WHO-PQ RECOMMENDED SUMMARY OF PRODUCT CHARACTERISTICS

This summary of product characteristics focuses on uses of the medicine covered by WHO's Prequalification Team - Medicines. The recommendations for use are based on WHO guidelines and on information from stringent regulatory authorities.[*]

The medicine may be authorised for additional or different uses by national medicines regulatory authorities.

[*]https://extranet.who.int/prequal/sites/default/files/document_files/75%20SRA%20clarification_Feb2017_newtempl.pdf

1. NAME OF THE MEDICINAL PRODUCT

[MA176 trade name]†

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

Each film-coated tablet contains 15 mg primaquine (as phosphate).

Each tablet contains 60 mg of lactose monohydrate.

For a full list of excipients, see section 6.1.

3. PHARMACEUTICAL FORM

Film-coated tablets

Pink, round, film-coated tablets. They are biconvex (rounded on top and bottom) with a flat edge. The tablets have a scoreline on one side and are plain on the other side.

4. CLINICAL PARTICULARS

4.1 Therapeutic indications

[MA176 trade name] is indicated for the radical cure (prevention of relapse) of *Plasmodium vivax* and *Plasmodium ovale* malaria.

Primaquine is used in conjunction with an effective blood schizonticide: either artemisinin-based combination therapy (ACT) or chloroquine for *P. vivax* or *P. ovale* malaria.

Testing for glucose-6-phosphate-dehydrogenase (G6PD) deficiency is needed beforehand to guide appropriate treatment.

[MA176 trade name] is also used in combination with an ACT to reduce the transmissibility of *Plasmodium falciparum* infections in low-transmission areas.

Treatment regimens should take into account the most recent official treatment guidelines (e.g. those of the WHO) and local information on the prevalence of resistance to antimalarial drugs.

4.2 Posology and method of administration

Posology

The recommended dose of [MA176 trade name] is based on the weight and G6PD status of the patient.

Radical cure of *P. vivax* or *P. ovale* malaria

Patients with normal G6PD function (see also section 4.4)

The recommended dose of primaquine is 1 mg/kg body weight, taken once daily for 7 days.

Typical doses of [MA176 trade name] to be taken per day are shown in the table below:

† Trade names are not prequalified by WHO. This is the national medicines regulatory agency's responsibility.

Patient bodyweight	Dose	Number of tablets
Less than 12 kg	*An alternative formulation should be used to supply the correct dose*	
12 to less than 21 kg	15 mg	1
Patient bodyweight	**Dose**	**Number of tablets**
21 to less than 26 kg	22.5 mg	1.5
26 to less than 36 kg	30 mg	2
36 to less than 51 kg	45 mg	3
51 to less than 71 kg	60 mg	4
71 to less than 101kg	75 mg	5

Patients with intermediate G6PD deficiency (see also section 4.4)

The recommended dose is 0.5 mg/kg body weight, taken once daily for 14 days.

Typical doses of [MA176 trade name] to be taken per day are shown in the table below:

Patient bodyweight	Dose	Number of tablets
Less than 12 kg	*An alternative formulation should be used to supply the correct dose*	
12 to less than 21 kg	7.5 mg	*0.5
21 to less than 36 kg	15 mg	*1
36 to less than 51 kg	22.5 mg	1.5
51 to less than 71 kg	30 mg	2
71 to less than 101 kg	37.5 mg	2.5

*Formulations containing less primaquine may also be used

Patients with severe G6PD deficiency (see also section 4.4)

The recommended dose of primaquine is 0.75 mg/kg body weight, taken once every week for 8 weeks.

Typical doses of [MA176 trade name] to be taken per week are shown in the table below:

Patient bodyweight	Dose	Number of tablets
6 to less than 12 kg	7.5 mg	* 0.5
12 to less than 21 kg	15 mg	*1
21 to less than 36 kg	22.5 mg	*1.5
36 to less than 51 kg	30 mg	2
51 to less than 71 kg	45 mg	3
71 to less than 101 kg	60 mg	4

*Formulations containing less primaquine may also be used

Reduction of transmission of *P. falciparum* malaria

In low-transmission areas, a single dose of 0.25 mg/kg bodyweight primaquine should be given with the first dose of artemisinin-based combination therapy (ACT) to patients with *P. falciparum* malaria unless contraindicated (see Section 4.3). G6PD testing is not required.

