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Magnolol Affects Cellular Proliferation, Polyamine Biosynthesis and Catabolism-Linked Protein Expression and Associated Cellular Signaling Pathways in Human Prostate Cancer Cells *in vitro*

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ABSTRACT:

Background: Prostate cancer is the most commonly diagnosed form of cancer in men in Canada and the United States. Both genetic and environmental factors contribute to the development and progression of many cancers, including prostate cancer.

Context and purpose of this study: This study investigated the effects of magnolol, a compound found in the roots and bark of the magnolia tree *Magnolia officinalis*, on cellular proliferation and proliferation-linked activities of PC3 human prostate cancer cells *in vitro*.

Results: PC3 cells exposed to magnolol at a concentration of 80 μM for 6 hours exhibited decreased protein expression of ornithine decarboxylase, a key regulator in polyamine biosynthesis, as well as affecting the expression of other proteins involved in polyamine biosynthesis and catabolism. Furthermore, protein expression of the R2 subunit of ribonucleotide reductase, a key regulatory protein associated with DNA synthesis, was significantly decreased. Finally, the MAPK (mitogen-activated protein kinase), PI3K (phosphatidylinositol 3-kinase), NFκB (nuclear factor of kappa-light-chain-enhancer of activated B cells) and AP-1 (activator protein 1) cellular signaling pathways were assayed to determine which, if any, of these pathways magnolol exposure would alter. Protein expressions of p-JNK-1 and c-jun were significantly increased while p-p38, JNK-1/2, PI3Kp85, p-PI3Kp85, p-Akt, NFκBp65, p-IκBα and IκBα protein expressions were significantly decreased.

Conclusions: These alterations further support the anti-proliferative effects of magnolol on PC3 human prostate cancer cells *in vitro* and suggest that magnolol may have potential as a novel anti-prostate cancer agent.

Key Words: prostate cancer cells, magnolol, polyamines, MAPK, PI3K, NFκB