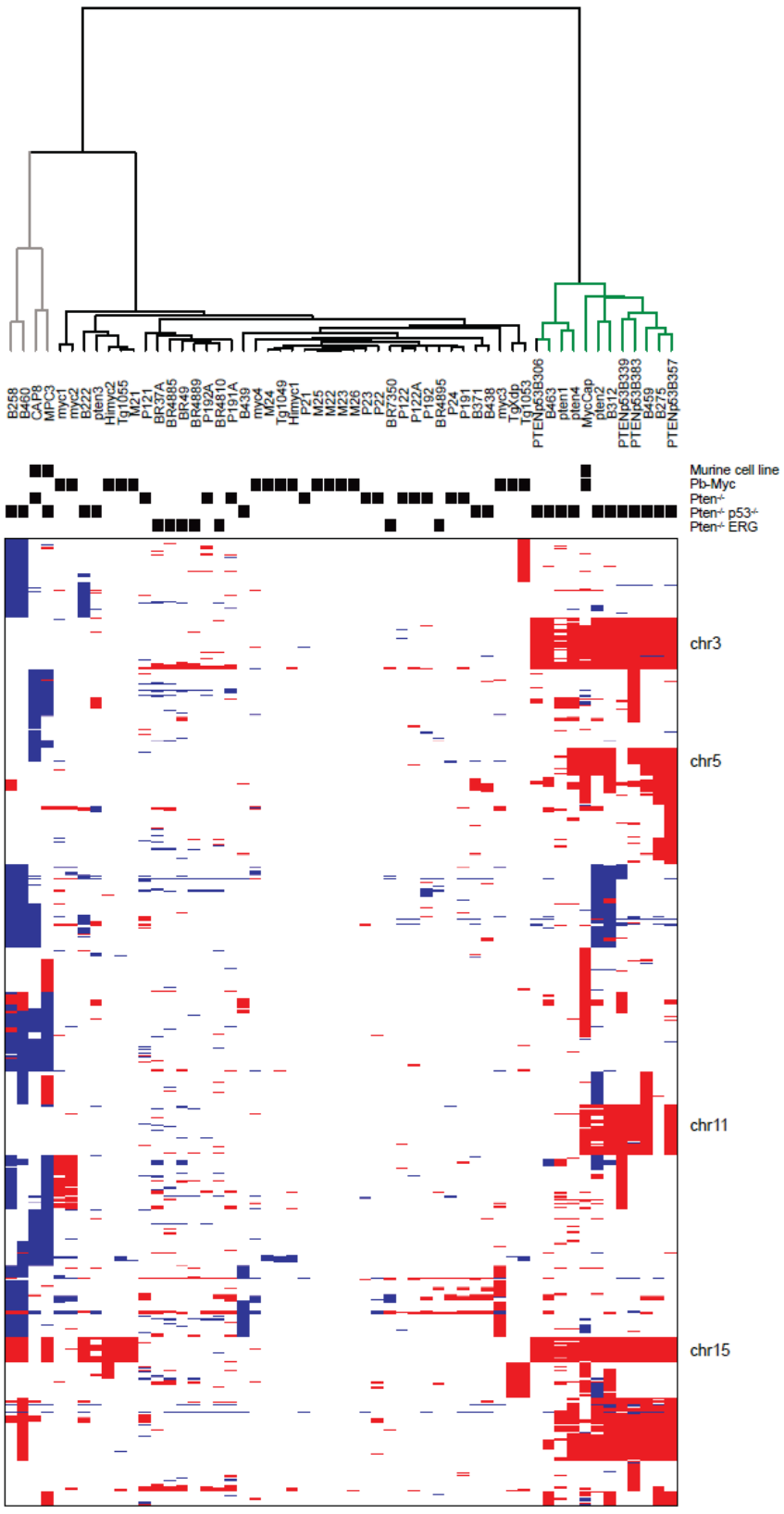


Figure S1. Unsupervised clustering of Genomic profiles of GEM models of prostate cancer. Unsupervised clustering of aCGH data of GEM models of prostate cancer. Blue color = copy number loss, Red color = copy number gain.

