(ELISA) we found no difference in the PGE2 production of the mutant strains compared to the wild type strain. Although the C. albicans *OLE2* gene proved to be participating in the PGE2 production, these results are intending that the *CpOLE2* gene do not play a role in the *C. parapsilosis* PGE2 biosynthesis. In order to identify genes that play role in the *C. parapsilosis* PGE2 biosynthesis we carried out qRT-PCR on several genes, chosen from literature and by *in silico* work, after treatment of arachidonic acid. The analysis revealed the significant up regulation of the potential multicopper ferro-O2-oxidoreductase (*CpFET3*) gene after the induction of arachidonic acid. Henceforth while we are creating the *CpFET3* homozygous deletion mutant, we are intending to identify further genes that participate in the PGE2 biosynthetic pathway by carrying out a micro-array analysis.

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Role of the HLTF in the tumorigenesis

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The helicase-like transcription factor (HLTF) belongs to the SWI/SNF family of chromatin-remodeling factors. Several SWI/SNF genes are disrupted in cancer, suggesting their possible role as tumor suppressors. Similarly, the HLTF gene was found to be inactivated by hypermethylation in a significant number of colon, gastric and uterine tumors, indicating that HLTF silencing may confer a growth advantage and that HLTF could be considered as a tumor suppressor gene.

Oncogenic activation of signaling pathways downstream of the EGFR, such as mutation of K-RAS and BRAF is central to the progression of colorectal cancer. According to published data the K-RAS mutation may be present in 35%-45% of patients with colorectal cancer.

The HLTF gene was silenced by hypermethylation in 45% of colon cancers. Recent studies did not examine the connection between HLTF and other tumor suppressor genes and oncogenes.

The K-RAS mutation and HLTF promoter hypermethylation can be detected equal frequency in colon cancers, but the correlation between them is not examined yet.

We examined about 100 colorectal cancer samples. We analyzed the K-RAS status, the microsatellite status and HLTF promoter hypermethylation. Our results indicate that there are some correlation between the K-RAS mutation and the HLTF promoter hypermethylation. Additionally there is correlation between HLTF hypermethylation and microsatellite instability.

The DNA Mismatch Repair (MMR) system plays an essential role in maintaining the fidelity of DNA replication by correcting single nucleotide mismatches and insertion/deletion (ID)-loops. Many components of eukaryotic MMR have been identified, the molecular mechanism of MMR has been largely demonstrated. Defects in the mismatch repair system result in a mutator phenotype, manifesting as microsatellite instability (MSI) in DNA of affected cells

Recently it has been published that many DNA repair genes (e.g. RecQ helicases, polymarese κ and HLTF) are possible new players of the MMR. We are developing a new useful research tool to verify the contribution of these potential candidates in the MMR process.

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The Role of *Candida parapsilosis* Secreted Aspartyl Proteinase 1 in Host-Pathogen Interactions

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Yeast of the genus *Candida* remain the most prevalent cause of human mycotic diseases worldwide and range in severity from superficial infections to life-threatening systemic diseases. *Candida parapsilosis* is currently the second most common cause of invasive candidiasis. Adhesion to host surface, cell morphology switching between yeast and filamentous growth, biofilm formation and secretion of hydrolytic enzymes such as lipases, phospholipases and aspartyl proteinases are considered to be the most important features for development of candidial disease. *C. albicans* possesses 10 genes encoding secreted aspartyl proteinases (Saps) where expression of a particular Sap isoenzyme depends on the type, site and stage of infection. In contrast little is known about the exact role of *C. parapsilosis* secreted aspartyl proteinases (Sapps) in the development of virulence in host-pathogen interactions. In *C. parapsilosis SAPP1* and *SAPP2* are the 2 annotated secreted aspartyl proteinase genes. Sapp1 and Sapp2 have been studied at the enzymological level. The production of Sapp1 in inducer medium is