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Review Article

Enzyme Glucose-6-Phosphate Dehydrogenase Deficiency And Its Effect On Human Health

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ABSTRACT

Not everyone has this disease, but those whose blood cells were deficient in an enzyme called (G6PD) Glucose 6 phosphate Dehydrogenase have this disease. This disease is present in those infected since birth, but its symptoms may not appear until years after the child's age. The disease symptoms appear if the patient eats peas, whether Green, cooked, or in any other form. The disease may occur even if the patient passes through a land planted with peas or inhales its flower. Some cases appeared in the baby after his mother ate the peas for transmission through milk to the child. It should be warned that eating falafel is also dangerous for these patients, as they usually mix the peas with chickpeas when preparing it. The disease, as we have said , is transmitted to the child genetically and is present in many countries such as the Americas, the Middle East , Greece, China, and other parts of the world, but it is rare in the countries of northern Europe, and what the bacillus does is affect the wall of the erythrocyte, weakening it by the absence of the enzyme Glucose 6 phosphate Dehydrogenase, the erythrocyte breaks and by breaking it, the child gets severe anemia, showing signs of pallor (anemia) and jaundice (as a result of breaking blood cells), and upon clinical examination of the patient's diuresis, we see it red in color as a result of blood coming out with it, And that the breakdown of blood cells may be rapid in one day, the child feels dizzy and throws and his strength weakens and his body turns pale and yellow, that is, if we look closely at the child, we will see two colors in tandem: the color of jaundice (yellowness) resulting from the rise of bile due to the breakdown of blood cells and their lack in the blood, and the breakdown of blood may be slow over a period of 5 – 4 days, the child does not suddenly weaken here, but he shows signs of heaviness in playing, lack of appetite and pain in some areas of the body.in both cases, the patient needs to be admitted to the hospital to give blood instead of his lost broken blood. the disease should not be underestimated, because if the disease is severe and is not treated in time, it may lead to death. god forbid, here it must be warned that people with this deficiency in their

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enzymes are not harmed by eating peas only, but there is a list of drugs that may cause hemolysis and break it down in them.

INTRODUCTION

Bacillus is unique in its rapid effect on the decomposition of erythrocytes when ingested by some people with special sensitivity to it, which leads to severe and dangerous anemia in children in particular and this phenomenon is called Bacillus allergy disease (Fauvism), The reason for this is an enzyme deficiency (G6PD) Glucose -6 – phosphate Dehydrogenase Which is one of the most important enzymes present in the human body[1-4]. This disease is one of the types of genetic diseases for which there is no cure yet, affecting more than 400 million people being aected worldwide, as it is transmitted by heredity from one generation to another, so the family should pay attention when feeding their children and investigate the presence of an infection in one of their members and to be more sure, a simple test can be done in the laboratory to make sure that there is no deficiency of this enzyme in the body[5-8]. The enzyme (G6PD) works to provide the full ability of the cell to protect red blood cells from decomposition and thus it preserves them from external influences, but when there is a shortage of this enzyme, the presence of some glucosidic compounds in the rest, such as Vicine and Convicine, will lead to an impact on the walls of red blood cells and thus their decomposition, As a result , the patient has some cases through which it is possible to know the occurrence of the infection, such as jaundice (yellowness) caused by high bile, as well as the color of pallor due to the breakdown of red blood cells and its lack, and the breakdown of red blood cells may be slow lasting for 5-4 days and its symptoms do not appear directly, and this is due to the variation in the occurrence of enzyme deficiency from case to case[1,2,5,6]. Therefore, the patient in this case should be admitted to the hospital not to give replacement blood to compensate for the shortage of decaying erythrocytes and the disease should not be underestimated when it occurs because there are some cases whose neglect and failure to treat them in time led to death, In addition, there are some medications that should be refrained from being taken by people with this type of allergy,which have a similar effect to the compounds contained in the bacillus, such as (aspirin, sulfate compounds and primacon), so the patient must tell the attending physician about the presence of an allergy to his Bacillus so that the attending physician can prescribe an alternative drug, as is the case for people with penicillin allergy[9,10].

