

SUPPLEMENTAL MATERIAL:

A

Cell line	Androgen receptor	Docetaxel resistance (nM)	Cabazitaxel resistance (nM)
IGR-CaP1	-	100	50
PC3	-	12	15
LNCaP	+	12	15
22RV1	+	100	50

B

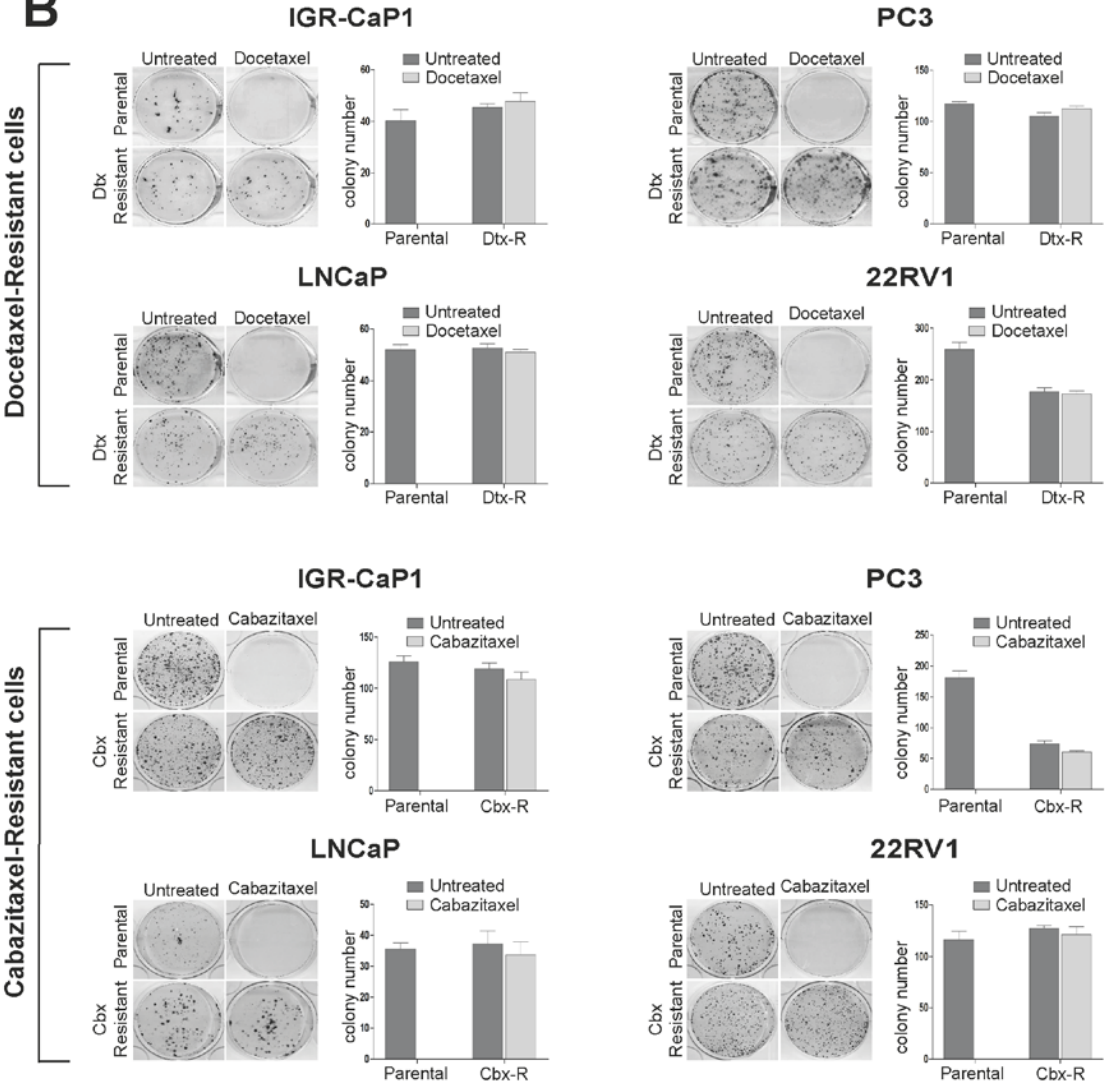


Figure S1. Characteristics of chemoresistant prostate cancer cells. (A) Table summarizing the androgen receptor status and the maximal dose of resistance obtained with docetaxel and cabazitaxel for IGR-CaP, PC3, LNCaP and 22RV1. **(B)** Clonogenicity assay; pictures of the representative stained clones obtained for parental or chemoresistant cells after control, docetaxel or cabazitaxel treatment. Colony count is represented as mean ± SD.

