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RESEARCH ARTICLE

Malaria and Modes of Transmission

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ABSTRACT

Background: Malaria is one of the most significant parasitic diseases, with a history dating back to ancient times, especially in tropical and subtropical regions. It remains a serious public health challenge today. The disease is caused by the *Plasmodium* parasite and is transmitted to humans through the bite of an infected female *Anopheles* mosquito. Clinical manifestations include intermittent fever, chills, anemia, splenomegaly, and, in severe cases, multiple organ failure and death. The growing resistance of the parasite to antimalarial drugs and the resistance of mosquitoes to insecticides have made disease control increasingly difficult. **Objective:** This study aims to examine the parasite's life cycle, transmission methods, clinical symptoms, and factors influencing malaria prevalence to propose effective strategies for its prevention and control. **Methods:** This study is a review based on the analysis of scientific articles, specialized books, reports from the World Health Organization, and other credible sources. Data were systematically collected from scientific databases and selected based on their relevance and credibility. **Results:** Malaria is caused by four *Plasmodium* species: *Plasmodium ovale*, *Plasmodium vivax*, *Plasmodium malariae*, and *Plasmodium falciparum*, with *P. falciparum* being the most pathogenic and lethal. The parasite's life cycle includes two main stages: Schizogony in the vertebrate host and sporogony in the mosquito vector. While the primary mode of malaria transmission is through *Anopheles* mosquito bites, alternative transmission routes include blood transfusion, organ transplantation, contaminated needles, and congenital transmission. Environmental factors such as temperature, humidity, and water sources, along with human factors such as race, age, occupation, and socioeconomic status, significantly influence malaria prevalence. **Conclusion:** Malaria remains a major global health threat. Effective control requires a multifaceted approach, including insecticide application, bed net utilization, stagnant water management, rapid diagnosis, and appropriate treatment. International collaboration, vaccine development, improved antimalarial drugs, and increased public awareness are crucial in reducing malaria transmission. Ultimately, with scientific advancements and comprehensive public health interventions, the incidence of malaria can be significantly reduced, and eradication may become a realistic goal in the future.

Keywords: *Anopheles*, malaria, modes of transmission, schizogony, sporogony

INTRODUCTION

Malaria is the most significant parasitic disease affecting humans and one of the oldest known human infections, with documented cases dating back over 3,000 years in ancient civilizations such as Iran, India, Egypt, and China.[1,2] The incidence of malaria is directly linked to factors such as forests, population migration, environmental sanitation conditions, and numerous other issues. It remains an endemic disease in many parts of the world, particularly in tropical and subtropical regions.[3-5] The probable origin of malaria is Africa, from where it spreads globally with human migration during the Neolithic era.[6] Clinically,

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malaria typically presents as an acute infection, which in most cases is severe and occasionally prolonged, characterized by intermittent fever, chills, anemia, and splenomegaly. It may also manifest with mild symptoms or severe, life-threatening complications.[7,8] The significance of malaria stems from its high prevalence and considerable mortality rate. The disease is transmitted to humans by mosquitoes of the *Anopheles* genus, which includes several species.[9] The term "malaria" originates from the Italian words Mal-Aria, meaning "bad air," reflecting the historical belief that the disease was caused by exposure to foul air in marshy areas.[1,2,8,9] Hippocrates classified malaria based on its fever patterns, coining terms such as "quotidian fever," "tertian fever," and "quartan fever" to describe its clinical presentation.[1]

Global warming has been associated with an increased prevalence of malaria.[9] At present, malaria is endemic in over 100 tropical countries, including Iran. A major concern in the modern era is the growing resistance of *Plasmodium* species to conventional antimalarial drugs and the increasing resistance of *Anopheles* mosquitoes to insecticides.[6] Throughout history, different names have been used to describe malaria in various regions, including "intermittent fever," "fever and chills," "Roman fever," "tropical fever," and "coastal fever."[8-10]

