GenomeDx Biosciences Laboratory
10355 Science Center Drive, Suite 240
San Diego, CA 92121
Tel 1 888 792 1601 | Fax 1 855 324 2768
GRIDsupport@genomedx.com | www.genomedx.com

TUMOR RNA EXPRESSION PROFILE

NAME:

RESEARCH USE ONLY

The information provided in this analysis has not been clinically validated and should not be used for clinical decision-making.

CLINICAL INFORMATION

Specimen Type: Resection
Patient Age:
PSA at Resection:
Gleason Score: 3 + 3
Pathologic features: not available
Biochemical Recurrence: No

RUO GRID INFORMATION

GRID ID:
GRID profile Date:
Ordering Physician:
Clinic/Hospital:

Clinic/Hospital Address:

GENOMIC PROFILE SUMMARY*

Molecular subtype signatures (P.2)

- ○ Neuroendocrine/small cell
- ● **Adenocarcinoma**
- ● **Luminal**
- ○ Basal
- ● **ERG**
- ○ ETS
- ○ SPINK1
- ○ TripleNeg

PREDICTIVE (P.3)

PERCENTILE RANK (%)** (0 – 100)

ADT response	99	HIGHER ADT RESPONSE
Radiation response	14	LOWER RT RESPONSE
Docetaxel sensitivity	92	HIGHER SENSITIVITY
Dasatinib sensitivity	7	AVERAGE SENSITIVITY

PROGNOSTIC (P.4)

Risk of metastasis (average of 18 signatures)*	64	AVERAGE METASTASIS RISK

TUMOR GRADE/STAGE (P.5)

Genomic Gleason grade	59	HIGHER GRADE

MOLECULAR PATHWAYS (P.5)

Tumor cell proliferation (average of 3 signatures)*	87	HIGH PROLIFERATION
AR signaling activity (average of 2 signatures)*	91	HIGH AR ACTIVITY

SELECT RNA MARKERS - TOP OUTLIERS (P.6)

		PERCENTILE RANK
RNA marker most over-expressed:	PDL3/B7H3	100%
RNA marker most under-expressed:	PDL1	1%

*RNA signatures and genes listed above are intended as a summary of the tumor profile, for which more detail is provided in the following pages. "Average of x signatures" is the average of the percentile ranks of the individual signatures.

**Percentile Rank indicates the percentage of tumor RNA profiles in the GRID (n=2,829) with lower scores than for this profile.

GenomeDx Biosciences Laboratory
10355 Science Center Drive, Suite 240
San Diego, CA 92121
Tel 1 888 792 1601 | Fax 1 855 324 2768
GRIDsupport@genomedx.com | www.genomedx.com

TUMOR RNA EXPRESSION PROFILE NAME:

RESEARCH USE ONLY
The information provided in this analysis has not been clinically validated and should not be used for clinical decision-making.

SECTION 1

GRID MOLECULAR SUBTYPE SIGNATURES

The clinically heterogeneous nature of prostate cancer can be partly explained by underlying molecular heterogeneity. Prostate cancer tumors can be subtyped based on their histological appearance, cell of origin and genomic alterations. These subtypes may be important to application of hormonal and systemic therapy.

NEUROENDOCRINE/SMALL CELL SUBTYPES
Several genomic models have been developed to discriminate histologic and phenotypic variants of prostate cancer. Neuroendocrine and small cell variants tend to have poor or transient response to androgen deprivation therapy.

LUMINAL/BASAL SUBTYPE
A genomic model developed from literature-curated signatures of basal and luminal cell of origin. Prostate cancer tumors with high expression of basal genes are associated with tumor invasion, stem cell-ness, neurogenesis and inactivity of the androgen receptor signaling axis.

GENOMIC ALTERATIONS
Gene expression models developed to detect genomic alteration of the ERG and PTEN genes. ERG gene overexpression is the most common genomic alteration in prostate cancer and is highly prevalent in men of European descent but less in men of African descent. The clinical implications of ERG overexpression are unclear. PTEN deletion is common in advanced stage prostate cancer but less prevalent in localized disease. Since, PTEN is a tumor suppressor gene, low expression or it's deletion is associated with aggressive disease.

