International Association of Athletics Federations (IAAF), Regional Training Centre for High-Performance Elite Athlete Development, University of Port Harcourt, Port Harcourt, Nigeria.

Authors’ contributions

This work was carried out in collaboration among all authors. Author IOO designed the study and wrote the protocol. Authors IOO, TIC, SAO and KCA managed the analyses of the study and performed the statistical analysis. Authors IOO and SAO managed the literature searches and wrote the first draft of the manuscript. Author IOO supervised the whole study which, Miss C. J. Anisiobi (CJA) used as part of her B.Sc. Project in the Department of Microbiology, University of Port Harcourt, Nigeria. All authors read and approved the final manuscript.

Article Information

DOI: 10.9734/AJBGMB/2020/v4i430115

Editor(s): (1) Dr. Theocharis Koufakis, Aristotle University, Greece.
Reviewers: (1) Hassan Rafieemehr, Hamadan University of Medical Sciences, Iran.
(2) Mojtaha Rasouli Gandomani, Islamic Azad University Khorasgan (Isfahan) Branch, Republic of Iran.
Complete Peer review History: http://www.sdiarticle4.com/review-history/60147

Received 02 June 2020
Accepted 08 August 2020
Published 18 August 2020

ABSTRACT

**Aim:** Athletes are not immune to human immunodeficiency virus (HIV) and Glucose-6-Phosphate Dehydrogenase (G-6-PD) deficiency, and these conditions do not cause any harm or damage to their body as long as the necessary precautions in term of medications and others are adhered to. This research’s main objective was to determine the prevalence rate of G-6-PD deficiency and HIV among the University athletes in Rivers State, Nigeria.

**Study Design:** Cross-sectional study.
**Place and Duration of Study:** Sports Institute, University of Port Harcourt (UNIPORT), Nigeria, between June 2012 and July 2015.

**Methods:** A total of 258 athletes were screened (134 females and 124 males) for HIV and G-6-PD deficiency. The athletes’ ages, gender and dates of birth were obtained and recorded. The presence of the HIV-1 and HIV-2 antibody was detected using the Uni-Gold™ Recombigen® HIV-1/2 for the detection of HIV. While Beutler Semi-quantitative G-6-PD Test Kit (BSA-3000) was utilized for the quantitative detection of G-6-PD deficiency in whole blood.

**Results:** Of the 258 athletes tested, 0.7% was G-6-PD deficient while 1.2% was positive for HIV. The G-6-PD deficient positive cases were found in the age range of 21-25 years while the higher prevalence of HIV was observed in the age range 31-35 years (4.8%), followed by 26-30 years (1.9%) and the least was observed in the age group 21-25 years (0.7%). Age and gender had no significant relationship with the positivity of the athletes to HIV and G-6-PD (p-value >0.05).

**Conclusion:** This study confirms the presence of HIV and G-6-PD deficiency among university athletes in Rivers State, Nigeria. This calls for routine testing of both the athletes and the general public for G-6-PD deficiency to prevent hemolysis causes by G-6-PD deficiency.

**Keywords:** Athletes; HIV; G-6-PD deficiency; Uni-Gold; Nigeria.

### 1. INTRODUCTION

The most common enzymopathy that occurs due to mutation in humans, is the Glucose-6-Phosphate Dehydrogenase (G-6-PD) deficiency. This causes a series of disease such as acute hemolysis, neonatal hyperbilirubinemia, while some individuals with this deficiency may be asymptomatic. G-6-PD encoded by the G-6-PD gene on chromosome X is an important enzyme that plays the role of keeping red blood cells (RBCs) safe from oxidative damage [1]. G-6-PD is an enzyme of the pentose phosphate pathway that catalyses the oxidation of glucose-6-phosphate to 6-phosphogluconate. It also plays an important role in catalyzing the reduction of nicotinamide adenine dinucleotide phosphate (NADP⁺) to nicotinamide adenine dinucleotide phosphatase (NADPH) via the pentose phosphate pathway. Red blood cells depend on G-6-PD activity since it is the only source of NADPH that protects the cells against oxidative stress created by excess glutathione [2].

