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The role of TRIM24 during prostate cancer progression

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Introduction

The steroid hormone androgen mediates a wide range of developmental, physiological and malignant responses by acting as a ligand for the androgen receptor (AR). AR in turn functions as a nuclear receptor transcription factor and executes specific gene expression programs in an androgen-dependent manner. Since the growth of prostate cancer cells initially depends on androgen, cancer therapy uses hormone-deprivation approaches to reduce the levels of serum androgen. While prostate cancer (PC) initially responds to androgen-ablation, most tumors progress to a castration resistant (CR) state insensitive to treatment. Both the androgen-dependent and the CRPC state depend on AR, which is probably activated by alternative molecular pathways in CRPC in response to low androgen levels.

Methods and results

Here, we assess the role of the transcriptional co-regulator TRIM24/TIF1α in mediating AR-dependent gene expression and growth. We find that TRIM24 expression is increased in different states of PC, particularly in CRPC. When we induce the drug-regulated knock-down of TRIM24, we find that cell growth in both our androgen-dependent (LNCaP) and in our CRPC model (LNCaP-abl) is affected. Therefore, we hypothesize that TRIM24 is involved in mediating both ligand-dependent and ligand-independent activation of AR. This is further consistent with our finding that TRIM24 and AR can physically interact in both conditions. We are currently identifying TRIM24-dependent gene expression programs by combining genome-wide binding analyses with microarray studies.

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Discussion

This will help us shed light on how the transcriptional co-regulator TRIM24 may reprogram AR activity in its progression from an androgen-dependent to a castration-resistant state. Current results from this study will be presented at the meeting.

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