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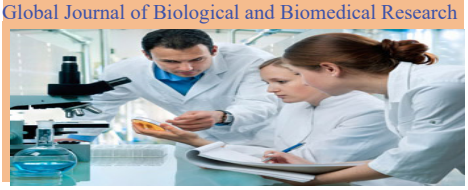

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Downregulation of *FERMT2* and the focal adhesion pathway

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ABOUT THE STUDY

One of the most prevalent malignancies among males in the United States is Prostate Cancer (PCa), which accounts for 21% of newly diagnosed cancer cases and the second-leading cause of cancer death (an estimated 333300 deaths in 2020). Prostatectomy or radiation therapy will usually cure PCa in its early stages, but a tiny percentage of patients will progress to castration-resistant prostate cancer, which can metastasize fatally.

A basic understanding of the molecular pathways underlying the onset and development of PCa has been disclosed by a few researches. It was once believed that the recurrent *TMPRSS2-ETS* family fusion was mostly unique to prostate cancer. The 5'-untranslated region of *TMPRSS2* was reported to be fused with ETS family genes, such as *ERG*, *ETV1* and *ETV4*, to increase prostate cancer growth and invasion. Three other androgen responsive genes, *SLC45A3*, *NDRG1*, and *HEPPUD1*, were found to fuse with *ERG* in addition to *TMPRSS2*. In contrast to one study's finding that only 5%–10% of samples were fused with ETS, the incidence of ETS fusion in prostate cancer was as high as 70%.

Although Prostate-Specific Antigen (PSA) is a prostate tissue specific biomarker rather than a PCa specific one, it has been discovered to be increased in prostate cancer and is used as a good standard of diagnosis or screening the early-stage prostate cancer. *PDGFR-β*, *HOX6C*, *ITPR3*, *Chromogranin A*, and *Sialyltransferase I* are five genes that have been linked to a positive relationship with prostate cancer recurrence. Histone3 and Histone4 acetylation and demethylation in primary PCa may be used to predict recurrence independently of PSA level. Prostate cancer patients' prognoses were discovered to be substantially linked with *EZH2*, a component of polycomb repression group complexes.

A technique for analysing the gene co-expression network is called Weight Gene Co-expression Network Analysis (WGCNA). In order to identify the hub genes, it primarily groups together genes with similar expression into one module and connects the module with clinical features. Here, we primarily employed WGCNA and Gene Score Enrichment Analysis (GSEA) to uncover the molecular basis of the PCa pathogenic process, and we identified the PCa-related hub gene *FERMT2*. Cell biology tests revealed that *FERMT2* prevented PC3's prostate cancer cell lines from proliferating and migrating. Potential biomarkers for PCa diagnosis and insight into the development of therapy strategies include *FERMT2*.

The main cause of morbidity and mortality in the US is Prostate Cancer (PCa), although it is still unclear how it develops on a molecular level. Here, by analysing PCa transcriptome data from the Gene Expression Omnibus (GEO) and The Cancer Genome Atlas (TCGA) datasets using bioinformatics methods, we found that the focal adhesion route was the primary problem pathway and that *FERMT2* was the critical gene in prostate carcinogenesis.

Cell Adhesion Mediated Drug Resistance (CAMDR) and cell adhesion mediated radioresistance are two examples of how the focal adhesion pathway plays a significant role in carcinogenesis. The cytoplasmic signalling necessary for cell survival, proliferation, and motility is maintained by focal adhesion pathway proteins, which also participated in the ECM connection and contributed in cell morphology maintenance. ErbB2 and STAT3 were downregulated when 4 integrin, an adhesion molecule for ECM proteins, was reduced. This prevented the onset of breast cancer. While overexpression of α2β1 induced differentiation and decreased proliferation in breast cancer cells.