

Supplementary Table S3: Functional characterization and association with prostate and other types of malignancy of the selected biomarkers.

Protein name	Gene Name	Molecular/biological function(s)	Classification and involvement in disease	Association with PCa (proteomics studies)	Associations with PCa (genomics and functional studies)	Association with cancer
Alpha-enolase	ENO1	Alpha-enolase is one of three enolase isoenzymes found in mammals. It functions as a glycolytic enzyme and as a structural lens protein (tau-crystallin) in the monomeric form. Alternative splicing of this gene results in a shorter isoform that has been shown to bind to the c-myc promoter and function as a tumor suppressor.	ENO1 overexpression and post-translational modifications could be of diagnostic and prognostic value in many cancer types [1].	ENO1 showed down-regulation in exosomes derived from PCa cells compared to normal cells [2]; Stromal ENO1 levels are increased in PCa compared with those in normal tissue [3]; Down-regulated in urine from PCa patients [4]; Down-regulated in urine from PCa patients compared to BPH [5]; Decreased methylation and increased expression of ENO1 have been detected in prostate cancer tissues [6]; ENO1 is up-regulated in prostate cancer tissue [7]; Anti-ENO1 antibodies are increased in serum from prostate cancer patients [8]; ENO1 is up-regulated in extracellular vesicles (serum) from PCa patients after acquiring cabazitaxel resistance [9];	Over-expression of ENO1 modulates the MAPK pathway by inhibiting MEK5 and inhibiting prostate cancer growth [10]; ENO1 knock-down results with reduced cell cycle progression in prostate cancer cells [11];	ENO1 mRNA and protein levels were upregulated in glioma tissues [12]; Upregulated in lung carcinoma [13-15]; Slightly elevated in serum and urine of renal cell carcinoma patients [16]; Down-regulation of ENO1 in nasopharyngeal carcinoma [17]; Upregulated in serum in cholangiocarcinoma [18]; ENO1 possesses tumor-suppressing effects in neurocytoma [19];
Fatty acid binding protein 5	FABP5	The fatty-acid-binding proteins (FABPs) are a family of carrier proteins for fatty acids and other lipophilic substances such as eicosanoids and retinoids. These proteins are thought to facilitate the transfer of fatty acids between extra- and intracellular membranes. FABPs roles include fatty acid uptake, transport and metabolism.	Involved in prostate cancer cell growth [20]; Novel molecular target in prostate cancer [21];	Up-regulated in tissue from prostate cancer patients [22]; FABP5 has been found to be up-regulated in tissue from prostate cancer patients with different racial disparities [23]; FABP5 is upregulated in prostate cancer tissue compared to BPH [24]; FABP5 is upregulated in PCa tissues compared to BPH and normal tissues [25]; FABP5 is up-regulated in urine extracellular vesicles from patients with high Gleason score PCa [26]; FABP5 has been found to be up-regulated in prostate cancer tissue, and could differentiate between lymph node metastatic PCa and localized from [27];	Up-regulated gene expression in prostate cancer tissue [28]; FABP5 gene expression was not linked to prostate cancer's clinical outcome but is associated with PPAR signaling, suggesting a drugable nature [29]; Co-expression of FABP5 with FASN and MAGL is associated with lipid-mediated metastasis [30]; CNFI26 is a potent FABP5 inhibitor [31]; Inactivated FABP5 suppresses malignant progression of prostate cancer cells [32];	Up-regulated in bladder squamous cell carcinomas tissue [33]; Up-regulated in bladder cancer urine [34]; Down-regulated FABP5 is associated with metastasis of squamous cell carcinoma of the oral tongue [35]; Family members of FABP5: Up-regulated in endometrial carcinoma [36]; Ubiquitous overexpression in endometrial cancers [37]; Up-regulated in breast cancer [38]; Up-regulated in PCa [25]; FABP family involved in cancer [39];

