OFFICIAL ABSTRACT and CERTIFICATION

Treating Post-HIV Infection Through Molecular Target of HIV TAT and PKC Regulation with Berberine and Curcumin

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Current drugs used for anti-retroviral therapy against HIV have a narrow spectrum of activity and associated adverse events, and possess vulnerability to viral mutation. The HIV TAT protein (TAT), a virus encoded protein required for efficient transcription of the HIV genome, hasn't been intensely researched as a target for developing therapeutics. This study examines Berberine's and Curcumin's effect at very low dosage on diminishing TAT's efficiency in activating Protein Kinase C (PKC), a critical step for HIV transcription.

Berberine is a known PKC inhibitor and Curcumin is previously shown to cause TAT degradation. The extent to which these drugs mitigate the level of TAT functionality is studied with cell migration and viability assays. Matrix Metallopeptidase-9 (MMP-9) levels were measured by Enzyme-linked Immunosorbent Assay (ELISA) as MMP-9 is downstream PKC and plays a role in cell migration and viability. Results showed that cells transfected by TAT had shorter migration bridges (MB), indicating a greater scale of cell migration and reduced viability when compared to the control. Dual-treatment with Berberine and Curcumin significantly restricted TAT induced MB shortening and maintained cell viability, resulting in 45.0% wider MB's (at 48hr) and 73.6% more survivorship in TAT transfected 3T3 cells, significantly exceeding the efficacy of single treatments. ELISA showed 50% greater MMP-9 levels with TAT transfection relative to control, and TAT + dual-treatment reduced that level to 49% of control. Such data shows MMP-9 expression can be explained by PKC activity, resulting in the changes seen with treatments.

This research demonstrates Berberine and Curcumin could work together to mitigate HIV TAT's efficiency in PKC activation.

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