I think that the life cycle of malaria plasmodium falciparum is too complex for the human body.

I estimate that each human gene have a genetic change each 30 year in the population (spontaneous mutation in prophase I of meiosis), so the genetic change in the population is too slow for modify the microbes genetic in so complex defence against antibodies.

I think that the malaria genetic change quickly to adapt to a host animal with a quick genetic change, an animal with liver and blood like human tissues: my hypothesis is that animal is the mouse (or, less likely, some bird with short life).

Each three or four month the mouse population change, then the genetic chromosome change is ~ 100 time more quickly of the human genetic, moreover the mouse population is greater of the human population, so there is a population multiplier that increase the genetic change.

If a virus, or a microbes, want to survive in the host animal it must to make a quick genetic evolution, so it can adapt to the quick genetic change (I think that this is like to aids disease, or seasonal fever, or plague, or malaria, or Kinetoplastid infectious disease): it is complex to obtain a laboratory cure for these disease because there is a complex interaction with the host, but the natural host animal defence permit to evaluate the cure with this complex interactions.

I think that the study of antibodies of the host animals with liver (not insects) in malaria zone is the perfect genetic laboratory that contrast - casually=genetically - the disease diffusion using random drug (proteins and antibodies modified by natural spontaneous mutation): the Nature in 65 million of year make drug research.

If you see the Earth 65 million year ago, there is hosts (mammal as mouse), vectors (insects), and microbes (blood diseases): the blood disease are not human disease, are all hosts mammal disease.

If you see the blood infectious disease in the world, seem that there is ever present the mouse host, because is the first mammal on the Earth: I think that the continuous genetic change in the mouse population can give some mouse with antibodies, and other without right antibody.

I think that is like the Fleming mould, that are cells that compete against microbes: their evolution is quickly, their number is huge, and their defence is the genetic change.