About AJPP

The African Journal of Pharmacy and Pharmacology (AJPP) provides African and International researchers a platform to highlight studies on drug delivery systems and composition, medication dispensation and management, natural and synthetic drug use and distribution. Other subjects covered include pharmacology, clinical pharmacy and medication counseling. The journal also emphasizes novel developments in drug design and its applications in allied fields.

Indexing

The African Journal of Pharmacy and Pharmacology is indexed in:

- African Index Medicus
- CAB Abstracts
- CABI’s Global Health Database
- Chemical Abstracts (CAS Source Index)
- China National Knowledge Infrastructure (CNKI)
- Dimensions Database
- Google Scholar
- Matrix of Information for The Analysis of Journals (MIAR)
- ResearchGate

Open Access Policy

Open Access is a publication model that enables the dissemination of research articles to the global community without restriction through the internet. All articles published under open access can be accessed by anyone with internet connection.

The African Journal of Pharmacy and Pharmacology is an Open Access journal. Abstracts and full texts of all articles published in this journal are freely accessible to everyone immediately after publication without any form of restriction.

Article License

All articles published by African Journal of Pharmacy and Pharmacology are licensed under the Creative Commons Attribution 4.0 International License. This permits anyone to copy, redistribute, remix, transmit and adapt the work provided the original work and source is appropriately cited. Citation should include the article DOI. The article license is displayed on the abstract page the following statement:

This article is published under the terms of the Creative Commons Attribution License 4.0 Please refer to https://creativecommons.org/licenses/by/4.0/legalcode for details about Creative Commons Attribution License 4.0

Article Copyright
When an article is published by in the African Journal of Pharmacy and Pharmacology, the author(s) of the article retain the copyright of article. Author(s) may republish the article as part of a book or other materials. When reusing a published article, author(s) should;

Cite the original source of the publication when reusing the article. i.e. cite that the article was originally published in the African Journal of Pharmacy and Pharmacology. Include the article DOI
Accept that the article remains published by the African Journal of Pharmacy and Pharmacology (except in occasion of a retraction of the article)
The article is licensed under the Creative Commons Attribution 4.0 International License.

A copyright statement is stated in the abstract page of each article. The following statement is an example of a copyright statement on an abstract page.
Copyright ©2016 Author(s) retains the copyright of this article.

Self-Archiving Policy
The African Journal of Pharmacy and Pharmacology is a RoMEO green journal. This permits authors to archive any version of their article they find most suitable, including the published version on their institutional repository and any other suitable website.
Please see http://www.sherpa.ac.uk/romeo/search.php?id=213&flDnum=|&mode=simple&la=en

Digital Archiving Policy
The African Journal of Pharmacy and Pharmacology is committed to the long-term preservation of its content. All articles published by the journal are preserved by Portico. In addition, the journal encourages authors to archive the published version of their articles on their institutional repositories and as well as other appropriate websites.
https://www.portico.org/publishers/ajournals/

Metadata Harvesting
The African Journal of Pharmacy and Pharmacology encourages metadata harvesting of all its content. The journal fully supports and implement the OAI version 2.0, which comes in a standard XML format. See Harvesting Parameter
Memberships and Standards

OPEN ACCESS

Academic Journals strongly supports the Open Access initiative. Abstracts and full texts of all articles published by Academic Journals are freely accessible to everyone immediately after publication.

Creative Commons

All articles published by Academic Journals are licensed under the Creative Commons Attribution 4.0 International License (CC BY 4.0). This permits anyone to copy, redistribute, remix, transmit and adapt the work provided the original work and source is appropriately cited.

Crossref

Crossref is an association of scholarly publishers that developed Digital Object Identification (DOI) system for the unique identification published materials. Academic Journals is a member of Crossref and uses the DOI system. All articles published by Academic Journals are issued DOI.

Similarity Check powered by iThenticate is an initiative started by CrossRef to help its members actively engage in efforts to prevent scholarly and professional plagiarism. Academic Journals is a member of Similarity Check.