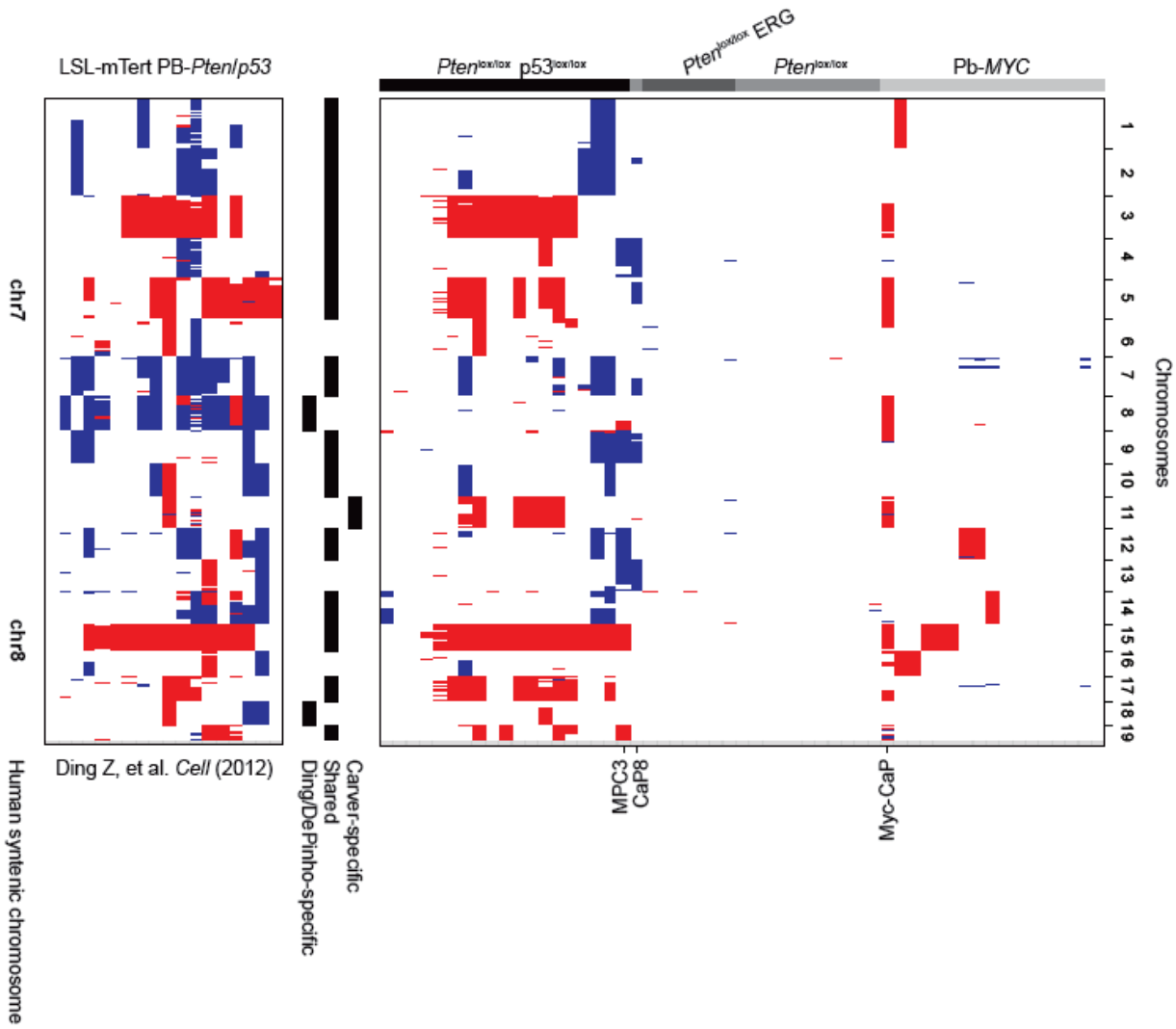
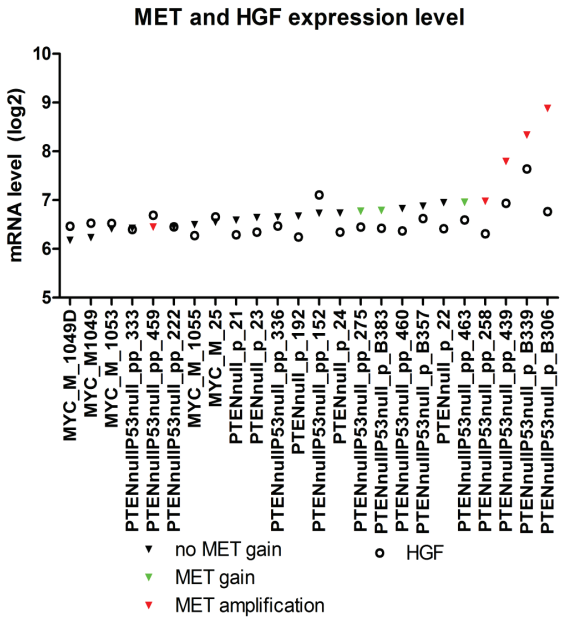


Figure S2. Genomic profiles of GEM models of prostate cancer. Comparison of aCGH data of GEM models of prostate cancer including a recently published data set from Ding et al, 2012 where prostates from mTert Pten p53 null mice were analyzed. Blue color = copy number loss, Red color = copy number gain.

