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ENHANCED RESISTANCE OF HIGHLY SUSCEPTIBLE BALB/C MICE TO INFECTION WITH TRYPANOSOMA CONGOLENSE AFTER INFECTION AND CURE

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ABSTRACT

Balb/c and C57Bl/6 mice were cured with Berenil after infection with cloned organisms of *Trypanosoma congolense* and challenged with homologous or heterologous variants. The mice were fully protected against infection with 10^3 but not 10^5 organisms of the homologous variant. Normal Balb/c mice infected with 10^5 organisms developed uncontrolled parasitemia and had a mean survival time of 8.4 days. Challenge of drug cured Balb/c mice with 10^5 organisms of the homologous variant established an infection associated with prolonged prepatent period, control of the first peak of parasitemia, and prolonged survival time (36 days). Indirect immunofluorescent and agglutination tests on live trypanosomes revealed that the "delayed" population of the first peak of parasitemia consisted of variants other than that used for challenge. No protection of drug-cured Balb/c mice was obtained following challenge with 10^3 - 10^5 organisms of a heterologous variant.

Passive transfer of variant-specific antiserum protected mice against infection with 10^3 organisms. Against infection with 10^5 organisms, it resulted in a prolonged prepatent period but had no effect on severity of parasitemia or duration of survival. There was no evidence for persistence of Berenil, which potentially could affect resistance. It was concluded that enhanced immunity in drug-cured Balb/c mice was due to (a) antibody to the variant surface glycoprotein and (b) another, yet unidentified, synergistically acting immune response to the parasite. Possible mechanisms are discussed.

Keyword