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Narrative Review of Co-Infection: Malaria in the Context of HIV, Tuberculosis, and Helminth Infections

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ABSTRACT

Malaria co-infection with HIV, tuberculosis (TB), and helminths presents a complex clinical and public health challenge, particularly in sub-Saharan Africa where all four infections are highly endemic. This narrative review synthesises existing evidence on the epidemiology, clinical outcomes, prevention, and research priorities concerning malaria co-infection with these major pathogens. Findings reveal that HIV infection compromises host immunity, exacerbating malaria severity and recurrence, while malaria infection accelerates HIV replication and progression. Co-infection with helminths produces contrasting effects, sometimes reducing malaria severity but increasing chronic susceptibility. Evidence on malaria–TB interactions remains limited, though coinfected individuals often present with altered clinical manifestations and poorer prognoses. The overlapping epidemiology and shared social determinants poverty, poor sanitation, and weak health systems compound disease burden and hinder effective management. Integrated prevention and control approaches, including combined vector control, harmonised diagnostic and treatment protocols, and strengthened health education, have shown promising results but remain under-implemented. Major challenges include poor adherence, drug resistance, and fragmented service delivery. Research gaps persist in understanding co-infection pathogenesis, immune mechanisms, and pharmacological interactions. Advanced methodologies such as geostatistical modelling, cohort studies, and multi-disease surveillance systems are needed to capture disease interactions at population and molecular levels. The review concludes that a unified One Health approach linking human, animal, and environmental health together with sustainable financing, innovation, and coordinated governance, is critical to mitigating the burden of malaria co-infection and improving global health outcomes.

Keywords: Malaria co-infection; HIV/AIDS; Tuberculosis (TB); Helminth infections; and One Health.

INTRODUCTION

In many malaria-endemic regions, particularly sub-Saharan Africa, co-infection with HIV, tuberculosis (TB), or helminths is highly prevalent. The epidemiological landscapes of malaria, HIV, tuberculosis, and helminth infections (e.g., schistosomiasis, soil-transmitted helminths, lymphatic filariasis) intersect in diverse contexts, with high levels of co-occurrence recorded at individual, household, and community levels [1-5]. The dynamic overlap between these infections has profound implications for public health, yet detailed consideration of specific co-infection combinations has been limited. Integrated approaches that incorporate malaria alongside HIV, tuberculosis, and helminths are critical to informing both clinical care and policy advocacy. Systematic epidemiological reviews indicate that these infections share socio-economic drivers, particularly poverty, urbanisation, and limited access to care [6-10]; a complementary analysis of pathophysiological mechanisms highlights their capacity to modulate each other's course [11-13]. Malaria, caused by parasitic protozoa of the genus Plasmodium (the most significant species are P. falciparum and P. vivax), is transmitted to humans through the bite of infected female anopheline mosquitoes [14-18]. The incubation period typically lasts from 7 to 30 days, depending on the species. Diagnostic confirmation by microscopy or rapid diagnostic tests is recommended before treatment initiation. In many endemic countries, malaria remains endemic, and the World Health Organization

estimates that 619 million clinical episodes and 627,000 deaths (primarily of children) occurred worldwide during 2021 [19-23]. Infection with the human immunodeficiency virus (HIV) leads to immune deficiency and acquired immunodeficiency syndrome (AIDS). HIV progresses through several clinical stages, beginning with an initial acute phase, followed by an asymptomatic stage that can last a decade or longer and finally AIDS. Infection is most commonly diagnosed through antibody detection [24-30].Tuberculosis (TB) is an infectious disease caused by the bacterium Mycobacterium tuberculosis, which primarily affects the lungs [31-35]. The time from infection to disease can span a few weeks to several years; however, infection may lead to a dormant state (latent TB) that presents no symptoms and is not transmissible. Diagnosis relies on microbiological confirmation, radiological examination, or use of clinical algorithms. In 2021, the World Health Organization reported nearly 10.6 million new TB cases and 1.6 million deaths globally, highlighting the need for expanded screening among vulnerable populations [36-39]. Helminths are multicellular parasites that inhabit the gastrointestinal tract, blood vessels, or tissue spaces [40-43]. Commonly encountered helminths include soil-transmitted helminths (Ascaris lumbricoides, Trichuris trichiura, and hookworm), Schistosoma species, and lymphatic filaria (Wuchereria bancrofti). Tuberculosis and helminth infections represent a much lower risk of morbidity or mortality among patients with well-managed malaria and HIV, even in cases of co-infection where cross-conditions exist [44-50]. A comprehensive review of clinical outcome signals across combinations can refine understanding of each interaction's relative impact. Programme constraints limiting access to antimalarial, antiretroviral, and anti-TB therapies are critical barriers to control; a recent global survey identified adherence problems (e.g., stockouts, late treatment initiation) as the principal obstacle for people with co-infection. Modulation of HIV and TB pathology co-infection drugs is another area of emphasis. A recent study in Côte d'Ivoire highlighted antagonistic effects of helminth and Plasmodium co-infections on malaria-related protection and fatality risk, suggesting increased risk following helminth deworming [51-56].