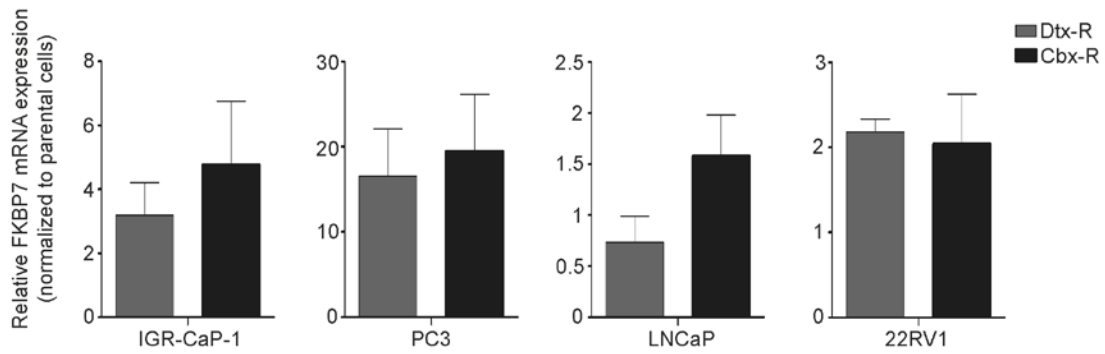


Figure S2. Upregulation of FKBP7 mRNA levels in chemoresistant prostate cancer cells. Quantitative RT-PCR (qRT-PCR) of FKBP7 mRNA level in docetaxel (Dtx-R) or cabazitaxel (Cbx-R) resistant cells, normalized to parental cells and GUSB. Data shows mean \pm SD (n=3).

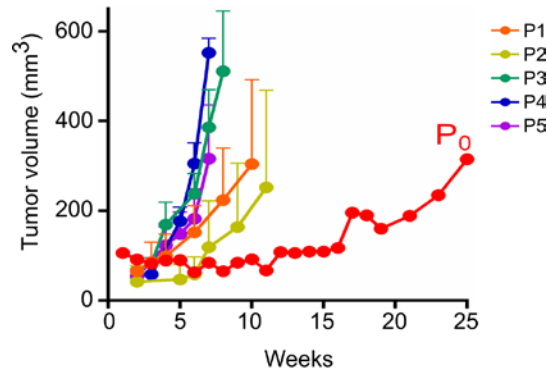


Figure S3: Increase of tumorigenicity of chemoresistant tumors through successive passages in mice. Tumor growth during the five passages done to generate the docetaxel resistant mice model. The data shows the mean \pm SEM.

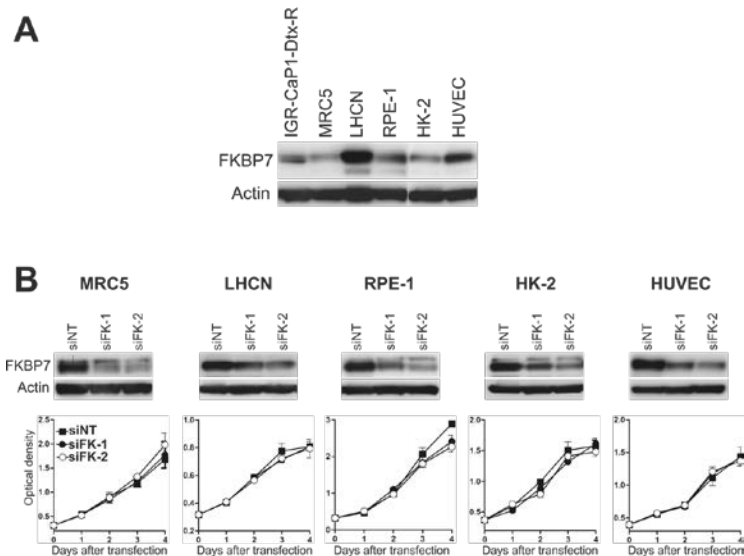


Figure S4: FKBP7 silencing does not affect cell growth in non-tumorous cells. (A) Immunoblot showing FKBP7 protein level in various non-cancerous cells, with IGR-CaP1-Dtx-R as a control of FKBP7 expression.