It was noted in one study that anemia resulting from the decomposition of erythrocytes in (10-5) % of American Negroes occurs when taking the antimalarial drug PREMAQUIN , where the severity of anemia depends on the dose of PREMAQUIN. And that there are many drugs besides PREMAQUIN cause erythrocytolysis for people suffering from a deficiency of the enzyme (G6PD), and cases of erythrocytolysis have been recorded if a person suffering from a deficiency of the enzyme (G6PD) ingested Bacillus, as the defect in the (Gene) the (G6PD) is distributed in different regions of the world, namely Central Africa, the Mediterranean region, the Middle East, India and Southeast Asia, where malaria has been endemic in these regions for several generations [3,5,7,11].

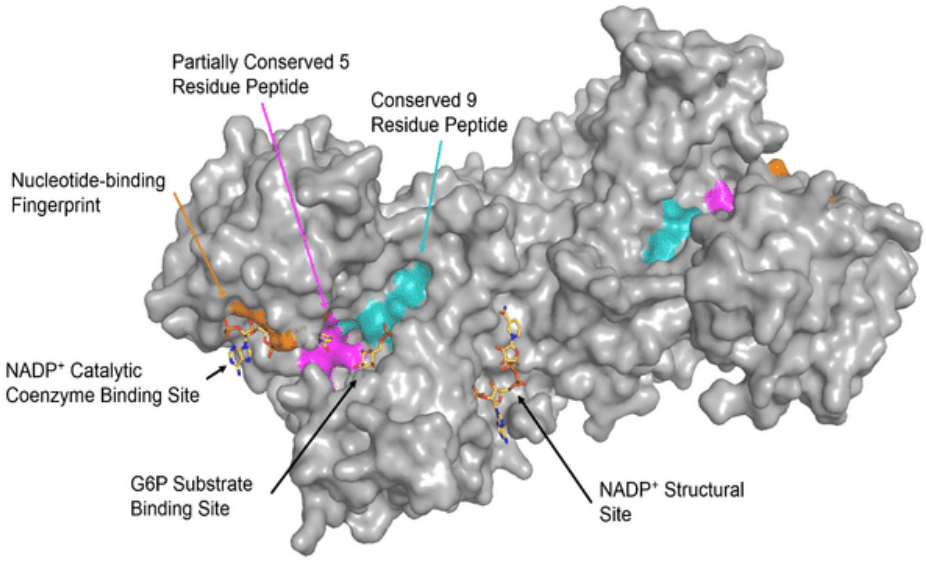

Figure 1. Glucose-6-phosphate dehydrogenase (G6PD)[12].

Aim of the study:

To determine the useful hematologic indicators of hemolysis, observe an early detection of G6PD enzyme deficiency (if any), and the available therapeutic measures.

MECHANISM OF ENZYME ACTION (G6PD):

The enzyme (G6PD) is one of the most important metabolic enzymes as a key to the pentose cycle Form Number (2) , as this enzyme oxidizes (Glucose 6 phosphate) to (6 – phosphorus – 8 – lactone) and reduces (NADP+) Nicotinamide adenine dinucleotide to (NADPH) Nicotinamide adenine dinucleotide phosphate hydrogen , then it decomposes (6 – phospho – 8-lacton) and the carboxyl group (- COH) is removed from it to produce (Ribulose – 5 – phosphate), which is necessary for the synthesis of the DNA strip form No. 3, so the enzyme (G6PD) plays an important role in regulating the level of (Ribulose-5 – phosphate is necessary for the synthesis of the DNA strand and cell growth and the enzyme (G6PD) maintains the cell through its production of (NADPH) necessary for reductive reactions and protection against oxidation force, and Figure 4 shows the role of the enzyme (G6PD) in the conversion of (NADP+) to (NADPH)[1,13,14].

