

## PRAD - Prostate adenocarcinoma

Subtype	Biology & Expression	Genomic Alterations	Clinical Features
<b>ETS-fusion</b>	Strong AR-driven program; upregulation of ERG (or other ETS) target genes; luminal epithelial markers	TMPRSS2-ERG fusions (~46%), plus rarer ETV1, ETV4, FLI1 fusions	Intermediate prognosis; elevated PSA; generally ADT-sensitive
<b>SPOP-mutant</b>	Heightened AR transcriptional activity; reduced DNA-repair and chromatin-remodeling gene expression	SPOP point mutations (~11%); frequent CHD1 deletions; genomic instability	Relatively favorable prognosis; lower Gleason scores; good response to ADT
<b>FOXA1-mutant</b>	Altered AR cistrome with luminal differentiation signatures; FOXA1 pioneer-factor dysregulation	FOXA1 hotspot mutations (~4%)	Variable outcome; some association with early biochemical recurrence
<b>IDH1-mutant</b>	CpG-island hypermethylation phenotype; metabolic reprogramming; neuroendocrine-like expression	IDH1 R132 mutations (~1%)	Rare; distinct epigenetic profile; potential sensitivity to IDH1 inhibitors
<b>Other/Unknown</b>	Mixed luminal and basal gene signatures; no dominant transcriptional driver	Lacks ETS fusions, SPOP, FOXA1 or IDH1 alterations (~35%)	Heterogeneous clinical course; may require additional molecular stratification