

# An Overview of G6PD Deficiency Testing for Guiding Radical Cure Treatment in Vivax Malaria

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## Introduction

Glucose-6-phosphate dehydrogenase deficiency is a genetic disorder that affects millions of individuals worldwide, particularly in regions where malaria is endemic. G6PD deficiency is of particular concern in the treatment of *Plasmodium vivax* malaria, as certain antimalarial drugs, such as primaquine, can cause severe hemolysis in individuals with this condition. In recent years, there has been a growing emphasis on G6PD deficiency testing to guide radical cure treatment in vivax malaria. This article provides an overview of G6PD deficiency testing methods, their limitations, and their role in informing treatment decisions for vivax malaria [1].

## Description

G6PD deficiency is an X-linked genetic disorder characterized by reduced activity of the enzyme glucose-6-phosphate dehydrogenase, which plays a crucial role in protecting red blood cells against oxidative stress. Individuals with G6PD deficiency are at risk of hemolysis, a condition in which red blood cells are destroyed at an accelerated rate when exposed to certain triggers, such as certain foods, drugs, or infections. In the context of malaria, G6PD deficiency poses a significant challenge due to the risk of hemolysis associated with primaquine, the only drug effective against the dormant liver-stage

Several methods are available for testing G6PD deficiency, ranging from qualitative screening tests to quantitative enzyme activity assays. Qualitative tests, such as the fluorescent spot test and the methemoglobin reduction test, provide a rapid assessment of G6PD enzyme activity but may lack sensitivity and specificity. Quantitative assays, such as the spectrophotometric assay and the quantitative fluorescent spot test, offer more precise measurements of enzyme activity but require specialized equipment and expertise. Additionally, genetic testing can identify specific mutations associated with G6PD deficiency, providing valuable information about an individual's risk of hemolysis. Despite advances in G6PD deficiency testing, several challenges remain [2].

One challenge is the variability in G6PD enzyme activity levels among individuals, which can complicate interpretation of test results. Additionally, G6PD deficiency testing may not be readily available or affordable in resource-limited settings where vivax malaria is endemic. Furthermore, the timing of testing and the choice of testing method can influence the accuracy of results, particularly in individuals with recent hemolysis or blood transfusions. G6PD deficiency testing plays a crucial role in guiding radical cure treatment for vivax malaria. By identifying individuals at risk of hemolysis, healthcare

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providers can tailor treatment regimens to minimize the risk of adverse effects while ensuring effective parasite clearance. For individuals with normal G6PD enzyme activity, primaquine can be safely administered to achieve radical cure and prevent relapse of vivax malaria. In contrast, individuals with G6PD deficiency may require alternative treatment regimens, such as extended dosing schedules or alternative drugs, to avoid hemolytic complications. G6PD deficiency testing plays a critical role in guiding radical cure treatment for vivax malaria. By accurately identifying individuals with G6PD deficiency, healthcare providers can tailor treatment regimens to minimize the risk of hemolytic complications while ensuring effective parasite clearance and preventing relapse [3,4].

For individuals with normal G6PD enzyme activity, primaquine can be safely administered to achieve radical cure by targeting the dormant liver-stage parasites (hypnozoites) of *Plasmodium vivax*. However, in individuals with G6PD deficiency, primaquine administration carries an increased risk of hemolysis. In such cases, alternative treatment regimens may be necessary, including extended dosing schedules, lower doses of primaquine, or alternative drugs such as tafenoquine. The accurate identification of G6PD-deficient individuals through testing enables healthcare providers to make informed treatment decisions, optimizing patient safety and treatment outcomes. Thus, G6PD deficiency testing serves as a crucial component of personalized medicine in the management of vivax malaria, ensuring that treatment is tailored to individual patient characteristics and minimizing the risk of adverse effects [5].

## Conclusion

G6PD deficiency testing is essential for guiding radical cure treatment in vivax malaria, particularly in regions where the prevalence of G6PD deficiency is high. By identifying individuals at risk of hemolysis, healthcare providers can optimize treatment regimens to achieve effective parasite clearance while minimizing the risk of adverse effects. Continued research and innovation in G6PD deficiency testing methods are needed to improve access to accurate and reliable testing, particularly in resource-limited settings where vivax malaria is endemic. Ultimately, the integration of G6PD deficiency testing into malaria treatment guidelines has the potential to improve patient outcomes and contribute to the global effort to eliminate malaria.

## Acknowledgement

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## Conflict of Interest

There are no conflicts of interest by author.

## References

1. Howes, Rosalind E, Katherine E. Battle, Kamini N. Mendis and David L. Smith, et al. "Global epidemiology of *Plasmodium vivax*." *Am J Trop Med Hyg* 95 Suppl (2016):15.

2. Battle, Katherine E., Tim CD Lucas, Michele Nguyen and Rosalind E. Howes, et al. "Mapping the global endemicity and clinical burden of Plasmodium vivax, 2000–17: A spatial and temporal modelling study." *The Lancet* 394 (2019): 332-343.
3. Nguitragool, Wang, Ivo Mueller, Chalermpon Kumpitaka and Teerawat Saeseu, et al. "Very high carriage of gametocytes in asymptomatic low-density Plasmodium falciparum and P. vivax infections in western Thailand." *Parasites & Vectors* 10 (2017): 1-9.
4. Rovira-Vallbona, Eduard, Juan José Contreras-Mancilla, Roberson Ramirez and Mitchel Guzmán-Guzmán, et al. "Predominance of asymptomatic and sub-microscopic infections characterizes the Plasmodium gametocyte reservoir in the Peruvian Amazon." *PLOS Negl Trop Dis* 11 (2017): e0005674.
5. Motshoge, Thato, Grace K. Ababio, Larysa Aleksenko and John Read, et al. "Molecular evidence of high rates of asymptomatic P. vivax infection and very low P. falciparum malaria in Botswana." *BMC Infect Dis* 16 (2016): 1-8.

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