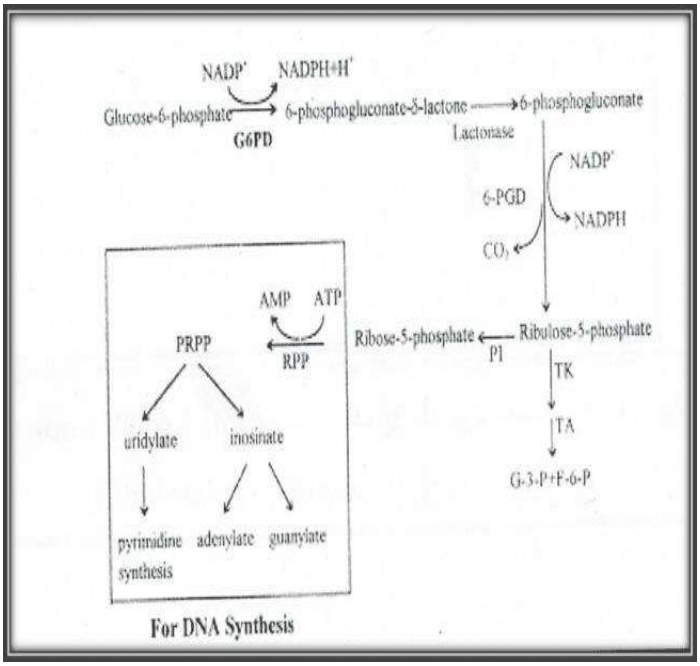

Figure 2. The pentose cycle in the cell and the importance of the enzyme (G6PD).

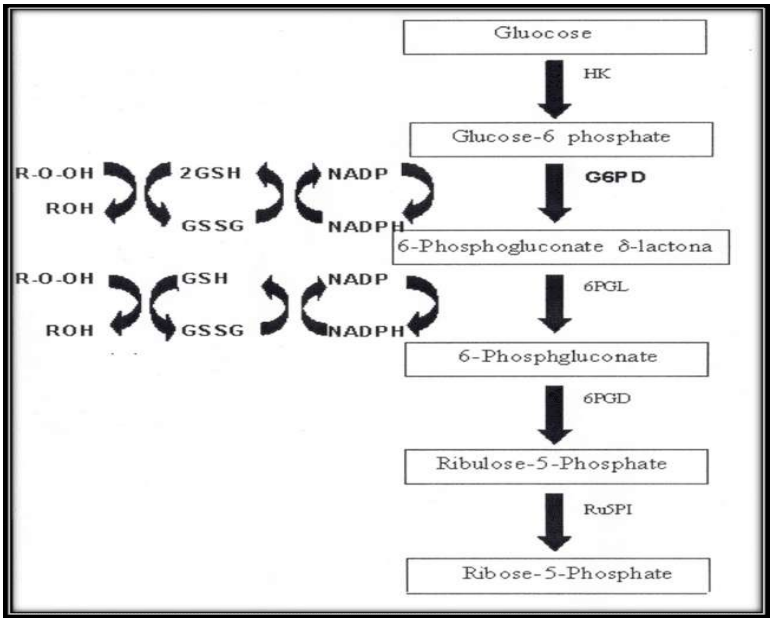

Figure 3. The importance of the enzyme (G6PD)by the oxidation of (Glucose - 6 – phosphate) in the cell and the production of (Ribulose-5-phosphate).

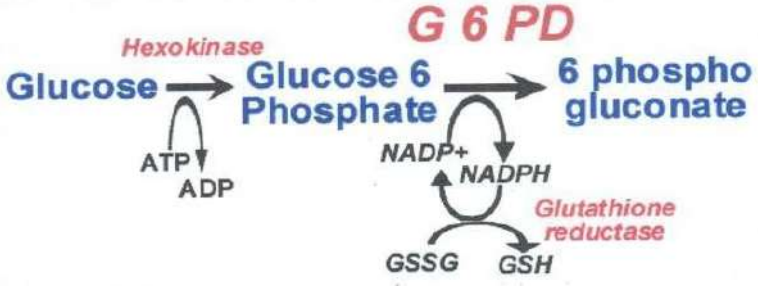

Figure 4. The role of the enzyme (G6PD) in the conversion of (NADP+) to .(NADPH).

