Glucose-6-Phosphate Dehydrogenase

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Featured ARUP Testing

Glucose-6-Phosphate Dehydrogenase 0080135
Method: Quantitative Enzymatic Assay

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is one of the most common enzymatic disorders. Pathogenic variants in the X-linked gene, *G6PD,* can increase the risk for red blood cell (RBC) hemolysis, anemia, and other complications like neonatal jaundice. Individuals with G6PD deficiency may remain asymptomatic until exposed to an exogenous hemolytic trigger that induces oxidative stress, such as bacterial or viral infections, ingestion of fava beans, or certain medications.[1] Complete G6PD deficiency is incompatible with life so affected individuals exhibit a spectrum of partial enzyme deficiency. G6PD deficiency occurs worldwide but is most prevalent in individuals from malaria-endemic regions, as G6PD deficiency may confer a selective advantage against malaria.[2]

Indications for Use

This is the preferred initial screening test for G6PD deficiency.

For genetic testing in individuals of African descent, refer to Glucose-6-Phosphate Dehydrogenase (*G6PD*) 2 Mutations (0051684); for genetic testing in individuals with other high-risk ethnic backgrounds, refer to Glucose-6-Phosphate Dehydrogenase Deficiency (*G6PD*) Sequencing (3004457).

Test Interpretation

Methodology

This quantitative assay measures enzymatic activity of G6PD in whole blood, with results normalized to hemoglobin concentration to account for variability in RBC counts. G6PD activity is interpreted by comparing results to age-specific reference intervals (Table 1). Additionally, enzyme activity can be compared to the percentage of the median normal enzyme activity for that age group (Table 2), which aligns with the World Health Organization's (WHO) criteria for correlating G6PD activity phenotype and *G6PD* genotype[2] (Table 3). However, confirmatory genetic testing is required to confirm the genotype and identify *G6PD* variants.

Interpretative Information

Table 1: Age-Specific Reference Intervals

Age Range	Reference Interval
<8 days	16.4-24.1
8-30 days	13.6-23.9
1-6 months	11.5-21.5
7-12 months	10.3-17.9
1-17 years	9.7-16.9
≥18 years	9.6-16.3

Measured in U/g Hb (units of G6PD activity per gram of hemoglobin)

Table 2: Percentages of Normal Activity			
Age	**100%**	**80%**	**30%**
<8 days	19.7	15.8	5.9
8-30 days	18.2	14.6	5.5
1-6 months	16.1	12.9	4.8
7-12 months	13.8	11.0	4.1
1-17 years	12.9	10.3	3.9
≥18 years	12.7	10.2	3.8

Measured in U/g Hb

Table 3: G6PD Activity-Based Phenotype and Presumed Genotype Interpretation			
	% of Normal Activity	**G6PD Activity Phenotype**	**Presumed *G6PD* genotype**
Males	<30%	Deficient	Hemizygous for a *G6PD* deficient allele
	≥30%	Normal	Hemizygous for a *G6PD* normal allele
Females	<30%	Deficient	Homozygous for *G6PD* deficient alleles **or** heterozygous for a *G6PD* deficient allele with a predominant G6PD deficient RBC population
	≥80%	Normal	Homozygous for *G6PD* normal alleles **or** heterozygous for a *G6PD* normal allele with a predominant G6PD normal RBC population
	30-80%	Intermediate	Heterozygous for a *G6PD* deficient allele and a *G6PD* normal allele

Source: WHO[2]

Factors Affecting Interpretation

- A recent transfusion with non-G6PD deficient donor RBCs may mask G6PD deficiency.
- Samples collected during or shortly after a hemolytic episode may yield falsely normal or elevated G6PD activity results as reticulocytes have significantly higher G6PD activity than mature RBCs. If no other cause of hemolysis is identified, repeat G6PD testing is recommended at least 2-3 months after a hemolytic episode.[1]

References

1. Roper D, Layton M, Rees D, et al. Laboratory diagnosis of G6PD deficiency. A British Society for Haematology Guideline. *Br J Haematol*. 2020 ;189(1):24-38.

2. World Health Organization. Guide to G6PD deficiency rapid diagnostic testing to support *P. vivax* radical cure. Published Jun 2018; accessed Oct 2025.

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