



RESEARCH ARTICLE

Effects of goat bile treatment on parasitemia, survival, and parasite-specific IgG in *Plasmodium berghei*-infected BALB/c mice

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<https://doi.org/10.33086/ijmlst.v8i2.8033>**Abstract**

Malaria remains a major global health problem. Immunoglobulin G (IgG) plays a key role in naturally acquired immunity and is associated with disease progression. Goat bile (GB) has traditionally been used as an antimalarial remedy; however, its effects on immunity and the balance between its efficacy and toxicity remain unclear. This study evaluated the effect of GB on anti-*Plasmodium berghei* IgG levels and their association with parasitemia and survival in infected mice. Fifty male mice were divided into five groups (n = 10 per group) and infected with *P. berghei*. Three groups received GB at 25% (GB25), 50% (GB50), and 100% (GB100). The positive control group (POS) received dihydroartemisinin-piperaquine (DHP: 187.2 mg/kg body weight), and the negative control group (NEG) received sterile water. Parasitemia was monitored daily, and IgG levels were measured by indirect ELISA (OD450) after 4 days of treatment. GB100 produced the lowest parasitemia ($16.76\% \pm 1.41$) but reduced survival (60%). GB25 and GB50 showed moderate parasitemia ($35.80 \pm 4.03\%$ and $31.21 \pm 4.31\%$, respectively) with 100% survival. IgG levels were highest in the untreated mice (NEG) group (3.70 ± 0.21) and significantly lower in all treated groups ($p < 0.01$). GB demonstrated anti-plasmodial activity, with greater parasite suppression at higher concentrations. However, GB100 was associated with reduced survival and diarrhea, indicating treatment-related toxicity. Lower parasite burden was accompanied by lower anti-*P. berghei*-specific IgG levels. These findings suggest that parasite suppression, survival, and parasite-specific IgG responses should be considered together when evaluating the therapeutic potential of goat bile.

1. INTRODUCTION

Malaria remains a critical global health problem, particularly in tropical and subtropical regions. In 2024, an estimated 282 million cases and approximately 610,000 deaths occurred worldwide, most of them in the WHO African Region, where children under five years of age are disproportionately affected. Despite substantial progress in prevention and treatment, the persistent burden of malaria, together with emerging drug resistance and limited access to care in endemic settings, underscores the need for alternative or adjunct therapeutic strategies (1). Experimental infection of mice with *Plasmodium berghei* is a well-established model for investigating host immune responses and evaluating antimalarial interventions (2).

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In Indonesia, consumption of intact goat gallbladder is traditionally believed to treat malaria and enhance physical stamina (3). Goat bile (GB) has also been used for centuries in Traditional Chinese Medicine (TCM) to treat various infectious and non-infectious diseases (3). Previous experimental studies have shown that GB exerts anti-plasmodial effects and modulates inflammatory cytokine responses in *P. berghei*-infected mice (4,5). However, toxicity studies indicate that high GB concentrations may induce adverse effects, including diarrhea, suggesting a concentration-dependent biological impact (6). Immunoglobulin G (IgG) is an important component of the immune response to blood-stage malaria, and parasite-specific IgG has been associated with control of parasitemia through antibody-mediated mechanisms (7-9). However, the magnitude of the IgG response may also reflect parasite exposure and does not necessarily indicate protective immunity, because malaria parasites can evade or modulate host immune responses (10). Because bile acids and bile-derived metabolites can influence immune cell function and immune homeostasis (11,12), GB may affect parasite-specific antibody responses in addition to exerting anti-plasmodial activity. Whether GB influences *P. berghei*-specific IgG responses remains unclear.

Although a previous study showed that GB modulated cytokine responses during *P. berghei* infection, but it did not determine whether GB also affected parasite-specific antibody responses. The IgG in that study was measured as total IgG using a commercial ELISA kit that was not specific to *P. berghei* (5). In contrast, the present study specifically measured anti-*P. berghei*-specific IgG using parasite antigen, which provides a more direct assessment of the parasite-specific humoral response and complement our earlier cytokine data (13). We hypothesized that GB exerts a concentration-dependent dual effect during *P. berghei* infection. Higher concentrations, previously shown to enhance parasite suppression, may compromise survival, whereas moderate concentrations may reduce parasitemia while maintaining survival and influencing parasite-specific IgG responses.

