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A Live *Plasmodium yoelii* 17XNL Vaccination Prevents the Development of Experimental Cerebral Malaria

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Introduction

Malaria is a parasite illness spread by mosquitos. According to the World Health Organization (WHO), there were 238,000,000 estimated malaria cases worldwide in 2000 and 241,000,000 in 2020. Malaria treatment and prevention have benefited from mass medicine administration, bed nets and indoor residual pesticide spraying. Despite this, the WHO estimates that 627,000 people will die from malaria by 2020. Furthermore, there has been a disturbing increase in the occurrence of insecticide-resistant mosquitoes and drug-resistant malaria parasites, as well as problematic linkages between malaria infections and the current COVID-19 pandemic.

A vaccination is one of the ways that might be utilised to manage malaria and address the issues outlined above. Malaria vaccine development has advanced in recent years. Malaria vaccines of several forms are being developed, including protein subunit vaccinations, DNA vaccines, viral vector or virus-like particle vaccines, whole parasite vaccines and genetically/chemically attenuated parasite vaccines, with some of them presently in clinical trials. The leading malaria vaccine, RTS, S/AS01 (Mosquirix), has been provided to 800,000 children in Ghana, Kenya and Malawi in an ongoing trial phase since 2019.

Description

The immune system's function during malaria is multifaceted. T cells, for example, respond differently to *Plasmodium yoelii* (Py) and Plasmodium berghei ANKA (PbA: deadly strain) in the blood stage in a C57BL/6 (B6) mouse malaria model. T lymphocytes (helper and killer T cells) are protective in Py but produce pathology in PbA, leading to the development of deadly experimental cerebral malaria (ECM). During the T cell-mediated immune response to Plasmodium, the immune system therefore operates as a "two-edged sword." Different Plasmodium strains, Py17XNL (non-fatal strain) and Py17XL (lethal strain), as well as different PbA species, exhibit distinct features [1].

PyNL parasites prefer to infect young RBCs (reticulocytes), but they can also infect RBC precursors, erythroblasts. Erythroblasts express MHC class I molecules that CD8T cells detect and parasitized erythroblasts are then destroyed by CD8T cells and macrophages. Some MHC class I molecules are expressed by reticulocytes but are not recognised by CD8T cells. PyL, on the other hand, infects both reticulocytes and mature RBCs, whereas PbA prefers to infect reticulocytes but does not infect erythroblasts. PyNL is comparable to *P. vivax* (Pv), which likes to infect reticulocytes. Because human reticulocytes contain MHC class I molecules, CD8T cells can detect and destroy parasitized RBCs. CD4T and CD8T cells are both engaged in ECM [2].

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The current investigation showed that live PyNL immunisation afforded sterile and long-term immunity against infection with a homologous fatal PyL strain but not against heterologous PbA infection. After cryopreservation, the PyL strain was generated from PyNL and is virtually antigenically similar to PyNL. This similarity in identity might have led in the development of sterile immunity. Although the vaccine did not protect animals against PbA infection, it did prevent ECM and MA-ARDS in live-vaccinated mice.Despite recent findings that an attenuated sporozoite vaccine containing Pb or Py protected certain mice against heterologous challenge infection, our live immunisation failed to generate sterile immunity against heterologous challenge infection [3].

Because a blood-stage *P. chabaudi* chemically attenuated vaccine gave protection against Py or P. vinckei, heterologous protection may be inducible in a mouse malaria model. Chimera Pf expressing PvCSP was created in and would be one of the contenders for a next-generation human cross-protective malaria vaccine. Malaria pathogenicity occurs during the erythrocytic cycle of malaria parasites and two basic pathological processes induce symptoms. One of these is parasite growth, often known as recurrent erythrocytic cycles. Severe anaemia can arise from the massive loss of RBCs. The other is immunopathology caused by overly strong host immunity to malaria parasites.

The combination of these two characteristics worsens malaria pathogenicity. PyL's high virulence is mostly due to parasitic proliferation, which results in mortality from severe anaemia with hyperparasitemia. In contrast, ECM found after PbA infection is immunopathologically reliant and develops while parasitemia is fairly low. Malaria is prevented by blocking one or both of these pathogenic pathways. Antibodies against parasite antigens and phagocytes are important in interrupting the erythrocytic cycle. Phagocytes, such as macrophages, absorb parasites and parasitized RBCs and are necessary for malaria parasite clearance [4].

Many studies have stressed the importance of antibodies in parasitemia control and practically all malaria vaccines are designed to generate particular antibodies. Antibodies inhibit merozoite motility and invasion, as well as egress from RBCs, allowing antibodies to cytolyze merozoites via antibody-dependent complement-mediated cytotoxicity and increase phagocytosis. PyNL immunisation generates antibodies that detect homologous PyL antigens, resulting in sterile immunity and limiting PyL's erythrocytic cycle. Although PyNL immunisation generated cross-reactivity against PbA, it may not be sufficient to interrupt PbA erythrocytic cycles. More research is needed to evaluate the amount of cross-reactivity of PyNL live vaccination antibodies against other strains.

A recent study found that the BCG (bacille Calmette-Guerin) immunisation prevented ECM. CD8T cells did not accumulate in the brains of PbA-infected mice in BCG-vaccinated animals. BCG had no effect on parasitemia and was unable to rescue mice lives after infection. This behaviour was observed in our studies of mice after live immunisation. The mechanism by which cerebral malaria develops in humans and animal models is still being debated. Post-mortem brain examination has traditionally been used to investigate the process of development of human cerebral malaria, with parasite or parasitized RBC sequestration assumed to be the predominant source of disease rather than immunopathology [5].

Sequestration of RBCs in brain BVs was reported in the current investigation. Another investigation found that a single PbA pRBC is enough to occlude blood vessels. According to recent research, invading CD8T cells were discovered in the brains of malaria patients. Because immunopathology has been observed in other infectious disorders, such as COVID-19, there is

more than enough evidence to rethink CM immunopathology. The processes of mouse ECM and human CM may not be as dissimilar as originally assumed.

The benefit of live vaccination is that it does not require booster vaccinations. Although live PyNL immunisation might impart significant immunity to PyL, vaccine-administered mice experienced adverse effects including as anaemia, fever and splenomegaly. As a result, more parasite attenuation is required to avoid undesirable outcomes.

Conclusion

Live PyNL immunisation offered homologous cross-strain protection and prevented cross-species ECM and MA-ARDS.

Acknowledgement

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Conflict of Interest

No potential conflict of interest was reported by the authors.

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