Impact of Dexamethasone on the Conversion of *T. gondii* within Neural Cells

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Toxoplasma gondii is an obligatory intracellular parasite and the causative agent of toxoplasmosis. The parasite exhibits two distinct infectious forms within hosts: tachyzoites, present in the acute phase of infection, and bradyzoites, which form tissue cysts in the chronic phase of infection. The EGS strain, isolated from amniotic fluid in MG, exhibits a recombinant genotype, both reflecting a phenotype of acute infection and the ability to form cysts spontaneously. Dexamethasone (DEX) is an immunosuppressive drug that can induce changes in lymphoid organs and, in patients chronically infected with *T. gondii*, the reactivation of the acute phase. This study aims to evaluate the conversion of the EGS strain *in vitro*. Initially, we analyzed the spontaneous or alkalinization-induced encystment of *T. gondii* in mouse neural cell lines. Neuro-2a cells were plated and infected with EGS strain at a MOI: 3, cultured in a medium at neutral or pH 8.0. Subsequently, they were fixed at 96-216 hours p.i. and incubated, with DBA-FITC (cyst wall marker), SAG-1 (tachyzoites maker) and BAG-5 (bradyzoite marker). We identified the predominance of bradyzoites mainly in samples cultured at alkaline pH. The effect of DEX [10μM] on the expression of stage-specific genes, SAG-1 and BAG-1, and on the egress of *T. gondii* was also evaluated. In the preliminary RT-qPCR results, no significant alterations were identified between the treated and untreated samples. However, the increase of extracellular parasites amount in the treated samples suggests that DEX induced the egress of the parasite from the host cell. Taken together, our early data suggest that alkalinization of the medium promoted an increase in the conversion to bradyzoites in Neuro-2a. In addition, the egress of *T. gondii* promoted by DEX treatment may provide clues about the cellular mechanism of action of DEX in the reactivation of the infection.

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