Preliminary *ex vivo* evaluation of a cyclic electromagnetic patch for reducing *Plasmodium falciparum* parasitemia among patients at Bon Samaritain Hospital in N'Djamena, Chad

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Conflict of interest: the authors declare no financial conflicts of interest related to this study. However, the patch used in the *ex vivo* experiments was kindly provided by collaborators who were also involved in the initial phases of testing. These collaborators did not influence the study design, data analysis, or interpretation of the results.

Ethics approval and consent to participate: this study was reviewed and approved by Ecologia-Ambiente-Biodiversita Urbana e Agraria-Entomologia, under Legal Ref: Section 148 Public Health Act, 2012, Act 851. The research was conducted in accordance with the Declaration of Helsinki and applicable local regulations. Human blood samples were used ex vivo only, with no additional risks imposed beyond standard clinical practice. The *ex vivo* results were obtained from residual blood samples collected from laboratory analyses of patients who gave consent for their use in research.

Availability of data and materials: the data underlying the findings of this study are available from the corresponding author upon reasonable request.

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Abstract

The emergence of artemisinin resistance in *Plasmodium falciparum* (*P. falciparum*) threatens malaria control efforts in Sub-Saharan Africa. Alternative, non-pharmacological adjunct therapies are urgently needed. This preliminary *ex vivo* study evaluated the effect of a cyclically activated electromagnetic patch on *P. falciparum* parasitemia. Venous blood samples from three adult patients with confirmed *P. falciparum* infection in N'Djamena, Chad, were exposed *ex vivo* to a patch device activated every 30 minutes over total durations of 4, 6, and 8 hours. Thick smears were examined by microscopy before and after activation and at 24 hours. Parasitemia reduction was calculated, and control samples were maintained for comparison. A time-dependent reduction in parasitemia was observed, with up to a 93% decrease after 8 hours of activation. After 24 hours, parasitemia remained lower in patched samples (160 parasites/µL) compared to controls (728 parasites/µL). Variability in parasitemia trends during intermediate cycles and a limited sample size were noted as study constraints. These preliminary findings suggest the potential utility of electromagnetic patch technology as a complementary tool in malaria management. However, larger studies using molecular quantification and mechanistic assays are required to confirm efficacy and clarify the underlying inhibitory pathways.

Introduction

Malaria remains one of the most urgent public health challenges in Sub-Saharan Africa, with *Plasmodium falciparum* (*P. falciparum*) being the most virulent species. Despite significant advances in our understanding of the biology of *P. falciparum*, malaria continues to pose a major global health challenge, resulting in truly high morbidity and mortality, especially in Sub-Saharan Africa.[1]

In Chad, malaria remains a major public health problem, with intense and stable transmission throughout the country. *P. falciparum* is responsible for over 90% of recorded cases, particularly affecting children under five and pregnant women.[2] Despite the efforts of the National Malaria Control Programme (NMCP), morbidity and mortality associated with this parasitic disease remain at concerning levels.

Artemisinin-based combination therapies (ACTs) are the current gold standard for treating malaria. However, the progressive emergence of resistance to artemisinin and its partner drugs, now documented in several regions of Africa, is raising increasing concern,[3] particularly in contexts like Chad where molecular surveillance capacities remain limited. In the face of this threat, the exploration of alternative or complementary therapeutic solutions

is imperative. Among these, biophysical approaches, specifically the use of electromagnetic fields, are attracting growing interest. Several studies have highlighted the sensitivity of *P. falciparum* to various types of electromagnetic waves, particularly due to the presence of paramagnetic hemozoin within infected red blood cells.[4,5] This sensitivity could be exploited to develop non-invasive devices capable of inhibiting parasitic growth without the direct use of an active compound.[6]

In the context of Chad, characterized by high endemicity of *P. falciparum* malaria and the likely emergence of artemisinin-resistant strains, exploring alternative, non-pharmacological therapeutic strategies is a priority. This study investigates the effect of a cyclically activated patch device, emitting electromagnetic signals, on *P. falciparum*-infected blood samples under *ex vivo* conditions. The patch, activated at regular intervals *via* a digital interface, is designed to induce a physical stimulation that may selectively inhibit parasite growth.

This study aims to evaluate the effects of a cyclic activation patch system on reducing *P. falciparum* parasitemia *ex vivo*. It is based on the hypothesis that the parasite may exhibit non-thermal sensitivity to electromagnetic signals, under conditions that do not harm host cells, thus opening the way to an innovative complementary therapeutic modality.

While the introduction reviews electromagnetic field effects on *P. falciparum*, it would benefit from adding African-centered or regional studies on alternative non-pharmacological malaria control strategies (*e.g.*, herbal extracts, environmental vector control innovations) for better contextual anchoring.

Materials and Methods

This *ex vivo* study was carried out on infected human blood samples at the Laboratoire des Grandes Épidémies Tropicales, Bon Samaritain University Hospital Complex (CHU-BS), N'Djamena, Chad. Residual venous blood was collected from the analysis laboratory at CHU-BS after obtaining informed consent from three adult male malaria patients diagnosed with *P. falciparum* by microscopy. For each experiment, 2 mL of blood was drawn and stored in ethylenediaminetetraacetic acid (EDTA) tubes. A control sample from each patient was set aside for comparison.