Molecular subtype signatures

- ○ Neuroendocrine/small cell
- ● **Adenocarcinoma**
- ● **Luminal**
- ○ Basal
- ● **ERG**
- ○ ETS
- ○ SPINK1
- ○ TripleNeg

SIGNATURE	TUMOR SCORE	PERCENTILE RANK (%)	TUMOR GENOMIC CLASS	PREDICTION ENDPOINT
NEUROENDOCRINE/ SMALL CELL SUBTYPE SIGNATURES				
Neuroendocrine (Kumar2016)	-0.11	9%	ADENOCARCINOMA	Adenocarcinoma vs Neuroendocrine
Small cell (Alshalalfa2016)	0.37	56%	ADENOCARCINOMA	Adenocarcinoma vs small cell carcinoma
LUMINAL/ BASAL SUBTYPE SIGNATURES				
Basal (Zhang2016)	-0.39	32%	LUMINAL-LIKE	Luminal vs Basal
GENOMIC ALTERATIONS				
ERG (Tomlins2015)	1.00	89%	ERG POSITIVE	ERG- vs ERG+
PTEN (Saal2007)	0.82	90%	PTEN-LOSS	Loss of PTEN expression

GenomeDx Biosciences Laboratory
10355 Science Center Drive, Suite 240
San Diego, CA 92121
Tel 1 888 792 1601 | Fax 1 855 324 2768
GRIDsupport@genomedx.com | www.genomedx.com

TUMOR RNA EXPRESSION PROFILE NAME:

RESEARCH USE ONLY
The information provided in this analysis has not been clinically validated and should not be used for clinical decision-making.

SECTION 2

RADIATION RESPONSE SIGNATURE

ADT RESPONSE SIGNATURE (ARS)

A gene expression signature derived from a panel of neuroendocrine (NE) genes has been developed to predict treatment failure from adjuvant androgen deprivation therapy (ADT) after radical prostatectomy. Patients with high ARS scores have been shown to have improved response to ADT (lower metastasis rate) whereas patients with low ARS scores have been shown to more rapidly fail adjuvant ADT (higher metastasis rate). This signature requires prospective validation.

RADIATION THERAPY RESPONSE (RTR)

A gene expression signature derived from a panel of DNA repair genes has been developed to predict treatment failure from radiation therapy (RT) after radical prostatectomy. Patients with high RTR scores have been shown to have improved response to RT (lower metastasis rate) whereas patients with low RTR scores have been shown to have lower response to RT (higher metastasis rate). This signature requires prospective validation.

DRUG RESPONSE SCORES (DRS)

Gene expression signatures were derived from in vitro screening of drugs in 60 pan-cancer tumor cell lines. A drug response score (DRS) is developed based on the expression profile for cell lines sensitive to a particular drug. Patients with high DRS (e.g. percentile rank > 90%) are predicted to be sensitive, whereas patients with low DRS (e.g. percentile rank < 10%) are predicted to be less sensitive to the drug. DRS is for research use only and has not been validated in human clinical trials.

SIGNATURE	TUMOR SCORE	PERCENTILE RANK (%)	PREDICTED RESPONSE	ENDPOINT DESCRIPTION
ADT RESPONSE SIGNATURE				
ADT Response (Karnes2016)	0.96	99%	HIGHER ADT RESPONSE	Response to adjuvant androgen deprivation therapy
RADIATION RESPONSE SIGNATURE				
RT Response (Zhao2016)	-1.23	14%	LOWER RT RESPONSE	Response to postoperative radiation
DRUG RESPONSE SIGNATURES				
Docetaxel (Lehrer2016)	0.31	92%	HIGHER SENSITIVITY	Sensitivity to docetaxel*
Dasatanib (Lehrer2016)	-0.26	7%	AVERAGE SENSITIVITY	Sensitivity to dasatinib*

*Based on similarity to expression profiles of sensitive and insensitive *in vitro* cancer cell lines.

GenomeDx Biosciences Laboratory
10355 Science Center Drive, Suite 240
San Diego, CA 92121
Tel 1 888 792 1601 | Fax 1 855 324 2768
GRIDsupport@genomedx.com | www.genomedx.com

TUMOR RNA EXPRESSION PROFILE NAME:

RESEARCH USE ONLY
The information provided in this analysis has not been clinically validated and should not be used for clinical decision-making.

SECTION 3

GRID PROGNOSTIC SIGNATURES

Several gene expression signatures have been developed to predict adverse pathology, biochemical recurrence, metastasis and prostate cancer-specific mortality. We have retrained these signatures to predict prostate cancer metastasis in 1,574 patients from a multi-institutional cohort (Karnes 2013, Den 2014, Klein 2014, Ross, 2015). The signatures are ranked ordered by their area-under-the curve values for predicting metastasis under cross-validation. Patients with higher scores for the majority of these signatures are at greater risk of developing metastatic disease. Patients with lower scores for the majority of these signatures have a lower risk of metastasis.