Mutation in the G-6-PD gene (which synthesizes G-6-PD enzyme) is responsible for G-6-PD deficiency. This mutation reduces the amount of G-6-PD enzyme or alters its structure in such a way the enzyme cannot perform its defensive role. By so doing, reactive oxygen species which are by-products of normal cell function begins to accrue and breakdown RBCs [3-4].

The G-6-PD gene responsible for encoding G-6-PD can be found at the long arm of the X chromosome (Xq28) consisting of 13 axons with a length of 18 kb [5]. The G-6-PD locus is polymorphic with almost 400 various alleles [6].

The World Health Organization (WHO) classified the alleles into 5 different variants. Based on the severity of enzymopathy and hemolytic response to oxidative stress these variants are grouped into 5 classes; Classes I to III have enzyme deficiencies associated with a hemolytic response, class IV have the “wild type” with no measurable difference, while class V is associated with an increase in enzyme activity. Therefore, only the first 3 classes are considered clinically relevant [7]. Even though most individuals with this condition are asymptomatic, factors such as several infections, some medications including various antimalarial drugs and fava beans ingestion can elicit acute hemolytic anaemia by exerting excess oxidative stress on the body [8]. Glucose-6-phosphate dehydrogenase (G-6-PD) deficiency affects approximately 400 million individuals globally [9-10]. WHO recommends the screening of the population in regions where G-6-PD deficiency prevalence is greater than 3-5% in males [7]. This is yet to be a routine check implemented by many places worldwide due to poor infrastructures and cost. Rate of prevalence is 15 to 26% in African regions with Sub-Saharan Africa has the highest prevalence due to its endemic malaria [10-11]. Information on its prevalence among athletes is very scanty. Due to paucity of information, it is important to detect Glucose-6-Phosphate Dehydrogenase (G-6-PD) deficient persons especially in areas where malaria and bacterial infections are pandemics before exposing them to oxidative stress to avoid an acute hemolytic attack. It is against this background that we examined G-6-PD deficiency among University athletes in Rivers state, Nigeria.
using a comparatively simple and cost-effective method with shorter incubation time.

HIV/AIDS persists as a health issue with millions of people affected around the world. Over the years since its discovery scientist in the medical community have learned more about HIV/AIDS, and improved its treatment helping people infected with the virus live longer and productive lives. Athletes are not exempted nor immune to the virus. Many athletes acquired the disease and are still active in sporting events. With the improvement in research and awareness about the history and treatment of this infection, people are now living a long and healthy way of life and sometimes engaging in athletic activities following protective guidelines [12]. This study aimed at determining the predominance of G-6-PD deficiency and HIV infection among the University athletes in Rivers State, Nigeria.

2. MATERIALS AND METHODS

2.1 Study Design

This was a cross-sectional study carried among athletes in the UNIPORT located in Choba community of Obio-Akpor Local Government Area, Rivers State, Nigeria. It is among the several tertiary institutions in Rivers State, Nigeria. Its layout has three campuses which are located at Choba Park, Delta Park, and University Park (Abuja Park). Ethical approval was sort from the Health Research and Ethics Committee of the University of Port Harcourt, Nigeria. The gender and age (15-35 years) of the athletes was obtained as the demographic factors.

2.2 Study Population

The survey was carried out in the University of Port Harcourt, Rivers State, Nigeria. The population comprising 258 athletes from the university – 134 females and 124 males.

2.3 Blood Sample Collection and Processing

The sampling technique used for drawing the samples in this study was the convenience sampling technique. Venepuncture technique was used for blood collection. The blood sample was then collected aseptically into EDTA bottle and taken to the Virus Research Unit, Department of Microbiology, University of Port Harcourt, Nigeria for the laboratory analysis.