Each infected blood sample was placed in sterile tubes and exposed to the patch device, which was activated in cycles. The patch was affixed to the exterior surface of the blood sample tube (to avoid any direct contact with the blood) using adhesive tape. Near-field communication (NFC) tools on a smartphone were used to read and activate the patch (each activation corresponded to four successive reads). The patch was activated in 30-minute cycles under the following test conditions: *Sample A*, total exposure time of 4 hours, with activation every 30 minutes; *Sample B*, total exposure time of 6 hours; *Sample C*, prolonged exposure of 8 hours. Between activations, samples were maintained upright at room temperature (25°C). Thick blood smears were prepared from each sample before and after exposure. Smears were stained with Giemsa (1:10 dilutions) and examined under optical microscopy at 1000×magnification.

Parasite density was calculated using the WHO standard formula: parasite density = (number of parasites counted/number of leukocytes counted) × 8,000. An average leukocyte count of 8,000 cells/µL was used in the absence of individual leukogram data. A final parasitemia count was performed 24 hours after the last activation cycle to assess the stability of parasite reduction.

Results

Sample A

As shown in Table 1, a sharp drop in parasitemia was observed from the first cycles (54% at cycle 1, 81% at cycle 3), followed by a plateau around 360-400 parasites/µL between cycles 4 and 8, before reaching a minimum of 220 parasites/µL (93%) at cycle 9. The occasional increases (in cycles 4 and 6) suggest a possible 'rebound' effect or heterogeneity in sampling/thick-smear analysis.

Results after 24-hour activation vs. control

After 24 hours, the parasitemia in the patched tube (160 parasites/µL) remains positive but is reduced by nearly 80% compared to the untreated control (728 parasites/µL). Interpretation: this suggests a sustained effect of the patch rather than a simple mechanical trapping, potentially indicating a prolonged action (*e.g.*, antibodies, photoactivation, *etc.*) (Table 2).

Table 1. Sample A: change in parasite density over the 9 cycles.

Cycles	PD (p/µL)	% reduction*
0	3120	—
1	1447	53.6%
2	941	69.8%
3	600	80.8%
4	880	71.8%
5	240	92.3%
6	360	88.5%
7	240	92.3%
8	400	87.2%
9	220	93.0%

PD, parasite density; *% reduction was calculated relative to PD_0: $(1–PD_x/PD_0)\times100$.

Table 2. Results after 24-hour activation *vs.* control.

Condition	PD (p/µL)	% reduction compared to control
Patch 24 h	160	78.1%
Control 24 h	728	—

PD, parasite density.

Table 3. Sample B and C: comparison of extended activation durations (6 h *vs.* 8 h).

Sample	PD_0	PD_1 (activation)	PD_2 (24 h)	PD_3 (PC*)
B (6 h)	224	256 (+14%)	160 (29%)	320
C (8 h)	258	188 (27%)	80 (69%)	300

PD, parasite density; *PC, positive control (initial blood sample retested).

Sample B and C

Sample B (6 h) shows a slight increase after activation, with 256 compared to 224, followed by a marked reduction to 160 parasites/µL at 24 h. Sample C (8 h) shows a continuous decrease, reaching 80 parasites/µL (-69%) after 24 h. A longer activation time (8 h) appears more effective at reducing parasitic density, whereas 6 h shows greater variability (possibly due to transient parasite release or counting bias) (Table 3 and Figure 1).

Rapid capture or inhibition of the parasite was observed from the very first cycles, with a progressive and cumulative reduction reaching 93% after nine passages. A persistent effect was noted at 24 hours, indicating a mechanism more complex than simple filtration, potentially involving immuno-adsorption or photoactivation of patch components. The data suggest favoring longer cycles (≥8 h) to achieve maximal reduction. The observed cyclical variability highlights the need to increase the number of replicates and employ automated quantitative methods (*e.g.*, quantitative polymerase chain reaction [qPCR]) to validate the effect. Only a single initial sample (specific to *P. falciparum*) was tested; extending the study to multiple isolates is necessary to assess generalizability. Further investigations are needed (for example, parasite apoptosis markers or electron microscopy) to elucidate the inhibitory pathway. When coupled with dried blood spots (DBS)/PCR protocols, this system could serve as an *ex vivo* tool for rapid antimalarial sensitivity screening.

Discussion

These results show a rapid and significant reduction in parasite density from the first cycle (54%), reaching up to 81% by the third cycle, then fluctuating between 71% and 92% through the ninth cycle, where it peaks at 93%. This pattern suggests a robust cumulative effect, likely driven by an active parasite elimination or inhibition mechanism.