A.



B.

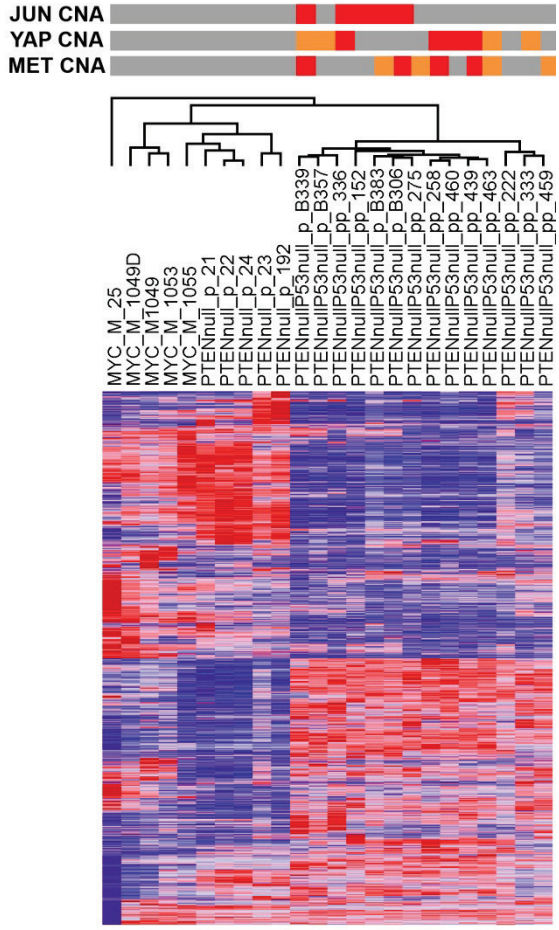
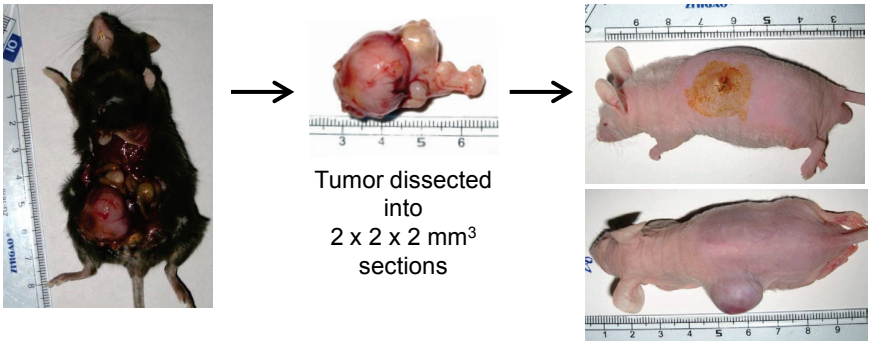


Figure S3. MET and HGF expression in GEM models of prostate cancer.
(A) mRNA expression of Met and Hgf in GEM models of prostate cancer, assayed by expression array and annotated by *Met* copy number status.
(B) Unsupervised hierarchical clustering of gene expression changes in 5 PB-*MYC, 5 Pten*$^{lox/lox}$ and 14 *Pten*$^{lox/lox}$ *p53*$^{lox/lox}$ *mice, annotated with Yap1, Met, and Jun* copy number gain status. Red indicates copy number amplification, orange indicates copy number gain, and gray indicates no copy number gain.

A.



B.

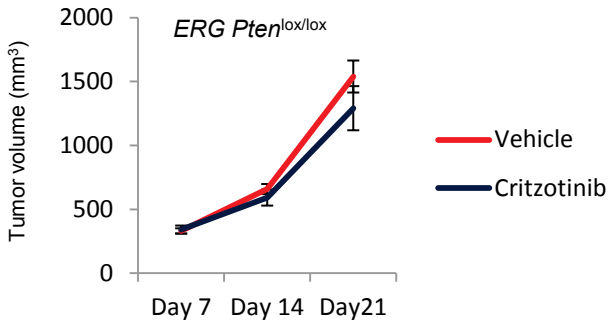


Figure S4. Preclinical models of prostate cancer for therapeutic studies. (A) Prostate cancer specimens were harvested from Pten p53 null mice, dissected, and transplanted into athymic mice to establish tumors. (B) Mice grafter with tumors derived from *Pten ERG* mice (no *Met* copy number gain) were treated with vehicle (n=8) or crizotinib (n=8, 50 mg/kg/day, Pfizer) for a total of 21 days and tumor volumes were measured on a weekly basis.