2.4 Determination of HIV

The qualitative detection of HIV-1/HIV-2 antibodies present in the whole blood of the athletes was determined by utilizing immunoassay technique using Uni-Gold™ Recombigen® HIV-1/2 rapid test kit.

2.5 Determination of G-6-PD

G-6-PD deficiency was determined quantitatively [13] using Beutler Semi-quantitative G-6-PD Test Kit (BSA-3000), which is a semi-automated biochemistry analyzer with Randox kit. The enzyme activity was determined by measuring the rate of absorbance change at 340nm due to the reduction of NADP+. Results were displayed in mU erythrocyte/ml and then converted to U/Hb with the formula provided by the manufacturer below.

\[
G-6-PD \ U/Hb = \frac{\text{mU} \ \text{erythrocyte}}{\text{ml}} \times \frac{100}{\text{Hb} \ \text{g/dl} \times 1000}
\]

Both tests were conducted following the manufacturers’ instructions.

2.6 Eligibility Criteria

Both male and female university athletes within the ages of 16 and 35 years were included in this study except for non-athletes and athletes above 35 years.

2.7 Statistical Analysis

Data collected for this study were registered in the computer by creating a spreadsheet and subjected to statistical analysis using a statistical package. Comparisons were assessed using the chi-square test. Quantitative data were presented as percentages and a level of significance set at \( P < 0.05 \).

3. RESULTS AND DISCUSSION

3.1 Results

Of the 158 athletes screened for G-6-PD deficiency and the 100 screened for HIV, 1 was HIV positive and 1 was G-6-PD deficient. The athletes that were HIV positive and G-6-PD...
deficient were within the age range of 21 – 30 years. The association between sex and HIV status as with G6PG deficiency were not significant with a p-value of 0.31 and 0.39 respectively. The distribution rate of HIV was 1.2%, while that of G6-PE deficiency was 0.7% among the athletes concerning sex as seen in Table 1. Whereas, Table 2 shows the distribution of HIV and G6-PE deficiency of the athletes and their age.

### 3.2 Discussion

We screened a total of 258 athletes for HIV and G6-PE deficiency among University athletes in Rivers State, Nigeria. They consisted of both males and females between ages 16-30 years. There was a prevalence of 1.2% HIV and 0.7% G6-PE deficiency in total. Both cases were found among the females only. None of the females was positive for both HIV and G6-PE deficiency.

HIV affects both genders with no exception to their ages. It has been proven that young women within the age group of 15-49 have higher HIV prevalence than men within the same age group [14]. Also, in 2019, there was a record of 19.2 million young women as compared to 17 million prevalence rates in young men [15]. There are no extensive studies on bloodborne pathogens such as HIV and Hepatitis virus among athletes existing in Nigeria presently. One of the limitations of this study is that the HIV positive result was not confirmed either through western blot or PCR as such the positive case per chance is false positive.

The study of Akanni et al. [16] recorded a prevalence of 19.5% G6-PE deficiency among males in Osogbo state, Nigeria. Whereas Jidda et al, reported a prevalence of 2.4% and 22.2% G6-PE deficiency in females and males respectively among students in Sokoto State, Nigeria [17] while Ademowo and Falusi [18] reported a prevalence of 4.6% females and 23.9% males having G6-PE deficiency. This was however higher than the 0.7% G6-PE deficient females of our study. The difference in prevalence can be explained by the difference in the study population (athletes) and population size. Although G6-PE deficiency is linked only to the X chromosome, it is also a hereditary disease that affects mainly men. Females can also have a silenced mutated X chromosome in the majority of their cells. In the presence of triggering factors (certain foods like fava beans, some antibiotics and infections), females exhibit symptoms similar to a G6-PE deficient male such as hemolysis [19]. Thou, enzymatic tests for G6-PE deficiency sometimes gives a false negative result especially in females due to the random inactivation of the X chromosome and X-linked heterozygosity [20].

