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Severe anemia and induction of extra-medullary erythropoiesis in murine malaria

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In malaria infection, induction of sufficient immunity alone is not enough for survival of the infected individuals or animals. Death because of malaria-associated anemia may occur even with ideal protective immune responses. Human complicated falciparum malaria is sometimes accompanied by severe anemia which is not replenished by compensatory erythropoiesis. The present study shows that anemia in *Plasmodium yoelii* 17XNL infected C57BL/6 mice occurs not only due to rupture of parasitized erythrocytes but also due to other unknown mechanisms. Especially hemolytic anemia because of induction of anti-erythrocyte autoantibodies resulting in destruction of both infected and uninfected erythrocytes leading to severe anemia. Interestingly, *Plasmodium yoelii* 17XNL infected C57BL/6 mice survive the anemia-related severity through massive erythropoiesis. Using reinfection of *Plasmodium yoelii* 17XNL infected C57BL/6 mouse model, we showed that HCT (hematocrit) came down from a normal value of 52% to as low as 33%, even when the peak parasitemia was only 0.25%, suggesting destruction of erythrocytes by other mechanisms rather than simply being parasite-mediated rupture of erythrocytes only. The study also offers an excellent opportunity to show how C57BL/6 mice escape death from malaria-associated severe anemia through replenishment of erythrocyte loss by extra medullary erythropoiesis in the spleen.

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