Rationale for Studying Co-Infections

Infection with Plasmodium spp., the causative agent of malaria, remains a major global health challenge [57-60]. An estimated 247 million new infections and 619,000 deaths occurred in 2021, almost entirely in low- and middle-income countries (LMIC), with 95% in Africa alone (WHO, 2023) [5]. In several regions with high malaria prevalence, co-infection with either human immunodeficiency virus (HIV), Mycobacterium tuberculosis (TB), or helminth parasites (e.g., Schistosoma, hookworm) represents additional disease burden on susceptible populations and has been associated with enhanced malarial morbidity [61-62]. These pathogens share several cross-cutting risk factors and interact along the clinical, therapeutic, and programmatic dimensions, which introduces both ambiguity and insight in epidemic response planning [3]. In settings where these pathogens co-occur, formal co-infection studies may never progress beyond initial exploratory analysis because cross-sectional surveys alone cannot disentangle sharing and impact determination from sampling in similarly vulnerable groups [4]. Parallel longitudinal measurements have indicated cumulative risk factors and therefore pathogen associations whose consistency and geographic coherence support an interdisciplinary integration of both pathogens alongside, or ultimately independent of, malaria [10].

Scope and Definitions (malaria, HIV, TB, helminths)

Malaria is defined as an infection of Plasmodium species, traditionally comprising P. falciparum, P. vivax, P. ovale, and P. malariae, but also including lesser-known zoonotic agents from simian hosts (primarily P. knowlesi) and rodents (P. eylesi, P. grranti) [4]. The term HIV encompasses the human immunodeficiency viruses HIV-1 and HIV-2, and TB refers to infection caused by Mycobacterium tuberculosis complex organisms (M. tuberculosis, M. bovis, M. bovis BCG, M. africanum). Helminths include nematodes (roundworms), cestodes (tapeworms), and trematodes (flukes) and share wating-associated lifecycles involving contact with infected water [6]. Co-infection with other malaria genera and species (particularly Plasmodium knowlesi), or with Leptospira, Rickettsia, giardia, Schistosoma, or Strongyloides, is recognized as warranting study and comment but has generally been excluded from quantitative summaries owing to scarce data [6]. The focus is primarily on malaria and Plasmodium falciparum in areas and settings bracketed by high burden from the other three pathogens: HIV in sub-Saharan Africa; TB in South Africa, India, and Southeast Asia; and helminth infection in tropical and/or impoverished regions [7]. This concentration allows the integration of longitudinal and cross-sectional evidence, calling attention to associated research gaps and strengthening policy- and care-related conclusions. Co-infections with high burden or known to influence clinical care are addressed, and previous reviews examining their effects on incidence, prevalence, diagnosis, and treatment provide the background [9].

Epidemiology and Burden

Co-infections remain a problematic public health issue despite years of study. Among them, malaria co-infection with human immunodeficiency virus (HIV), mycobacterium tuberculosis (TB), and helminths represents one of the most pressing issues in public health today [2]. Each of the three pathogens is emergent within the epidemiology

of malaria and in nearly every geographic region where malaria exists [1]. Malaria alone is responsible for an estimated fifty-eight million cases annually, leading to an estimated 627,000 deaths [5]. The impact of each pathogen is increased due to the unique gender, sexual, and socioeconomic misunderstandings surrounding them [6]. Countries afflicted with malaria, HIV, tuberculosis, and helminths suffer co-infection with regional variation. The epidemiological study of co-infection supports the calls for more integrated services, whether through coherent delivery of prevention messages or focused expertise on disease interactions [7]. Tackling these accents provides an opportunity to ameliorate the plight of six hundred million people at risk of malaria and forty-four million people with existing infections [8].

Global and Regional Prevalence

In endemic regions worldwide, malaria remains a leading cause of morbidity and mortality. In tandem, a variety of infectious diseases have emerged with similar epidemiological profiles, resulting in significant co-infection dynamics at both the individual and population levels [8]. Countries with a high burden of malaria are also often heavily afflicted by human immunodeficiency virus (HIV) infection, tuberculosis (TB), and helminth parasites. Congruently, these diseases have been identified in recent decades as the four highest-priority global public health threats, yet their interactions are poorly understood and neglected in the medical literature [1]. This narrative review synthesizes the relationship of co-occurring malaria infections with HIV, TB, and helminths in the context of pathophysiology, clinical presentation, treatment, epidemiology, and broader health systems. Insights gleaned from dedicated studies on all four pathogens indicate an integrated approach to prevention and management of co-infection would enhance the quality of life for the vulnerable groups and communities most affected by these interrelated diseases [9]. Co-infections of malaria with multiple diverse pathogens remain understudied, especially in developing economies heavily burdened by these diseases. Reports published in 2015 and 2019 identified substantial co-infections of malaria with both HIV and helminths in sub-Saharan Africa, yet a more recent overview of concurrent management between malaria and HIV reported a lack of extensive prevalence data for both pathogens in any region [5]. Limited co-infection data are available at sub-country scales, where only select zones are designated as high-burden. Human interactions with other vector-borne diseases, waterborne diseases, neglected tropical diseases, and zoonotic pathogens are also deemed important focal points for further understanding the underlying aetiologies and cross-species symptomatology of co-infections in endemic regions [2].

