Human immunodeficiency virus (HIV) is responsible for the onset of acquired immunodeficiency syndrome (AIDS). The risk of adults becoming infected through the oral route appears to be low and it is generally assumed that HIV transmission does not occur through casual oral contact, even among patients with high viral load. People with advanced HIV infection are vulnerable to life-threatening malignancies and opportunistic infections such as candidiasis most commonly caused by the organism Candida albicans. While Candida spp are a normal part of the oral microbiome they can react to certain cues and switch into an invasive multicellular filamentous form Oropharyngeal candidiasis affects up to 50% of untreated HIV-1 subjects and 90% of AIDS patients. Interestingly, HIV-1 directly interacts with Candida spp and this interaction modulate the production of HIV-1 by infected macrophages.

Objective: The aim of the study was to investigate the pathways by which Candida spp interfere with HIV-1 pathogenesis and to elucidate cell-signaling pathways of Candida pathogen associated molecular pathways. The pathways by which Candida spp affect HIV-1 infection involve the upregulation and downregulation of genes.

Methods

Human monocytes (THP-1) were cultured in RPMI-1640 medium with 10% fetal bovine serum and 0.05mM of 2-mercaptoethanol at 37ºC in 5% CO2. The cells were exposed to a variety of fetal bovine serum and 0.05mM of 2-mercaptoethanol at 37ºC in 5% CO2. The cells were analyzed using EPI2ME software to determine modulation of genes.

Results

There is modulation of genes in response to candida infection in the toll-like receptor pathway which primarily involve the PI3K and MAPK signaling pathways. Inflammatory cytokines are upregulated and result in proinflammatory effects possibly increasing viral replication.

Future direction: Third generation sequencing done by the Oxford Nanopore M1CK MINION has given a long read of the genetic sequence which we have narrowed for this study. In the future these long reads can be utilized to further explore other pathways.

Conclusions

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References