2. MATERIALS AND METHODS

2.1. Preparation of Goat Bile

Gall bladders from Java goats (*Capra aegagrus hircus*) were obtained from a local slaughterhouse. Goat bile (GB) was used to mimic the goat gallbladder fluid traditionally consumed by some communities in Indonesia to treat malaria. The gallbladders were surface cleaned with 70% alcohol and the GB was aspirated with a syringe and transferred into a centrifuge tube. Undiluted GB served as 100% GB (GB100). Lower concentrations were prepared by diluting GB with sterile distilled water (v/v) to obtain 50% (GB50) and 25% (GB25) solutions (6).

2.2. Animal Model and Treatment Protocol

A post-test-only control group design was used, in which outcomes were measured post-treatment and compared across groups. Fifty male BALB/c mice were intraperitoneally infected with 1×10^6 *P. berghei*-infected erythrocytes and randomly allocated to five groups (n=10 per group). Curative treatment began on day 3 post-infection. The treatment groups received GB at concentrations of 25% (GB25), 50% (GB50), and 100% (GB100) at 8 mL/kg body weight. The positive control group (POS) received dihydroartemisinin-piperaquine (DHP; 187.2 mg/kg body weight), and the negative control group (NEG) received sterile water only. All treatments were administered by oral gavage once daily for four consecutive days.

2.3. Measurement of Parasitemia

Parasitemia was assessed daily by microscopic examination of Giemsa-stained thin tail blood smears and expressed as the percentage of infected erythrocytes (14). Parasitemia on day 4 of treatment (day 7 post-infection) was used to analyse its correlation with IgG levels across all groups. Clinical signs and mortality were recorded for survival analysis.

2.4. Antigen Preparation

Parasites were collected from 20 male BALB/c mice infected with *P. berghei* when parasitemia exceeded 20% and were stored at -80°C until use. Parasites were lysed with 0.15% saponin and sonicated for 5 minutes in the presence of 1 mM phenylmethylsulfonyl fluoride (PMSF), followed by centrifugation at 5000 rpm and 4°C for 20 minutes. The supernatant was used as the crude antigen, and its protein concentration was measured using a NanoDrop spectrophotometer (Syaf Unica, Indonesia).

2.5. Measurement of Anti-*P. berghei* IgG Levels

After the 4-day treatment, blood was collected by cardiac puncture. Parasite-specific IgG levels were determined by indirect enzyme-linked immunosorbent assay (ELISA) with minor modifications to a previously described protocol (15). Briefly, 96-well microplates were coated with crude *P. berghei* antigen (200 µg/mL) overnight at 4°C, washed three times with phosphate-buffered saline containing 0.05% Tween 20 (PBST), and blocked with creamer at 37°C for 1 hour. After washing, bound antibodies were detected with horseradish peroxidase (HRP)-conjugated goat anti-mouse IgG (1:3000), followed by washing as above. Tetramethylbenzidine (TMB) substrate (0.3 mg/mL) was added and incubated in the dark

at room temperature for 30 minutes. The reaction was stopped with 1N H₂SO₄, and absorbance was read at 450 nm using a microplate reader (Bio-Rad, USA). All samples were assayed in duplicate on a single plate, and the mean optical density (OD) was used for analysis. The intra-assay coefficient of variation (CV) was calculated from the duplicate OD values. Because no standard curve was used, results are expressed as relative OD₄₅₀ values representing parasite-specific IgG levels.

2.6. Statistical Analysis

Parasitemia and anti-*P. berghei* IgG levels were summarised using descriptive statistics. Differences among groups were assessed by one-way ANOVA. Pairwise comparisons were performed with the Games-Howell post hoc test, which does not assume equal variances. The Bonferroni post hoc test was used for pairwise comparisons among the treated groups to control the family-wise error rate when multiple comparisons were performed. The association between individual parasitemia and IgG levels was evaluated using Spearman's rank correlation. Survival probability was estimated by Kaplan–Meier analysis and compared between groups using the log-rank test. Statistical significance was set at $p < 0.05$.