CrossRef Cited-by Linking (formerly Forward Linking) is a service that allows you to discover how your publications are being cited and to incorporate that information into your online publication platform. Academic Journals is a member of CrossRef Cited-by.

International Digital Publishing Forum (IDPF)

Academic Journals is a member of the International Digital Publishing Forum (IDPF). The IDPF is the global trade and standards organization dedicated to the development and promotion of electronic publishing and content consumption.
COUNTER (Counting Online Usage of Networked Electronic Resources) is an international initiative serving librarians, publishers and intermediaries by setting standards that facilitate the recording and reporting of online usage statistics in a consistent, credible and compatible way. Academic Journals is a member of COUNTER

Portico is a digital preservation service provided by ITHAKA, a not-for-profit organization with a mission to help the academic community use digital technologies to preserve the scholarly record and to advance research and teaching in sustainable ways.

Academic Journals is committed to the long-term preservation of its content and uses Portico

Academic Journals provides an OAI-PMH(Open Archives Initiatives Protocol for Metadata Harvesting) interface for metadata harvesting.
Contact

Editorial Office: ajpp@academicjournals.org
Help Desk: helpdesk@academicjournals.org
Website: http://www.academicjournals.org/journal/AJPP
Submit manuscript online http://ms.academicjournals.org

Academic Journals
73023 Victoria Island, Lagos, Nigeria
ICEA Building, 17th Floor, Kenyatta Avenue, Nairobi, Kenya
Editors

Prof. Zhe-Sheng Chen
College of Pharmacy and Health Sciences
St. John's University
New York,
USA.

Prof. Mahmoud Mohamed El-Mas
Department Pharmacology and Toxicology
Faculty of Pharmacy
Alexandria University
Egypt

Prof. Mohammed Abdur Rashid
Department of Pharmaceutical Chemistry
Faculty of Pharmacy
University of Dhaka
Dhaka. BANGLADESH.

Dr. Fulya Ustun Alkan
Department of Pharmacology and Toxicology,
Faculty of Veterinary Medicine, Istanbul
University,
Turkey.

Dr. Amel Hashim
Department of Biochemistry and Molecular Biology
Faculty of Pharmacy
Helwan University
Egypt.

Dr. Kavitha Balaji
Investigational Cancer Therapeutics,
Anderson Cancer Center
USA.

Dr. Huma Ikram
Department of Biochemistry,
Neurochemistry and Biochemical Neuropharmacology Research Unit,
University of Karachi
Karachi, Pakistan

Dr. Prakash Srinivasan Timiri Shanmugam
Department of Biochemistry and Molecular Biology
Louisiana State University Health Sciences Center
New Orleans, USA.

Dr. Yao Dai
Division of Cardiology
Department of Internal Medicine
School of Internal Medicine,
University of Arkansas for Medical Sciences
USA.

Dr. Hazem Shaheen
Department of Pharmacology
Faculty of Veterinary Medicine
Damanhour University
Egypt.
Editors

Dr. Doaa Ibrahim
Clinical Pharmacy and Pharmacy Practice,
University of Science and Technology
Yemen-Sana’a.

Editorial Board Members

Abiy Habtewold Eyakem
School of Pharmacy, Union University
(Jackson, Tennessee, USA) and School of
Medicine, Addis Ababa University (Addis Ababa,
Ethiopia)

Prof. Kittisak Sawanyawisuth
Department of Medicine
Faculty of Medicine
Khon Kaen University
Khon Kaen
Thailand.

Dr. Subhalakshmi Ghosh
Department of Pharmaceutical Technology
Jadavpur University
Kolkata
India.

Dr. Riyanto Teguh Widodo,
Pharmaceutical Sciences, Pharmacy,
Institution University of Malaya,
Malaysia

Dr. Ravi Shankar Shukla
Preformulation and Exploratory Research R&D
Amneal Pharmaceuticals
USA.

Dr. Ying-Yong Zhao
Department of Traditional Chinese Medicine
The College of Life Sciences
Northwest University
Xi’an
China.