There are four species of *Plasmodium* that cause malaria in humans, discovered between 1880 and 1922. However, their complete life cycle and detailed characteristics remained unknown until 1948.[1] Malaria infection begins with the bite of an *Anopheles* mosquito. The female *Anopheles* mosquito, which feeds at night, transmits *Plasmodium* parasites into the human bloodstream through its saliva while taking a blood meal.[6] Once inside the body, the parasites travel to the liver, where they rapidly multiply.[6] Each invading parasite can produce approximately 10,000 new parasites within liver cells.[6] As the parasite population increases, the infected liver cells rupture, releasing thousands of parasites into the bloodstream.[6] These parasites then invade red blood cells (RBC), where they continue their replication cycle.[6] Inside the RBCs, *Plasmodium* parasites consume hemoglobin as a nutrient and multiply. Within 2–3 days, the hemoglobin is depleted, and the infected RBCs become packed with parasites. At this stage, the RBCs rupture, releasing a new wave of parasites into the bloodstream, which then invade fresh RBCs, perpetuating the cycle.[6] When the parasite burden reaches a critical level, the patient develops fever. The onset of fever coincides with the rupture of infected RBCs, and as the parasites invade new RBCs, the fever subsides. This cycle repeats every 2 or 3 days, leading to a characteristic pattern where the patient remains fever-free for 1 or 2 days before experiencing another fever episode on the 3rd day.[6] The life cycle of *Plasmodium* consists of two stages. First, an infected human transmits the parasite to a female *Anopheles* mosquito during a blood meal. Inside the mosquito's gut, the parasite undergoes reproduction and multiplication before migrating to the mosquito's salivary glands. The parasite remains there until the mosquito takes another blood meal, at which point it is transmitted to a new human host, initiating a new cycle of infection.[1,2,6,9]

Ambient temperature plays a crucial role in the transmission of malaria. In warm climates, *Plasmodium* parasites within the *Anopheles* mosquito rapidly reproduce and develop, becoming infectious more quickly and increasing the likelihood of transmission to healthy individuals. In contrast, in cooler temperatures, the parasite's replication and maturation within the mosquito take significantly longer. Given that mosquitoes have a relatively short lifespan, many do not survive long enough to complete the parasite's development cycle, leading to a slowdown or interruption in malaria transmission.[6]

METHODS

This study is a review article that examines malaria and its transmission methods. Data collection was conducted through the analysis of credible scientific sources, including peer- reviewed journal articles, specialized textbooks, World Health Organization (WHO) reports, and other relevant scientific documents. The process of literature search and source selection was carried out as follows:

Findings

Malaria symptoms

Patients with malaria experience a wide range of symptoms throughout the course of the disease. The most common clinical manifestations include:

1. Intermittent fever (ranging from mild to high, often accompanied by chills)
2. Sudden and severe chills
3. Profuse sweating
4. Severe headache
5. Generalized muscle pain
6. Nausea and vomiting, sometimes accompanied by diarrhea
7. Extreme fatigue and generalized weakness
8. Abdominal pain
9. Shortness of breath
10. Confusion and delirium
11. Seizures
12. Anemia
13. Jaundice (yellowing of the skin and eyes).[1-3,5,6,9-11]

The intensity and type of malaria symptoms can vary among individuals. The onset of the disease is accompanied by fatigue, weakness, headache, and body aches, which closely resemble common viral infections such as the flu. Before treatment begins, patients experience high and periodic fevers (sometimes exceeding 40°C) along with chills, which can persist for several hours before subsiding. This rise in body temperature serves as a defense mechanism to eliminate malaria parasites, as they cannot survive in high temperatures. Another defense response of the body is splenomegaly (enlargement of the spleen), which aids in the destruction of infected RBCs, ultimately leading to anemia. In cases of severe malaria, infected RBCs develop protrusions that cause them to adhere to each other and to the vascular endothelium, obstructing blood flow. Cerebral vessel occlusion can result in drowsiness and coma. In addition, one of the complications of malaria is hypoglycemia, which is particularly dangerous in children and pregnant women, potentially leading to severe complications and mortality.[6]

Types of Malaria Parasites (*Plasmodium* Species)

Plasmodium vivax

This is the most prevalent species of *Plasmodium* worldwide and is widely distributed in endemic regions and temperate zones. *P. vivax* primarily invades larger, immature, and hypochromic RBCs. The disease caused by this parasite is referred to as benign tertian malaria (malaria with a 48-h cycle).

Plasmodium falciparum

P. falciparum can infect all stages of RBCs, and in severe infections, ring forms and gametocytes can be observed in peripheral blood smears. A hallmark feature of this species is the presence of multiple rings within a single RBC and double-chromatin ring forms. *P. falciparum* malaria is almost exclusively found in tropical and subtropical regions and is responsible for malignant tertian malaria, which is more severe and life-threatening.