3. RESULTS AND DISCUSSION

3.1. Effect of GB on Parasitemia

On day 4 after treatment initiation, parasitemia levels showed an inverse trend (Figure 1). Among the goat bile (GB)-treated groups, GB100 showed the lowest parasitemia ($16.76 \pm 1.41\%$), followed by GB50 ($35.80 \pm 4.03\%$) and GB25 ($31.21 \pm 4.31\%$). These results demonstrate the effectiveness of GB in reducing parasitemia in a concentration-dependent manner. The positive control (POS; dihydroartemisinin–piperaquine, DHP) showed complete parasite clearance ($0.00 \pm 0.00\%$), confirming its antimalarial activity, whereas the negative control (NEG) group had the highest parasitemia ($38.20 \pm 4.03\%$), indicating uncontrolled parasite growth. One-way ANOVA followed by Games–Howell post hoc comparisons showed significant differences among groups ($p < 0.001$). Although GB100 produced the greatest parasite suppression, the response was not strictly dose-dependent, as parasitemia was higher in GB50 than in GB25. Moreover, GB100 was associated with reduced survival, whereas moderate concentrations (25%, 50%) achieved parasite control without compromising survival. These findings suggest that the biological activity of GB is not linearly beneficial and may reflect a balance between antiparasitic efficacy and systemic toxicity. Previous studies have reported that high concentrations of GB may induce adverse physiological effects (6,14) supporting the interpretation that excessive dosing limits therapeutic applicability.

This finding is consistent with previous studies demonstrating the effectiveness of GB in reducing parasitemia in a concentration-dependent manner (4,14). However, this should be considered together with the survival outcomes, particularly in the GB100 group.

3.2. Survival Rates

The effect of GB treatment on survival was analysed using the Kaplan–Meier method (Figure 2). In the GB100 group, one mouse died on 2-day post-treatment (90% survival), and three additional mice died by day 4, reducing survival to 60%. Mice in this group also developed diarrhoea, which was not observed in the GB50 group. No deaths occurred in the GB50, GB25, or POS groups (100% survival). In the NEG group, five mice died by day 4 post-infection, when parasitemia was high, resulting in 50% survival. Log-rank tests showed significant differences in survival rates between NEG and GB25, GB50, and POS ($p < 0.05$).

Differences in survival between the present study and our previous studies may reflect differences in experimental models and treatment conditions. The acute and subacute toxicity study by Arwati et al. (6) used non-infected BALB/c mice and therefore did not involve malaria-associated disease progression. The suppressive study by Arwati et al. (14) used *P. berghei*-infected mice but a suppressive-treatment design, whereas the present study used a curative-treatment design. Differences in animal cohorts, infection conditions and treatment protocols may therefore contribute to the variation in survival outcomes, although the available data do not allow any single factor to be identified as the definitive cause.

The present study has several limitations. First, the absence of biochemical or histopathological toxicity assessments were performed. Although the reduced survival in the GB100 group is consistent with previous reports of adverse effects of high-concentration GB (6), the absence of liver, intestinal, and other tissue assessments means that the pathological basis of the diarrhoea and reduced survival could not be confirmed. Second, the crude GB was not chemically characterised or quantified, so the constituents responsible for the observed anti-plasmodial activity or adverse effects cannot be determined. Future studies should combine biochemical and histopathological assessments with chemical characterisation and fractionation of GB to identify its active and potentially toxic constituents and to better define its safety profile.