Patient bodyweight	Single dose
Less than 50 kg	*
50 kg or over	1 tablet

* For these patients, other formulations containing less primaquine should be used.

Special populations

Elderly patients

There are no specific studies in the elderly. In general, dose selection for an elderly patient should be cautious, usually starting at the low end of the dosing range reflecting the greater frequency of decreased renal, hepatic, or cardiac function, and of concomitant disease or other drug therapy in the elderly; monitoring of efficacy and adverse reactions is needed.

Children

Population pharmacokinetics analysis performed in a limited paediatric population indicate that in children over 15 kg, no major age or body weight effect was evidenced in primaquine pharmacokinetics parameters when doses were adjusted to body weight.

Hepatic insufficiency

No data are available in patients with hepatic impairment. It is not known whether in patients with hepatic impairment, accumulation of primaquine and its metabolites could occur or if there could be an impact on generation of metabolites contributing to pharmacological activity.

Renal insufficiency

Single dose pharmacokinetics studies performed in patients with stages IV and V chronic kidney disease indicate higher primaquine C_{max} (up to 1.7-fold higher as compared to healthy subjects) but no evidence of major difference in AUC or $t_{1/2}$. It is not known whether after repeated dosing there could be an accumulation of metabolites that are mainly excreted by the renal route.

Genetic polymorphism:

- G6PD deficiency: The pharmacokinetic parameters in G6PD-deficient patients were not different from non-deficient patients.
- CYP2D6 polymorphism: Based on experiments in mice, primaquine activity probably depends on the formation of CYP2D6 metabolite(s). CYP2D6 polymorphism may be associated with variability in clinical response to primaquine.

Missed dose

If a dose is missed, it should be taken as soon as possible. However, if it is time for the next dose, the regular dosing schedule should be resumed until the course is completed. A double dose should not be taken.

Method of administration

[MA176 trade name] should be taken after a meal to reduce abdominal pain and cramping associated with ingestion of the medicine. The tablets are preferably swallowed whole. If the patient cannot swallow tablets, they may be crushed and mixed with a sweet food such as apple sauce or chocolate pudding and taken immediately.

4.3 Contraindications

Hypersensitivity to primaquine or to any of the excipients listed in Section 6.1.

[MA176 trade name] is contraindicated in the following situations:

- Pregnant women.
- Women who are breastfeeding infants aged less than 1 month, or breastfeeding infants whose G6PD status is unknown.
- Infants aged less than 1 month.
- Acutely ill patients with systemic disease associated with granulocytopenia, such as rheumatoid arthritis and systemic lupus erythematosis (SLE).
- Patients receiving concurrent treatment with other potentially haemolytic drugs or depressants of the myeloid elements of the bone marrow.
- Patients who are taking or have recently taken quinacrine.

4.4 Special warnings and precautions for use

[MA176 trade name] is not indicated as monotherapy for the treatment of malaria. Patients suffering from an attack of *P. vivax* or *P. ovale* malaria or who have parasitised red blood cells should initially be treated with a blood schizonticide to eliminate the erythrocytic parasites. Primaquine should then be administered to eradicate the exo-erythrocytic parasites.

G6PD deficiency

Primaquine may cause haemolytic anaemia in individuals with glucose-6-phosphate dehydrogenase (G6PD) deficiency. Before starting treatment, G6PD status should be assessed using a semi-quantitative near-patient test where possible. This will enable differentiation of enzyme activity thresholds relevant for selecting the appropriate treatment regimen (see section 4.2).

G6PD activity may be classified as follows:

- **Normal G6PD activity: At least 70%** of normal enzyme activity
  Patients with *normal G6PD activity* are at the lowest risk of haemolysis and may be treated with high-dose (1 mg/kg), short-course regimens.
- **Intermediate G6PD deficiency: 30% to 70%** of normal enzyme activity
  Patients with *intermediate G6PD deficiency* may be at increased risk of haemolysis, although the risk is generally lower than in patients with severe deficiency. This group includes, in particular, **heterozygous females**, in whom G6PD activity may vary and may not be reliably identified by qualitative tests.
- **Severe G6PD deficiency: Less than 30%** of normal enzyme activity
  Patients with severe G6PD deficiency are at high risk of haemolysis and if primaquine is given it should only be in a once-weekly regimen; appropriate monitoring and strict medical supervision, for haemolysis is recommended.

