The role of B-1 cell and cytokines involved in the pathogenesis of microsporidiosis

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Microsporidia are unicellular organisms that infect a wide range of vertebrate and invertebrate species. Encephalitozoon cuniculi is one of the most common microsporidian species in human. These pathogens were considered as protozoa but recently have been reclassified phylogenetically as fungi. Our knowledge about the immunity during microsporidiosis is not fully understood and we have decided to study the in vitro role of B-1 cells and cytokines involved in the immune response against E. cuniculi infection. Supernatants from cultures of adherent peritoneal cells (APerC) from BALB/c mice and APerC from XID mice (B-1 cells deficient) infected and not with E. cuniculi after 30 min, 1h, 48h, 96h and 144h was collected and cytokines were measured. In the supernatants of APerC from BALB/c mice there were levels of MCP-1 and TNF detected, and they decreased after 48 hours in both groups (uninfected and infected with E. cuniculi). Besides that, increased levels of IL-6 were observed in infected group in 96 and 144 hours and IL-10 in 144 hours. In supernatants of APerC from XID there were low levels of MCP-1 and TNF in both groups and levels of TNF decreased after 96 hours and MPC-1 were not found after this time. There was an increased in levels of IL-10 in the infected group with 30 minutes and 1 hour. The cytokine IL-12 was tested but detectable levels were not found. These preliminary results of our research can help to better understand the role of B-1 cell and cytokines involved in the pathogenesis of E. cuniculi infection.

Biography

Adriano Pereira is working as a Teacher in the areas of Health and Biological Sciences at São Camilo University, São Paulo, Brazil. He has obtained his Master’s Degree in Veterinary Medicine and is currently pursuing his PhD studies in Environmental and Experimental Pathology. His research involves studying microsporidia with a focus on biology and immune response against this emerging and opportunistic pathogen.

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