Dr. Maria Ondina Paganelli
Department of Pharmaceutical Sciences,
Faculty of Pharmaceutical Sciences of
Ribeirão Preto University of São Paulo
(USP),
Brazil.
Table of Content

Modern approach of treatment on destroyable pathogenicity of malaria parasite: A review article
Sourav Das, Sabahuddin Siddique, Ahmed M Shehata, Mohamed A. Shaker, Mohi Iqbal Mohammed Abdul, Asis Bala, Pallab Mandal, Shubhasis Dan and Anirbandeep Bose
Review

Modern approach of treatment on destroyable pathogenicity of malaria parasite: A review article

Sourav Das¹, Sabahuddin Siddique², Ahmed M Shehata³,⁴, Mohamed A. Shaker⁵,⁶, Mohi Iqbal Mohammed Abdul³, Asis Bala⁷, Pallab Mandal⁸, Shubhasis Dan⁸ and Anirbandeep Bose⁸

¹Guru Nanak Institute of Pharmaceutical Science and Technology, Kolkata, India.
²Patel College of Pharmacy, Madhyanchal Professional University, Bhopal, M.P., India.
³Department of Pharmacology and Toxicology, College of Pharmacy, Taibah University, Al-Madinah Al-Munawarah, Kingdom of Saudi Arabia.
⁴Department of Pharmacology and Toxicology, Faculty of Pharmacy, Beni-Suef University, Beni-Suef, Egypt.
⁵Pharmaceutics and Pharmaceutical Technology Department, College of Pharmacy, Taibah University, Al-Madinah Al-Munawarah, Kingdom of Saudi Arabia.
⁶Pharmaceutics Department, Faculty of Pharmacy, Helwan University, P. O. Box 11795, Cairo, Egypt.
⁷Faculty of Pharmacology and Toxicology at National Institute of Pharmaceutical Education And Research, Hajipur, India.
⁸TAAB Biostudy Services, Jadavpur, Kolkata, India.

Received 14 November, 2018: Accepted 18 December, 2018

Plasmodium is the malaria parasite that completes the life cycle between two different hosts, such as human and Anopheles mosquito. These parasites go through several developmental stages like exoerythrocytic stage that is absent in Plasmodium falciparum, so relapses do not occur. The drugs which contain three P’s like proguanil, primaquine, and pyrimethamine kill schizonts in the liver. Due to prolonged treatment of high dose of chloroquine, there may be serious side effects named as Bull’s eye maculopathy. Atovaquone is rapidly acting blood schizonticide that acts by collapsing the parasite’s membrane. Artemisinins are the fastest acting drugs against malaria. Mepacrine is an anti malarial drug which concentrates in collagen tissue. Infection by P. falciparum is the most lethal form of malaria, in this case agglutination of the infected RBC occurs and these agglutinised RBCs block the capillary vessels of the internal organs. Tafenoquine is a single dose medication for radical cure of P. vivax malaria. People with an enzyme problem G6PD deficiency can cause severe anaemia. At least two genes affecting red cells which are resistant to P. falciparum are autosomal gene for haemoglobin S (HbS) and the gene linked to sex G6PD variant gene. Anaemia is the main result due to malaria by haemolysis of infected and uninfected erythrocytes, dyserythropoiesis, splenomegaly and depletion of folate stores. Cerebral malaria is the most urgent complication that is manifested by confusion or coma by clusters of parasitized red blood cells to form large size cells of the capillary circulation which adhere to the vascular endothelium and block the circulation causing cerebral hypoxia and resulting to neurological symptoms and diagnosed cerebral malaria. Blackwater fever associated with falciparum malaria is mostly common with individuals that have taken antimalarial treatment irregularly or deficient in G6PD deficiency. Tropical splenomegaly is another symptom in falciparum malaria. HbAS was the genetic variant which associated with protection against malaria incidence and other variants such as alpha thalassemia, G6PD deficiency, polymorphism of genes encoding NOS₂A and TNF, as well as protection against uncomplicated malaria.

