

Technology: Mortalin/HSPA9 and DNaseK antagonists for the treatment of cancer, inflammation, and infections

Morehouse School of Medicine (MSM) Case No.: IP-0015

Patent Applications: US 12/783,829, US 13/267,977, US 13/327,244, CN 201080032113.X, EP 10786562.8, IN 8365/CHENP/2011, JP 2012-514976, PCT US2011/055219, and PCT/US11/065137

Abstract:

GP120 is a glycoprotein exposed on the surface of HIV envelope. Interaction between CXCR4 and gp120-IIIB mediates apoptosis of both immune and endothelial cells. Nef (Negative Regulatory Factor) is a protein expressed by primate lentiviruses and a virulence factor able to manipulate host cell machinery. Regions interact with multiple host factors including CXCR4 and mortalin. SMR is a virus-ended secretion modification region that inhibits an intracellular target called mortalin (aka, HSPA9, HSP70, glucose-regulated protein 75 [GRP75], and peptide-binding protein 74 [PBP74]), which plays an important role in cell proliferation. Importantly, this peptide binds with pM affinity to mortalin and nM affinity to bacteria DNase K. MSM was the first to demonstrate efficacy of HIV-1 derived proteins and peptides as therapeutics for cancer, viral replication, inflammation and bacterial infection.

Applications:

- A dominant and expanding patent position for the composition and use of SMR (& derivatives)
- Treatment of cancer, viral infected, and inflammatory cells that over express mortalin
- Treatment of antibiotic resistant bacteria that express DNase K.

Value Proposition:

- Demonstrated efficacy i) mortalin-SMR binding; ii) reduction in exosome secretion; iii) induction of cancer cell, but not “normal” cell death, iv) reduction in HIV particle secretion; and v) bacterial growth inhibition.
 - Demonstrated effective dose in mouse models of breast and prostate cancer.
 - Eliminated CD4+ T cell depletion during productive as well as Hu/NOD-SCID mouse model of HIV infection with an intact human immune system.
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