If G6PD testing is not available, treatment may be considered based on a careful assessment of the benefit–risk balance and should be undertaken with caution.

Patients should be advised to discontinue treatment and seek immediate medical attention if symptoms of haemolysis occur. These may include fatigue, weakness, shortness of breath, dark urine, or jaundice.

Cardiovascular effects

Due to a potential for QT interval prolongation, [MA176 trade name] should be used with caution in

- patients with heart disease, a history of ventricular arrhythmias, uncorrected hypokalaemia and/or hypomagnesaemia or bradycardia (< 50 bpm)
- patients receiving concomitant treatment with QT interval prolonging agents (see Sections 4.5).

Haematological effects

Anaemia, methaemoglobinaemia and leukopenia have been observed following administration of large doses of primaquine (see Section 4.9). The recommended dose should not be exceeded. It is advisable to do routine blood cell counts and haemoglobin measurements during therapy.

Methaemoglobinaemia:

Primaquine may cause a transient increase in methaemoglobin levels up to 10% in patients without risk factors (see Section 4.8). Methaemoglobinaemia may be severe in patients who are deficient in nicotinamide adenine dinucleotide (NADH) methaemoglobin reductase or treated with methaemoglobinaemia-inducing drugs (such as dapsone or sulfonamide, see Section 4.5). In these cases, close blood monitoring is required.

All patients should be advised to seek immediate medical attention if signs of methaemoglobinaemia occur (such as bluish lips or nails).

CYP2D6 phenotype

Non-clinical data suggest that primaquine activity depends on the formation of CYP2D6 metabolites. CYP2D6 polymorphism may therefore be associated with variability in clinical response to {DotWPProductName}.

Limited clinical data report higher treatment failure rates in patients with CYP2D6 poor or intermediate metaboliser status than in patients with normal or extensive metaboliser status.

In case of treatment failure, after checking the patient's compliance with treatment, potential concomitant use of CYP2D6 inhibitors should be checked (see Section 4.5). The patient's CYP2D6 status should be assessed if feasible. For poor CYP2D6 metabolisers, alternative treatment should be considered.

Excipients

[MA176 trade name] contains lactose. Patients with congenital lactase deficiency, galactosaemia or glucose-galactose intolerance must not be given this medicine unless strictly necessary.

The small amount of lactose in each dose is unlikely to cause symptoms of lactose intolerance in other patients.

Patients who are allergic to cow's milk proteins must not be given this medicine unless strictly necessary.

It is important to consider the contribution of excipients from all the medicines that the patient is taking.

4.5 Interaction with other medicinal products and other forms of interaction

Pharmacodynamic interactions

Co-administration of quinacrine (mepacrine) and primaquine is contraindicated due to increased toxicity of both drugs.

Concurrent administration of other medicines that can cause haemolysis, or methaemoglobinaemia-inducing drugs (such as dapsone or sulfonamides) should be avoided. If the association cannot be avoided, close blood monitoring is required.

Caution is required if primaquine is used concurrently with other drugs that prolong the QT interval, such as class IA and III antiarrhythmics, some tricyclic antidepressants, some antipsychotics, and some drugs from other classes.

Pharmacokinetic interactions

Effect of other agents on primaquine:

Caution is required with concurrent administration of potent CYP2D6 inhibitors, such as some SSRIs, as these may impact the formation of active metabolites of primaquine (see Section 5.1).

Primaquine exposure is slightly increased following co-administration with mild to moderate CYP2D6 inhibitors or with mild to moderate CYP3A inhibitors. However, there is no evidence that these interactions are clinically significant.

Primaquine pharmacokinetics are not significantly affected by the presence of mefloquine, artemether, or quinine.

Effect of primaquine on other agents:

Primaquine inhibits CYP1A2 potentially resulting in increased exposure of CYP1A2 substrates, such as duloxetine, theophylline and tizanidine. The data are limited, and no predictions can be made regarding the extent of the pharmacokinetic impact on CYP1A2 substrates. Caution is advised when CYP1A2 substrates are co-administered with primaquine.

The effect of primaquine on the pharmacokinetics of permeability glycoprotein (P-gp) substrates *in vivo* is unknown. However, *in vitro* observations suggest that primaquine inhibits P-gp, and has the potential to increase concentrations of P-gp substrates. Caution is advised when P-gp substrates with narrow therapeutic index, such as digoxin and dabigatran, are coadministered with primaquine.