Alpha-2-glycoprotein 1, zinc-binding	AZGP1	It is a secretory protein, with a structure similar to the MHC I molecules with strong affinity for zinc and fatty-acid binding. It exerts a role of lipid-mobilizing adipokine involved in fat loss by lipid degradation in adipocytes [40].	Heavily involved in cachexia, a wasting syndrome associated with cancer [41]; AZGP1 has been found to inhibit cancer invasion by inhibiting TGF- β mediated epithelial-mesenchymal transition, facilitating its role as a tumor suppressor [42];	AZGP1 is down-regulated in PCa tissue compared to BPH [43]; AZGP1 has been found to be up-regulated in tissue from PCa patients [44]; AZGP1 is up-regulated in seminal plasma of PCa patients [45]; AZGP1 is up-regulated in serum but did not show a similar pattern in tissues of PCa patients [46]; AZGP1 is up-regulated in urine of PCa patients, showing predictive power as a solo and as a panel biomarker together with PSA[47]; AZGP1 shows a down-regulated trend in epithelial prostate cancer cells, and negative correlation with Gleason grading [48]; AZGP1 is down-regulated in tissue from PCa patients [24]; AZGP1 is down-regulated in aggressive compared to non-aggressive PCa tissues [49, 50];	Low AZGP1 expression was associated with metastasis or death from prostate cancer [51]; Up-regulated AZGP1 gene expression is associated with decreased risk of PCa recurrence [52]; Low expression of AZGP1 is an independent predictor of recurrence in margin-positive localized PCa [53]; Through meta-analysis, low expression of AZGP1 is associated with a higher risk of aggressive time-dependent outcomes in PCa patients undergoing radical prostatectomy [54];	Up-regulated in urine of bladder cancer patients [55]; Up-regulated in serum of colorectal cancer patients [56]; Up-regulated in urine of endometrial cancer patients [57]; Up-regulated in multiple myeloma [58]; Up-regulated in cancer cachexia in human urine [59]; Up-regulated in saliva in head and neck squamous cell carcinoma patients [60]; Tumor suppressor in pancreatic cancer [42];
Glutathione S-transferase mu 2 (muscle)	GSTM2	Glutathione S-transferases comprise a family of enzymes that are critical for the inactivation of toxins and carcinogens. Glutathione S-transferase mu2 is a phase II detoxification enzyme. Proposed role in cancer as tumor suppressor.	GSTM2 is associated with metabolism of xenobiotics, such as carcinogens, catalyzing the conjugation of these compounds with glutathione [61]; Glutathione S-transferases have been associated with resistance to anticancer drugs [62] and have been shown to have a regulatory role in the proliferation and differentiation of tumor cells [63];	GSTM2 has been shown to be down-regulated in tissue from prostate cancer patients [24]; GSTM2 has been shown to be down-regulated in tissue from prostate cancer patients [64];	Loss of expression of all GST subclasses results in the progression of prostatic neoplasia to carcinoma [65]; Epigenetic silencing of GSTM2 is a molecular marker for PCa [66]; Simultaneous genetic silencing of GSTM2 and MYCL2 is proposed as a molecular marker panel to predict biochemical recurrence of PCa after radical prostatectomy of high-risk localized PCa [67]; Multigene panel, consisting of GAS6, GSTP1 and HAPLN3, has been created and validated for the classification of benign and malignant PCa [68];	GSTM2 differential expression in breast cancer cells [69]; Low expression of GSTM2 in lung cancers is due to hypermethylation of its promoter [70]; Association with kidney and bladder tumors [71];