Enzyme Glucose-6-phosphate dehydrogenase(G6PD) and anti-oxidant defence:

Glucose 6-phosphate (G6P) is converted to ribulose-5-phosphate (R5P), where NADP+ is reduced to NADPH through G6PD and 6-phosphogluconate dehydrogenase (6PGD) enzymes. NADPH is a major cellular reductant involving in the balance between oxidized glutathione (GSSG) and reduced glutathione (GSH) as redox couple system (Figures 3,4). GSH is a major antioxidant molecule involving in the various cellular processes such as detoxification, anti-oxidant defence mechanism and cell proliferation through maintaining the intracellular redox homeostasis.

Enzyme classification (G6PD):

The enzyme (G6PD) was classified by the enzyme Classification Committee of the scientific Union of chemistry as one of the enzymes of the oxidation-reduction group (Oxidoreductase) and was given the systematic designation .(EC :1.1.1.49) [13,15,16].

Form and structure of the enzyme (G6PD):

The enzyme molecule (G6PD) exists in two effective forms, the binary form (Dimer) and the Quaternary form (Tetramer) and these forms are determined by the influence of PH and ionic strength depending on the surrounding conditions , as the high ionic strength and the base ocean increase the presence of the binary form while the acidic ocean and the high ionic strength increase the presence of the Quaternary form, it was noted that the fixed form of the enzyme molecule is the Quaternary form and through electron microscope analysis studies, it was found that the Union of two molecules of the enzyme (G6PD) dimeric (dimeric) for the formation of the Quaternary form and the decomposition of the latter into the binary form again takes place very quickly not exceeding fractions of a second , Therefore, if the enzyme is found in a certain environment where the formed NADPH is not oxidized quickly, this will lead to the decomposition of the Quaternary form into the Quaternary form and then the monoclinic, and it was concluded that the ionic bonds are the basis for the stability of the double form inside the Quaternary form of the enzyme, while the hydrophobic bonds maintain the stability of the ionization and PH on the synthetic form of the enzyme (G6PD) extracted from human erythrocytes, And that the enzyme molecule purified from human blood contains about (18) sulfhydryl group (-SH), and that the enzyme contains the amino acid (Lysine) in its active position sequentially between the amino acid (100 – 93) in the enzyme molecule, and the group (-amino) of this acid forms the binding point between (NADP+) and the base substance (G6P), and the enzyme is of the type glycoproteins (Glycoproteins Figure 5 shows the three-dimensional structure of the enzyme (G6PD) purified from various sources [2,11,17].

The applied importance of the enzyme (G6PD):

The enzyme (G6PD) is one of the most important enzymes and is used in the field of medical and laboratory analyzes due to the many properties of this enzyme that have earned it that importance, as the enzyme is used in many quantitative estimation methods, such as its use in estimating the enzyme (Hexokinase) and estimating the compound (ATP) adenosine triphosphate, it is also used in estimating hexoses (hexoses) as an estimate of glucose , sucrose, lactose and raffinose, and in the field of food industries enzyme in glucose estimation to detect legal sugar additives to fruit juice and wine products, The enzyme is also used in the estimation of starch (Starch) after converting it into units of glucose by the enzyme (Gluco amylase) in addition to its use in the estimation of glycogen (Glycogen) , the enzyme was also used in the preparation of enzyme Electrode enzyme electrodes for the detection of glucose sugar in the blood and administration for people with diabetes

, and the enzyme (G6PD) acquires medical importance through its use in the investigation of the effect of oxidative drugs and studying their effect on humans , the enzyme is also used in detection of biotin and avidin