Disease Interactions and Shared Risk Factors

Malaria, HIV, and TB remain leading preventable causes of mortality in low-income countries despite numerous global eradication programs [1]. The interactions among specific diseases of global health significance allow for greater understanding of how they impact those co-infected. Poverty and urbanization contribute to the co-occurrence of malaria, HIV, and TB, and access to healthcare services also shapes the risk of co-infection [1]. Three-quarters of malaria deaths occur in parts of Africa where HIV is hyperendemic, with women, truck drivers, and low-income urban residents at particular risk [2]. Malaria especially falciparum malaria and helminth parasites frequently co-infect the same individual due to their overlapping target demographics [3].

Pathophysiology and Immunology of Co-infections

In Malaria-Endemic Countries These co-infections lead to devastating interactions that widen the gap for at-risk communities further undermining decades of health advancement and eroding human dignity[9]. Priority should be given to a layered set of investments including the strengthening of health-system infrastructure to protect populations from multiple infectious disease threats while addressing the underlying structural inequalities underpinning the emergence and spread of successive infectious diseases [10]. Co-Infection Pathophysiological Mechanisms Co-infections with HIV, tuberculosis (TB), and neglected tropical helminth parasites compromise immunity, pathology, and therapies for malaria at the root of much morbidity and mortality in co-infected individuals [5]. Although co-infections involving HIV or TB remain exceptional in less-resource-limited settings, they exert an outsized toll in malaria-endemic countries [7]. Malaria promotes pre-existing HIV infections by dramatically increasing exposure and by enhancing viral load through the host inflammatory response. Conversely, during the subsequent preventive and therapeutic delivery of antiretroviral (ARV) therapies, malaria poses additional burdens [10]. Malaria incubation has a shorter duration than that of other pathogens thereby favoring either long-standing or clear-cut co-infection by these bacteria at either end of the malaria episode. Active malaria infections considerably alter the dynamic of the pre-existing tuberculosis (TB) infection within the host influencing the extent and the severity of manifestations by dramatically modifying the host immune response. Among neglected tropical diseases, schistosomiasis and lymphatic filariasis emerge as the most frequent helminth co-infections with HIV [11]. Helminth co-infections with malaria exist yet are less commonly studied. Although urban settings reduce exposure to malaria and helminths, populations infected are often more impoverished face food insecurity and regularly make long-distance journeys into rural transmission zones [6]. Historically co-

infections involving helminths and malaria have generally been excluded from the mainstream literature. Helminths have large human and economic burdens for labour then turning toward helminth-scale systems analyses where concurrent observations of malaria co-infection are evident. Schistosomiasis is the highest ranked neglect tropical disease worldwide but remains fundamentally neglected. Co-Infection Cohorts Pathogenic co-infections and co-morbidities occur globally but interact chiefly in large swathes of Sub-Saharan Africa thought to possess 37 million HIV+ individuals are also exposed to malaria[9]. Furthermore, when specifically focused on malaria substantially more is learnt of both HIV and helminth minimise epidemiological enquiries on tuberculosis instead. Research across the Pathophysiology sub-section captures more relevant ideas in each domain when, in turn, shaping the Epidemiology section. Individual attempts at modelling co-infections between malaria and either TB or helminths repeatedly indicate that grounding activities proceeds most beneficially across the highly influential and yet poorly described Interaction Mechanisms [9]. Much of human complement remains a rich tapestry of vampiric viruses spiriting away genetic material under cover of arbovirus, helical, and nucleus-forming global transportation networks yet larger even than the total viral narrative the mycobacterium-genus likewise extends a quite different invitation across vicinity scarcely overlapping the other entire [[10].

Malaria–HIV Interactions

HIV acquisition and disease progression are greater among patients with malaria, and HIV is progressively acquired more quickly in persons with malaria than in those without; thus, malaria infection translates into more rapid AIDS progression [1]. Individuals already infected with HIV receive treatment to suppress the virus; however, in the context of malaria they are able to avoid a severe febrile episode. Although HIV treatment is known to offer patients protection against malaria, through the significant reduction of HIV-1 viral load, malaria remains a high-risk disease in this population [5]. Therefore, in the context of primary infection, the urgent need continues for novel methods to interact with antiretroviral therapy or drug regimens that minimize side effects [1].

Malaria and TB Co-infection Dynamics

Co-infections with Mycobacterium tuberculosis (MTB) and Plasmodium species present a well-documented public health challenge in many settings [9]. Regions with pre-existent high burdens of malaria and tuberculosis documented that co-infection with malaria worsens clinical outcomes in tuberculosis treatment cohorts [10]. It can increase both MTB bacillary burden and impairment of specific T cell responses. Control of malaria transmission therefore influences the course of tuberculosis disease during the period directly after infection with MTB. M. tuberculosis (MTB) remains a formidable public health challenge in many countries [11]. Co-infections with Mycobacterium tuberculosis (MTB) and Plasmodium species present a well-documented public health challenge in many settings. Regions with pre-existent high burdens of malaria and tuberculosis documented that co-infection with malaria worsens clinical outcomes in tuberculosis treatment cohorts [10]. It can increase both MTB bacillary burden and impairment of specific T cell responses. Control of malaria transmission therefore influences the course of tuberculosis disease during the period directly after infection with MTB [11].

