Genes	Differentially expressed genes (Wilcoxon Rank Test)			
	Resistant DU145 Samples	**Sensitive DU145 Samples**	**Resistant PC3 Samples**	**Sensitive PC3 Samples**
CENPF	KPNA2, DKK1, NEAT1	DKK1, TOP2A, NEAT1	HIF1A, HSPA5, KPNA2, TFRC, HIST1H4C, KRT18, GARS	HIF1A, HSPA5, KPNA2, TFRC, HIST1H4C, KRT18, GARS
DKK1	HIF1A, HSPA5, KPNA2, TFRC, CENPF, HIST1H4C, KRT18, GARS, TOP2A	KPNA2, TFRC, CENPF, HIST1H4C, KRT18, GARS, TOP2A	HIF1A, HSPA5, KPNA2, TFRC, HIST1H4C, KRT18, GARS, NEAT1	HIF1A, HSPA5, KPNA2, TFRC, HIST1H4C, KRT18, GARS
GARS	HIF1A, HSPA5, DKK1, NEAT1	HIF1A, DKK1, NEAT1	HIF1A, HSPA5, KPNA2, TFRC, CENPF, DKK1, HIST1H4C, TOP2A, NEAT1	HIF1A, HSPA5, KPNA2, TFRC, CENPF, DKK1, TOP2A, NEAT1
HIF1A	KPNA2, TFRC, DKK1, HIST1H4C, KRT18, GARS, TOP2A, NEAT1	KPNA2, TFRC, KRT18, GARS, TOP2A, NEAT1	CENPF, DKK1, KRT18, GARS, TOP2A, NEAT1	CENPF, DKK1, KRT18, GARS, TOP2A, NEAT1
HIST1H4C	HIF1A, HSPA5, DKK1, NEAT1	DKK1, TOP2A, NEAT1	CENPF, DKK1, KRT18, GARS, TOP2A, NEAT1	KPNA2, TFRC, CENPF, DKK1, TOP2A, NEAT1
HSPA5	KPNA2, DKK1, HIST1H4C, KRT18, GARS, TOP2A, NEAT1	KPNA2, KRT18, TOP2A, NEAT1	CENPF, DKK1, KRT18, GARS, TOP2A, NEAT1	CENPF, DKK1, KRT18, GARS, TOP2A, NEAT1
KPNA2	HIF1A, HSPA5, TFRC, CENPF, DKK1, NEAT1	HIF1A, HSPA5, DKK1, NEAT1	CENPF, DKK1, KRT18, GARS, TOP2A, NEAT1	CENPF, DKK1, HIST1H4C, KRT18, GARS, TOP2A, NEAT1
KRT18	HIF1A, HSPA5, TFRC, DKK1, NEAT1	HIF1A, HSPA5, DKK1, NEAT1	HIF1A, HSPA5, KPNA2, TFRC, CENPF, DKK1, HIST1H4C, TOP2A, NEAT1	HIF1A, HSPA5, KPNA2, TFRC, CENPF, DKK1, TOP2A, NEAT1
NEAT1	HIF1A, HSPA5, KPNA2, TFRC, CENPF, HIST1H4C, KRT18, GARS, TOP2A	HIF1A, HSPA5, KPNA2, TFRC, CENPF, HIST1H4C, KRT18, GARS, TOP2A	HIF1A, HSPA5, KPNA2, TFRC, DKK1, HIST1H4C, KRT18, GARS	HIF1A, HSPA5, KPNA2, TFRC, HIST1H4C, KRT18, GARS
TFRC	HIF1A, KPNA2, DKK1, KRT18, NEAT1	HIF1A, DKK1, NEAT1	CENPF, DKK1, KRT18, GARS, TOP2A, NEAT1	CENPF, DKK1, HIST1H4C, KRT18, GARS, TOP2A, NEAT1
TOP2A	HIF1A, HSPA5, DKK1, NEAT1	HIF1A, HSPA5, CENPF, DKK1, HIST1H4C, NEAT1	HIF1A, HSPA5, KPNA2, TFRC, HIST1H4C, KRT18, GARS	HIF1A, HSPA5, KPNA2, TFRC, HIST1H4C, KRT18, GARS

Table 2. The differentially expressed genes obtained from Wilcoxon Rank Test

Table 2 shows the differentially expressed genes according to Wilcoxon Ranked Sum Test for resistant and sensitive samples in both cell lines. There are a lot more differentially expressed genes for all cell lines compared to Table 1 and **it's interesting to see that the set of genes which are differentially expressed from one another is similar for both sensitive and resistant samples and there is a large number of genes which are differentially expressed from one which reflects the complexity of prostate cancer.** Using CENPF as an example we can see that it is differentially expressed from KPNA2, NEAT1 and DKK1. KPNA2 is associated with biomedical recurrence in prostate cancer, NEAT1 promotes prostate cancer growth through the IGF1R/AKT signaling pathway and DKK1 is involved in tumor growth but also promotes bone metastasis (D'Antonio, A., Caputo, A., Fraggetta, F.,

Pepe, P., Insabato, L., et al., 2020 & Xiong, W., Huang, C., Deng, H., Jian, C., Zen, C., et al., 2018 & Thudi, N. K., Martin, C. K., Murahari, S., Shu, S. T., Lanigan, L. G., et al., 2011). The reason why these genes may exhibit differential expressions from CENPF could be due to the fact that they have different roles in prostate cancer and thus have differential expression patterns based on the response state and the cell line. **The functionality of these genes is still not well known within literature and thus it is difficult for us to explain the reasoning why certain genes have differential expression from one another in different response states.** This difference could be attributed to the limitations of our study as our data could not be representative of the actual complexity of the genes within prostate cancer and thus our WIlcoxon analysis will unlikely generate correct results.

1. D'Antonio, A., Caputo, A., Fraggetta, F., Pepe, P., Insabato, L., Barra, E., ... & Zeppa, P. (2020). KPNA2/ERG coexpression is associated with early recurrence in advanced prostate cancers. Applied Immunohistochemistry & Molecular Morphology, 28(1), 62-66. 10.1016/j.canlet.2012.12.013
2. Xiong, W., Huang, C., Deng, H., Jian, C., Zen, C., Ye, K., ... & Zhu, L. (2018). Oncogenic non-coding RNA NEAT1 promotes prostate cancer cell growth through the SRC3/IGF1R/AKT pathway. The International Journal of Biochemistry & Cell Biology, 94, 125-132. https://doi.org/10.1016/j.biocel.2017.12.005
3. Thudi, N. K., Martin, C. K., Murahari, S., Shu, S. T., Lanigan, L. G., Werbeck, J. L., ... & Rosol, T. J. (2011). Dickkopf-1 (DKK-1) stimulated prostate cancer growth and metastasis and inhibited bone formation in osteoblastic bone metastases. The Prostate, 71(6), 615-625. https://doi.org/10.1002/pros.21277