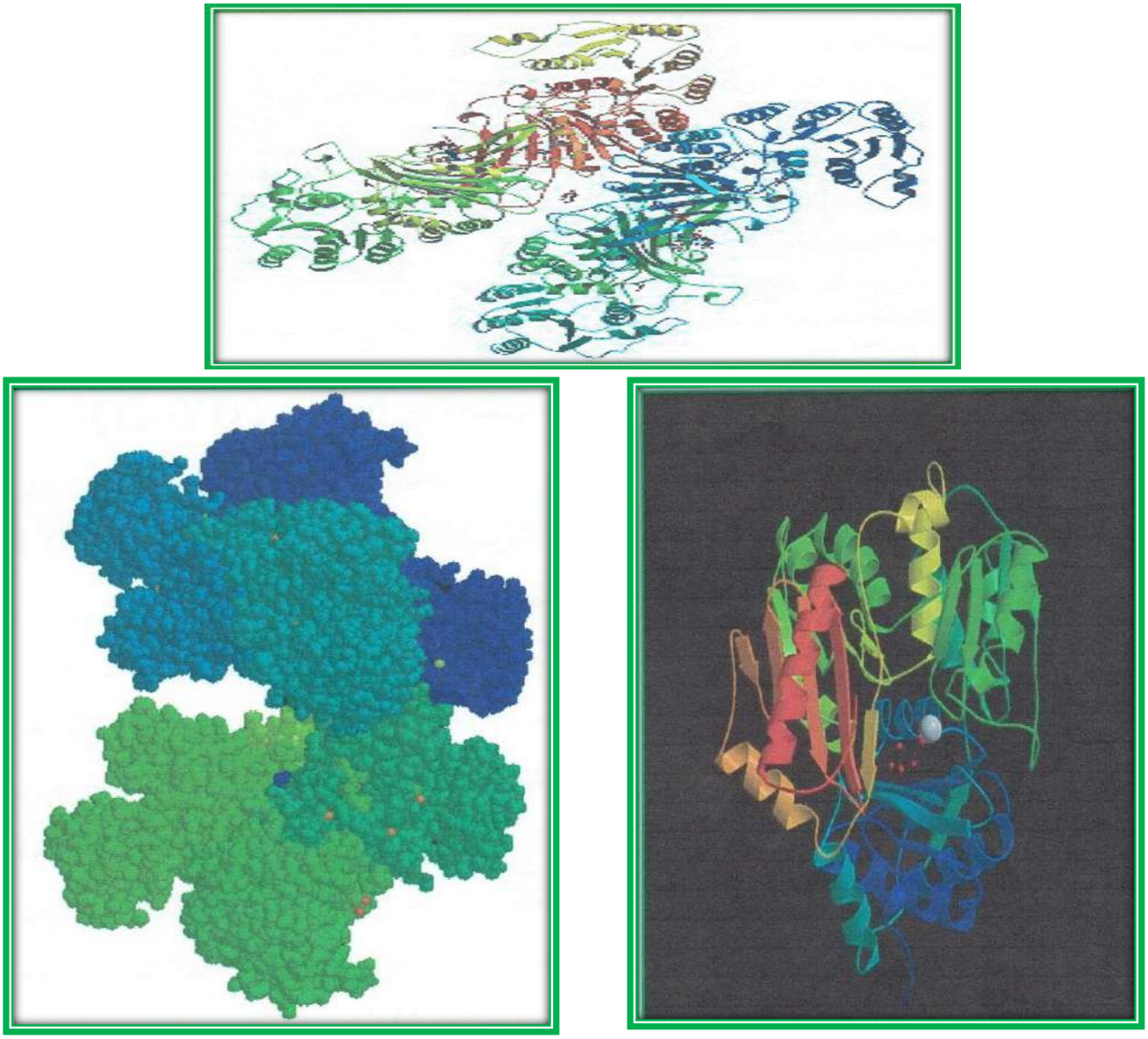

Figure 5. Three-dimensional structure of the enzyme (G6PD) purified from various sources