Malaria and Helminth Co-infections

Governments and international organizations are making considerable investments in HIV/AIDS and tuberculosis (TB) control; sensitization and health education programs have been developed to make the public aware of these diseases. Campaigns to increase the demand for treatment of malaria (the primary treat-and-control disease) have unintentionally increased the demand for TB and HIV/AIDS co-treatments [5]. Consequently, the evidence for the co-occurrence and of simultaneous co-infection of HIV/AIDS, TB, and malaria has greatly expanded [8]. In endemic areas, Plasmodium infections may affect the dynamics of tissue-dwelling helminth parasite infections. Like many other countries with endemic malaria (e.g., Ethiopia), most people in South Africa (and in most developing countries) live in abject poverty and lack drug-based preventive measures against malaria [6]. Malaria and helminth co-infections may have important implications for disease severity and intervention outcomes [6]. For instance, co-infections influence immune response, which in turn may have vaccination consequences for both pathogens. Similarly, in co-infected individuals, optimal clearance of tissue-dwelling helminths may confer protection against subsequent malaria episodes (which can be taken as indirect evidence of immunological interaction between these pathogens) [8]. Additionally, many coincide well with inter-epidemic periods when malaria transmission returns to low levels, permitting mosquitoes to colonize new insecticide-treated nets prepared for the newly born in several stillbirth sites [9].

Clinical Manifestations and Diagnostic Challenges

Overlapping clinical signs and symptoms complicate the differential diagnosis of co-infection; situations arise where various combinations lead to only minor modifications, whereas other combinations can modify presentations severely [2]. Symptom combinations that reinforce malarial cases and reinforce other co-infectious cases have been documented, while combinations that reinforce malarial cases but contradict the case for other co-

infections are also frequent. In light of this complexity, standardized diagnostic algorithms can allow thorough screening instead of relying only on sign combinations [7]. Adaptive diagnostic algorithms that leverage data from screening tests strategically applied to the clinical picture demonstrate higher performance than fully sequenced approaches [9]. Three testing strategies stand out: deploying multiple point-of-care tests simultaneously; conducting sequential testing based on pre-established priorities of suspected diseases; and sequentially applying tests directed towards the next most probable candidate starting from a vetted reservoir [9]. The second strategy appears particularly suitable for resource-limited settings: tests and strategies targeted to the co-infected population can be determined ahead of time, and wide access to safe third-line treatment permits confident completion of the algorithm in the face of uncertainty [9].

Symptom Overlap and Atypical Presentations

Malaria co-infections with HIV, tuberculosis (TB), and helminths modify the clinical presentation of malaria. Co-infected patients with malaria tend to exhibit a greater severity of symptoms and atypical clinical manifestations of the additional infections [7]. In co-infected patients, the accurate identification of malaria can be challenging due to the overlap of signs and symptoms, which can complicate treatment and contribute to high morbidity and mortality [7]. Diagnostic algorithms have been recommended to screen for co-infections in patients presenting with fever and malaria in co-endemic areas: a straightforward point-of-care test for the additional infection, followed by a sequential testing approach for non-screenable co-infections, and an algorithmic decision tree that accounts for the likelihood of malaria co-infection in the absence of tests [7]. Co-infection with HIV and malaria can lead to altered disease progression and increased severity of both illnesses. HIV-infected individuals are more susceptible to severe malaria due to their compromised immune status, leading to higher parasite levels and more severe clinical outcomes [7]. Malaria can accelerate the progression of HIV by increasing viral replication and reducing the effectiveness of antiretroviral therapy [1]. The coexistence of HIV and malaria poses challenges in treatment strategies, as drugs used for one condition may interact with those for the other, requiring careful consideration [1]. Co-infected individuals may exhibit a wide spectrum of clinical manifestations, including more severe malaria symptoms and atypical presentations of HIV-related illnesses. Complications such as anemia, neurological disorders, and increased susceptibility to opportunistic infections are more prevalent in co-infected individuals, necessitating vigilant clinical management. The combined impact of HIV and malaria on disease outcomes can result in poorer prognosis and increased mortality rates [1].

Diagnostic Algorithms in Co-infected Patients

In patients with multiple infections of public health concern, diagnostic algorithms that incorporate symptom patterns are needed to facilitate timely identification of the most deadly or treatable illness[1]. Malaria co-infection with either tuberculosis (TB) or helminths presents particular diagnostic challenges. Fever, cough, and weight loss are key symptoms of both malaria and TB, while acute febrile illness is a common presentation of malaria in individuals with helminth infections [4]. In areas where TB is prevalent, the possibility of co-infection must always be considered. Treatment of co-infected patients is further complicated by important overlaps in drug interactions and pharmacokinetics [7].