Prognostic

Risk of metastasis* — 0 — PERCENTILE RANK (%) — 100 — 64 — AVERAGE METASTASIS RISK

SIGNATURE	INSTITUTION NAME	TUMOR SCORE	PERCENTILE RANK (%)	METASTATIC RISK
Wu 2013	Massachusetts General Hospital	0.53	73%	AVERAGE
Bismar 2006	Dana Farber Cancer Institute	0.69	92%	HIGHER
Penney 2011	Dana Farber Cancer Institute	0.24	45%	LOWER
Agell 2012	Hospital del Mar-Mar Health Park	0.20	16%	LOWER
Ramaswamy 2003	Dana Farber Cancer Institute	0.37	60%	LOWER
Varambally 2005	University of Michigan	0.60	86%	AVERAGE
Bibikova 2007	UC San Diego	0.35	59%	LOWER
Talantov 2010	Garvin Institute	0.34	72%	LOWER
Nakagawa 2008	Mayo Clinic	0.23	23%	LOWER
Stephenson 2005	Memorial Sloan Kettering Cancer Center	0.32	83%	LOWER
Lapointe 2004	Johns Hopkins	0.24	48%	LOWER
Yu 2007	University of Michigan	0.41	76%	AVERAGE
Long 2011	Emory University	0.53	91%	AVERAGE
Long 2014	Emory University	0.22	87%	HIGHER
Singh 2002	Dana Farber Cancer Institute	0.51	62%	AVERAGE
Klein 2014	Cleveland Clinic	0.39	77%	LOWER
Cuzick 2011	King's College	0.60	85%	HIGHER
Larkin 2012	Queen Alexandria Hospital	0.44	92%	AVERAGE

*Average of signatures

GenomeDx Biosciences Laboratory
10355 Science Center Drive, Suite 240
San Diego, CA 92121
Tel 1 888 792 1601 | Fax 1 855 324 2768
GRIDsupport@genomedx.com | www.genomedx.com

TUMOR RNA EXPRESSION PROFILE NAME:

RESEARCH USE ONLY
The information provided in this analysis has not been clinically validated and should not be used for clinical decision-making.

SECTION 4

TUMOR GRADING/STAGING GENOMIC MODELS

Several genomic signatures have been developed to predict adverse pathology such as high Gleason grade and tumor stage. At needle biopsy, these signatures may be useful in addition to prognostic scores for improved staging of the tumor. Patients with high scores may harbor aggressive prostate cancer and a second line of treatment may be recommended, while patients with low scores across all signatures may be suitable candidates for active surveillance.

Tumor Grade/Stage

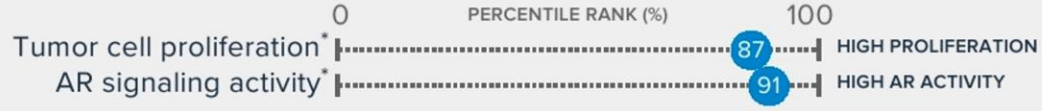
SIGNATURE	TUMOR SCORE	PERCENTILE RANK (%)	TUMOR CLASS	PREDICTION ENDPOINT
Genomic Gleason (Abdollah)	0.80	59%	HIGHER GRADE	Primary pattern 4 or 5 at RP
Genomic Gleason (Penney2011)	0.99	76%	HIGHER GRADE	Primary pattern 4 or 5 at RP
Genomic CAPRA-S (Abdollah2016)	0.93	83%	HIGHER GRADE/STAGE	High CAPRA-S (>5) score
pT3 Disease (Abdollah2016)	0.87	89%	NON-ORGAN CONFINED	Pathologic stage T3

SECTION 5

MOLECULAR PATHWAYS

A hallmark of cancer cells is their loss of cell cycle control, which enables uncontrolled proliferation and growth. Highly correlated cell cycle progression genes have been used to provide a robust measurement of cell proliferation (Cuzick 2011). Tumors with high expression of proliferation genes are associated with biochemical recurrence and a worse prognosis after radical prostatectomy but may also be more sensitive to anti-mitotic chemotherapy. Androgen Receptor (AR) signaling is a key regulator of prostate tumor development where tumors with very low or very high AR signaling have poor prognosis and may be insensitive to hormonal suppression (Kumar 2016).

