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CYP19A1 is regulated by BRD4 and suppresses castration-resistant prostate cancer cell invasion and proliferation by decreasing AR expression

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Castration-resistant prostate cancer (CRPC) is the final stage of prostate cancer. Thus far, there is no effective method for CRPC treatment. Androgen and androgen receptor (AR) play a vital role in CRPC occurrence and drug tolerance. Testosterone is the main androgen in males. CYP19A1 can encode aromatase, a key enzyme that can catalyze the conversion of testosterone to estrogen, and may affect both androgen and AR. CYP19A1 may play a role in the occurrence of CRPC; however, the function of CYP19A1 in CRPC remain unclear. In this study, we found that CYP19A1 was downregulated in CRPC samples and cells. CYP19A1 overexpression decreased CRPC cell invasion and proliferation. In addition, CYP19A1 expression was negatively correlated with AR expression. CYP19A1 affected CRPC cell invasion and proliferation ability by suppressing the expression of AR, and this may be attributed to the metabolism of testosterone by CYP19A1. Moreover, the BRD4 inhibitor-JQ1 induced the expression of CYP19A1 and suppressed the expression of AR. Following BRD4 knockdown, CYP19A1 showed a higher expression level, and, AR expression was decreased. Taken together, our findings demonstrated that CYP19A1 could reduce CRPC cell invasion and proliferation by targeting AR, and this process could be regulated by BRD4. The BRD4-CYP19A1-AR pathway may be a potential cause of CRPC occurrence. Therefore, CYP19A1 may be a potential therapeutic target for treating CRPC.

Biography

Xi Chen is affiliated from Tongji University, China. His research interest includes Cancer Genetics and Epigenetics, Prostrate Cancer.

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