Treatment and Therapeutic Considerations

Antimalarial therapy for HIV and TB co-infection [11]. The selected therapy should reflect the broader context of treatment for co-infecting pathogens. Attention to drug resistance and patient safety during pregnancy is advisable for the choice of antimalarial agents [8]. Important pharmacokinetic interactions between antimalarials, ART, and second-line TB therapy also merit consideration [2]. In malaria-endemic areas co-infected with TB–HIV, appropriate timing of TB treatment initiation relative to malaria treatment and ART initiation affects severity of TB disease 1. In areas with moderate to high malaria transmission, baseline malaria treatment—ideally at the time of ART start might limit the multiplicative effect on viral load of TB co-infection. In co-endemic settings with TB and helminth interactions, whether deworming is beneficial or detrimental for co-morbidities, including malaria needs further investigation [7]. Interference between malaria and other infections can arise from shared risk factors, overlapping clinical stages, and pathogen-additive or synergy interactions. Clarity on co-infection epidemiology is thus crucial for decision-making, especially regarding prevention and vaccination within extended HAART initiatives targeting other pathogens [9].

Antimalarial Therapy in HIV/TB/Helminth Contexts

Longitudinal and cross-sectional studies demonstrate associations between HIV-1 and malaria [1]. HIV infection increases susceptibility to malaria infection, thickens the malaria parasite cycle in the blood, and modulates the effectiveness of anti-malarial treatment [2]. A dense malaria infection can increase the HIV-1 circulating viral load and accelerate the HIV-1 disease progress. Various treatments for HIV and malaria exhibit either antagonistic or synergistic effects [7]. Therefore, a comprehensive approach that incorporates HIV and anti-malarial co-treatment is imperative to control both diseases for the benefit of public health [7]. Tuberculosis (TB)

and malaria are two major infectious diseases impacting public health globally, especially in developing countries. Literature documents interactions between TB and malaria. Active malaria augments TB bacillary replication in infected cells. TB similarly enhances malaria parasitic burden [8]. The immune response modulation by either pathogen exacerbates the disease [8]. Co-infection with TB and malaria confounds clinical diagnosis and hampers effective treatment [2]. Schistosoma haematobium and other helminth infections are associated with HIV-1 acquisition and pertub immune status in HIV-infected individuals [1]. Deworming therapy may improve HIV-1 viral load [6]. In malarious regions, consideration of various anthelminthic treatment timing options is crucial due to interference with malaria management. Current quantitative treatment assessment tools are limited; development of validated questioning tools for coverage, compliance, and compliance monitoring is urgent; further, treatment interruption often arises due to tolerance, necessitating an evaluation of the overall treatment impact [1].

Antiretroviral Therapy Interactions

Antiretroviral therapy (ART) is central to HIV management. Nevertheless, the high burden of malaria in HIV-endemic regions complicates ARV use. Antimalarial agents can affect ARV pharmacokinetics and vice versa. Such interactions may also occur with co-administration of anti-tuberculosis treatments, especially rifampicin [8]. HIV infection is a strong risk factor for severe malaria. Consequently, co-infected patients may experience higher exposure to antimalarial drugs [9]. Patients receiving rifampicin may therefore require higher quinine doses to achieve therapeutic plasma concentrations. Several other antimalarials including atovaquone, chloroquine, piperaquine, mefloquine, and doxycycline are also known to interact with rifampicin and should be used with caution. Due to complex, variable, and insufficiently quantified simultaneous interactions between rifampicin and CYP450-metabolised antiretroviral drugs, an integrated approach to managing these co-infections remains elusive. Relatively little is known about the potential effects of malaria co-infection on ARV pharmacokinetics [10]. The CYP450 pathway is involved in the metabolism of several antimalarials, including artemether, lumefantrine, and piperaquine, making these drugs candidates for further research in this area [11].

TB treatment Considerations in Co-infection

Co-infection with Mycobacterium tuberculosis (M. tuberculosis) and malaria remains a significant global health challenge, particularly in sub-Saharan Africa, where HIV and tuberculosis (TB) are endemic [10]. The co-infection with both pathogens increases the mortality rate among co-infected individuals. Compounding the challenge further, an estimated one billion people worldwide are affected by helminth (parasitic worms) infections, with co-infection of TB and helminths being reported in many malaria-endemic countries [11]. The emergence of clear evidence for such co-infections and constant surveillance activities in many of these areas necessitates not only a deeper understanding of the immunity of the host against co-infection with TB/malaria and helminth/malaria but also the time relationship between treatments of these diseases [9].

Helminth Co-Infections and Immune Modulation

Helminth infections often coexist with other pathogens in individuals residing in endemic areas, and their potential to influence the course of disease, particularly Plasmodium infections, has been projected [7]. Co-infection with Schistosoma spp. and lymphatic filariae has been linked to HIV-1 susceptibility and transmission, while other helminths such as intestinal nematodes may interfere with HIV control by stimulating broad immune responses [5]. Helminth treatments can alter immunity to both exogenous and endogenous pathogens, and this may bear relevance for the malaria–HIV dynamic [8]. In addition to direct effects on P. falciparum parasitism, helminth infections and their treatment have been reported to modulate immune responses to malaria antigens and to key pathways involved during P. falciparum infection [10]. Such interactions may therefore impact susceptibility to Plasmodium along with the progression and outcome of co-infection, with practical implications in deworming strategies and vaccine development [9].

