Reactivation of the Brazilian *Toxoplasma gondii* EGS Strain in an Immunosuppressed Murine Model.

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Toxoplasma gondii, an obligate intracellular parasite of the phylum Apicomplexa, is the etiological agent of toxoplasmosis. The clinical outcome of toxoplasmosis is associated with the parasite strains and the immunological status of the host. Strains of *T. gondii* isolated in Brazil exhibit significant genetic variability and deviate from the clinical patterns seen in other countries. The EGS strain, isolated in 1998 from the amniotic fluid of a patient in Minas Gerais/Brazil, presents a recombinant genotype (I/III). In the chronic phase of infection, tissue cysts form mainly in the central nervous system and muscle tissue. Immunosuppressed individuals, whether due to genetic factors, HIV infection, or immunosuppressive treatments are at risk of infection reactivation, which can culminate in fatal cases. Despite its clinical significance, the mechanisms underlying toxoplasmosis reactivation are poorly understood. Our study aims to establish *T. gondii* reactivation using a murine immunosuppression model infected with the EGS strain. C57BL/6 mice, 30 days post-infection, were treated with dexamethasone at 5 or 10 mg/kg. After 14 days, dexamethasone treatment led to a reduction in $CD4^+$ T, $CD8^+$ T, and B cells in the spleen, validating the immunosuppression status. Additionally, serological data indicated reactivation of the infection, with elevated IgM levels in the blood of treated animals. The treatment increased parasite load, as evidenced by higher numbers of tachyzoites in the ventral region assessed using the IVIS Lumina imaging system. The PCR analysis of the SAG-1 gene in the brain showed an increase in tachyzoites, particularly at the 5 mg/kg dose. These results demonstrate that DEX is capable of promoting immunosuppression in mice during infection with the EGS strain.

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