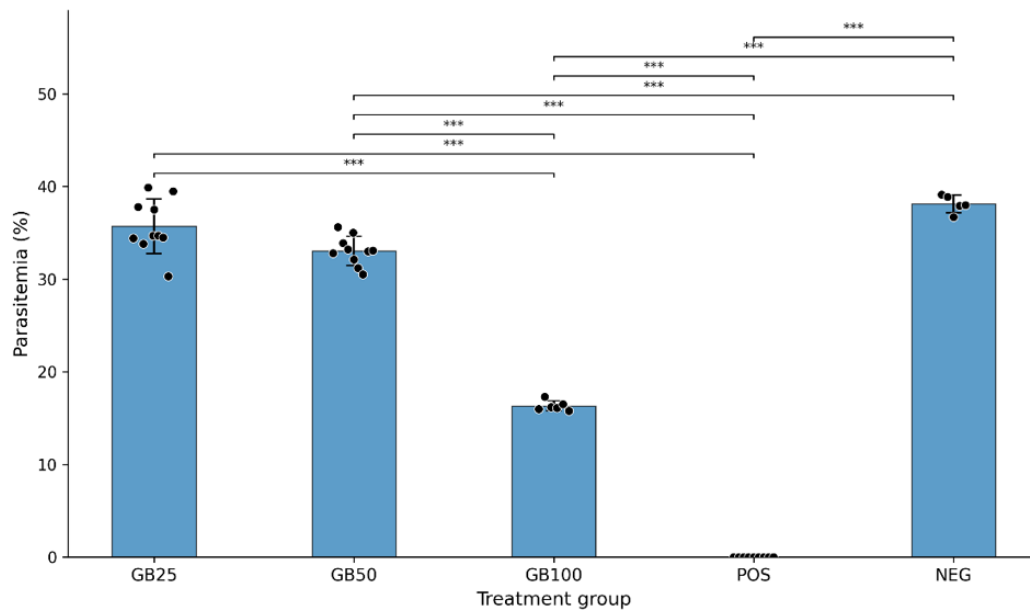


Figure 1. Mean percentage parasitemia on day 4 post-treatment initiation in *P. berghei*-infected BALB/c mice treated orally once daily with GB25, GB50, GB100 (8 mL/kg), dihydroartemisinin-piperaquine (DHP; 187.2 mg/kg body weight; POS), or sterile water (NEG). Individual points represent individual mice, and bars show mean $\pm$ SD. Statistical analysis was performed using one-way ANOVA followed by the Games–Howell post hoc test. *** $p < 0.001$.

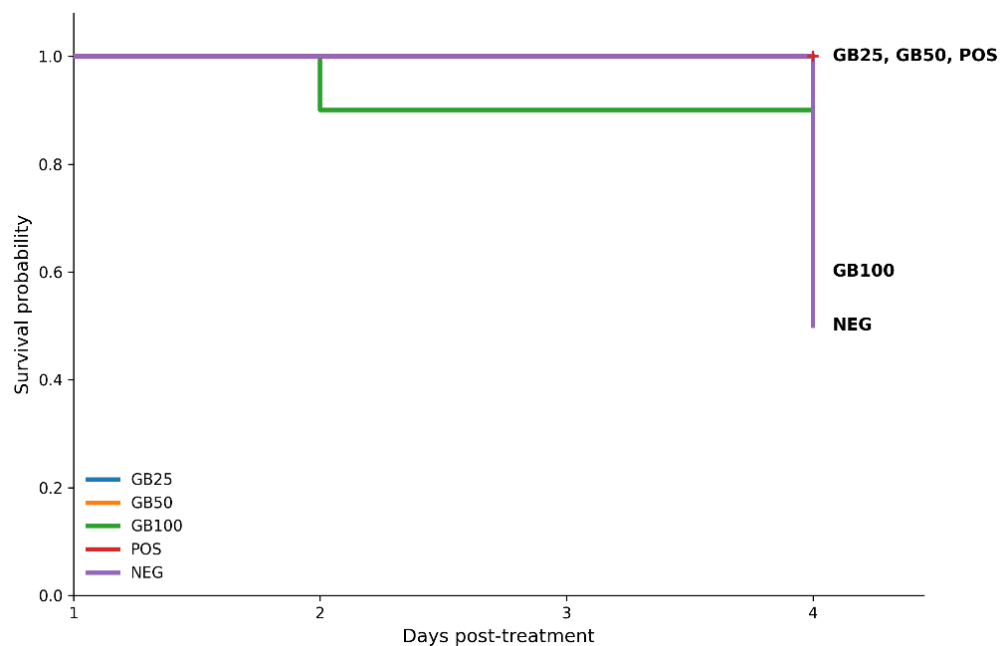


Figure 2. Kaplan–Meier survival estimate of *P. berghei*-infected mice. Survival remained at 100% in the GB25-, GB50-, and POS groups, while it declined to 60% in GB100 and 50% in untreated infected controls (NEG) by day 4 post-treatment initiation. Log-rank tests indicated significant differences between NEG and GB25, GB50, and POS ($p < 0.05$).

3.3. Effect of GB on the Anti-*P. berghei* IgG levels

IgG analysis was conducted using all available serum samples collected at the terminal time point. Because mortality differed among groups, sample sizes were unequal: GB25 ($n = 10$), GB50 ($n = 10$), GB100 ($n = 6$), POS ($n = 10$), and NEG ($n = 5$). Parasite-specific IgG levels were measured by indirect ELISA and expressed as OD450 values (Figure 3). Mean anti-*P. berghei* IgG levels (OD450) were 3.698 ± 0.208 , 3.046 ± 0.138 , 2.774 ± 0.240 , 3.004 ± 0.120 , and 2.722 ± 0.305 in the NEG, GB25, GB50, GB100, and POS groups, respectively. The intra-assay coefficient of variation, calculated from duplicate measurements (OD1 and OD2), was 2.44%. Bonferroni-adjusted comparisons excluding the NEG group showed significant

differences among the treated groups ($p < 0.001$). When the NEG group was included, the Games-Howell test showed that IgG levels differed significantly between NEG and the treated groups ($p < 0.05$), as well as between GB25 and GB50 ($p = 0.024$), GB50 and GB100 ($p = 0.006$), and GB25 and POS ($p = 0.024$).