Plasmodium malariae

This species is less prevalent than *P. falciparum* and *P. vivax* and is commonly found in subtropical and temperate regions. It primarily infects smaller RBCs and causes benign quartan malaria (malaria with a 72-h cycle), distinguishing it from other species, which typically exhibit a 48-h erythrocytic cycle.

Plasmodium ovale

Predominantly found along the West African coast, *P. ovale* is responsible for benign tertian malaria. Similar to *P. vivax*, it preferentially invades immature RBCs but at a lower frequency. Infected RBCs often appear oval-shaped with irregular, serrated edges, resembling a comet-like structure. The parasitized RBCs exhibit pallor, and *P. ovale* infections are associated with a higher likelihood of spontaneous recovery.[1-3,5]

Life Cycle of Malaria Parasites (*Plasmodium* spp.)

Among the 483 *Anopheles* mosquito species identified worldwide, approximately 70 are capable

of transmitting malaria. Of these, 40 species serve as primary malaria vectors in different regions. In Iran, 29 *Anopheles* species have been recorded, with 7 species confirmed as definitive vectors of malaria.

The *Plasmodium* life cycle occurs in two hosts: A vertebrate and an invertebrate. The asexual cycle, known as schizogony, takes place in the vertebrate host, while the sexual cycle, which culminates in sporogony within the invertebrate host, is referred to as sporogony.

Sporogony

Various species of *Anopheles* mosquitoes serve as the definitive hosts for *Plasmodium*, the causative agent of malaria. When a female *Anopheles* mosquito feeds on an infected individual, both male (microgametocytes) and female (macrogametocytes) gametocytes may be ingested along with the blood. Unlike other parasite forms, gametocytes are not digested in the mosquito's midgut; instead, they undergo a maturation process known as gametogonia, preparing them for fertilization. In microgametocytes, this maturation process is termed exflagellation, during which the nucleus divides into multiple smaller nuclei, each migrating to the periphery of the microgametocyte. The cytoplasm then divides, giving rise to 8–12 slender, flagellated male gametes, known as microgametes. Simultaneously, the female macrogametocyte matures into a macrogamete. Following this process, fertilization occurs when a microgamete fuses with a macrogamete, forming a zygote. Approximately 12–24 h after the mosquito's blood meal, the zygote transforms into a motile, elongated ookinete. The ookinete penetrates the midgut wall and lodges beneath its outer lining, developing into an oocyst. As the oocyst matures, it produces thousands of sporozoites, which are thread-like infectious forms. Upon rupture of the oocyst, sporozoites are released into the mosquito's hemolymph and eventually migrate to the salivary glands, making the mosquito capable of transmitting malaria. The duration of the sporogony cycle varies among *Plasmodium* species: *P. vivax*: ~8 days. *P. falciparum*: ~11–12 days. *P. malariae*: ~35 days. In addition to parasite species, factors such as the *Anopheles* mosquito type and environmental temperature influence the length of the sporogonic cycle. For instance, the minimum temperature required for sporogony in *P. vivax* and *P. falciparum* is 16°C and 18°C, respectively. This explains why *P. vivax* can persist in temperate regions.

Schizogony

Upon the mosquito's blood meal, sporozoites enter the human bloodstream and tissues, initiating the exoerythrocytic (pre-erythrocytic) cycle. Within 20–40 min of inoculation, infectious sporozoites exit circulation and invade hepatocytes. This marks the beginning of the exoerythrocytic stage, as RBCs have not yet been targeted. Within liver cells, asexual replication of schizonts leads to the production of thousands of delicate merozoites, which are eventually released into the bloodstream. The erythrocytic cycle begins when these merozoites invade RBCs. Inside the RBCs, merozoites mature into trophozoites, which subsequently develop into schizonts. Upon further replication, RBCs rupture, releasing newly formed merozoites that continue infecting other erythrocytes. The intraerythrocytic schizogony lasts between 48 and 72 h, yielding 4–36 new parasites per infected RBC. The rupture of infected RBCs leads to the release of parasitic metabolic byproducts, contributing to malaria symptoms. In the early stages of infection, RBC rupture is asynchronous, meaning the number of toxic substances released into circulation is insufficient to trigger a full malarial paroxysm. Consequently, fever may appear irregular or intermittent rather than following a strict cyclic pattern. However, as the infection progresses, the developmental cycles of the parasites become synchronized, leading to simultaneous RBC rupture and the characteristic periodic febrile episodes, occurring every 48–72 h, depending on the *Plasmodium* species. In mixed infections, fever episodes may occur daily or even twice per day due to overlapping schizogony cycles. Notably, some merozoites differentiate into gametocytes before the onset of overt clinical symptoms. These gametocytes appear morphologically similar to asexual merozoites but do not undergo further division. Instead, they mature into male (microgametocytes) and female