Key words: Malaria, pathogenicity of malaria, parasite of malaria.
INTRODUCTION:

The innumerable microscopic and macroscopic forms which after attacking the human body manifest different diseases are known as pathogens and their activity are known as pathogenicity. Over the century, malaria has been the sustained thread to the urban areas of so many countries. Though according to the Greek mythology, it was so believed that this diseased condition accompanied by high fever and inflammation in spleen was often seen in local communities with poor hygienic condition, with this disease highly communicable. The term originated from Greek word *malaria*, which refers to polluted environment (Ali et al, 2011). Later on, in the year 1880, the Charles Louis Alphonse Laveran discovered that the disease was caused by protozoan parasite (*Plasmodium vivax*) infection which is transmitted by female *Anopheles* species mosquitoes.

Eventually, microbiological research revealed that five different species of *Plasmodium* genus protozoas, namely, *P. falciparum*, *P. vivax*, *P. ovale*, *P. knowlesi*, and *P. malariae* have been identified as causes of malaria in humans (Anne et al, 2013). Among them it was reported that *P. falciparum* and *P. vivax* registers majority (>60%) of death cases in human. As per the WHO malaria report 2015, African countries are prone (70-80%) to be affected by malaria; whereas the south Asian countries registers (10%) of overall deaths worldwide. In 2015 worldwide, 214 million cases of malaria were identified, and 438 000 malaria deaths were reported in this survey (Beatriz et al, 2015). According to a survey report conducted in Ethiopia, malaria was considered the most common communicable disease in the country, as 75% of the total population are reportedly victims of malaria (Clark et al, 2004).

The parasite named as plasmodium genus can manifest the malaria in the human body in a serious manner. The rate of manifestation of the malaria depends on how fast the parasite replicates. Over 100 types of plasmodium parasites are able to spread the diseases in various species. Five different types of parasite named plasmodium can spread the diseases in human (Clark et al, 2004, Doolan et al, 2009; Francis et al, 2010).

MALARIAL LIFE CYCLE

*Plasmodium* genus parasites are transmitted into human blood stream after a bite from female *Anopheles* species mosquitoes; this parasite initiates the infections which thereafter travel to the liver and attacks hepatocytes, of thousands of merozoites in the blood stream. Merozoites in the bloodstream undergoes another asexual multiplication invading the RBCs (Red Blood cells) and forms mature schizonts, which in turn further releases merozoites that attacks new erythrocytes (Doolan et al, 2009; Smith et al, 1995). The highest mortality rate of malaria is reported by *P. falciparum* and *P. vivax*, where *P. vivax* causes benign malaria, whereas *P. falciparum* causes most of the malignant malarials among young people (Eastman et al, 2011; Eltahir et al, 2010; Cox, 2010). *P. falciparum* contains trophozoites rings formed by cytoplasm and two chromatin dots, which is responsible for erythrocyte damage and facilitates malignant malaria.

Usually, all type of malaria is characterized by the incidence of anaemia, as it causes destruction of RBCs. A survey was carried out in Ethiopian young subjects who were admitted in hospital; the results obtained from the respective disciplines showed that the subjects with *P. falciparum* malaria had a high level of lymphocytes, other WBCs (White Blood cells) were within range, but the hemolysis was greater in patients having *P. falciparum* type of malaria when compared to other malarial parasites attacks. Due to impaired cytokine level, the healing of tissue damage also takes more time than the merozoites attack to the new cells (Clark et al, 2004).