Spearman's rank correlation of individual animal data ($n = 41$) showed a moderate positive correlation between parasitemia and anti-*P. berghei*-specific IgG levels ($\rho = 0.571$, $p < 0.001$). When the NEG group was excluded ($n = 36$), the correlation was weaker but remained significant ($\rho = 0.394$, $p = 0.018$). Mice with higher parasite burdens therefore tended to show higher anti-*P. berghei*-specific IgG levels (16), suggesting that antibody magnitude was associated with parasite burden in this model, possibly because greater antigenic stimulation drives stronger B cell responses (17). This is consistent with previous reports of dynamic parasite-specific antibody responses during malaria infection (18,19). In human *P. falciparum* infection, IgG responses increase after infection, although their magnitude and timing vary with parasite antigen and prior exposure (18,19). Longitudinal studies describe distinct kinetic patterns, with rapid induction followed by decline for some antigens and longer persistence for others (19-21). In *P. berghei* models, parasite-specific IgG is detectable early and increases during later stages of infection (22). In the present study, however, IgG was measured at a single terminal time point, so antibody kinetics could not be determined.

Correlations among parasitemia, anti-*P. berghei* IgG, and survival are shown in Figure 4. In our previous study, goat bile suppressed total serum IgG levels in infected mice (5). However, total IgG does not necessarily reflect antigen-specific protective immunity. The present study evaluated anti-*P. berghei*-specific IgG and found that its magnitude was associated with parasitemia rather than with survival (23), highlighting the need to distinguish global immunoglobulin responses from parasite-specific humoral responses.

Importantly, indirect ELISA with crude *P. berghei* antigen detects antigen-binding antibodies but does not distinguish functional or protective activity. Binding does not necessarily reflect opsonization capacity, neutralizing function, or growth-inhibitory activity. The IgG levels reported here therefore represent relative humoral reactivity rather than direct evidence of protective immunity. Functional assays would be needed to determine whether GB influences antibody quality as well as magnitude.

Beyond its antiparasitic effect, GB treatment was associated with changes in anti-*P. berghei*-specific IgG levels. Bile acids are increasingly recognized as signaling molecules that modulate immune cell function through receptors such as farnesoid X receptor (FXR) and Takeda G protein-coupled receptor 5 (TGR5). These pathways regulate macrophage activation and inflammatory signaling and may indirectly influence B-cell differentiation and antibody production (12,19-21). However, these pathways or immune-cell responses were not assessed here, and B-cell phenotype, germinal center responses, or antibody subclass distribution were not examined. The observed IgG changes therefore do not establish a direct effect of GB on adaptive immunity. Further studies are needed to determine whether GB directly influences immune responses or whether the changes mainly reflect differences in parasite burden.

