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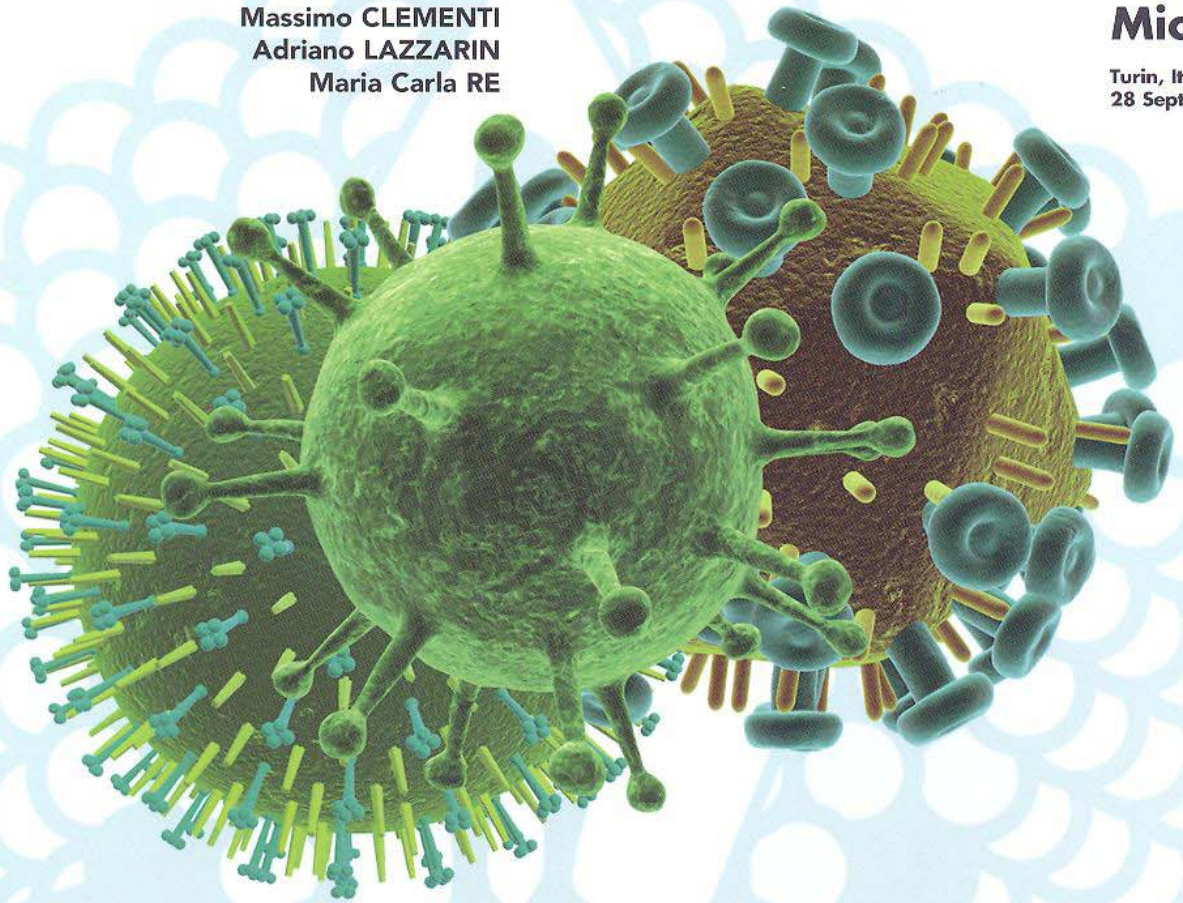

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IMMUNOREACTIVITY OF MICROGLIAL CELLS TO IN VITRO INFECTION BY *CANDIDA ALBICANS* ISOLATES WITH DIFFERENT GENOMIC BACKGROUNDS

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Introduction: *C. albicans* is an important opportunistic yeast causing superficial, as well as invasive infections in susceptible host, while behaving as an harmless commensal on healthy individuals. A complex interplay between host immune defence and fungal virulence mechanisms accounts for the outcome of infection. In this respect, we have previously shown that microglia, the resident phagocytes of the brain, plays a crucial role in preventing experimental meningoencephalitis by *C. albicans*. Recently, a differential adaptation of *C. albicans* strains *in vitro* and *in vivo* has been demonstrated. This capacity largely depends on the genetic background of the strains, their ability to adapt to host carbon sources and the host environment. In this study, we investigated the immune response of microglia against two different *C. albicans* isolates.

Materials and Methods: The *C. albicans* strains YL1 and YQ2 were used in our *in vitro* infection model, that employed the BV2 microglial cell line, as prototype of host brain effector cells. In particular, the fungal isolates were evaluated for their susceptibility and killing to the microglia, and phagosome maturation in these cells.

Results: Although comparable in their susceptibility to phagocytosis by BV2 cells, the YL1 and YQ2 strains showed striking differences in term of intracellular survival. The YL1 isolate, in contrast to YQ2, resisted indeed to intracellular killing and eventually replicated inside the microglia. Moreover, we found a significantly lower percentage of YL1-containing acidic phagosomes, as compared to those observed in the YQ2-infected BV2 cells.

Discussion and Conclusions: these results indicated that both *C. albicans* isolates were successfully ingested by microglia, but only the YL1 strain was resistant to the killing, suggesting that phagocytic uptake and intracellular killing were two independent phenomena. Although the mechanisms responsible for the observed resistance are not currently known, these data suggest that YL1 may impair bactericidal activities of the microglia by inhibiting phagosome maturation. The increased virulence of YL1 shown in our *in vitro* model appears to correlate with a different genetic makeup of this strain, particularly in genes involved in the pathogenesis of *C. albicans*.