SEVERITY AND THREATS FEATURED BY MALARIAL PARASITE

Apart from conventional and regular diseased state in vulnerable population living in poor hygienic places, there are several more severe threats accomplished with the attack of *Plasmodium* parasite specie. Blood circulation comprises RBC movement through the heart followed by passing through the spleen. Therefore, spleen hygiene is majorly dominated by blood cells, as it regulates the filtration of impaired or infected RBCs. However, focussing the pathogenicity of *Plasmodium* species on RBC, gives rise to the *P. falciparum* Erythrocyte Membrane Protein 1 ( PfEMP1) in the iRBC membrane surface, resulting to elicitation of different inflammatory responses (Su et al, 1995; Baruch et al, 1995). Among them, TNF-α plays a key role as a cytokine, facilitates ICAM-1 expression, and enables followed by asexual multiplication resulting to production cytoadhesion of pRBC. Apart from TNF-α, (IL)-1b, IL-6 and IL-10 and endogenous NO (Nitric Oxide) also involves in the pathogenesis of malaria. NO is subjected to developing the host defence, as well as maintaining both the vascular permeability and microenvironment of organs. Splenomegaly, considered as the marker of *P. falciparum* transmission of infected areas indicates severe malaria caused by parasite, which in turn results to spleen enlargement. During the acute infection phase, uRBC,
knob positive and negative iRBC accumulated in spleen (Prommano et al, 2005, Imbert et al, 2010) Moreover, *P. falciparum* infection causes cerebral malaria; a neurological disorder found in African countries. Generally, according to previous reports, children are the most vulnerable to the brain injury which can even lead to coma, caused by neurological disruption infected by *P. falciparum* parasite (Henry et al, 2012).

**ANTIMALARIAL DRUGS AND DRUG RESISTANCE**

The biggest threat to counteract malaria has been the drug resistance shown by parasites (more specifically by *P. falciparum*). The first line treatment against malaria parasite was designed by giving chloroquine with sulfadoxine/pyrimethamine combination. Chloroquine enters the haemoglobin, followed by protonation, causing acidification and binds with heme group, and ultimately causes lysis of parasite cell (Geleta and Ketema, 2016; Giha et al, 2005). Sulfadoxine/pyrimethamine causes enzyme (DHFR) inhibition in parasite cell, resulting to DNA damage. However, the recent manifestation against anti malarial treatment regimen is resistance to chloroquine. From a genetic point of view, *P. falciparum* parasites become spontaneously multigenic and are conferred primarily by mutations in a transporter (PICRT) gene (Hall AP et al, 1975). This genetic postulation for resistance by *P. vivax* is not employed because the genetic mutation does not occur like PICRT gene. Resistance to few more drugs belonging to quinine derivatives like Amodiaquine, Mefloquine, Piperaquine have developed resistance evidentially by *P. falciparum* (Hart and Naunton, 1964; Heinonen et al, 1977). A treatment schedule was designed by China and that attempt as anti malarial therapy was awarded Nobel prize in 2015, with the application of Artemisinin derivatives along with quinine derivatives and Sulfadoxine/pyrimethamine. Artemisinin causes protein damage of parasite cell by activating free radicals in heme/parasite cell by activating free radicals in heme acidification and bind with heme group, and ultimately causes lysis of parasite cell (Geleta and Ketema, 2016; Giha et al, 2005). Sulfadoxine/pyrimethamine causes enzyme (DHFR) inhibition in parasite cell, resulting to DNA damage. However, the recent manifestation against anti malarial treatment regimen is resistance to chloroquine. From a genetic point of view, *P. falciparum* parasites become spontaneously multigenic and are conferred primarily by mutations in a transporter (PICRT) gene (Hall AP et al, 1975). This genetic postulation for resistance by *P. vivax* is not employed because the genetic mutation does not occur like PICRT gene. Resistance to few more drugs belonging to quinine derivatives like Amodiaquine, Mefloquine, Piperaquine have developed resistance evidentially by *P. falciparum* (Hart and Naunton, 1964; Heinonen et al, 1977). A treatment schedule was designed by China and that attempt as anti malarial therapy was awarded Nobel prize in 2015, with the application of Artemisinin derivatives along with quinine derivatives and Sulfadoxine/pyrimethamine. Artemisinin causes protein damage of parasite cell by activating free radicals in heme group of erythrocytes. The therapy facilitates a better efficacy against *P. falciparum* parasites, because Artemisinin have shorter half life whereas quinine derivatives as combination therapy have longer elimination half-life. However, from a recent survey, acceptance of this therapy is still debatable, because among people from the south east Asian countries, and due to low immunity level in community and greater risk associated with mutation of parasite genotype have conflicted so many arguments (Hodder et al, 2009; Kaiser et al, 2004; Kochar et al, 1995).