Glutathione S-transferase Pi 1	GSTP1	GSTP1 is thought to function in xenobiotic metabolism and play a role in susceptibility to cancer and other diseases.	Glutathione S-transferases play tumor suppressive, cyto-protective roles and interact and modulate several pathways involving cell growth, differentiation and cell death and as such are interconnected in the process of tumorigenesis [72];	GSTP1 is down-regulated in PCa tissue compared to BPH (Garbis, 2008) GSTP1 has been found as down-regulated in tissue biopsy from PCa patients [22]; GSTP1 is down-regulated in PCa tissue compared to benign prostate hyperplasia [73]; GSTP1 has been shown to have a down-regulated expression in prostate cancer tissue [74]; GSTP1 is down-regulated in prostate cancer tissue [64]; GSTP1 is down-regulated in prostate cancer tissue [6];	The promotor methylation of GSTP1 have high implications in early PCa initiation and development [75]; Hyper methylation of the GSTP1 promotor leads to silencing in PCa [76]; GSTP1 is part of a gene panel used in urine for non-invasive detection and risk stratification for PCa [77]; GSTP1 hyper methylation can be used for selecting PCa patients for active surveillance [78]; ProCURE, a non-invasive urinary methylation assay, consisting of LASSO, HOXD3 and GSTP1 is can assist in the early detection and prognosis of PCa [79]; GSTP1 methylation in combination with 4 other genes can help with risk assessment of PCa [80]; GSTP1 methylation can predict missed PCa [81]; GSTP1 has been shown to be hyper methylated selectively in PCa tissue [82];	Hyper methylation of GSTP1 has been shown in: lung cancer [83]; breast cancer [84]; hepatocellular carcinoma [85]; bladder cancer [86];
Malate dehydrogenase 2	MDH2	The protein encoded by this gene is localized to the mitochondria and may play pivotal roles in the malate-aspartate shuttle that operates in the metabolic coordination between cytosol and mitochondria.	As a mitochondrial enzyme, MDH2 has an association with mitochondrial diseases such as early-onset severe encephalopathy [87];	MDH2 is up-regulated in PCa tissue [7]; MDH2 is down-regulated in highly metastatic variant LNCaP-LN3 cell line [88]; MDH2 is up-regulated in PCa tissue, that continues to increase with the cancer progression [6]; MDH2 has been detected as up-regulated in prostate cancer tissue both with a proteomic approach and a gene expression microarray [74]; MDH2 is down-regulated in PCa patients which experience biochemical relapse compared to patients who do not [89];	MDH2 overexpression decreases the relapse-free period of prostate cancer patients. MDH2 knockdown in prostate cancer cell lines increases sensitivity to docetaxel [90]; Treatment with Alternol results in reduced cell respiration and ATP production in PCa cells [91];	MDH2 up-regulation is associated with poor responses to radiation treatment in colorectal cancer [92];
Ezrin	EZR	As a member of the Ezrin/Radixin/Moesin protein family, this protein serves as an intermediate between the plasma membrane and the actin cytoskeleton.	Ezrin (EZR) is involved in cell network regulation by linking the actin cytoskeleton to the cell membranes and also control signaling transduction by interacting with adhesion molecules and various growth factor receptors [93];	EZR is up-regulated in PCa tissue compared to BPH [43]; EZR overexpression has been correlated with PCa and adverse prognosis [95]; EZR has been found as up-regulated in tissues from lymph node metastatic PCa compared to localized PCa [27];	EZR is involved in androgen-induced cell invasion [98]; EZR in MCF7 and PC3 were shown to form a complex with podocalyxin, which implicates its role in aggressive forms of prostate and breast cancer [99]; c-Myc regulates EZR expression in the presence of androgens, inducing cell invasion [100];	EZR has been associated with several types of cancer such as: lung cancer [103], breast cancer [104], ovarian cancer [105], cervical cancer [106], gastric cancer [107].

			In cancer cells, the relative membrane localization of EZR is increased which facilitates the process of cancer progression and invasion [94];	EZR is up-regulated in urine-derived extracellular vesicles from PCa patients [96]; EZR has been detected as overexpressed in high-grade prostatic intraepithelial neoplasia compared to PCa in a less aggressive stage [97];	EZR is linked with modification of cellular adhesion and cellular motility [101]; EZR hadn't shown differential gene expression in prostate cancer compared to benign tissue [102];	
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