Co-administration of primaquine with antimalarials, ethinyl-estradiol/levonorgestrol or acetaminophen has no significant impact on their pharmacokinetics.

4.6 Fertility, pregnancy and breastfeeding

Pregnancy

The safety of primaquine in human pregnancy has not been established. The use of primaquine is contraindicated during pregnancy due to the potential for G6PD deficiency in the fetus.

Preclinical data show a potential risk of genotoxicity and a potential embryo-fetal developmental toxicity. Although no clinical consequences have been identified, human data are limited.

Patients must be informed of the potential genotoxic risk. Effective contraception is recommended while on treatment and for the following period after the end of treatment:

- in treated women of childbearing potential, until completion of 2 menstrual cycles.
- in treated males whose partner may become pregnant, for 3 months.

Breast-feeding

Small amounts of primaquine are secreted into breast milk. Although the estimated infant exposure is less than 1% of a 0.5 mg/kg daily dose, there are very limited safety data in breastfed infants.

Because of the potential of primaquine or its metabolites to produce serious haematological adverse reactions in breastfed infants, especially those who may be G6PD deficient, primaquine should not be used during breastfeeding unless the breastfed infant has been tested and confirmed not to have G6PD deficiency. In addition, primaquine is contraindicated in women breastfeeding infants aged less than 1 month.

Fertility

There are no data on the effect of primaquine on fertility in humans.

4.7 Effects on ability to drive and use machines

Dizziness may occur during treatment with primaquine. This may impair the patient's ability to concentrate and react while driving or operating machinery.

4.8 Undesirable effects

Summary of the safety profile

While primaquine is generally well tolerated, it may cause dose-related gastrointestinal discomfort, including abdominal pain, nausea, and vomiting. Administration with food reduces these symptoms.

The most important adverse effect is haemolysis in patients with G6PD deficiency. The degree of haemolysis is dependent on the dose, duration of exposure and extent of G6PD deficiency (see Section 4.4).

Tabulated list of adverse events

Adverse events are listed under system organ class and ranked by headings of frequency. Within each frequency grouping, adverse reactions are presented in the order of decreasing seriousness. Frequencies are

defined as very common (at least 1 in 10), common (1 in 100 to 1 in 10), uncommon (1 in 1000 to 1 in 100), rare (1 in 10 000 to 1 in 1000), very rare (less than 1 in 10 000) or not known (frequency cannot be estimated from available data).

The table in this section is for adult patients only. The safety profile in children over 4 years is similar to adults with the exception of a higher frequency of gastrointestinal disorders.

Blood and lymphatic system	
Very common	Haemolysis in patients with G6PD deficiency
Uncommon	Haemolysis in patients without G6PD deficiency. Methaemoglobinaemia.
Rare	Leukopenia‡
Nervous system disorders	
Uncommon	Dizziness, headache
Cardiac disorders	
Uncommon	QT interval prolongation§
Rare	Arrythmias[2]
*Gastrointestinal disorders***	
Very common	Abdominal pain
Common	Nausea, vomiting, epigastric distress
Skin and subcutaneous tissue disorders	
Uncommon	Pruritis
Rare	Maculopapular rash

Reporting of suspected adverse reactions

Health care providers are asked to report adverse reactions that may be linked to a medicine, to the marketing authorisation holder, or, if available, to the national reporting system. Reports of suspected adverse reactions to a medicine are important for the monitoring of the medicine's benefits and risks.

4.9 Overdose

Symptoms

Abdominal cramps, vomiting, jaundice, burning epigastric distress, CNS disturbances (including headache and insomnia), cardiovascular disturbances (including cardiac arrhythmia and QT interval prolongation), methemoglobinaemia (indicated by cyanosis), moderate leukocytosis or leukopenia, granulocytopenia, and anaemia. Acute haemolysis may occur and will be severe in G6PD deficient patients.

Treatment

After acute intoxication, activated charcoal may be used where practical, to limit absorption in the gut. General supportive measures include airway management and cardiac monitoring.

‡ Leukopenia reported in patients with rheumatoid arthritis or SLE
§ With higher doses
** Incidence is reduced when primaquine is administered with food

Symptomatic methaemoglobinaemia in patients with normal G6PD activity may be treated with 1 to 2 mg/kg of methylene blue. It is contraindicated in patients with G6PD deficiency as it requires the enzyme to be effective and it may cause additional haemolysis in these patients.

