

INTERNALIZATION OF *CRYPTOCOCCUS NEOFORMANS* AND ITS  
MAJOR CAPSULE COMPONENT BY HUMAN BRAIN PHAGOCYTES

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The encapsulated fungus *Cryptococcus neoformans* is the etiological agent of cryptococcosis, a respiratory syndrome that can evolve to a lethal meningoencephalitis. The internalization of this yeast-like pathogen occurs after its adhesion to host cells, frequently mediated by the capsular polysaccharide. Capsular antigens are associated to the fungal surface, but are also released to the extracellular environment. In the present study, we observed that surface-associated capsule polysaccharides did not interfere with the adhesion of *C. neoformans* to U87 cells a human astroglia-derived cell line. The endocytosis of soluble glucuronoxylomannan (GXM), the major cryptococcal capsule component, by these cells was also evaluated. Polysaccharide internalization occurred after 30 minutes of its interaction with host cells. Immunofluorescence microscopy using GXM-binding antibodies suggested that aggregation can occur in specific surface sites. Microscopic analysis associated to the lactate dehydrogenase assay indicated that GXM damaged host cells, although cell lysis was only observed in higher polysaccharide concentrations. We also evaluated the effect of GXM on the expression of gangliosides by U87 cells, since these glycosphingolipids are key elements in endocytic processes. The polysaccharide induced an increased ganglioside expression. The present results indicate that the major capsular antigen of *C. neoformans* is internalized by brain cells. As a consequence, host cells can be damaged, which may be associated to the polysaccharide-mediated regulation of their sphingolipid metabolism.