## Name: Awulu Mary Inikpi

## Matric No: 17/mhs02/027

## Course title: Pharmacology

## Answers

## Mefloquine

**Mechanism of Action:**

Unknown, chemically related to quinidine. Has strong blood schizonticidal activity against P. falciparum and P. vivax, but not against hepatic stages or gametocytes.

## Pyrimethamine + Sulfadoxine

**Mechanism of Action:**

**Folic acid antagonists.** The rationale for there combination is a synergistic effect to inhibit folic acid synthesis, and a differential requirement between host and parasite for nucleic acid precursors involved in growth.

This activity is highly selective against plasmodia and Toxoplasma gondii.

Pyrimethamine is chemically related to trimethoprim. It acts slowly against erythrocytic forms of susceptible strains of all four human malaria species. It is not adequately gametocidal or effective against liver stages.

Arthemeter and Artesunate

**Mechanism of Action:**

**Produces a free radical when it undergoes an iron-catalyzed cleavage of an endoperoxide bond in the parasite food vacuole**.

It is a rapidly acting blood schizonticide, with some activity against gametocytes, but no activity against the hepatic stages of the malarial parasite.

Primaque

**Mechanism of Action:**

Active against hepatic stages **of all human malarial parasites** Some gametocytes are destroyed while others cannot undergo maturation division in the gut of the mosquito.

Primaquine’s cellular mechanism of action is still poorly understood:

Fourteen primaquine metabolites have been detected, and few have been fully assessed for their biological activity.

Evidence suggests that one or more highly reactive metabolites of primaquine inflict extensive oxidative damage that interferes with mitochondrial electron transport in parasites(NOTE:primaquine is also known to increase the oxidative stress on human red blood cells, an effect that contributes to its hemolytic side effects) (Butterworth et al, 2013).

Quinine and Quinidine for malaria

**Mechanism of Action:**

Its precise mechanism as an antimalarial is poorly understood.

In Plasmodium falciparum quinine has been found to inhibit nucleic acid synthesis, protein synthesis, and glycolysis; it also binds with hemazoin in parasitized erythrocytes.

Quinine is effective as a malarial suppressant and in control of overt clinical attacks. Its primary action is schizontocidal, no lethal effect is exerted on sporozoites or pre-erythrocitic tissue forms.

Quinine blocks cardiac K & Na channels similar to quindine