BI-19
^{60}CO IONIZING RADIATION EFFECTS IN THE PHYSIOLOGY AND CELLULAR INVASION OF *TOXOPLASMA GONDII* TACHYZOITES

Hiramoto RM, Galisteo Jr. AL*, Cardoso RPA*, Andrade Jr. HF*
Serviço de Radiobiologia IPEN/CNEN *Laboratório de Protozoologia, IMTSP, Av. Dr.E.C.Aguiar 470, 05403-000 São Paulo, SP, Brasil (E-mail: hfandrad@usp.br)

Toxoplasma gondii is an intracellular obligatory protozoon, with a complex life cycle, with felids as definitive host, and warm-blooded mammalian and birds as intermediate host. The infection is transmitted through the consumption of cysts on infected meat by or oocysts of cat's feces in contaminated food or water. Widespread among humans and generally asymptomatic, this agent could induce devastating disease in fetus, AIDS patients and recipients of organ transplants. The ionizing radiation was used to sterilized meat and immunized animals against *T. gondii*, with encouraging results, and here we study the physiological alterations and cellular invasiveness of 200 Gy irradiated tachyzoites. After the irradiation, we study the physiology of the agent by precursor incorporation in short term cultures, aside to metabolic assays using MTT(3-[4,5-Dimethylthiazol-2-yl]-2,5-diphenyltetrazolium bromide; Tyazolyl blue) as revealing agent of oxidative metabolism, and morphological study of its invasiveness on LLC-MK2 and human fibroblasts. The irradiated parasites presented the same oxidative response, protein synthesis(^{3}H-prolina) and nucleic acid(^{3}H-hypoxantina) incorporation as their non-irradiated counterparts. These irradiated parasites had the same capability of cell invasion and parasitic vacuole formation on both cells tested, as compared to non-irradiated agents. No further growth of the parasite was observed in those cultures, with some clearly degeneration of the irradiated agent after invasion, suggesting that the irradiation induced a mitotic death by double strand breaks in DNA. Those data reinforces the fact that, at those level of radiation, the only effect was the reproductive blockade of the parasite, with preservation of most of their metabolic and physiologic activity, an desirable effect in vaccine development.

RM Hiramoto is a fellow of CNPq. AJ Galisteo Jr. is a fellow of Fapesp (98/1681-0).
Fapesp (96/5875-8) and LIMHCFMUSP-49 supported this work.

BI-20
A NEW MODEL FOR THE STUDY OF THE DAMAGE AND RECOVERY OF SYMPATHETIC NERVE TERMINALS INDUCED BY *TRYPANOSOMA CRUZI* AND OTHER PRO-INFLAMMATORY AGENTS USING THE RAT DUCTUS DEFERENS

Arantes RME, Galvão LMC, Santos MMF, Machado CRS
Departamentos de Morfologia e de Parasitologia, Instituto de Ciências Biológicas, UFMG, Belo Horizonte, MG, Brasil

We have developed and standardized an experimental model for studying inflammatory processes induced directly in the smooth muscle layer of the rat ductus deferens aiming at assessing their effects on the sympathetic innervation. Besides an easy surgical accessibility, the rat vas deferens has a rich post-ganglionic sympathetic innervation that remains unaffected by *Trypanosoma cruzi* infection (Y strain) via the peritoneal cavity. Adult Holtzman rats (180 animals) comprised the following groups according to the material injected directly in the ductal wall: alive trypomastigotes (Y strain), dead trypomastigotes, supernatant of *T. cruzi* culture, carrageenan and methylated albumin. In this later case sensitized and non-sensitized animals were used. Controls were provided by sham-operation, inoculation of sterile saline or culture medium. Ductus deferens fragments were withdrawn at 48 hours, 4, 8,10, 12, 14, 20 days and processed for histological study and for the histochemical demonstration of catecholamines by a glyoxylic acid technique. In groups inoculated with *T. cruzi* forms or culture supernatant the hearts were also histologically studied. Regarding the experiments with alive trypomastigotes, the kinetic of the inflammation was faster in the ductal smooth muscle than in heart. In the ductus, the most intense mononuclear exudate and parasitism occurred at days 8-12. Afterwards there was a fast recovery, the normal histological pattern being reached at day 20. By this time the heart still exhibited intense inflammatory infiltrate. The glyoxylic-acid-induced fluorescence showed intense and diffuse reduction of nerve terminals in the ductus at days 8 to 14. At day 20, the recovery of the normal pattern of innervation was still incomplete, in spite of the normal histology. All other procedures induced focal rarefaction of nerve terminals restricted to tissue damage, with faster recovery. We conclude that the nerve lesions occurred in parallel with the inflammatory process and that the regeneration of the smooth muscle was faster than that of the nerve terminals. Our model is suitable for *in vivo* interventions aiming at inhibiting hypothetical mechanisms supposed to be implicated in the pathology of Chagas' disease. This is our next objective.

Supported by Pronex-1996, Fapemig and CNPq.