Molecular pathways



SIGNATURE	TUMOR SCORE	PERCENTILE RANK (%)	PREDICTED CLASS	PREDICTION ENDPOINT
Proliferation (Cheville2007)	0.52	81%	AVERAGE PROLIFERATION	Tumor cell proliferation
Proliferation (Cuzick2011)	0.06	87%	AVERAGE PROLIFERATION	Tumor cell proliferation
Proliferation (Glinsky2005)	0.64	93%	HIGHER PROLIFERATION	Tumor cell proliferation
AR Activity (Faisal2015)	15.92	91%	AVERAGE AR ACTIVITY	Expression of AR-dependent genes
AR Activity (Kumar2016)	1.31	91%	AVERAGE AR ACTIVITY	Expression of AR-dependent genes

*Average of signatures

GenomeDx Biosciences Laboratory
10355 Science Center Drive, Suite 240
San Diego, CA 92121
Tel 1 888 792 1601 | Fax 1 855 324 2768
GRIDsupport@genomedx.com | www.genomedx.com

TUMOR RNA EXPRESSION PROFILE NAME:

RESEARCH USE ONLY

The information provided in this analysis has not been clinically validated and should not be used for clinical decision-making.

SECTION 6

SELECT RNA MARKERS

Decipher GRID contains RNA expression values covering approximately 46,000 coding and non-coding genes. The list below represents genes currently evaluated for their prognostic & predictive power in prostate cancer. GRID will be updated as new markers are studied and evaluated. High and low expression of PCA3 are defined by segmenting a bimodal distribution. For all other markers, high and low outliers are defined by 2.2 median absolute deviations greater or lower than the median of the reference GRID population (n=2,829). A full list of the 36 genes in this profile, relevant research findings and references are updated regularly on www.DecipherGRID.com.

		PERCENTILE RANK (%)	TUMOR SCORE	OUTLIER STATUS*
ANDROGEN SIGNALING	AR	78%	0.90	-
	KLK2	70%	4.34	-
	KLK3	62%	4.41	-
	PCA3	26%	1.08	LOW
	NKX3-1	97%	1.60	-
	SRD5A1	54%	0.31	-
NEUROENDOCRINE/SMALL-CELL	pRB1	67%	0.74	-
	CCND1	97%	1.39	-
	CHGA	30%	-0.25	-
	AURKA	8%	-0.26	-
	NEAT1	93%	4.44	-
	MYCN	20%	-0.19	-
PROLIFERATION/GROWTH	Ki67	90%	0.12	-
	TOP2A	83%	0.15	-
	EGFR	49%	0.43	-
	HER2/NEU	89%	0.75	-
	ERBB3	85%	1.04	-
	c-MET	11%	-0.01	-
DNA REPAIR	ATM	43%	0.47	-
	ATR	83%	0.57	-
	RAD21	25%	0.90	-
	DNAPK	52%	0.62	-
	NBN	30%	0.61	-
	PARP1	83%	0.41	-
INVASION/ANGIOGENESIS	SChLAP1	90%	1.86	HIGH
	EZH2	70%	0.02	-
	SPARCL1	27%	1.86	-
	GSTP1	36%	0.30	-
	VEGFR2	16%	-0.01	-
	HIF1A	55%	1.24	-
IMMUNO-ONCOLOGY	PD1	39%	0.49	-
	PDL1	1%	-0.38	LOW
	PDL2	5%	-0.13	-
	PDL3/B7H3	100%	1.43	HIGH
	CTLA4	61%	0.04	-
	IDO1	19%	-0.13	-

*Outlier status is based on the expression level of each individual gene relative to the total patient population (2,829). Therefore, this is not an absolute value, but rather a comparative level of expression based on the normal distribution observed for that gene marker.

RESEARCH USE ONLY

The information provided in this analysis has not been clinically validated and should not be used for clinical decision-making.

REFERENCES

Agell L, Hernández S, Nonell L, Lorenzo M, Puigdecanet E, de Muga S, Juanpere N, Bermudo R, Fernández PL, Lorente JA, Serrano S, Lloreta J. A 12-gene expression signature is associated with aggressive histological in prostate cancer: SEC14L1 and TCEB1 genes are potential markers of progression. Am J Pathol. 2012 Nov;181(5):1585-94. doi: 10.1016/j.ajpath.2012.08.005. Erratum in: Am J Pathol. 2013 Feb;182(2):610.

