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Investigating the Role of Ca2+ and the Acto-myosin Mechanism on the Human MCF-7 Breast Cancer Cell Line

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**ABSTRACT**

In our study, we chose to investigate enhanced potential to metastasize via increased actomyosin crossbridges in cancer cells. The inhibition of PI-3 kinase and restriction of Akt activation necessary for cell survival and division. PI3-Kinase/Akt pathway is critical for cell survival and division.

**TARGETED HYPOTHESIS OF PHARMACOLOGICAL AGENTS**

Nifedipine is a VGCC inhibitor.

ML-7 is an MLCK inhibitor.

Application of Nifedipine with varying doses will decrease cell viability because calcium is necessary for the formation of the actin-myosin crossbridge and closing the channels deprives the cells of the necessary calcium for cell attachment, viability, and migration.

**FUTURE AIMS**

Analyze the effect of Wortmannin – a PI-3 Kinase inhibitor on MCF-7 cells

Transfection of MCF-7 cells with siRNA for VGCC knockout for further verification

Proceed with PCR and Western blotting