Actin is the loading control. (B) Immunoblot shows FKBP7 knockdown efficiency 48h after transfection with either two different siRNA sequences targeting FKBP7 (siFK-1 (●) or siFK-2 (○)) or control siRNA (siNT, ■). Cell viability with cells transfected with either two different siRNA sequences targeting FKBP7 or control siRNA is determined daily with WST1.

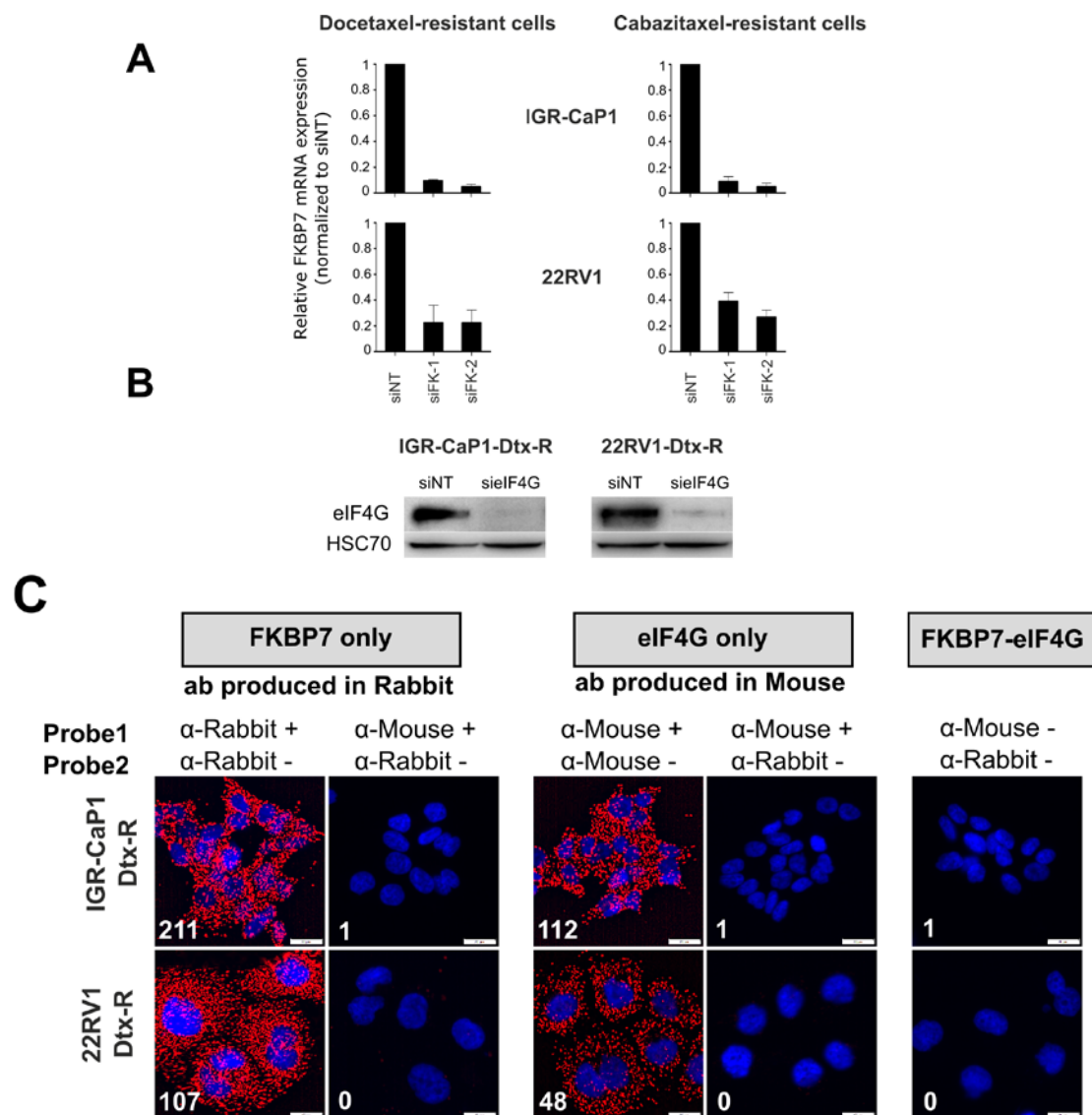


Figure S5: FKBP7 regulates the formation of eIF4F translation initiation complex through a direct interaction with eIF4G. (A) Quantitative RT-PCR (qRT-PCR) showing the FKBP7 knockdown efficiency 48h after transfection with either two different siRNA sequences targeting FKBP7 (siFK-1 or siFK-2) or control siRNA (siNT) in docetaxel- or cabazitaxel-resistant IGR-CaP1 and 22RV1. FKBP7 mRNA level was normalized to siRNA control and with GUSB. (B) Immunoblots showing eIF4G knockdown efficiency 48h after transfection with either siRNA targeting eIF4G (sielF4G) or siRNA control (siNT) in docetaxel resistant IGR-CaP1 and 22RV1 (IGR-CaP1-Dtx-R and 22RV1-Dtx-R respectively). HSC70 is the loading control. (C) Proximity ligation assay (PLA) performed in order to prove the