(macrogametocytes) gametocytes, which circulate freely in the bloodstream. When ingested by a mosquito during a blood meal, they undergo gametogonia and sporogony within the mosquito's gut, completing the malaria transmission cycle. Merozoite invasion of RBCs requires the recognition of specific surface receptors on the erythrocyte membrane. The apical end of the merozoite must properly orient itself before invasion. Specialized organelles within the merozoite facilitate this process, inducing structural changes in the RBC membrane. Subsequently, the merozoite enters the RBC through an invagination of the erythrocyte membrane, ensuring successful intracellular parasitism.

Complications of Malaria

1. Miscarriage and preterm birth: In pregnant women, malaria can lead to miscarriage, preterm labor, and low birth weight in newborns.
2. Low birth weight and malnutrition: In children, malaria may cause low birth weight, malnutrition, and growth impairment.
3. Cerebral malaria: This is the most common and severe complication of *P. falciparum* infection and the leading cause of malaria-related mortality. Cerebral malaria may manifest suddenly, sometimes as the first symptom of infection. In severe cases, *Plasmodium* parasites invade the brain, causing seizures, abnormal drowsiness, coma, and ultimately death. In some instances, coma is the only prominent symptom. Long-term neurological complications may include cortical blindness, hemiparesis, generalized seizures, and hypotonia (muscle weakness and loss of function).
4. Neurological damage: Malaria can cause temporary or permanent neurological and muscular impairment.
5. Severe anemia: Severe anemia results from high parasite loads of *P. falciparum*. If hematocrit drops below 20% and parasitemia reaches 5% or higher, immediate treatment is required. Care must be taken to prevent fluid overload and pulmonary edema.
6. Renal disease: Acute renal complications are common in severe *P. falciparum* malaria. Chronic infections with *P. malariae*, particularly in children, may also contribute to kidney dysfunction. Acute renal failure can occur during severe *P. falciparum* attacks, likely due to tubular necrosis from RBC sequestration and renal hypoxia. However, renal function usually recovers once the infection is controlled.
7. Metabolic acidosis: Clinical manifestations include elevated serum lactate, arterial blood gas disturbances, and deep, rapid breathing (Kussmaul respiration).
8. Severe anemia: Marked by normochromic-normocytic anemia, defined as hemoglobin levels below 5 mg/dL.
9. Pulmonary edema: In malaria, lung tissues become pigmented, and capillaries contain pigmented leukocytes, phagocytes, and infected RBCs. Acute pulmonary edema can arise from severe anemia, high parasitemia, or cerebral malaria, leading to respiratory distress and lung congestion. In *P. falciparum* malaria, pulmonary edema may also result from bacterial sepsis, excessive fluid administration, or acute respiratory distress syndrome.
10. Hypoglycemia: Elevated insulin levels may result from treatment with quinine or quinidine, leading to hypoglycemia, which is particularly dangerous in the third trimester of pregnancy, contributing to fetal distress or stillbirth. Hypoglycemia has also been observed in adults with severe *P. falciparum* malaria who were not treated with quinine.

Algid Malaria (Shock Malaria)

This term describes severe *P. falciparum* malaria, characterized by sudden hypotension and vascular permeability disturbances. It presents with a rapid drop in body temperature, altered mental status, and delirium. This syndrome may be triggered by Gram-negative sepsis, pulmonary edema, severe gastrointestinal bleeding, splenic rupture, or dehydration.

