The p53-SP1 Axis Regulates the Immune Checkpoint Molecule CD276 in Prostate Cancer

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Introduction

- TP53, a tumor suppressor gene, incurs deletions or mutations in more than 50% of cancers.
- Cluster of Differentiation 276 (CD276), also known as B7 Homolog 3 (B7-H3), is an immune checkpoint molecule.
- Recent findings have shown that CD276 inhibits T cells to promote tumor proliferation and invasion.
- Previous studies using CD276 as a target for immunotherapy have found promising anti-tumor effects with CD276 antibodies.
- These anti-CD276 antibodies are currently undergoing clinical trials as immunotherapy agents.

Cell Lines:
- Human prostate cancer cell lines: LNCaP, 22RV1, DU145, and PC3
- Mouse prostate cancer cell lines: PP7777 and DX1

Methods:
- Western blotting analysis
- Quantitative reverse transcription PCR
- Flow cytometry
- CRISPR/Cas9 and shRNA
- Chromatin immunoprecipitation assay
- Dual-luciferase reporter assay

Results

- Figure 1. Immune checkpoint molecule alterations in patients with tumor suppressor gene deficiency. Patient data taken from the cBioPortal for cancer genomics database.

Conclusions

The immune checkpoint molecule CD276 was identified as a promising therapeutic candidate in prostate cancer. Our study demonstrated that the p53-SP1 axis is involved in the transcriptional regulation of CD276 in prostate cancer, suggesting the potential of immunotherapy targeting CD276 in p53-deficient cancers.

References