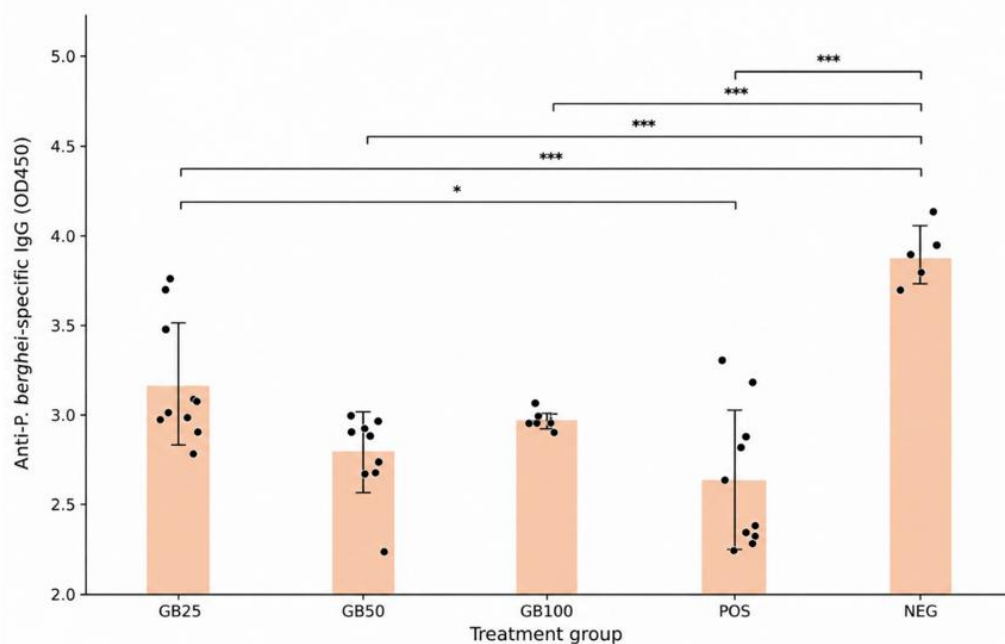


Figure 3. Anti-*P. berghei* IgG levels in mice treated with GB in groups of GB25 ($n=10$), GB50 ($n=10$), GB100 ($n=6$), with 182.7 mg/mL dihydroartemisinin-piperaquine in the POS group ($n=10$), and NEG (infected mice with no treatment, $n=5$). Data represent mean $\pm$ SD. Statistical analysis was performed using one-way ANOVA followed by the Games-Howell post hoc test. Individual points represent individual mice; * $p < 0.05$. *** $p < 0.001$

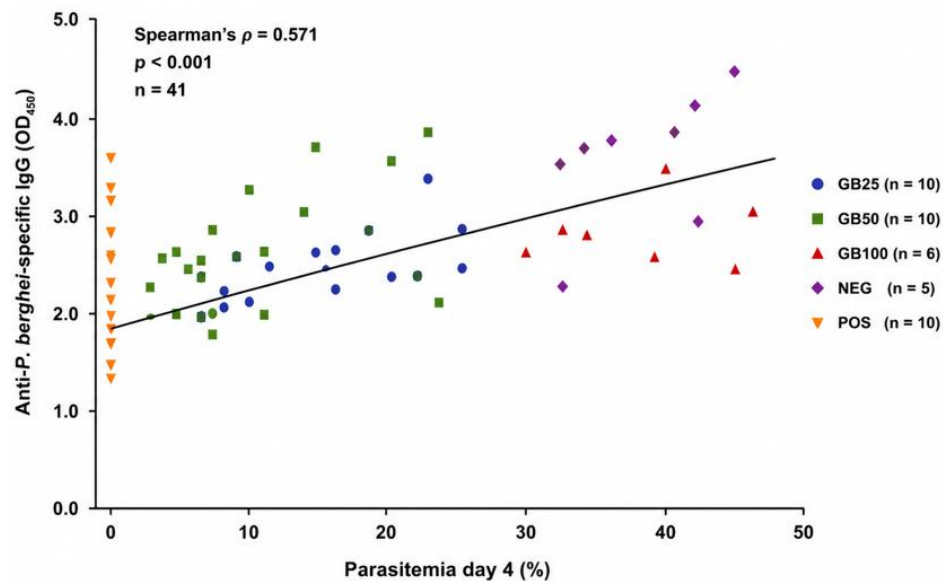


Figure 4. Relationship between parasitemia on day 4 and anti-*P. berghei*-specific IgG (OD450) in individual mice (n = 41). Each point represents one mouse, with colors and symbols indicating the treatment groups as shown in the legend. Spearman correlation analysis showed a significant positive correlation between parasitemia and anti-*P. berghei*-specific IgG ($\rho = 0.571$, $p < 0.001$)

4. CONCLUSIONS

Goat bile (GB) treatment was associated with reduced parasitemia and altered anti-*P. berghei*-specific IgG levels in *P. berghei*-infected BALB/c mice. Parasitemia and anti-*P. berghei*-specific IgG were positively correlated, suggesting that antibody levels reflected parasite burden in this model. However, these findings do not establish a direct immunomodulatory or immune-protective effect of GB. Further studies are needed to determine whether GB directly influences adaptive immune responses and to elucidate the mechanisms underlying the observed changes in parasite-specific IgG.

Author contributions: HA: Designed and led the research project and drafted and finalized the manuscript. CC: Performed *P. berghei* infection and mouse treatments. ENF, ML: Prepared thin blood smears, counted parasitemia, sacrificed mice, collected blood, and organized and analyzed the data. All authors read and approved the final manuscript.

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Ethics statement: The proposal of this study has been reviewed and approved by the Research Ethical Committee of the Faculty of Dental Medicine, Universitas Airlangga, Surabaya, Indonesia, as stated in certificate number 531/HRECC.FODM/XII/2020.

Conflict of interest: The authors declare no competing conflicts of interest.

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