Outcomes, Mortality, and Health System Implications

Among HIV, TB, helminths, and malaria, the most extensive literature pertains to malaria–HIV co-infection, with emerging papers on malaria and TB [8]. Co-infections of malaria with helminths, TB, or both typically alongside HIV contribute to substantial morbidity, especially in southeastern Africa. Although detailed co-infection epidemiology in these regions remains scarce, malaria's extensive coverage parallels infection with HIV or another pathogen [1]. Malaria–HIV co-infection adversely affects malaria-specific outcomes, reducing quality of life, increasing mortality, and establishing persistence and resistance in endemic settings. In co-infected patients, the incidence of uncomplicated malaria can exceed that for HIV mono-infection by 40%, rising to 70% for severe malaria [7]. Conversely, advanced malaria increases the rate of mortality associated with HIV disease. Similarly, the burden of TB in malaria-endemic regions substantially exceeds that in similar areas without malaria [5].Programmatic factors adherence, supply, and integration influence disease control in resource-limited settings,

with co-infected individuals facing significant obstacles [6]. The requirement to balance multiple treatments raises the risk of suboptimal compliance [9]. Stock-outs of any essential medicine lead to treatment interruptions and disease progression, necessitating an effective cross-commodity supply system [8]. A further facet of co-infection mitigation addresses the integration of HIV and TB services into malaria programs. In high-burden countries, incorporating these diseases into existing healthcare delivery and prevention models represents the most viable intervention approach.

Clinical Outcomes in Co-Infected Individuals

Information on co-infection with HIV, tuberculosis (TB), and helminths, and malaria is often fragmented [8]. In South Africa, incidence rates of malaria, TB, and HIV are among the highest in the world yet little is known about the possible interactions between these diseases. HIV co-infection is known to compromise the immune response to malaria, increasing infection severity. Co-infection with helminths (in South Africa primarily Schistosoma spp. and Ascaris lumbricoides), renders patients less susceptible to severe disease, reducing parasite burden and delaying reinfection [5]. The presence of helminths has therefore contradictory effects, modifying the outcome of co-infection [3]. Significant discrepancies exist in the published literature regarding malaria–TB co-infection. Evidence indicates that co-infected patients differ from non-co-infected patients with regard to clinical signs. Ongoing research aims to investigate the capacity of certain anti-TB treatment and antimalarial agents to influence HIV virus and HIV protozoa replication and infectivity, with a view to disinfecting the body of these parasites—without risk of spreading resistance while enhancing the health of the co-infected individual. National scale-up of ART, TB, and malaria services, together with the concomitant introduction of biomedical neo- and biotechnologies, the simplification of the ART regimen to once daily and the introduction of decentralised treatment of TB and as well as other innovative delivery approaches promise to improve the management of individuals who are co-infected[3].

Adherence, Resistance, and Programmatic Challenges

Care-seeking behaviour in co-infected individuals is often hampered by multiple barriers, including social, economic, logistical, and environmental determinants. Such factors also limit the success of integrated HIV/tuberculosis/malaria programmes, which often rely on lessons learnt from the HIV/tuberculosis paradigm [7]. Stock-outs, supply chain disruptions, and service integration that compromises the continuity of care further complicate treatment retention in co-infected patients [7]. Attention to adherence-related challenges and their connections across clinical disciplines is, therefore, essential to maintaining meaningful interactions between HIV, tuberculosis, and malaria [1]. Malaria remains widespread in many settings where human immunodeficiency virus (HIV) transmission is ongoing, and the dynamics of both infections are influenced by economic and demographic trends [11]. Co-infection may impede the treatment of either illness [7]. For example, the initiation of antiretroviral therapy (ART) to treat HIV, where recommendations denote early treatment, is frequently delayed in individuals co-infected with Mycobacterium tuberculosis because of concerns regarding the safety of ART regimens concomitant with bactericidal antituberculosis therapy[8].M. tuberculosis, once acquired, may become dormant and reactivate even after anti-tuberculosis therapy is completed, especially in individuals with immune suppression due to co-infection with HIV. Nevertheless, the co-administration of antimalarial therapy, besides the first-line therapy for both uncomplicated and complicated malaria, remains a matter of concern. The vast global burden of HIV, combined with high prevalence rates of various proxy infections with substantial public health relevance and policy interest, align these interactions with the wider understanding of M. tuberculosis and microbiome interactions [2].