Primaquine is not significantly removed by haemodialysis.

5. PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Pharmacotherapeutic group: Aminoquinoline anti-protozoal agent.

ATC code: P01BA03.

Mechanism of action

The precise mechanism of action has not been established. The presumed mechanisms are inhibition of the mitochondrial electron transport system of the parasite and the generation of reactive oxygen species in infected calls. Primaquine may also bind to and alter the properties of protozoal DNA.

Primaquine is active against the dormant liver form (hypnozoite) of *P. vivax* and *P. ovale* and the secondary exoerythrocytic stages (schizonts and schizogonies). In humans, primaquine activity is probably related to hydroxylated metabolites generated intrahepatically by CYP2D6.

Primaquine is also active against the primary exoerythrocytic forms of *P. falciparum* and has a gametocytocidal effect on all plasmodia gametocytes, particularly *P. falciparum* gametocytes.

Clinical studies

The multicentre, before and after implementation, staged Short COurse PrimaquinE (SCOPE) study is an ongoing study of a revised case management approach that incorporates point of care semi-quantitative G6PD testing and assesses the cost effectiveness, safety and feasibility of short course high dose primaquine (7 mg/kg total dose adjusted) based on G6PD enzyme activity and the risk of haemolysis.

In patients with normal G6PD activity (≥ 70%) a total dose of 7 mg/kg is administered, given as 1 mg/kg once daily for 7 days. For patients with intermediate G6PD activity (≥ 30% to < 70%), primaquine is given at the same total dose of 7 mg/kg but given as 0.5 mg/kg once daily for 14 days.

Patients with G6PD severe deficiency (< 30%) may be administered at a dose of 0.75 mg/kg once weekly for 8 weeks under close medical supervision with monitoring for signs of haemolysis.

5.2 Pharmacokinetic properties

No pharmacokinetic data are available for [MA176 trade name].

Pharmacokinetics of primaquine

Absorption	
Absorption	t_{max} 1-3 hours
Absolute bioavailability	96%
Oral bioavailability	At least 96%
Food effect	C_{max} ↑ 26%, AUC ↑ 15%
Distribution	
Volume of distribution (mean)	2.9-7.9 L/kg
Plasma protein binding *in vitro*	75% (mainly alpha-1 glycoprotein)

Tissue distribution	Extensive, with high erythrocyte concentrations. Crosses the placenta and passes into breast milk in small amounts.
Elimination	
Mean systemic clearance (Cl/F)	0.31-1.19 L/h/kg
% of dose excreted in urine	< 4% unchanged excreted in urine. 64% of dose recovered over 143 hours
% of dose excreted in faeces	Biliary excretion. Appears in faeces within 24 hours of administration.
Pharmacokinetic linearity	Dose proportional between 15 and 45 mg
Drug interactions *(in vitro)*	
Transporters	Not a substrate of P-gp or BCRP Inhibitor of P-gp
Metabolising enzymes	Primaquine is metabolized by MAO-A into the main inactive metabolite carboxyprimaquine. Hydroxylation by CYP2D6 generates the reactive species responsible for the antimalarial effects and haemolytic toxicity. CYP1A2, 2C19, 3A4 and direct conjugation of primaquine play a smaller role in metabolism.
	Low potential to inhibit any of the major MAO or CYP450 isoforms, except CYP1A2.

PQN: primaquine; BCRP: breast cancer resistance protein; P-gp: permeability glycoprotein

Special populations

Hepatic insufficiency:

No data are available in patients with hepatic impairment. It is not known whether in patients with hepatic impairment accumulation of primaquine and its metabolites could occur or if there could be an impact on generation of metabolites contributing to pharmacological activity.

Renal insufficiency:

Single dose pharmacokinetics studies performed in patients with stages IV and V chronic kidney disease indicate higher primaquine C_{max} (up to 1.7-fold higher as compared to healthy subjects) but no evidence of major difference in AUC or $t_{1/2}$. It is not known whether after repeated dosing there could be an accumulation of metabolites that are mainly excreted by renal route.

Paediatrics:

Population pharmacokinetics analysis performed in a limited paediatric population indicate that in children > 15 kg, no major age or body weight effect was evidenced in primaquine pharmacokinetics parameters when doses were adjusted to body weight.

