

To investigate the role of ZBTB10 in the development of castration-resistant prostate cancer

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Prostate cancer (PCa) is the second leading cause of cancer-related deaths in men worldwide. Patients with local advanced prostate cancer will be received androgen deprivation therapy (ADT) and generally have a good prognosis. Unfortunately, some PCa patients will develop into a status called castration-resistant prostate cancer (CRPC) which means their tumors will still progress even under low androgen level. Numerous studies have shown that abnormal activation of androgen receptor (AR) including AR mutation, AR copy number gain, and AR splice variants are the major underlying mechanisms causing transition from castration-sensitive prostate cancer (CSPC) to CRPC. Besides AR, other critical factors involved in the development of CRPC are still unknown. According to our bioinformatics analysis, Zinc Finger and BTB Domain Containing 10 (ZBTB10), a transcription factor, was identified to highly overexpress in the prostate cancer cell lines, particularly in CRPC cells. Furthermore, its abnormal expression was observed in the higher percentage of CRPC specimens after analyzing public dataset. Since there is no literature to report it yet, we are interesting to investigate the role of ZBTB10 in the development of CRPC. There are three specific aims to verify our ideas. First, specific aim one is to investigate the role of ZBTB10 in the development CRPC. Second, specific aim two is to study the underlying mechanism of ZBTB10 isoform switching during the transition from CSPC to CRPC. Finally, specific aim three is to verify whether ZBTB10 can be used as an index for applying novel therapy to CRPC patients. Hopefully, we can identify that ZBTB10 might be an alternative mechanism causing CRPC development and innovate a novel therapy for CRPC patients through our works.