Prevention, Vaccination, and Public Health Strategies

The interactions between vector control measures remain poorly examined, yet integrated approaches hold promise for substantial impact [1]. Integrated vector control combines multiple interventions to attack unified transmission pathways and increase overall impact. Co-occurrence of malaria and HIV, tuberculosis (TB), or helminths can increase risk of progression to disease and/or exposure to additional pathogens; effective co-infection prevention may increase resilience to multiple morbidity and mortality threats [6]. Integrated approaches consist of complementary interventions and delivery methods unified by common transmission pathways [8]. In the Malaria–HIV context, environmental management, larviciding, active/inactive focal spraying, source reduction, insecticide-treated nets, application of insecticides to livestock and cattle, health education, communication, and mass distribution of condoms, contraceptives, and anti-HIV drugs have been integrated with successful results [1]. For TB, attention to source/active case finding, improved case management of clinical TB with preventive treatment, household contacts tracing, and education to improve care-seeking and adherence within community-managed short-course regimens among smear-positive TB patients has been accomplished for HIV, radio/telecommunications, HIV self-testing services, TB preventive treatment education, and distribution of peak-flow meters (TB treatment/monitoring) in Senegal[5]. A co-initiated method

to facilitate coordination among disconnected prevention and treatment strategies, alerts upon incident malaria, TB, and HIV infections, conducts serial monitoring, anticipates their joint consequences, and recommends nationally-approved actions according to the prevailing respective risk over an extended time range would be more suitable to the Helminth and related systemic changes co-infections and could be a step towards the responsible self-formulated primate co-prioritizer formulation to jointly tackle the Malaria–HIV section and the malaria–helminth co-infected section[2]. Malaria–HIV targeted and TB–HIV targeted preventive measures may jointly increase the likelihood of shifting from focus to elimination through non-temporal natural inter-goal synergistic decision-support enhancement via 'a' prevented-detection temporal-irrelevance prioritizer [7]. Guidance on primary prevention for malaria co-infections within regions displaying low local population immunity remains absent, although awareness of early suspension of periodic interventions is crucial once population immunity threshold is anticipated to be incessantly, sustainedly, and invariably attained for the benignant strains and similar attention demands for the parasites of the corresponding remnants (aligned with A4)[11].

Integrated Prevention Approaches

Globally, the co-existence of HIV and malaria cuts across multiple multiplex challenges, crosspollenated by socio-economic and environmental determinants, reproductively projecting the disease burden and aggravating morbidity across populations [6]. HIV co-infection worsens malarial endemicity in communities; conversely, the presence of malaria heightens incident and prevalent burdens of HIV. Consequently, there is a growing philosophic consensus that quality health systems must synergistically integrate control policy-measures for HIV, malaria and other relevant infectious diseases [11]. National HIV/prevention of mother-to-child transmission (PMTCT) initiatives also increasingly views malaria as a key co-infection, suggesting that the two diseases should be assessed together at the design phase of health systems and national control strategies [1]. Target-group with universal access to free HCT is highly transportable across public-health sectors, so concurrent management of the two microbicides-such as use of d-hydroartemisinin (DHA) candles together with 0.05% agrochemical insecticides, and/or 2000-cycles insecticide-treated bed-nets (ITB-N) must be added. All conventional anti-malarials are also combined with the same frontline microbicide impulse [11]. The multi-stakeholder challenge of finding prospective synergies-followed by delivery integration in actual practice-conducts fresh priority-setting both for global knowledge and resource-positioning, still under-explored in conventional policy and strategic processes [4].

Vaccine Considerations and Research Gaps

Currently, the malaria/HIV/TB vaccine agenda remains focused on the needs of individual pathogens. Concerted efforts to establish and sidestep critical or limiting co-infection research gaps across different transmission and epidemiological settings grounded in the determination of future co-infection vaccine-eligibility are therefore warranted [7]. Large-scale cohort studies designed to assess the dynamics of HIV, TB, and helminth co-infection in the context of malaria control and elimination represent a methodological priority [7]. Enhanced access to existing co-infection datasets, purposive data-harmonisation efforts, and a reliance on mathematical modelling offer other avenues to stimulate the generation and dissemination of co-infection-relevant knowledge that will ultimately bolster malaria control efforts [1].

Research Gaps and Future Directions

Despite the extensive research on the substantial burden imposed by HIV, tuberculosis (TB), and helminths, along with the co-infection with malaria there remains an insufficient investigation of the pathogenesis and chronicity of these infections, as well as associated immunity [7]. Although several questions remain to be solved, mainly due to the complexity of such interactions in humans, the study of co-infection offers the opportunity to evaluate emerging patterns of malaria which will be valuable for the selection of study cohorts, modelling or epidemiology, longitudinal studies, and harmonization [1]. Methods of standardization should aim to bring together the broad range of approaches used to investigate co-infection studies across various science fields[3]. The most promising approaches include the estimation of the extent to which critical determinants of malaria disease, such as reinforcement of infection/chronicity, parasite burden, and underlying immunity, become integrated into complex, multiscale, epidemiological, or population-dynamical frameworks [9]. Such an advanced portrayal would improve our capacity to identify entry points for preventive interventions. Sufficient coupling of basic molecular/conceptual knowledge with epidemiological explanation and practical applications will facilitate the development of mathematical models to analyse co-infection [7]. Subdivision of general clinical investigations into comprehensive categories enables a better comprehension of the relevance of each and provides a straightforward path for selecting studies. Emphasis in epidemiological studies is predominantly placed on particular junctions that have consistently remained prominent in malaria co-infection investigations [5]. The general level of formalisation adopted by either subject provides complementary perspectives, with minimal overlap, on the links between

parasite-specific effects of malaria and at least four general properties of the disease: spatial meta-dynamics of both malaria and the alternative infection; the structure of the host-parasite interaction, although essentially analogous to the single-pathogen case some extra complications arise; parasite-dynamical effects of malaria mediated by co-exposing a host to more than one species; and potential vulnerability to other infections in the post-malarial period as a result of time elapsed since that treatment 2. Nevertheless, no indication exists concerning the direction of influence between population dynamics and primarily pathogen-driven perspectives [6]. Rather, integration of cross-infection, acquired immunity, and reservoir promotion into models of TB or helminth transmission appears a far more advanced step towards broadening upon the details of particular anti-malarial effects. Nevertheless, tremendous progress has been made both experimentally and theoretically on clarifying and conceptualizing the interaction between concurrent helminth and malaria infections [8].

