Dynamics of B cell subpopulations and immunoglobulin repertoire during experimental infection with *Plasmodium yoelii* 17XNL

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Malaria, caused by *Plasmodium* parasites, is a major global health burden. Although B lymphocytes and antibodies are crucial for controlling parasitemia, infection alters B cell function, potentially leading to dysregulated immune responses and autoreactive antibodies. Understanding these dynamics is essential for elucidating protective immunity and pathogenic mechanisms during malaria. This study aimed to characterize dynamic alterations in B lymphocyte subpopulations and the antigen-specific antibody repertoire following *Plasmodium yoelii* 17XNL infection in a non-lethal murine model. To assess antigen-specific responses, the $MSP1_{19}$ protein was recombinantly expressed in bacteria and validated by Western blot. C57BL/6 mice were intraperitoneallly infected with $1x10^6$ *P. yoelii* 17XNL-infected red blood cells (iRBCs) or healthy RBCs as controls. Parasitemia was monitored daily by GFP expression using flow cytometry. Hematological analyses were performed in parallel to evaluate infection-associated alterations in peripheral blood parameters. For B cell profiling, splenocytes were collected at 10 days post-infection (p.i.) and analyzed by flow cytometry. Parasitemia peaked approximately 5 days p.i. and gradually declined, leading to complete infection control by day 20. Hematological evaluation revealed significant alterations at day 14 p.i., including a macrocytic, hypochromic erythrogram with anisocytosis, accompanied by leukocytosis and thrombocytopenia, consistent with infection-associated anemia. Infected mice exhibited significant splenomegaly. Flow cytometric analysis revealed an increase in the frequency and absolute number of various B lymphocyte subsets in infected mice compared to uninfected controls. Notably, a fraction of atypical memory B cells co-expressed FAS and GL7. These findings demonstrate pronounced hematological alterations and significant remodeling of B lymphocyte populations during experimental *P. yoelii* 17XNL infection.

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