In our study, there was no subject with both HIV and G6-PE deficiency. However, there has been a prevalence between 6.8% and 13% of the HIV population having G6-PE deficiency [21-23]. The replication of HIV in itself has not been proved to clinically produce a trigger in G6-PE deficient patient, but the medications used in the prophylaxis and treatment of opportunistic infections in HIV patients are known to induce
hemolysis precipitation [21,24]. This is also an aspect that is understudied in Nigeria.

Individuals diagnosed with HIV are likely to inculcate athletic activities as they adapt to healthy lifestyles. As positive psychological and physical benefits of exercise have been proven to be beneficial in other health conditions such as fatigue, nausea, depression and anxiety in patients with, Hodgkin’s disease, cardiovascular disease, fibromyalgia, cancer and chronic fatigue syndrome [25-26]. This proves that exercising can have positive effects in individuals with HIV and so it does not have any adverse effect on athletes with this condition and risk of transmitting it to other athletes is near impossible as long as necessary precautions are taken.

According to the research of Stone et al. [27] which was based on G-6-PD deficiency in athletes, they concluded that G-6-PD deficient athletes are not physically limited. Rather, physicians should be mindful of potential problems, especially with medication [27].

4. CONCLUSION

In conclusion, the incidence of HIV and G-6-PD deficiency although low does exist among athletes. Having these conditions does not cause an impediment to exercise but instead, it encourages. More studies on bloodborne pathogens and enzymatic deficiencies among Nigerian athletes is needed.

CONSENT

All authors declare that written informed consent was obtained from the participants for the publication of this study.

ETHICAL APPROVAL

All authors hereby declare that all experiments have been examined and approved by the University of Port Harcourt Research Ethics committee and have, therefore, been performed following the ethical standards laid down in the 1964 Declaration of Helsinki.

ACKNOWLEDGEMENTS

The authors would like to acknowledge the support obtained from the management and staff of O.B. Lulu Briggs Medical Centre, the University of Port Harcourt and University of Port Harcourt Sports Institute during the enrollment and collection of samples used in this study. The authors also acknowledge the contributions of Miss C. J. Anisiobi (CJA) during the laboratory analysis which CJA used as part of her B.Sc. Project in the Department of Microbiology, University of Port Harcourt, Nigeria. The authors are grateful to the University athletes for their willingness to be part of the study.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

1. Prchal JT, Gregg XT. Red cell enzymes. Hematology. American Society of Hematology. Education Program. 2005; 19–23.
2. Mehta A, Mason PJ, Vulliamy TJ. Glucose-6-phosphate dehydrogenase deficiency. Baillieres Best Practice and Research: Clinical Haematology. 2000;13:21-38.
3. Steiner LA, Gallagher PG. Erythrocyte disorders in the perinatal period. Seminars in Perinatology. 2007;4:254-261.
4. Frank JE. Diagnosis and management of G-6-PD deficiency. American Family Physician. 2005;72(7):1277-1282.
5. Chen EY, Cheng A, Lee A, Kuang WJ, Hillier L, Green P, Schlessinger D, Ciccodicola A, D’Urso M. Sequence of human glucose-6-phosphate dehydrogenase cloned in plasmids and a yeast artificial chromosome. Genomics. 1991; 10(3):792-800.
6. Beutler E, Vulliamy TJ. Hematologically important mutations: Glucose-6-phosphate dehydrogenase. Blood Cells, Molecules & Diseases. 2002;28(2):93–103.
7. Glucose-6-phosphate dehydrogenase deficiency. WHO Working Group. Bulletin of the World Health Organization. 1989; 67(6):601–611.
8. Luzzatto L, Mehta A, Vulliamy TJ. (Glucose 6-phosphate dehydrogenase deficiency. In The Metabolic and Molecular Bases of Inherited Disease, 8th edition (Scriver CR, Beaudet AL, Sly WS, Valle D, Eds.), McGraw-Hill, New York. 2000;4517–4553.
9. Cappellini MD, Fiorelli G. Glucose-6-Phosphate dehydrogenase deficiency. Lancet. 2008;371(9606):64-74.
10. 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, and Diseases. 2009;42(3):267–278.