1. Choleric malaria: This form presents with vomiting and persistent coughing.

2. Malarial dysentery: A rare but severe complication of *P. falciparum* malaria, characterized by bloody diarrhea, abdominal pain, dizziness, vomiting, and upper gastrointestinal bleeding. It may be associated with focal ischemic changes in the intestinal vasculature. In some cases, patients develop a tender, enlarged liver, jaundice, and bile-stained urine (referred to as fluctuating biliary fever). Malabsorption and reduced hepatic blood flow have also been observed in severe *P. falciparum* malaria.
3. Tropical splenomegaly syndrome (TSS): During acute malaria episodes, the spleen enlarges but later returns to its normal size. Persistent splenomegaly is uncommon in adults. However, idiopathic splenomegaly is frequently observed in tropical malaria-endemic regions, historically recognized as TSS.
4. Hyper parasitemia: The presence of high parasite loads (10–20% of RBCs infected) is associated with high mortality rates, even with prompt and intensive treatment. In cases of cerebral malaria, parasitemia levels as low as 5% can result in severe complications.[1-3,5,9]

Modes of Malaria Transmission

The transmission and prevalence of malaria, whether in endemic regions or during epidemic outbreaks, are influenced by numerous factors, including *Plasmodium* species and strain, human immune response, mosquito vector behavior and ecology, human lifestyle and habits, environmental conditions such as temperature, humidity, rainfall, and vegetation; vector control strategies and interventions, these factors can be categorized into three key components: The infected and susceptible human hosts (as recipients and transmitters of the disease), the *Plasmodium* parasite (as the causative agent), and the *Anopheles* mosquito (as the biological vector). Malaria can be transmitted through the following routes:

1. Natural transmission: Transmission through the bite of an *Anopheles* mosquito
2. Acquired transmission: Transmission through inoculation.

Natural Transmission: (Transmission through the bite of an *Anopheles* mosquito)

The primary mode of malaria transmission is through the bite of an infected female *Anopheles* mosquito. These mosquitoes are found in most temperate and tropical regions, wherever suitable habitats for them exist. The transmission process occurs as follows: when a female mosquito feeds on the blood of an infected person, it ingests the parasite. After a specific incubation period within the mosquito's body, the parasite transforms into an infective form, which is then transmitted to a healthy person during the mosquito's subsequent blood meal through its saliva. This cycle continues until it is interrupted by a specific factor. For mosquitoes to become infected, not only is a suitable vector required, but also a human host with a sufficient number of gametocytes in their blood is also necessary. Therefore, individuals who have recently been infected cannot transmit malaria.

Acquired Transmission (Transmission through Inoculation)

When malaria is transmitted by means other than the bite of an *Anopheles* mosquito, it is referred to as inoculated malaria. In regions where malaria transmission by *Anopheles* mosquitoes occurs, but where natural transmission has been interrupted, malaria can still spread through carriers of the parasite. If the malaria-causing parasite is *P. falciparum* and is not diagnosed and treated in time, it can lead to the death of the patient. The mechanism of this type of transmission is as follows: In individuals who have malaria, if adequate or complete treatment has not been given, after developing relative immunity, the parasite count in the blood decreases to a point where clinical symptoms do not manifest. Sometimes, the parasite load in the blood becomes so low that it cannot be detected in a routine microscopic blood smear. However, if the blood of these individuals, who are carriers of the parasite, is inoculated into healthy individuals, the schizont stage parasites in the RBCs begin to multiply, and symptoms of malaria appear in the recipient. Inoculated malaria includes the following cases:

1. Transmission through blood transfusion
2. Transmission through organ transplantation
3. Transmission through malaria therapy
4. Transmission through hemotherapy
5. Transmission from mother to fetus
6. Transmission through contaminated needles
7. Accidental transmission (occupational exposure).[1,6,9]

Human Factors Influencing Disease Transmission

These include race, age, gender, and occupation, which all play a role in disease transmission.

Race

Black individuals are less susceptible to *P. vivax* malaria than Caucasians, and this resistance is closely related to the absence of the Duffy blood group antigen in these individuals. The RBCs of individuals with Duffy-negative blood groups are resistant to *P. vivax*, while Duffy-positive RBCs are more prone to infection. In West Africa, the Duffy-negative phenotype is found in 90% of the native population.

Age

Although malaria is more prevalent in children than adults in areas with high transmission rates, the parasitemia in children under 6 months is less than that in children aged 6 months and older. This is likely due to maternal antibodies or possibly abnormal hemoglobin (such as fetal hemoglobin) or breastfeeding.

