Open Access Research Journal of
Chemistry and Pharmacy
Journals home page: https://oarjpublication/journals/oarjcp/
ISSN: 2783-0276 (Online)

(RESEARCH ARTICLE)

Antioxidant activity and levels of lipid peroxidation in glucose -6- phosphate deficient neonates

I. Akinlua *

Department of Biochemistry, Ekiti State University, Ado Ekiti, Nigeria.

Open Access Research Journal of Chemistry and Pharmacy, 2024, 05(01), 001–005

Publication history: Received on 09 August 2023; revised on 16 February 2024; accepted on 19 February 2024

Article DOI: https://doi.org/10.53022/oarjcp.2024.5.1.0072

Abstract

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is a genetic abnormality that results in inadequate amount of this all important protein that help regulate key physiological processes in the body. This study was carried out to evaluate plasma activity of antioxidants and level of lipid peroxidation in neonates with G6PD deficiency. A total of forty neonates delivered at Wesley Guide Hospital, Ilesa, Osun State were used for this study. They were divided into two groups; group 1 consists of 20 G6PD deficient neonates as the test while group2 consists of 20 G6PD positive neonates as control. 5mls of venous blood was taken from both test and control and prepared to obtain the plasma. The plasma was stored at room temperature prior analysis. The plasma activity of antioxidant enzyme [superoxide dismutase, catalase, glutathione, glutathione peroxidase (Gpx)], non-enzymatic antioxidant (vitamin E, vitamin C, beta-carotene) and plasma level of malonyldialdehyde (MDA) was carried out in both test and control using standard methods. The results of the study revealed no significant difference ($p<0.05$) in the plasma activity of most of the antioxidant enzymes with the exception of GPx which was significantly raised in the test subjects when compared with the control.. However, the plasma activity of both vitamin C and beta-carotene increased significantly ($p<0.05$) in the test when compared with the control. The plasma level of MDA was also significantly raised in the test when compared with the control group. This study revealed the presence of lipid peroxidation and possibly oxidative stress in G6PD deficient neonates.

Keywords: Neonates; Glucose -6- phosphate dehydrogenase; Antioxidants; MDA

1. Introduction

Glucose-6-phosphate dehydrogenase (G6PD) is a protein found in the cytoplasm and is responsible for the production of the reduced form of nicotinamide adenine dinucleotide phosphate (NADPH), a co-enzyme which maintains the intracellular pool of reduced glutathione (GSH) which protect cells from oxidative damage (Pes *et al*., 2019). The protein is coded for by the Gd gene which is located on the long arm of X chromosome and therefore follows a X-linked inheritance. G6PD deficiency may result from mutations to this gene that change the protein structure and therefore reduce its activity or the amount of enzyme produced (Lauden *et al*., 2019; Puthumana *et al*., 2019). G6PD deficiency therefore present a significant public health burden in certain region of the world especially in tropical Africa where the prevalence ranges from 15% - 26%. G6PD deficiency affects more than 200 million people round the world and is the most common human enzymopathy (Cappellini *et al.,* 2008). G6PD deficiency leads to neonatal jaundice often accompanied by hyperbilirubinemia which put infants at risk of kernicterus within the first few days of life, which is a cause for concern in some populations, especially in the Mediterranean countries and also in various racial groups in the Far East (Howes *et al.,* 2012). This condition further leads to hearing defects, behavioral problems and permanent neurologic damage in such infants.

Neonates with G6PD deficiency may appear healthy at first, but could later face the challenge of red blood cells hemolysis as a result of rapid imbalance in the redox status, following exposure to a pro-oxidants such as anti-malaria drugs substantially used in sub-Sahara Africa. Red blood cell hemolysis in such infants could lead to severe anemia,

* Corresponding author: I. Akinlua

heart failure and even death if not diagnosed early.. Challenge and barriers to screening include cost, underestimation of the public health impact of G6PD deficiency and lack of awareness of its deficiency among the people. Neonatal screening and health education can reduce the incidence rate of G6PD deficiency clinical manifestations.