**ANTI MALARIAL DRUG IN PREGNANCY**

Evidently, anti-malarial drug Chloroquine causes impaired fetal toxicity during third trimester, due to slow clearance rate from plasma (Korenromp et al, 2003, Lewis and Ponnampalam, 1975). It is also relatable that the awful thread to the African and SE Asian countries as the Chloroquine is becoming resistant to *P. falciparum* parasites, the breakdown of erythrocytes may worsen the fetal health; thus, treatment options are becoming much intricated (Li et al, 2002). Quinine was the first invented anti-malarial drug, without having any reported teratogenicity, but the patient compliance is very much poor after the completion of total course because it causes hypoglycaemia. This can be indirectly harmful to pregnancy (Marsh et al, 1998, Matuschewski et al, 2002) also, some study report on animals revealed that the quinine dose may cause nerve damage in cranial nerve (Miller et al, 2002). Another drug combination which have been popular over the years (sulfadoxine and pyrimethamine), have also shown some dose related embryo toxicity in pregnant rats in animal study. This is a fundamental fact that 5-methyl tetrahydrofolate demethylated to form the active forms of folate (tetrahydrofolate) is independent of enzyme dihydrofolate reductase which is inhibited by pyrimethamine (Mutabingwa et al, 1991; White et al, 1985). However, several studies on human female volunteers clinically postulated that the treatment procedure resulted to an increased risk of malformations, kernicterus or any other severe effect on the fetus (Pauflque and Magnard, 1969; Phillips-Howard and Wood, 1996; Phillips, 1991). Artemisinin derivatives are the recently innovated group of drugs which has been very popular in SE Asia and part of Africa due to the fact that till now there has not been any noticeable resistance by parasites. These derivatives include Artesunate, Artemether, and Arteether used in severe or complicated malaria. But the biggest concern of this drug is regarding the susceptibility towards pregnant population. Few animal studies reported that the high dose formulation produced embryo-fetal toxicity, cardiovascular malformations and skeleton abnormalities (Plowe et al, 2003).

**GENETIC MODIFICATION AS AN APPROACH FOR MALARIA TREATMENT**

One of the recent advanced approach to treat malarial parasite involves determination of specific genes responsible for encoding in protozoans cell and present during pre-erythrocyte and liver stage. The salient feature of this attempt would be tricky and advantageous avoiding drug resistance and minimizing the probable toxicity caused by combination therapy. Few genes (e.g. UIS) are expressed in pre-erythrocytic stage in sporozoites which causes infections in erythrocytes in the mammalian host. However, it can be postulated that, targeting the UIS proteins at erythrocyte levels could be a sharp approach which may lead to attenuation of the liver-stage parasite. In an independent study, a protein
UIS3 was identified which encodes a transmembrane of parasite sporozoites. The protein structure was altered and the alteration could be checked by RT-PCR. This will contribute to the inability of the host-cell invasion capacity (Rustaiyan et al, 2009; Baragaña et al, 2015). Another study was carried out in Kenya, where an attempt was taken to find out genetic diversity and prevalence of malaria drug-resistant mutations in different geographical regions (Schlagenhauf et al, 2004). Different patients, suffering from malaria were randomly chosen based on different treatment groups they are getting. Different genes were analysed (like Poly a, Pfg377, 2490, TA 81, TA 87, Ara2, TA1, PIPK2, Ta109, and TA42) isolated from P. falciparum positive samples (Schultz et al, 1994). PCR was carried out during the analytical phase in specific DNA template, specific volume and given temperature, to obtain proportion of multiclonal infections and number of infections with more than one allele at ≥8. Different mutations were observed emphasizing the drug resistant malaria mutations (Bala et al, 2018). Nine codons in four genes for resistance to chloroquine and Sulfadoxine/ pyrimethamine: pfCRT (K76T), pfmdr1 (N86Y, N1042D, and D1246Y), pfdhfr (N51I, C59R, and S108N), and pfdhps (A437G and K540E). Genomic DNA from P. falciparum clones HB3, W2, and DD2 (MR4, Manassas, VA) were used as positive control. The overall genetic diversity was studied and compared, and the obtained result showed that, Sulfadoxine/pyrimethamine-resistant mutants at the pfdhfr codon 51, the pfdhps codons 437 and 540 were significant (Schlagenhauf et al, 2004). However, modification in the mutation could be a remarkable approach to counter drug resistant malaria (Schultz LJ et al, 1994- Sourav et al, 2018).