Gender and occupation

Gender does not directly influence susceptibility or resistance to malaria, but occupation or attire may have an impact. In some areas, women, unlike men, must wear full clothing at night, or men are more exposed to mosquito bites due to their occupations (such as farm guarding or fishing), which increases their risk of being bitten by mosquitoes.[6,9]

Environmental Factors Affecting Disease Transmission

These include physical, biological, social, and economic environments that play a role in disease transmission.

Physical environment

This includes factors such as temperature, humidity, rainfall, and surface water availability, all of which impact the epidemiology of the disease. The sexual cycle of *P. vivax* does not occur at temperatures below 16°C, and the sexual cycle of *P. falciparum* does not occur below 19°C. Temperature also affects the duration of the sexual cycle. For instance, the sexual cycle of *P. vivax* lasts 50 days at 16°C and only 9 days at 26°C. Relative humidity influences the lifespan and activity of mosquitoes, with transmission risk decreasing if the relative humidity is below 60% at 8 a.m.

Biological environment

Factors such as the presence of specific plants in mosquito breeding sites or the presence of livestock in the area play a significant role in the proliferation of disease vectors and the subsequent transmission of malaria.

Social-economic environment

Social and economic characteristics, along with the daily activities, habits, and customs of people, influence disease transmission. Factors such as where people live, the installation of mosquito nets and screens, and the use of bed nets can reduce the risk of malaria. Illiteracy and poor economic conditions are contributing factors that increase disease incidence. Poor nutrition can impair the body's ability to combat symptoms, leading to higher rates of relapse. Wars also contribute to an increase in malaria cases. Migration, transportation, water resources and their usage, housing conditions, and building infrastructure all play crucial roles in the prevalence of the disease.[9]

DISCUSSION AND CONCLUSION

Malaria is a significant parasitic disease whose transmission is influenced by various factors, including the biological characteristics of the parasite, the behavioral habits of the vector mosquito, environmental conditions, and human factors. The disease can be transmitted through two main routes: natural transmission (through the bite of an infected *Anopheles* mosquito) and acquired transmission (through the inoculation of contaminated blood). Human factors such as race, age, gender, and occupation play a crucial role in the severity and prevalence of the disease. For instance, individuals with Duffy-negative blood groups have higher resistance to *P. vivax*. In addition, environmental conditions such as temperature, humidity, rainfall, and the presence of water sources are vital in the survival and multiplication of the disease vector. Furthermore, social and economic factors, including housing conditions, literacy, nutrition, and migration, influence the disease transmission patterns. Consequently, controlling malaria requires a comprehensive approach that involves preventive measures, rapid diagnosis, effective treatment, and improvements in environmental and social conditions.

Malaria remains one of the most serious diseases in many parts of the world, especially in tropical and subtropical regions. This disease, caused by *Plasmodium* parasites and transmitted through the bite of infected mosquitoes, can lead to severe complications, including anemia, organ failure, and even death. Despite advancements in medical and healthcare technologies, many regions continue to struggle with the disease.

Fever and headache are among the most common symptoms of malaria, with 35.1% of malaria cases reported from neighboring countries. Preventive measures such as the use of bed nets, spraying, the use of cooling devices, proper management of stagnant water, as well as early treatment, history of previous malaria infections, and education level are key preventive factors. Travel history within the last month is also identified as a major risk factor for the disease. Based on the findings of studies and the preventive measures reviewed, it can be concluded that the most important and effective approach for combating malaria is the integrated management of the vectors, using multiple preventive methods. Ongoing research into the development of effective vaccines and the improvement of antimalarial drugs holds promise for better control of the disease in the future. Furthermore, international collaborations and the efforts of health organizations such as the WHO and the Global Fund to Fight Acquired Immunodeficiency Syndrome, Tuberculosis, and Malaria play a key role in reducing malaria-related morbidity and mortality. Raising public awareness, providing health education, and improving access to healthcare facilities and medications can significantly contribute to controlling this disease.

Ultimately, the fight against malaria requires a multifaceted approach, including prevention, early diagnosis, effective treatment, and vector control. With continued scientific research and the application of novel methods, there is hope that, in the near future, malaria prevalence will decrease, and the disease will eventually be eradicated.

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