

Ecdysoneless Protein (ECD): A Novel Androgen Receptor Target Promoting Prostate Cancer Tumorigenesis by Regulating Androgen Response Elements (AREs)

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OBJECTIVE

To identify whether **Ecdysoneless protein (ECD)** functions as a novel transcriptional coactivator for the **androgen receptor (AR)** and to determine its role in amplifying AR-driven gene expression at canonical **Androgen Response Elements (AREs)** in prostate cancer.

METHODOLOGY

1 Protein-protein interaction

Co-immunoprecipitation and GST pull-down assays in AR-positive prostate cancer cell lines.

2 Transcriptional activity

ARE-driven luciferase reporter assays with ECD overexpression or knockdown.

3 Chromatin occupancy

ChIP for AR and ECD at AREs following dihydrotestosterone (DHT) stimulation.

4 Target gene expression

qPCR quantification of downstream AR target genes.

RESULTS

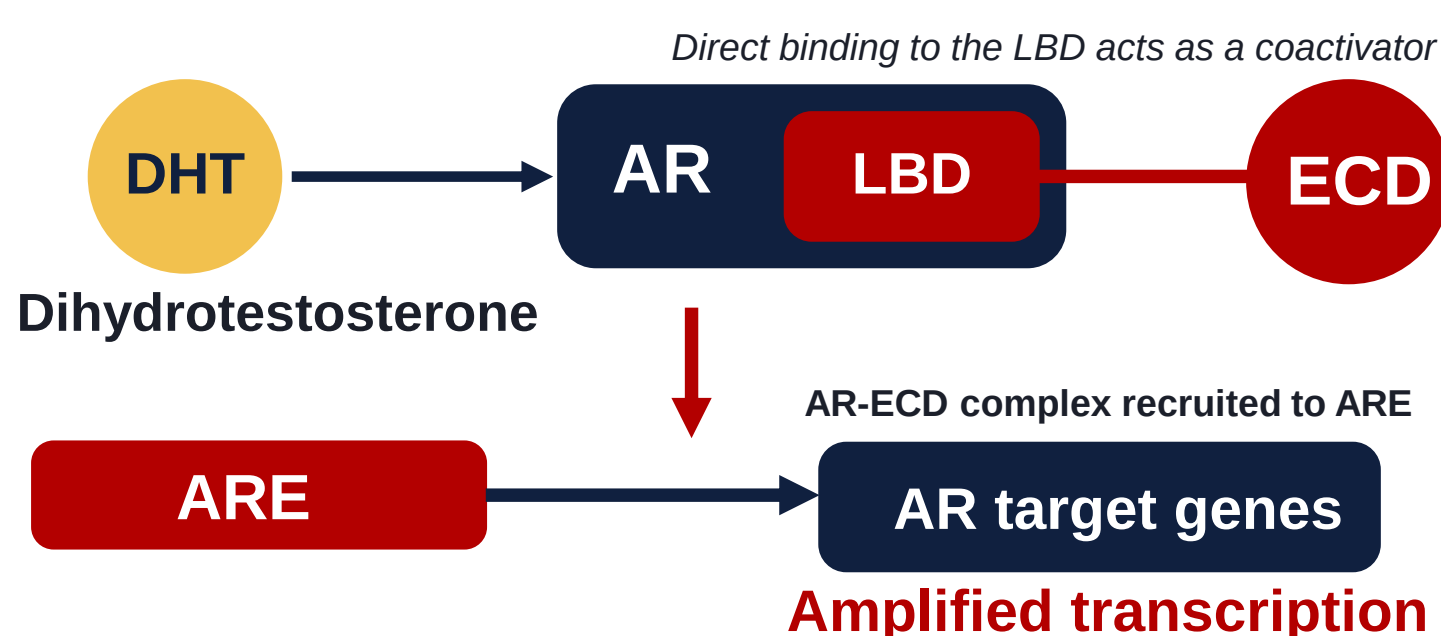
1 ECD physically interacts with AR, specifically through its **ligand-binding domain (LBD)**.

2 ECD overexpression **robustly enhanced ARE-driven luciferase activity** in a dose-dependent manner; ECD silencing significantly reduced it.

3 ChIP confirmed **ECD is recruited to AREs** in an AR-dependent manner upon DHT activation.

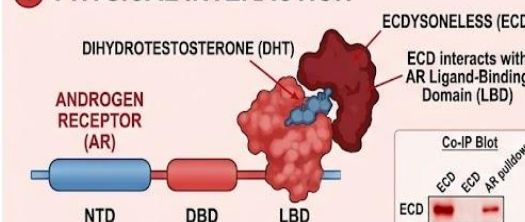
4 ECD depletion **markedly suppressed endogenous expression** of classical AR target genes - ECD is required for full transcriptional output.

PROPOSED MODEL

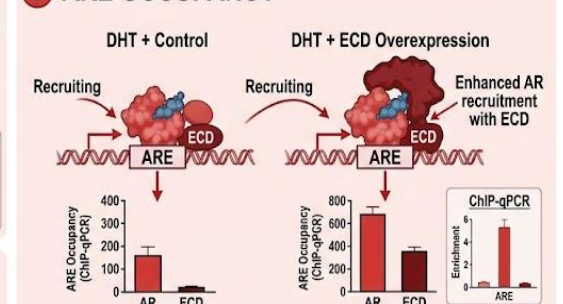


ECD: A NOVEL AR COACTIVATOR AMPLIFYING ARE-DRIVEN TRANSCRIPTION

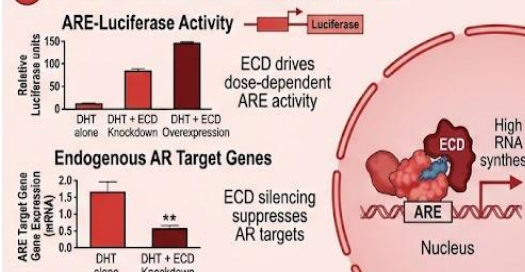
1 PHYSICAL INTERACTION



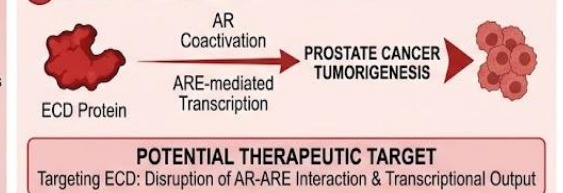
2 ARE OCCUPANCY



3 TRANSCRIPTIONAL OUTPUT



4 MECHANISM & IMPLICATION



CONCLUSION

ECD is a previously unrecognized AR coactivator that directly interfaces with the receptor to amplify ARE-driven transcription. By enhancing AR signaling at the genomic level, ECD plays a critical role in sustaining prostate cancer progression, making it a **promising therapeutic target** for disrupting AR transcriptional activity beyond conventional ligand-binding antagonists.

Acknowledgment

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