Knowledge Gaps in Pathogenesis and Immunity

A major challenge in addressing co-infections between malaria and other pathogens is the limited understanding of the broader immunological and pathophysiological effects of each infection, as well as how these effects may be modified by systemic interactions with co-infecting organisms [8]. For example, the co-infection of malaria and HIV (human immunodeficiency virus) is extensively documented, yet dozens of important questions remain the relative impact of acute and chronic malaria on HIV acquisition and disease progression, the persistence of HIV in individuals after successful treatment of the malaria infection, whether tissue-dwelling helminths that affect systemic immune modulation influence HIV acquisition in malaria-endemic regions, and the potential interactions between them [5].Malaria co-infection with other pathogens such as tuberculosis (TB), helminths, and the more nefarious viruses (such as HIV-1, hepatitis B and C viruses, and human T cell leukemia virus type 1) introduces corresponding gaps in knowledge regarding the origin of the host immune response in each co-infection scenario, whether acquired immunity can be maintained or is invariably lost, the influence of maternal infections on immune responses, the nature of adaptive responses that develop over time, and how reinfection interferes with maintenance of acquired immunity [2].

Methodological Priorities for Co-Infection Research

Future work on co-infection with malaria should adopt a combination of different methodological approaches that exploit the size of existing datasets [6]. Co-infection studies often rely on data generated as part of other research efforts. This practice is advantageous both ethically and financially, as it allows researchers to take research gaps into consideration without incurring the expenses associated with independent data collection [9]. Through such means, co-infection studies may rigorously adjust for the potential influence of many spatially varying factors, including political stability, completion of basic education, use of improved drinking water, access to electricity, and access to telephone services [7]. Such adjustments are crucial given that many socio-economic, environmental, behavioural, and governance factors are known to exert a powerful influence on co-infection rates 5. Identifying the factors that drive co-infection requires the ability to relate them to a large variety of epidemiological and operational factors, such as malaria elimination [1]. Co-infection studies can thus be understood as a form of the wider investigation of financial, spatial, or other multi-dimensional epidemiological modeling [2]. The geographical dependence of a large number of diseases and co-morbidities encourages the implementation of geostatistical models that accommodate spatially structured variation in the underlying risk of infection. Geostatistical approaches are able to account for spatially structured individual factors while also permitting prediction in areas where prevalence has not been measured [10]. Co-infection studies that employ geostatistical modelling allow for interpolation of epidemiological measures into unobserved locations; consequently, they support analysis of the degree to which co-infection accompanies conditions met at the individual level [11]. In addition, the co-distribution of many diseases permits co-infection studies that exploit a joint modelling approach to capture inter-dependencies between the different risks. Such models are capable of identifying the extent to which disease co-distribution arises from association between the effects of common risk factors and the degree to which co-distribution is explained by a direct link between diseases, thereby allowing better understanding of the initiation and development of diseases[11-16].

CONCLUSION

Malaria co-infection with HIV, tuberculosis, and helminths remains a significant impediment to disease control and elimination efforts in endemic regions. The overlapping distribution of these pathogens, coupled with their synergistic effects on immune function, contributes to heightened morbidity, mortality, and socioeconomic vulnerability. HIV, malaria co-infection amplifies viral replication and weakens antimalarial responses, while helminth–malaria interactions produce both protective and adverse immunological outcomes. Tuberculosis co-infection, though less extensively studied, further complicates diagnosis, treatment, and long-term recovery. Persistent clinical and programmatic challenges including drug interactions, poor adherence to multidrug regimens, resistance development, and limited diagnostic capacity underscore the need for integrated care models.

Effective management requires harmonised protocols that coordinate malaria, HIV, TB, and helminth programs within a strengthened primary healthcare framework. Integration of community-based prevention, targeted health education, and novel technologies such as digital adherence monitoring can enhance treatment continuity. The absence of cross-pathogen vaccine strategies and the scarcity of longitudinal cohort studies highlight major research gaps. Future work should prioritise the study of immune mechanisms underlying co-infection, evaluate the effects of concurrent treatment regimens, and employ geostatistical and joint-modelling approaches to predict co-infection hotspots. A unified One Health strategy, incorporating environmental control, social determinants of health, and cross-sectoral collaboration, is essential to reduce co-infection burden sustainably. Long-term success will depend on political commitment, equitable access to therapies, and the translation of research insights into actionable, community-responsive policy frameworks capable of addressing the multifaceted nature of malaria co-infection.

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