Toxoplasmic Retinochoroiditis in the Hamster

To the Editor:

The article by Pavesio and colleagues (IOVS 1995;36:2166–2175) concerning the clinico-pathological features of Toxoplasma-associated retinochoroiditis in a hamster model is of interest, particularly when the need to identify and test novel treatments for this ocular infection is considered. The value of their study was clearly stated, as the ease of production of ocular and protozoological manifestations in both eyes of the infected animal after a relatively short post-infection period, when overt clinical signs of systemic toxoplasmosis, had not become apparent. There are a number of issues, however, that merit clarification.

Many of the histopathologic features described are reminiscent of those recorded from a murine model of congenital ocular toxoplasmosis, particularly selective outer retinal destruction and vasculitis. Such lesions are similar architecturally to those observed in experimental autoimmune uveoretinitis. This observation has led us to postulate that Toxoplasma, in some circumstances, may act as a type of adjuvant in the induction of retinal inflammation.

The absence of Toxoplasma antigen without intact tissue cysts confirms our own light microscope immunohistochemical observations using the mouse model. We have, however, detected such antigen in brain tissue from our model using more sensitive immunoelectron microscopy.

The authors draw attention to differences in the intracellular status of tissue cysts between the ME 49 and Beverley (RRA) strains of Toxoplasma, suggesting that the former can exist extracellularly in murine brain tissue. Detailed ultrastructural and immunohistochemical studies have consistently shown that in the eye and brain of the mouse model, intact tissue cysts of the RRA strain always are located intracellularly. In the retina, they reside within neurons or Müller cells.

It has been suggested that the murine model of ocular toxoplasmosis would enhance the expedient evaluation of novel agents for efficacy in specific disease states. The hamster model may be more useful in this context, with the proviso that if the intact tissue cysts are truly “extracellular” within the retina of these animals, the potential usefulness of candidate drugs would have to be examined further in situations in which there is definite evidence of an intracellular location. This is of special importance for the prevention of recrudescence of ocular toxoplasmosis, which may involve activation of the intact tissue cyst.

For these reasons, we contend that the quest for a satisfactory animal model of ocular toxoplasmosis must be continued. This is especially so in the investigation of factors associated with the immunopathology of the infection. In this context, it would have been useful if the authors had recorded the age and sex of their experimental animals, factors that are known to influence the outcome of a Toxoplasma infection in rodents.

John Hay, PhD
Gordon N. Dutton, MD
Tennent Institute of Ophthalmology
University of Glasgow
United Kingdom

References