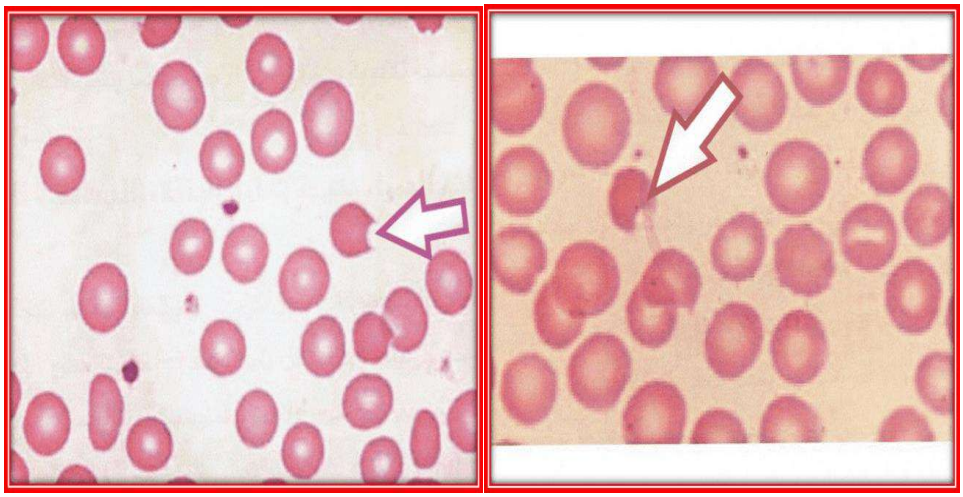

Figure 6. Erythrocyte lysis as a result of a deficiency of the G6PD enzyme[17].

MATERIALS AND METHODS

This review was made possible by conducting a search of the literature available in the following databases: Scopus, journals, PubMed, and Google Scholar. The keywords used were glucose-6-phosphate dehydrogenase (G6PD), deficiency, disease, exercise, and antioxidants. The research was limited to English-language publications, conducted on humans.

RESULTS AND DISCUSSION:

The high risk for G6PD deficiency disease result from great lack of this biochemical enzyme in the red blood cells. It is known that glucose-6-phosphate dehydrogenase catalyzes the pentose phosphate pathway in carbohydrate metabolism, so any biological and chemical disorder in this enzyme will lead to different complications in the physiological role belonging to red blood cells (RBC). Some people are exposed to serious breakdown of red blood cells (RBC) as a result of eating uncooked or partially cooked beans. This defect occurs only in cases of enzyme deficiency (G6PD). It most often occurs in the western Mediterranean basin, but not all of them suffer from a deficiency of the enzyme (G6PD). (G6PD) are susceptible to hemolytic anemia if they eat beans. In addition to the decrease in the activity of the enzyme (G6PD), there is evidence that there is another factor, which is the genetic factor, for the occurrence of severe lysis of red blood cells (RBC) as a result of eating beans.

CONCLUSIONS:

The present study was established to investigate and estimate some enzymatic and non-enzymatic antioxidants that have a biochemical relationship with G6PD deficiency disease. It was found that G6PD concentrations significantly decreased in favism patients. Catalase as an enzymatic antioxidant proved the biochemical correlation between this enzyme with G6PD deficiency therefore CAT has important action for investigation of the severity of favism disease. GPT had no effect on the lack of G6PD enzyme in both males and females. Non-enzymatic antioxidants represented by vitamins A and E indicated a great relationship with G6PD deficiency in blood serum for male and female patients. In conclusion, catalase, vitamins A, and E are considered important markers for diagnosis of favism disease severity.

RECOMMENDATIONS:

The Screening of all family members of affected patients for enzyme status to prevent acute hemolysis and inform the families of affected patients about the types of food and drugs that can cause hemolysis. Great need for newborn screening for early diagnosis of enzyme deficiency (G6PD).