Genetic polymorphism:

- G6PD deficiency: The pharmacokinetic parameters in G6PD-deficient patients were not different from non-deficient patients.
- CYP2D6 polymorphism: Based on experiments in mice, primaquine activity probably depends on the formation of CYP2D6 metabolite(s). CYP2D6 polymorphism may be associated with variability in clinical response to primaquine.

5.3 Preclinical safety data

Primaquine is a weak genotoxic agent which elicits both gene mutations and chromosomal/DNA breaks. The *in vitro* reverse gene mutation assays using bacteria (Ames test) and the *in vivo* studies using rodents (mouse bone marrow cell sister chromatid exchange, mouse bone marrow cell chromosome abnormality, and rat DNA strand-breaks in multiple organs) gave positive results.

In studies in rats, adverse effects on the fetus (visceral anomaly, skeletal variation, etc.) were observed at dose levels toxic to the dams.

No fertility studies have been conducted with primaquine.

6. PHARMACEUTICAL PARTICULARS

6.1 List of excipients

Core tablet: microcrystalline cellulose
lactose monohydrate
pregelatinized starch
povidone
magnesium stearate

Film coat: hypromellose
titanium dioxide
macrogol/polyethylene glycol
talc
polysorbate
iron oxide red

6.2 Incompatibilities

Not applicable

6.3 Shelf life

24 months

6.4 Special precautions for storage

Do not store above 25°C. Avoid excursions above 30°C.

Discard the product 50 days after initial opening (HDPE bottle packs).

6.5 Nature and contents of container

Blister pack:

Aluminium (OPA/Alu/PVC laminate) on aluminium foil blister cards, each containing 10 tablets. Available in cartons of 10 x 10 tablets.

HDPE container:

Round, opaque white plastic (HDPE) bottle containing 100 tablets. The bottle has a sachet of dessicant (drying material). The bottle has a printed plastic/aluminium foil seal and a white, childproof plastic (polypropylene) screw cap with a pulp (cardboard-type material) liner.

6.6 Special precautions for disposal and other handling

Any unused product or waste material should be disposed of in accordance with local requirements.

7. SUPPLIER

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8. WHO REFERENCE NUMBER (WHO Prequalification Programme)

MA176

9. DATE OF PREQUALIFICATION

22 December 2023

10. DATE OF REVISION OF THE TEXT

June 2026

References

WHO Guidelines for malaria:13 August 2025. https://iris.who.int/server/api/core/bitstreams/26a6af2d-060c-4449-8207-1f25e63c6cc3/content, accessed 26 November 25)

Short COurse PrimaquinE for the radical cure of *P. vivax* (SCOPE), https://www.vivaxmalaria.org/projects/pave-feasibility-studies/scope-short-course-pq-radical-cure-p-vivax accessed 18 January 2026

WHO. Malaria case management – *Plasmodium vivax* malaria: a field guide. Geneva: World Health Organization; 2026 (https://iris.who.int/server/api/core/bitstreams/d577a1ad-ae11-465a-be4d-7b865fa2b71c/content, accessed 23 March 2026)

WHO policy brief: Testing for G6PD deficiency for safe use of primaquine in radical cure of *P. vivax* and *P. ovale* 2016 (http://apps.who.int/iris/bitstream/handle/10665/250297/WHO-HTM-GMP-2016.9-eng.pdf;jsessionid=514237439B564F5A88C54908FCA01C66?sequence=1)

Primaquine (primaquine phosphate tablets): product monograph, Sanofi-Aventis Canada, June 25, 2025 (https://pdf.hres.ca/dpd_pm/00080891.PDF accessed 27 November 25)

Treatment of Malaria: CDC Guidelines for Clinicians (United States) Last reviewed May 11, 2020 https://www.cdc.gov/malaria/resources/pdf/Malaria_Treatment_Guidelines.pdf

Baird K.J. and Hoffman S.L. Primaquine Therapy for Malaria. *Clin Infect Dis* (2004) 39:1336-1245 https://academic.oup.com/cid/article/39/9/1336/404549

Hill, DR; Baird, JK; Parise, ME; et al. Primaquine: report from CDC expert meeting on malaria chemoprophylaxis I. *Am J Trop Med Hyg* (2006) 75: 402-15.