However, the observed rebounds during cycles 4 and 6 may reflect natural biological fluctuations in parasite life stages or limitations of manual microscopic counting (including sampling bias or inconsistent scoring). These variations underscore the importance of using standardized and automated quantitative methods (*e.g.*, qPCR, volumetric flow cytometry, or automated microscopy) to increase precision and better understand parasitemia dynamics. The analysis comparing the activated patch with the untreated control shows a 78% suppression of parasitemia after 24 h, suggesting a sustained effect from the patch. This outcome goes beyond simple mechanical trapping of parasites: it indicates a prolonged active action, potentially through immuno-adsorptive or photoactive mechanisms of patch components. This opens avenues toward a durable inhibitory mechanism, but complementary studies (such as immunofluorescence, parasite apoptosis assays, and functional tests) are necessary to confirm this hypothesis. Comparative analysis between 6 and 8 hours of activation indicates that prolonged exposure results in more effective parasite elimination: a slight reduction is observed after 6 hours of activation, followed by a 29% decrease at 24 hours; in contrast, 8 hours of activation exhibits a continuous decline throughout the activation period, culminating in a 69% reduction at 24 hours. These data suggest that an 8 h (or possibly longer) activation threshold may be optimal for maximizing the effect and underscore that exposure duration is a critical parameter to standardize in future protocols.

Perspectives

The preliminary results of this study indicate that an activated electromagnetic patch device may serve as a novel complementary strategy for reducing *Plasmodium falciparum* parasitemia *ex vivo*. Its non-invasive, device-based approach shows promise, especially in Sub-Saharan African settings where therapeutic innovation and resistance management are public health priorities.

In clinical contexts, the concept of integrating this technology into wearable devices, such as bracelets for children with severe malaria, is worth exploring. Such an approach could deliver continuous, adjunctive parasite suppression alongside standard antimalarial treatment. This dual-therapy strategy may improve parasite clearance rates, potentially reduce severe complications, and contribute to mitigating selective drug pressure in regions facing emerging artemisinin resistance.

Ultimately, integrating non-pharmacological adjunct therapies into malaria control programs could broaden the tools available to national programs and local health services, particularly where

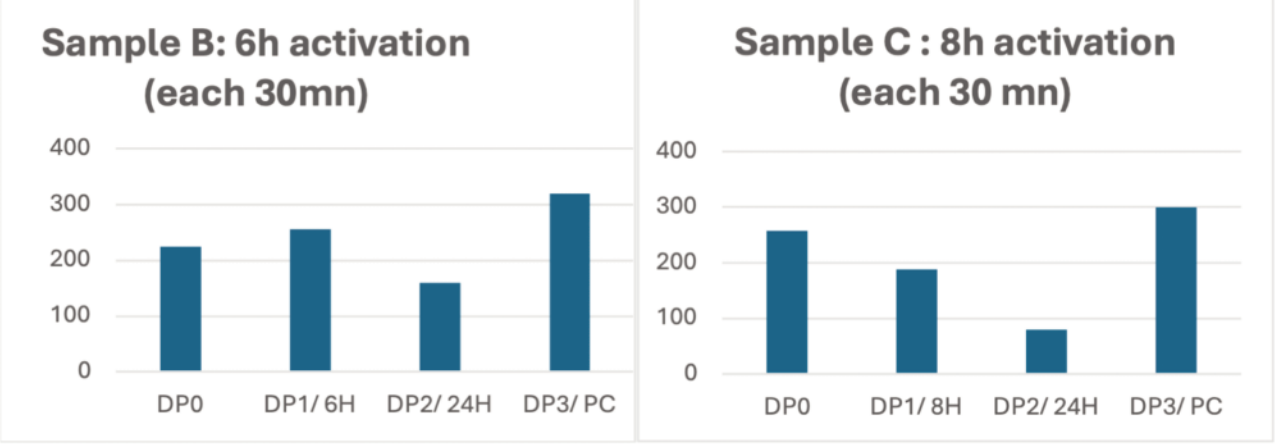

Figure 1. Variation in parasite density in samples B and C.

access to advanced drugs or molecular surveillance remains limited. Furthermore, in the context of increasing artemisinin resistance, the patch could serve as an innovative tool in the fight against malaria. By maintaining sub-therapeutic, controlled concentrations of active molecules, it may help reduce selective pressure on the parasite and delay the emergence of resistant variants.

Finally, as a non-invasive device, it enhances pediatric safety and adherence (fewer repeat vein punctures, simplified hospital management), thereby strengthening the overall effectiveness of severe malaria care in children.

Conclusions

This preliminary *ex vivo* study demonstrated a rapid and sustained reduction in *Plasmodium falciparum* parasitemia following exposure to a cyclically activated electromagnetic patch device. The findings indicate that prolonged activation, particularly beyond 8 hours, enhances parasitemia suppression. While the results are promising, the small sample size, the absence of molecular quantification, and the lack of mechanistic data limit the strength of conclusions that can be drawn. Further studies involving larger patient cohorts, advanced parasitemia assessment methods, and investigations into the biological mechanisms of parasite inhibition are needed. If validated, this approach could represent an innovative adjunctive tool for malaria management in endemic African settings, contributing to efforts to contain drug resistance and improve clinical outcomes, particularly in vulnerable populations such as children with severe malaria.

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