REFERENCES:

1. Mehta A, Mason PJ, Vulliamy TJ (2000) Glucose-6-phosphate dehydrogenase deficiency. Best Practice & Research Clinical Haematology 13: 21-38.
2. Beutler E (2001) Glucose-6-phosphate dehydrogenase deficiency and other red cell enzyme abnormalities. Williams hematology 8: 647-674.
3. Nkhoma ET, Poole C, Vannappagari V, Hall SA, Beutler E (2009) The global prevalence of glucose-6-phosphate dehydrogenase deficiency: a systematic review and meta-analysis. Blood Cells, Molecules, and Diseases 42: 267-278.
4. Khalaf DK, ALsaadi LAK, Lkreeshy AK (2018) Age of Spontaneous Regeneration of Glucose-6-Phosphate Dehydrogenase Among Children in Baghdad. Iraqi Postgraduate Medical Journal 17.
5. Georgakouli K, Fatouros IG, Draganidis D, Papanikolaou K, Tsimeas P, et al. (2019) Exercise in glucose-6-phosphate dehydrogenase deficiency: Harmful or harmless? A narrative review. Oxidative Medicine and Cellular Longevity 2019.
6. Frank JE (2005) Diagnosis and management of G6PD deficiency. American family physician 72: 1277-1282.
7. Demir AY, van Solinge WW, van Oirschot B, van Wesel A, Vergouwen P, et al. (2009) Glucose 6-phosphate dehydrogenase deficiency in an elite long-distance runner. Blood, The Journal of the American Society of Hematology 113: 2118-2119.
8. Al-Toumaa FJ, Frankoolb WM (2010) Incidence of Erythrocyte Glucose-6-Phosphate Dehydrogenase Deficiency in Male Neonatal Hyper-bilirubinemia in Kerbala Region: Iraq. KUFA MEDICAL JOURNAL 13.
9. La Vieille S, Lefebvre DE, Khalid AF, Decan MR, Godefroy S (2019) Dietary restrictions for people with glucose-6-phosphate dehydrogenase deficiency. Nutrition reviews 77: 96-106.
10. Alalh NA (2012) Study of relationship of Glucose-6-Phosphate Dehydrogenase deficiency and its effect on neonatal jaundice. Al-Anbar Journal of Veterinary Sciences 5.
11. Cappellini MD, Fiorelli G (2008) Glucose-6-phosphate dehydrogenase deficiency. The lancet 371: 64-74.
12. Özcan E, Selvi SS, Nikerel E, Teusink B, Toksoy Öner E, et al. (2019) A genome-scale metabolic network of the aroma bacterium Leuconostoc mesenteroides subsp. cremoris. Applied microbiology and biotechnology 103: 3153-3165.
13. Aydemir D, Ulusu NN (2020) The possible role of the glucose-6-phosphate dehydrogenase enzyme deficiency in the polyneuropathies. Journal of Basic and Clinical Health Sciences 4: 212-217.
14. Dore MP, Parodi G, Portoghese M, Pes GM (2021) The controversial role of glucose-6-phosphate dehydrogenase deficiency on cardiovascular disease: a narrative review. Oxidative Medicine and Cellular Longevity 2021.
15. Ulusu NN, Gok M, Erman B, Turan B (2019) Effects of timolol treatment on pancreatic antioxidant enzymes in streptozotocin-induced diabetic rats: An experimental and computational study. Journal of Medical Biochemistry 38: 306.
16. Al-Takay DT, Kashmola MA (2012) Survey for glucose-6-phosphate dehydrogenase enzyme deficiency among premarital attendanceinniava. Tikrit Journal of Pharmaceutical Sciences 8: 118-122.
17. Luzzatto L, Nannelli C, Notaro R (2016) Glucose-6-phosphate dehydrogenase

deficiency. Hematology/Oncology Clinics 30: 373-393.

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