Alshalalfa M, Tsai H, Haddad Z, Ross A, Karnes RJ, Davicioni E, Schaeffer EM, Lotan TL, Deciphering the genomic fingerprint of small cell prostate cancer with potential clinical utility, J Clin Oncol 34, 2016 (suppl 2S; abstr 303)

Bibikova M, Chudin E, Arsanjani A, Zhou L, Garcia EW, Modder J, Kostelec M, Barker D, Downs T, Fan JB, Wang-Rodriguez J. Expression signatures that correlated with Gleason score and relapse in prostate cancer. Genomics. 2007 Jun;89(6):666-72. Epub 2007 Apr 24.

Bismar TA, Demichelis F, Riva A, Kim R, Varambally S, He L, Kutok J, Aster JC, Tang J, Kuefer R, Hofer MD, Febbo PG, Chinnaiyan AM, Rubin MA. Defining aggressive prostate cancer using a 12-gene model. Neoplasia. 2006 Jan;8(1):59-68.

Cheville JC, Karnes RJ, Therneau TM, Kosari F, Munz JM, Tillmans L, Basal E, Rangel LJ, Bergstralh E, Kovtun IV, Savci-Heijink CD, Klee EW, Vasmatzis G. Gene panel model predictive of outcome in men at high-risk of systemic progression and death from prostate cancer after radical retropubic prostatectomy. J Clin Oncol. 2008 Aug 20;26(24):3930-6. doi: 10.1200/JCO.2007.15.6752.

Cuzick J, Swanson GP, Fisher G, Brothman AR, Berney DM, Reid JE, Mesher D, Speights VO, Stankiewicz E, Foster CS, Møller H, Scardino P, Warren JD, Park J, Younus A, Flake DD 2nd, Wagner S, Gutin A, Lanchbury JS, Stone S; Transatlantic Prostate Group. Prognostic value of an RNA expression signature derived from cell cycle proliferation genes in patients with prostate cancer: a retrospective study. Lancet Oncol. 2011 Mar;12(3):245-55. doi: 10.1016/S1470-2045(10)70295-3. PMID: 21310658

Faisal FA, Sundi D, Tosoian JJ, Choeurng V, Alshalalfa M, Ross AE, Klein E, Den R, Dicker A, Erho N, Davicioni E, Lotan TL, Schaeffer EM. Racial Variations in Prostate Cancer Molecular Subtypes and Androgen Receptor Signaling Reflect Anatomic Tumor Location. Eur Urol. 2016 Jul;70(1):14-7. doi: 10.1016/j.eururo.2015.09.031. Epub 2015 Oct 9.

Glinsky GV, Berezovska O, Glinskii AB. Microarray analysis identifies a death-from-cancer signature predicting therapy failure in patients with multiple types of cancer. J Clin Invest. 2005 Jun;115(6):1503-21.

Karnes RJ, Ashab HA, Trock B, Ross A, Tsai H, Tosoian J, Erho N, Alshalafa M, Choeurng V, Yousefi K, Haddad Z, Takhar M, Abdollah F, Klein E, Nguyen P, Feng F, Dicker A, Den R, Davicioni E, Jenkins RB, Lotan T, Schaeffer E. Development and validation of an ADT resistance signature that predicts adjuvant hormone treatment failure. 22nd Annual PCF Scientific Retreat. Oct 8-10, 2015.

Klein EA, Cooperberg MR, Magi-Galluzzi C, Simko JP, Falzarano SM, Maddala T, Chan JM, Li J, Cowan JE, Tsiatis AC, Cherbavaz DB, Pelham RJ, Tenggara-Hunter I, Baehner FL, Knezevic D, Febbo PG, Shak S, Kattan MW, Lee M, Carroll PR. A 17-gene assay to predict prostate cancer aggressiveness in the context of Gleason grade heterogeneity, tumor multifocality, and biopsy undersampling. Eur Urol. 2014 Sep;66(3):550-60. doi: 10.1016/j.eururo.2014.05.004. Epub 2014 May 16. PMID: 24836057.

