INNATE IMMUNITY TO *RHINOSPORIDIUM SEEBERI* IN GUINEA PIGS

W.M. TILAKARATNE AND S.N. ARSECULARATNE*

*Department of Oral Pathology, Faculty of Dental Sciences, *Department of Microbiology, Faculty of Medicine, University of Peradeniya*

No farm or laboratory animal has supported the growth of R. Seeberi after experimental inoculation. Inocula in congenitally immuno deficient mice also failed to reproduce the disease, suggesting that specific adaptive immunity was not involved in preventing the establishment of infection. The testing of the possibility that non-specific immune mechanisms, notably the phagocytes, were responsible for the elimination of experimental inocula was planned with the guinea pigs skin as the test tissue, with H & E examination of sections of the intrademally-injected sites at serial intervals. PAS stained sections of these sites for the rhinosporidial bodies were performed. Identification of the specific lymphocytes with fluorescent antibody markers for macrophages, B and T cell subsets was planned as the second step.

Method

Intradermal inocula of 0.1 ml of suspensions of purified rhinosporidial endospores and juvenile sporangia (containing endospores 550/ml and sporangia 365/ml) were injected into the flanks of two guinea pigs (Hartley strain), and 1 injected site (total skin thickness) was excised from each animal under local anesthesia at intervals of 1.3.5.7.10.15.18.21 days. The tissue was fixed in buffered formal-saline and processed for H &E and PAS staining.

Results

The tissues showed host cell infiltration beginning around the 3rd day, consisting of neutrophils, macrophages and lymphocytes. Some free endospores were seen. Between the 7th – 15th days, the macrophages infiltration was marked, with some containing PAS debris possibly of endospore origin. Sporangia were not seen. In later stages, the endospores were absent.

Conclusion

The tentative conclusion is that rhinosporidial endospores are phagocytosed and destroyed by macrophages. Further detailed tests on the guinea pig skin were abandoned on account of the failure to obtain the specific markers for specific cells from abroad. Alternative experiments are planned with mouse macrophages in vitro for tests of phagocytosis and with liposome-blockaded macrophages in vivo in mice for the ability of these mice to support rhinosporidiosis.