*ENOX1,*  *CCDC122* and *LACC1* role in progression of prostate cancer.

In this study analysing prostate cancer progression, I leveraged multiple bioinformatics techniques and databases to reveal novel mechanisms and therapeutic targets. By examining age-stratified genomic alterations in nearly 500 patients, I identified the genes ENOX1, CCDC122, and LACC1 as having increased deletion rates with later diagnosis.

Through integrated analysis, I demonstrated these three genes' combined effects in enriching pathways promoting cancer cell proliferation when deleted. The top implicated signalling cascades were oestrogen biosynthesis, KSRP mRNA regulation, omega-3/6 metabolism, and RAP1 signalling. My approach provided a multidimensional perspective on how prostate cancers strategically dysregulate systems biology to their advantage.

After mapping enriched genes to curated pathways using Enrichr and Reactome, I highlighted targetable molecules in prostate cancer progression pathways. My study exemplified the insight bioinformatics workflows can offer into elusive disease mechanisms. I also displayed dedicated perseverance by overcoming initial technical difficulties with statistical analysis of patient data in cbioportal.

This work spearheading an innovative methodology showcases skills in leveraging public databases, analysing big datasets, interpreting enrichment patterns, identifying actionable targets, and ultimately furthering personalised oncology. My proactive drive to master bioinformatics and think creatively positions me to continue making impact in prostate cancer research through a PhD investigating immunotherapy biomarkers.