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

[MA196 trade name]†

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

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

Excipients with potential clinical effect

Each tablet contains 43.15 mg of lactose.

3. PHARMACEUTICAL FORM

Film-coated tablets.

Brown to light brown, round, film-coated tablets. They are biconvex (rounded on top and bottom) with a flat edge. The tablets have a break line on one side and are plain on the other side.

The break line can be used to divide [MA196 trade name] into equal doses

4. CLINICAL PARTICULARS

4.1 Therapeutic indications

[MA196 trade name] is indicated for the radical cure (prevention of relapse) of *Plasmodium vivax* and *Plasmodium ovale* malaria, in adults and children.

It is also used to reduce the transmissibility of *Plasmodium falciparum* infections in low-transmission areas.

Except in primary prophylaxis, primaquine is used in conjunction with an effective blood schizonticide: either artemisinin-based combination therapy (ACT) or chloroquine for *vivax* or *ovale* malaria.

Consideration should be given to official treatment guidelines for malaria (e.g., those of the WHO).

4.2 Posology and method of administration

Posology

The recommended dose of [MA196 trade name] is based on the weight and G6PD status of the patient, and on the strain of the parasite.

Radical cure of P. vivax or P. ovale malaria

Patients without G6PD deficiency

The recommended dose of primaquine is 0.25-0.5 mg/kg body weight, taken once daily for 14 days. Doses of 0.25 mg/kg are considered appropriate for infections with temperate strains; doses of 0.5 mg/kg may be required for the tropical, frequently relapsing strains of *P. vivax* prevalent in East Asia and Oceania.

Typical doses of [MA196 trade name] to be taken per day are shown in the table below:

Patient bodyweight	Dose
Less than 30 kg	*
30 to less than 60 kg	1 tablet daily for 14 days
60 to less than 100 kg	2 tablets daily for 14 days
100 kg or over	3 tablets daily for 14 days

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

Alternatively, patients may be given a shorter course, comprising primaquine 0.5 mg/kg daily for 7 days.

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

Patients with mild to moderate G6PD deficiency (see also Section 4.4)

The recommended dose is 0.75 mg/kg body weight taken once a week for 8 weeks, up to a maximum unit dose of 45 mg primaquine base. The number of tablets of [MA196 trade name] to be taken per day is shown in the table below:

Patient bodyweight	Dose
Less than 20 kg	*
20 to less than 30 kg	1 tablet once a week for 8 weeks
30 to less than 60 kg	2 tablets once a week for 8 weeks
60 kg or over	3 tablets once a week for 8 weeks

* For these patients, other formulations containing less primaquine should 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. There are no data on the toxicity of primaquine in infants below 6 months.

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.

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.

Method of administration

[MA196 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.

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.

4.3 Contraindications

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

[MA196 trade name] is contraindicated in the following situations:

- Patients with severe G6PD deficiency (< 30% of the normal mean red cell activity).
- Pregnant women.
- Women who are breastfeeding unless the G6PD status of the infant is known.
- Infants aged less than 6 months.
- 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

[MA196 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

Any patient (male or female) with red cell G6PD activity < 30% of the normal mean has G6PD deficiency and will experience haemolysis on exposure to primaquine. Heterozygous females with higher mean red cell activity may still develop significant haemolysis. The severity of haemolysis depends on the dose of primaquine and on the variant of the G6PD enzyme. Primaquine is eliminated rapidly, so haemolysis is self-limiting once the drug has been stopped.

The G6PD status of patients should be used to guide administration of primaquine for preventing relapse.

Concurrent administration of haemolytic agents should be avoided (see Section 4.5).

Screening for G6PD deficiency is not widely available outside hospitals, but rapid screening tests that can be used at points of care are becoming increasingly available.

- In patients known to be G6PD deficient, the decision to give or withhold primaquine should depend on the availability of close medical supervision, with ready access to health facilities with blood transfusion services.
- In people with mild-to-moderate G6PD deficiency, a regimen of 0.75 mg/kg primaquine given once weekly for 8 weeks is preferred (see section 4.2).
- Some heterozygous females who test as normal or not deficient in qualitative G6PD screening tests have intermediate G6PD activity and are susceptible to haemolysis. Intermediate deficiency (30-80% of normal) and normal enzyme activity (> 80% of normal) can be differentiated only with a quantitative test. In the absence of quantitative testing, all females should be considered as

potentially having intermediate G6PD activity and given the 14-day regimen, with counselling on how to recognise the symptoms and signs of haemolytic anaemia. They should be advised to stop primaquine and be told where to seek care should these signs develop.

