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ADME	Absorption Distribution Metabolism Excretion
AMACR	Alpha-methylacyl-CoA racemase.
AR	Androgen Receptor
ATM	Ataxia Telangectasia Mutated
BPH	Benign Prostatic Hypertrophy
CDC2	Cell Division Cell Cycle 2
CDKS	Cyclin Dependent Kinase
CHK	Checkpoint Kinase
DAPI	4', 6-Diamidino-2-Phenylindole Dihydrochloride
DCFH-DA	2, 7-Dichlorodihydrofluorescein Diacetate
DHT	Dihydrotestosterone
DMSO	Dimethyl Sulfoxide
DTT	Dithiothreitol
EGCG	Epigallocatechin Gallate
ER	Estrogen Receptor
FAS	Fatty Acid Synthase
FBS	Fetal Bovine Serum
FITC	Fluorescein Isothiocyanate
FTIR	Fourier Transforms Infrared Spectroscopy
MTT	3-(4, 5-Dimethylthiazol-2-yl)-2, 5-Diphenyltetrazolium Bromide
PBS	Phosphate Buffered Saline
PC3	Prostate Cancer Line
PCa	Prostate Cancer
PDB	Protein Data Bank

PI	Potassium Iodide
PIN	Prostatic Intraepithelial Neoplasia
PSA	Prostate Specific Antigen
RB	Retinoblastoma Protein
ROS	Reactive Oxygen Specific
UV	Ultraviolet
WCRF	World Cancer Research Fund
XP	Extra Precision
^{13}C NMR	Carbon Nuclear Magnetic Resonance
^{1}H NMR	Proton Nuclear Magnetic Resonance

ABSTRACT

Prostate cancer is the second most common malignancy in the human reproductive system. Eupalitin is one of the O-methylated flavonol-exhibited enhanced cancer chemopreventive agents. The current study highlights anti cancer activity against prostate cancer cell line (PC3) andits underlying mechanism. Eupalitin and cirsilineol structure was determined by using FTIR, 1H NMR, and 13C NMR. PC3 cells were treated with increasing concentrations of eupalitin, followed by analysis of the cell viability with an MTT assay. The results demonstrated that eupalitin markedly inhibited the proliferation of PC3 cells in a concentration-dependent manner. The results from fluorescent microscopic analysis of nuclear condensation and intracellular ROS generation determined that progression as a percentage of cells in G0/G1 phase decreased whereas S phase increased. Caspase-3 immunofluorescence analysis confirms the efficacy of eupalitin-inducing apoptotic pathway and cell death. Flow cytometry analysis of Annexin V and PI stain revealed that eupalitin greatly enhanced late apoptotic cells in cells population. Molecular study using protein blot study revealed that eupalitin greatly up regulated the proapoptotic marker bax expression while down regulated the expression of antiapoptotic bcl2 protein. Furthermore, *in silco* study revealed strong binding affinity of ligand eupalitin with androgen receptors of prostate cancer. The cytotoxic response of the cancer cells to eupalitin was dose dependent. Our data indicate that eupalitin inhibits growth and induces apoptosis in human prostate carcinoma cells that may provide additional molecular targets for cancer therapy.

Further spectral analysis of cirsilineol revealed the structural confirmation of the candidate. Anti proliferative study of cirsilineol revealed that ROS induction and nuclear apoptosis were enhanced slightly less than eupalitin.

Thus, our study is helpful in understanding the mechanism underlying these effects in prostate cancer and it may provide novel molecular targets for prostate cancer therapy.