11. Howes RE, Piel FB, Patil AP, Nyangiri OA, Gething PW, Dewi M, Hogg MM, Battle KE, Padilla, CD, Baird JK, Hay SI. G-6-PD deficiency prevalence and estimates of affected populations in malaria-endemic countries: A geostatistical model-based map. PLoS Medicine. 2012;9(11):e1001339.

12. Clem KL, Borchers JR. HIV and the athlete. Clinics in Sports Medicine. 2007;26:413-424.

13. Shah SS, Diakite SAS, Traore K, Diakite M, Kwiatkowski DP, Rockett KA. A novel cytofluoro-metric assay for the detection and quantification of glucose-6-phosphate dehydrogenase deficiency. Scientific Report. 2012;2:29-30.

14. NACA. End of Term Desk Review Report of the 2010-2015 National HIV/AIDS Strategic Plan; 2015.

15. UNAIDS. Global facts sheet: AIDInfo; 2019. Available: http://aidsinfo.unaids.org/ [Accessed 22/07/2020]

16. 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.

17. Jidda M, Ibrahim K, Aiki G, Ngaski AA, Blessing J, Asiya U, Nwachukwu C. The Prevalence of Glucose-6-Phosphate Dehydrogenase (G-6-PD) Deficiency in Students of Sultan Abdurrahaman School of Health Technology Gwadabawa, Sokoto, North-Western Nigeria. International Blood Research & Reviews. 2017;7:1-6. DOI: 10.9734/IBRR/2017/31901

18. Ademowo OG, Falusi AG. Molecular epidemiology and activity of erythrocyte G-6-PD variants in a homogeneous Nigerian population. East African Medical Journal. 2002;79(1):42-45.

19. Luzzatto L. Glucose-6-phosphate dehydrogenase deficiency: From genotype to phenotype. Haematologica. 2006;91:1303-1306.

20. Beutler E. Glucose-6-phosphate dehydrogenase deficiency: A historical perspective. Blood. 2008;111(1):16-24.

21. Serpa JA, Villarreal-Williams E, Giordano TP. Prevalence of G-6-PD deficiency in a large cohort of HIV-infected patients. Journal of Infection. 2010;61:399–402.

22. Xu JZ, Francis RO, Lerebours Nadal LE, Shirazi M, Jobanputra V, Hod EA, Jhang JS, Stotler BA, Spitalnik SL, Nicholas SW. G-6-PD deficiency in an HIV clinic setting in the Dominican Republic. American Journal of Tropical Medicine and Hygiene. 2015;93:722–729.

23. Tungsiripat M, Drechsler H, Sarlone C, Amyot K, Laffey E, Aberg J. Prevalence and significance of G-6-PD deficiency in patients of an urban HIV clinic. Journal of the International Association of Physicians in AIDS Care. 2008;7:88–90.

24. Rapezz D, Porqueddu EM, Fenu L, Racchi O, Ferraris AM, Gaetani GF, Aceti A. Survival of people who are HIV-1-positive and G-6-PD-deficient is unaffected by virus-induced oxidative stress. Lancet. 1998;351:264–265.

25. Ciccolo JT, Jowers EM, Bartholomew JB. The benefits of exercise for quality of life in the post HAART era. Sports Medicine. 2004;34(8):487–99.

26. Burnham TR, Wilcox A. Effects of exercise on physiological and psychological variables in cancer survivors. Medicine and Science in Sports and Exercise. 2002;34(12):1863–1867.

27. Stone SN, Reisig KV, Saffel HL, Miles CM. Management of athletes with G-6-PD deficiency: Does missing an enzyme mean missing more games? Sports Health. 2020;12(2):149–153.

© 2020 Okonko et al.; This is an Open Access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Peer-review history:
The peer review history for this paper can be accessed here:
http://www.sdiarticle4.com/review-history/60147