specificity of FKBP7-eIF4G interaction. The interactions studied are noted in grey rectangles and the probes used are listed. The expression of FKBP7, of eIF4G and the interaction FKBP7-eIF4G were detected in docetaxel-resistant IGR-CaP1 (IGR-CaP1-Dtx-R) and 22RV1 (22RV1-Dtx-R). The interactions were visualized as red dots and the nuclei in blue. The white numbers indicate the mean calculated on at least 100 cells. Bars represent 20µm.

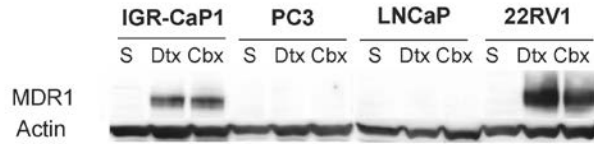


Figure S6: Expression of MDR1 in prostate cancer cell lines. Immunoblots shows MDR1 level in parental (S) and docetaxel (Dtx) or cabazitaxel (Cbx) resistant IGR-CaP1, PC3, LNCaP and 22RV1. Actin is the loading control.

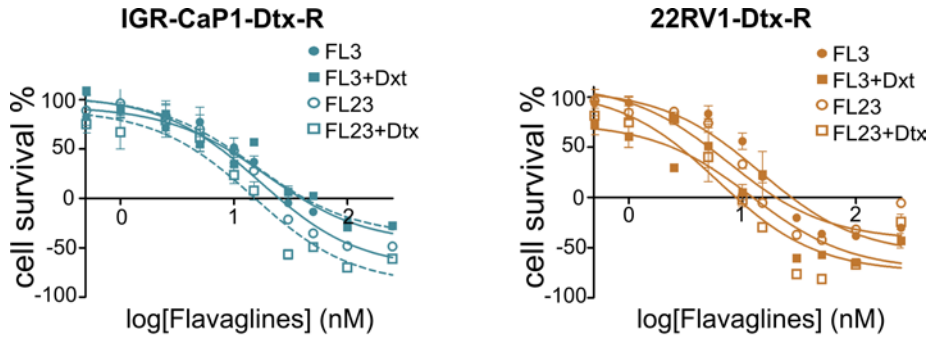


Figure S7. Effect of the combination of Docetaxel with Flavaglines on cell proliferation of chemoresistant cells. Cell proliferation assay; cell viability (calculated relatively to control treatment) of docetaxel-resistant cells IGR-CaP1 and 22RV1 (IGR-CaP1-Dtx-R and 22RV1-Dtx-R respectively) treated either with flavagline 3 or 23 alone or in association with docetaxel (50nM). The data are represented as the mean \pm SD.