Oxidative stress and non-immune hemolytic anemia could be a serious challenge for subjects with G6PD deficiency due to reduced level of cytosolic NADPH and reduced form of glutathione that protects red blood cells from oxidative damage (Prcha *et al.,* 2005). Oxidative stress itself results from an imbalance between increased level of oxidants and decreased antioxidant defenses in the body.

This study aimed to evaluate the activity of antioxidants and levels of lipid peroxidation in G6PD deficient neonates.

2. Material and methods

2.1. Participants

The subjects were twenty live birth neonates of healthy mothers delivered at the obstetrics and gynecology department of Wesley Guide Hospital in Ilesa, Osun State, Nigeria. They were divided into two groups. Group 1were ten G6PD positive neonates which serve as control while group 2 consists 10 G6PD deficient neonates which serve as the test group.

2.2. Data Collection

Informed consent was obtained from the mothers of the neonates used for this study after the study guidelines had been explained to them

2.3. Sample Collection and Processing

Venous blood samples of were collected from each of both test and control into heparinized bottles. The sample was centrifuged using a bucket centrifuge at 4000rpm for 3 minutes to obtained the plasma which was used for biochemical analysis.

2.4. Biochemical Analysis

Determination of Superoxide dismutase (SOD) activity.

2.4.1. Determination of Superoxide Dismutase(SOD) activity

Plasma activity of SOD was determined using the method of Ookawara *et al.* (1998)

2.4.2. Determination of catalase activity

Catalase activity was determined colorimetrically according to the methods of Sinha (1972).

2.4.3. Determination of Glutathione peroxidase activity.

Glutathione peroxidase activity was determined by the method described by Rotruck et al. (1973)

2.5. Estimation of reduced glutathione (GSH) activity

Reduced glutathione (GSH) activity was measured by the method of Anderson (1985)

2.6. Statistical analysis

Values were presented as mean ± SD. Data were analyzed using one-way analysis of variance (ANOVA). P-value less than 0.05 ($p<0.05$) was considered to be statistically significant.

3. Results

Table 1 Showing the result of plasma level of malondialdehyde (MDA) in both test and control.

3.1. Activity of MDA

Parameter	G6pd positive (control)	G6pd deficient group (test)	P-value
MDA (nmol/L)	1.31± 3.21	7.5±5.62	0.039*

MDA- Malondialdehyde, G6PD = Glucose-6-phosphate dehydrogenase; Values with superscript are significantly different *= p<0.05.