Approaches with phytomolecules as anti-malarial treatment

Choice of phytoconstituents instead of synthetic compounds was always preferable to treat diseases, as it causes less toxicity and cheaper too (Sourav et al, 2018, Asis et al, 2017, Naskar et al, 2011). Due to geographical variance and genetic manifestation, a lot of synthetic compounds have grown resistance. There are also few groups of drugs which can be harmful to pregnant women (Mohammed et al, 2018) Thus, scientists are in search of such molecule which should have moderate safety profile and also should be new entities that would not show resistance. The basic mechanism of action of the plant extract or molecule should be concentrated on how the growth of parasite is inhibited or the mechanism of transforming the biochemistry which in turn causes death of parasite (Schultz et al, 1994). Previously, in the mid eighteenth century, the revolutionary discovery by French scientist was the isolation of alkaloidal moiety of quinine from Cinchona species of plant (Sutherland et al, 2010). Afterwards, it was found that quinine derivatives developed resistance, in the SE Asian and few parts of African countries. Another invention of active compound of Artemisinin from Artemisia annua was a breakthrough in medical research, which was useful in condition where there was chloroquine resistant malaria (Plowe et al, 2003). It was currently found that, in two different studies (Sutherland et al, 2010; Verhoeff et al, 1998) Artabotrys hexapetalus, a plant along with another plant found in Iran (Artemisia diffusa) contains endoperoxide; a compound from sesquiterpene group is present which provides a synergistic action as anti malarial treatment when used in combination with chloroquine. However, the chloroquine induced resistance against P. falciparum can be overcome and several in vivo studies revealed that it showed relatively low toxicity (West and Wichita, 1938; Naskar et al, 2011).

Modern emerging tools to treat malaria

New therapeutics invention has sparked spontaneous interest among scientists, and still there is so much scope on malarial drug research. As a new therapeutic tool, a specific papain-like proteins SERA and its analogs was targeted. From a backround study, the P. falciparum invades erythrocytes very rapidly, where specific proteins play a key role in parasite life cycle. A specific antigen SERA, is expressed in malarial cell parasitophorous vacuole, which protects parasites from host cell phagolysosome. SERA5 an analogue of SERA is broken down by SUB1 enzyme during asexual blood stage (White et al, 1999; White et al, 2004; Ruecker et al, 2012). Disruption of both SERA4 and SERA5 proteins causes impaired replication, as well as invaded rupture of host cell (World Health Organisation, 2015). This strategy of modification of SERA proteins could be a significant tool to control malaria.

Another approach was attempted (Wright et al, 2009) where a new molecule was designed (DDD107498), whose molecular mechanism of action was unique from other anti malarial drugs. This reportedly can show activity against different life cycle stages of different malarial parasites. The molecule targets a specific translation of protein elongation factor 2 (eEF2), which enables translocation of ribosome analogue messenger RNA, which is an important tool for protein synthesis.

Conclusion

As medical research is developing rapidly by the course of time, new molecules and strategies are being employed to explore more opportunities. Malarial research has been one of such concern for so many years. The different species of parasites is modifying their genetic morphology predominantly, and is randomly challenging the older treatment options. Drug resistance
have also been observed as another issue. Some drugs cause toxicity in individuals. Thus, new treatment remedies with phytocompounds or gene-based therapy would be beneficial.

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

REFERENCES


PMID: 22984267; PubMed Central PMCID: PMC3488066
Related Journals:

- Clinical Reviews and Opinions
- Journal of Medicinal Plant Research
- African Journal of Pharmacy and Pharmacology
- Journal of Dentistry and Oral Hygiene
- Journal of Parasitology and Vector Biology
- Journal of Pharmacognosy and Phytotherapy
- Journal of Medical Laboratory and Diagnosis
- Journal of Diabetes and Endocrinology
- Medical Practice and Reviews

www.academicjournals.org