Kumar A, Coleman I, Morrissey C, Zhang X, True LD, Gulati R, Etzioni R, Bolouri H, Montgomery B, White T, Lucas JM, Brown LG, Dumpit RF, DeSarkar N, Higano C, Yu EY, Coleman R, Schultz N, Fang M, Lange PH, Shendure J, Vessella RL, Nelson PS. Substantial interindividual and limited intraindividual genomic diversity among tumors from men with metastatic prostate cancer. Nat Med. 2016 Apr;22(4):369-78. doi: 10.1038/nm.4053. Epub 2016 Feb 29.

Lapointe J, Li C, Higgins JP, van de Rijn M, Bair E, Montgomery K, Ferrari M, Egevad L, Rayford W, Bergerheim U, Ekman P, DeMarzo AM, Tibshirani R, Botstein D, Brown PO, Brooks JD, Pollack JR. Gene expression profiling identifies clinically relevant subtypes of prostate cancer. Proc Natl Acad Sci U S A. 2004 Jan 20;101(3):811-6. Epub 2004 Jan 7.

S E T Larkin, S Holmes, I A Cree, T Walker, V Basketter, B Bickers, S Harris, S D Garbis, P A Townsend, C Aukim-Hastie. Identification of markers of prostate cancer progression using candidate gene expression. Br J Cancer. 2012 January 3; 106(1): 157–165. Published online 2011 November 10. doi: 10.1038/bjc.2011.490.

Lehrer J, Alshalalfa M, Erho N, Davicioni E, Knudsen KE, Kelly WK, Hoffman-Censits J, Lin J, Trabulsi E, Lallas C, Gomella LG, Dicker AP, Chellapagounder T, Haber A, Karnes RJ, Tomlins S, Feng FY, Schaeffer E, Den RB. Coupling in-vitro drug sensitivity data with whole-transcriptome expression profiles for the prediction of patient-specific response to individual drugs. SFU NSERC CREATE Workshop. April 28, 2016.

Long Q, Johnson BA, Osunkoya AO, Lai YH, Zhou W, Abramovitz M, Xia M, Bouzyk MB, Nam RK, Sugar L, Stanimirovic A, Williams DJ, Leyland-Jones BR, Seth AK, Petros JA, Moreno CS. Protein-coding and microRNA biomarkers of recurrence of prostate cancer following radical prostatectomy. Am J Pathol. 2011 Jul;179(1):46-54. doi: 10.1016/j.ajpath.2011.03.008. Epub 2011 May 3.

Long Q, Xu J, Osunkoya AO, Sannigrahi S, Johnson BA, Zhou W, Gillespie T, Park JY, Nam RK, Sugar L, Stanimirovic A, Seth AK, Petros JA, Moreno CS. Global transcriptome analysis of formalin-fixed prostate cancer specimens identifies biomarkers of disease recurrence. Cancer Res. 2014 Jun 15;74(12):3228-37. doi: 10.1158/0008-5472.CAN-13-2699. Epub 2014 Apr 8.

Nakagawa T, Kollmeyer TM, Morlan BW, Anderson SK, Bergstralh EJ, Davis BJ, Asmann YW, Klee GG, Ballman KV, Jenkins RB. A tissue biomarker panel predicting systemic progression after PSA recurrence post-definitive prostate cancer therapy. PLoS One. 2008;3(5):e2318. doi: 10.1371/journal.pone.0002318. Epub 2008 May 28.

GenomeDx Biosciences Laboratory
10355 Science Center Drive, Suite 240
San Diego, CA 92121
Tel 1 888 792 1601 | Fax 1 855 324 2768
GRIDsupport@genomedx.com | www.genomedx.com

RESEARCH USE ONLY

The information provided in this analysis has not been clinically validated and should not be used for clinical decision-making.

REFERENCES

Penney KL, Sinnott JA, Fall K, Pawitan Y, Hoshida Y, Kraft P, Stark JR, Fiorentino M, Perner S, Finn S, Calza S, Flavin R, Freedman ML, Setlur S, Sesso HD, Andersson SO, Martin N, Kantoff PW, Johansson JE, Adami HO, Rubin MA, Loda M, Golub TR, Andrén O, Stampfer MJ, Mucci LA. mRNA expression signature of Gleason grade predicts lethal prostate cancer. J Clin Oncol. 2011 Jun 10;29(17):2391-6. doi: 10.1200/JCO.2010.32.6421. Epub 2011 May 2.

Ramaswamy S, Ross KN, Lander ES, Golub TR. A molecular signature of metastasis in primary solid tumors. Nat Genet. 2003 Jan;33(1):49-54. Epub 2002 Dec 9.