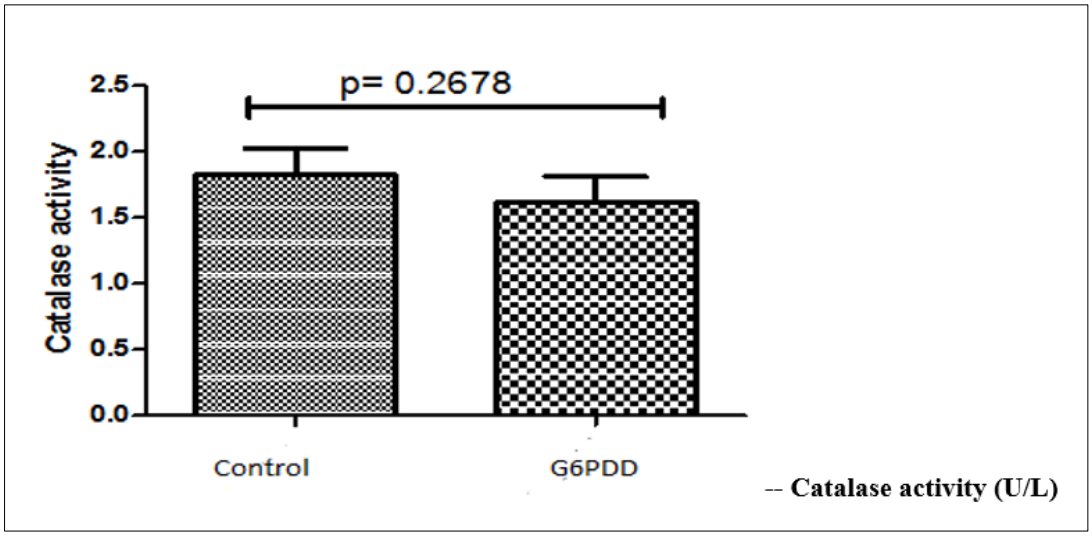
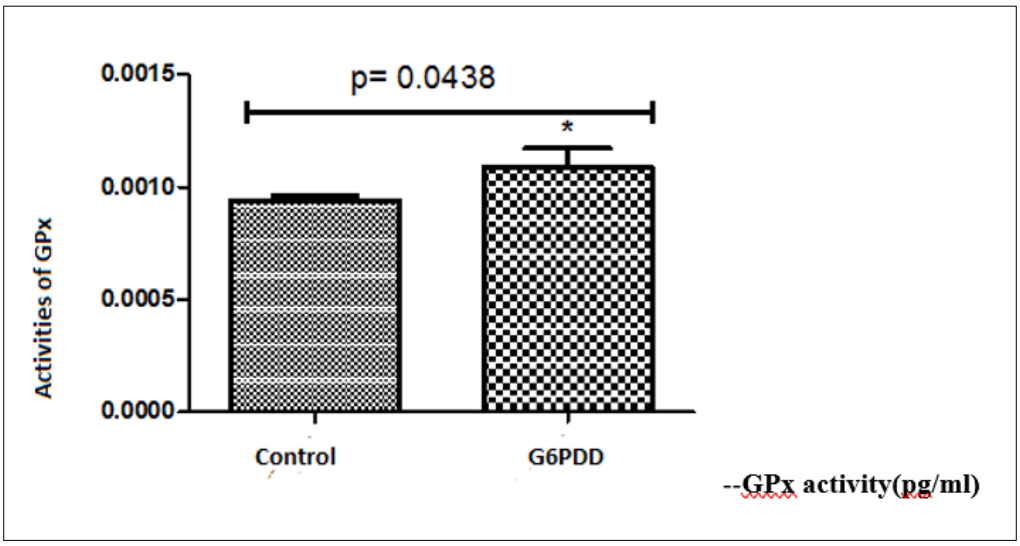


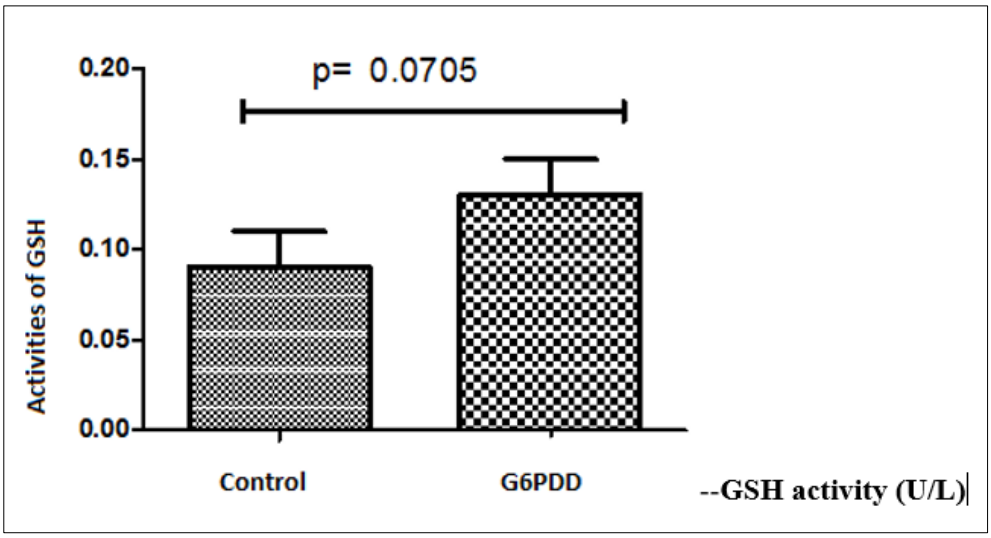
Figure 1 Result showing the activity of catalase in both G6PD deficient neonates and G6PD positive neonates (control)