- If G6PD testing is not available, the decision to prescribe or withhold primaquine should be based on the balance of the probability and benefits of preventing relapse against the risks of primaquine-induced haemolytic anaemia. This will depend on the population prevalence of G6PD deficiency, the severity of the prevalent genotypes and on the capacity of health services to identify and manage primaquine-induced haemolytic anaemia.

Cardiovascular effects

Due to a potential for QT interval prolongation, [MA196 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 4.8 and 4.9).

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

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, a decision should be made whether to interrupt breastfeeding or to delay maternal primaquine treatment until breast-feeding has finished.

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

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

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.

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

Based on many years of clinical experience, reviewed in several meta-analyses for *P. vivax*, primaquine provides efficient radical cure for both *P. vivax* and *P. ovale* infections (cure rate of relapses > 60 to 100%) in patients receiving regimens of 15 mg or 30 mg daily for 14 days after an appropriate schizonticidal treatment. Factors affecting the efficacy are: the total dose over a sufficient treatment duration, the bodyweight of the patient, adherence to therapy and the type of *P. vivax* strain.

Dose optimisation

The recommended doses for radical cure of *P. vivax* and *P. ovale* malaria, presumptive anti-relapse therapy remain unchanged:

- 0.25 mg base/kg per day for 14 days for temperate strain infections
- 0.50 mg base/kg per day for 14 days for tropical, frequently relapsing infections

There seems to be no difference in recurrence of parasitaemia, the safety or the tolerability of a standard dose of 0.5 mg/kg/day for 7 days compared with 0.25 mg/kg/day for 14 days.

The WHO now recommends a single low-dose of 0.25 mg/kg to reduce onward transmission of *P. falciparum* in programmes to eliminate *P. falciparum* malaria and in areas threatened by resistance of *P. falciparum* to artemisinins. The lower dose is safer and considered to be as effective as 0.75 mg/kg body weight in reducing transmissibility based on limited data from assessments of direct transmission blocking in mosquito feeding studies, which is considered therapeutically more relevant than gametocyte clearance.

5.2 Pharmacokinetic properties

Absorption of [MA196 trade name]

The absorption characteristics of [MA196 trade name] have been determined after administration of single dose primaquine 15 mg tablet in healthy volunteers in the fasting state as follows:

Pharmacokinetic variable'	**Mean value* (± standard deviation)**
	Primaquine
Maximum concentration (C_{max})	66 ± 26 ng/mL
Area under the curve ($AUC_{0-\infty}$), a measure of the extent of absorption	645 ± 243 ng.h/mL
Time to attain maximum concentration (t_{max})	2.80 ± 0.79 h

* arithmetic mean

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 secreted in breast milk.
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 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 : Lactose

Microcrystalline cellulose

Pregelatinised starch

Purified Talc

Magnesium stearate

Film coat : Hypromellose

Ethyl cellulose

Triacetin

Titanium dioxide

Iron oxide red

6.2 Incompatibilities

Not applicable

6.3 Shelf life

24 months

6.4 Special precautions for storage

Do not store above 30ºC. Protect from light.

6.5 Nature and contents of container

Aluminium blisters

[MA196 trade name] is provided in aluminium foil blister packs, each containing 7 tablets. Available in cartons of 1 x 7 or 10 x 7 tablets.

6.6 Special precautions for disposal and other handling

No special requirements.

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)

MA196

9. DATE OF PREQUALIFICATION

17 July 2025

10. DATE OF REVISION OF THE TEXT

September 2025

References

WHO Guidelines for malaria:16 October 2023. Geneva: World Health Organization; 2023. (https://iris.who.int/bitstream/handle/10665/373339/WHO-UCN-GMP-2023.01-Rev.1-eng.pdf, accessed 20/11/23)

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)

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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.

Detailed information on this medicine is available on the World Health Organization (WHO) website: https://extranet.who.int/prequal/medicines/prequalified/finished-pharmaceutical-products