Saal LH, Johansson P, Holm K, Gruvberger-Saal SK, She QB, Maurer M, Koujak S, Ferrando AA, Malmström P, Memeo L, Isola J, Bendahl PO, Rosen N, Hibshoosh H, Ringnér M, Borg A, Parsons R. Poor prognosis in carcinoma is associated with a gene expression signature of aberrant PTEN tumor suppressor pathway activity. Proc Natl Acad Sci U S A. 2007 May 1;104(18):7564-9. Epub 2007 Apr 23.

Singh D, Febbo PG, Ross K, Jackson DG, Manola J, Ladd C, Tamayo P, Renshaw AA, DAmico AV, Richie JP, Lander ES, Loda M, Kantoff PW, Golub TR, Sellers WR. Gene expression correlates of clinical prostate cancer behavior. Cancer Cell. 2002 Mar;1(2):203-9.

Stephenson AJ, Smith A, Kattan MW, Satagopan J, Reuter VE, Scardino PT, Gerald WL. Integration of gene expression profiling and clinical variables to predict prostate carcinoma recurrence after radical prostatectomy. Cancer. 2005 Jul 15;104(2):290-8.

Talantov D, Jatkoe TA, Böhm M, Zhang Y, Ferguson AM, Stricker PD, Kattan MW, Sutherland RL, Kench JG, Wang Y, Henshall SM. Gene based prediction of clinically localized prostate cancer progression after radical prostatectomy. J Urol. 2010 Oct;184(4):1521-8. doi: 10.1016/j.juro.2010.05.084. Epub 2010 Aug 17.

Tomlins SA, Alshalalfa M, Davicioni E, Erho N, Yousefi K, Zhao S, Haddad Z, Den RB, Dicker AP, Trock BJ, DeMarzo AM, Ross AE, Schaeffer EM, Klein EA, Magi-Galluzzi C, Karnes RJ, Jenkins RB, Feng FY. Characterization of 1577 primary prostate cancers reveals novel biological and clinicopathologic insights into molecular subtypes. Eur Urol. 2015 Oct;68(4):555-67. doi: 10.1016/j.eururo.2015.04.033. Epub 2015 May 8.

Varambally S, Yu J, Laxman B, Rhodes DR, Mehra R, Tomlins SA, Shah RB, Chandran U, Monzon FA, Becich MJ, Wei JT, Pienta KJ, Ghosh D, Rubin MA, Chinnaiyan AM. Integrative genomic and proteomic analysis of prostate cancer reveals signatures of metastatic progression. Cancer Cell. 2005 Nov;8(5):393-406.

Chin-Lee Wu, Brock E. Schroeder, Xiao-Jun Ma, Christopher J. Cutie, Shulin Wu, Ranelle Salunga, Yi Zhang, Michael W. Kattan, Catherine A. Schnabel, Mark G. Erlander, W. Scott McDougal. Development and validation of a 32-gene prognostic index for prostate cancer progression. Proc Natl Acad Sci U S A. 2013 April 9; 110(15): 6121–6126. Published online 2013 March 26. doi: 10.1073/pnas.1215870110.

Yu J, Yu J, Rhodes DR, Tomlins SA, Cao X, Chen G, Mehra R, Wang X, Ghosh D, Shah RB, Varambally S, Pienta KJ, Chinnaiyan AM. A polycomb repression signature in metastatic prostate cancer predicts cancer outcome. Cancer Res. 2007 Nov 15;67(22):10657-63.

Zhang D, Park D, Zhong Y, Lu Y, Rycaj K, Gong S, Chen X, Liu X, Chao HP, Whitney P, Calhoun-Davis T, Takata Y, Shen J, Iyer VR, Tang DG. Stem cell and neurogenic gene-expression profiles link prostate basal cells to aggressive prostate cancer. Nat Commun. 2016 Feb 29;7:10798. doi: 10.1038/ncomms10798. PubMed PMID: 26924072; PubMed Central PMCID: PMC4773505.

Zhao SG, Chang SL, Spratt DE, Erho N, Yu Menggang, Ashab HA, Alshalalfa M, Speers C, Tomlins SA, Davicioni E, Dicker AP, Carroll PR, Cooperberg MR, Freedland SJ, Karnes RJ, Ross A, Schaeffer EM, Den RB, Nguyen PL, Feng FY. A 24-gene Predictor of Response to Post-operative Radiation Therapy in Prostate Cancer. The Lancet Oncology. 2016.