G6PDD = Glucose-6-phosphate dehydrogenase deficiency



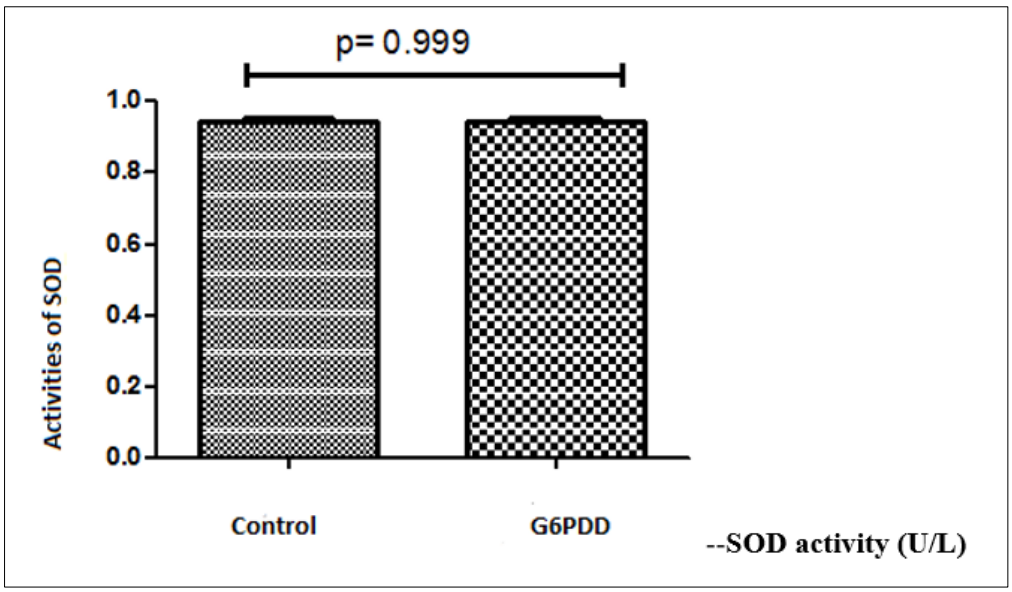
G6PDD = Glucose-6-phosphate dehydrogenase deficient neonates(Test)

Figure 2 Result showing the activity of glutathione peroxidase (GPx) both G6PD deficient neonates and G6PD positive neonates (control)



G6PDD = Glucose-6-phosphate dehydrogenase deficient neonates(Test)

Figure 3 The activity of reduced glutathione (GSH) both G6PD deficient neonates and G6PD positive neonates (control)



G6PDD = Glucose-6-phosphate dehydrogenase deficient neonates(Test)

Figure 4 The activity of superoxide dismutase (SOD) both G6PD deficient neonates and G6PD positive neonates (control)

4. Discussion

Glucose-6-phosphate dehydrogenase (G6PD) deficiency, will definitely affects some key physiological processes which are vital to the general wellbeing of the body. The enzyme is responsible for maintaining the physiological level of GSH which help the body against oxidative stress. The result of this study shows a significant increase ($p<0.05$) in the plasma level of MDA in G6PD deficient neonates while there is no significant difference ($p<0.05$) in the plasma activity of GSH and other antioxidants considered in this study when compared with that of G6PD positive neonates. The presence of high level of MDA in G6PD deficient neonates however indicates possible oxidative stress in these neonates which could be as a result of high production of free radicals which may not be matched with normal activity of antioxidants. The test and control used for this study are newborn whose body system has not fully matured functionally which might explain the insignificant difference in the plasma activity of antioxidants in both. However an earlier study Yasser *et al.*,

(2013) reported a decrease in the activity of SOD and presence of oxidative stress in subjects with G6PD deficiency which also agrees with the outcome of this study Swastika *et al.,* (2020) , Yasser *et al.,* (2013).

5. Conclusion

This study reveals the presence of oxidative stress in G6PD neonates considered. There is therefore need for expectant mothers to take food rich in antioxidants to improve the nutritional status of their unborn child which can possibly militate against oxidative stress especially in children born in regions susceptible to high occurrence of G6PD deficiency.

Compliance with ethical standards

Acknowledgments

I appreciate the contributions of Dr. A. T. Ogundajo and other staffs of the Chemical pathology department of Wesley Guide Hospital Obafemi Awolowo University Teaching Hospital, Ilesa, Osun State, Nigeria in the area for their contributions in the area of sampling and analysis.

Statement of ethical approval

Ethical approval for the study was obtained from the board of the hospital where the study was carried out.

Statement of informed consent

Informed consent was obtained from the mothers of the neonates used for this study after the study guidelines had been explained to them.

References

[1] Anderson M. E. Determination of glutathione and glutathione disulfide in biological samples.Methods Enzymol 1985; 113: 548-555.

[2] Cappellini M.D, Fiorelli G. Glucose6-phosphate dehydrogenase deficiency. Lancet, 2008; 5; 64-74.

[3] Howes R.E., Piel F.B., Patil A.P., Nyangiri O.A., Gething P.W., Dewi M, et al. G6PD deficiency prevalence and estimates of affected populations in malaria endemic countries: a geostatistical model-based map. *PLoS Med.* 2012; 9:e1001339. 10.1371/journal.pmed.1001339Swastika, M., Harahap, AR., Panggalo, LV. et al. Determining a critical threshold forG6PD activity below which red blood cell response to oxidative stress is poor. Malar J2020; 19, 208.

[4] Lauden SM, Chongwain S, Achidi A, Helm E, Cusick SE, Krug A, Slusher TM, Lund TC. Prevalence of glucose-6-phosphate dehydrogenase deficiency in Cameroonian blood donors. BMC Res Notes. 2019; 12(1):195. [PMC free article] [PubMed]]

[5] Ookawara T, Imazeki N, Matsubara O, Kizaki T, Oh-Ishi S, Nakao C, et al. Tissue distribution of immunoreactive mouse extracellular superoxide dismutase. Am J Physiol Cell Physiol. 1998; 275(3):C840-7. PubMed | CrossRef

[6] Pes GM, Parodi G, Dore MP. Glucose-6-phosphate dehydrogenase deficiency and risk of cardiovascular disease: A propensity score-matched study. Atherosclerosis. 2019; 282:148-153. [PubMed]

[7] Prchal J.T., Gregg X.T. Red Cell Enzymes. Hematol, 2005; 1: 19-23.

[8] Puthumana JS, Regenold WT. Glucose-6-phosphate dehydrogenase activity in bipolar disorder and schizophrenia: Relationship to mitochondrial impairment. J Psychiatr Res. 2019; 112:99-103. [PubMed]

[9] Rotruck, J.T., Pope, A.L., Ganther, H.E., Swanson, A.B., Hafeman, D.G. and Hoekstra, W.G. Selenium: Biochemical Role as a Component of Glutathione Peroxidase. Science, 1973; 179, 588-590. https://doi.org/10.1126/science.179.4073.588

[10] Sinha, A.K. Colorimetric Assay of Catalase. Analytical Biochemistry, 1972; 47, 389-394. https://doi.org/10.1016/0003-2697(72)90132-7

[11] Yasser E.N., Hanan A.F., Alaa A., Manal A.H., Gihan A.F. Evaluation of G6PD activityand antioxidants status in jaundiced Egyptian neonates. International Journal of Medicine and Medical Sciences. 2013; Vol. 5(12), 550-559.