Role of Estrogen Receptor in Cancer

Jemma S. Braun*

Royal College of Surgeons of England, London, UK

Corresponding Author*

Jemma S. Braun

Royal College of Surgeons of England, London, UK

E-mail: jemma.braun@gmail.com

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Brief Report

Estrogen Receptor (ER) signaling is essential for breast cell homeostasis and transformation, and it is the major target for therapeutic intervention in Breast Cancer (BC). ER-mediated transactivation is often tightly regulated by a slew of cofactors that regulate the receptor's transcriptional competence at several levels. Several lines of evidence suggest that chromatin deposition of the pioneer factors FOXA1 and GATA3 at ER-target loci is important for the receptor's proper DNA interactions. Subsequent recruitment of enzymes that regulate the chromatin environment, such as Histone Acetyl Transferase (HAT) p300, enhances ER signaling even further by increasing recruitment of the RNA polymerase machinery. Additional downstream trans-acting factors, such as the Bromodomain and Extra-Terminal (BET) protein BRD4, which promotes mRNA elongation, are also required for ER-induced gene expression.

Ovarian Cancer (OC) is a heterogeneous tumor that has traditionally been classified based on histologic and differentiation grade characteristics. High-Grade Serous Ovarian Cancer (HGSOC) is the most aggressive and lethal form of epithelial ovarian cancer, and despite responding to platinum-based therapies, the majority of patients relapse after developing resistance to first-line therapy. Estrogen Receptors (ERs) are expressed in several OC histotypes, with the highest expression in serous ones, but endocrine therapy has been used with modest and variable results in the treatment of OC due to the expression of both ER and ER receptor subtypes, which behave in opposite ways after anti-estrogen administration. Targeting specific ER subtypes may thus aid in the identification of personalised therapeutic approaches and improve survival. ER is expressed in more than 50% of OCs and in approximately 80% of HGSOC, where it is associated with a poor prognosis. Given its ability to promote cell proliferation and platinum resistance, ER is worth investigating in OC. A direct action of oestrogen in OC growth, metastasis, and progression mediated by ER has been shown to occur through specific pathways such as VEGF and MAPK signaling. Furthermore, through its involvement in lymphovascular space invasion, ER has been implicated as a promoter of metastasis in HGSOC.

Despite diagnostic, clinical, and therapeutic advances, prostate cancer (PC) remains a major urological disease with significant morbidity. In Western society, this cancer is the most common malignancy and the second leading cause of cancer death among men. Significant progress has been made in PC diagnostic tools and therapeutic approaches. They are based on the use of novel radiotracers for imaging and patient tracking.

Furthermore, new potent androgen synthesis inhibitors or Androgen Receptor (AR) antagonists, such as abiraterone and enzalutamide, have improved PC patient survival. Despite this, PC frequently develops resistance to these treatments and progresses to the Castration-Resistant PC (CRPC) stage, which is distinguished by adaptation to the castrate condition and invasiveness. Metastatic spread of CRPC cells is still the leading cause of PC-related death, and novel compounds such as taxanes, poly (adenosine diphosphate-ribose) polymerase inhibitors, and Programmed Cell Death 1 (PD-1) inhibitors are in clinical trials. A precise understanding of the molecular basis for cell spreading and drug-resistance patterns in PC, on the other hand, remains a challenge. Lysine methylation of histone H3 lysine 4 (H3K4me) and 36 (H3K36me), which is primarily associated with transcriptional activationlysine The dynamic and selective methylation of lysine on histones H3 and H4 adds an important layer of epigenetic regulation to gene transcription. Unlike methylation of histone H3 lysine 9 (H3K9me) and 27 (H3K27me) is enriched Although it is commonly acknowledged that EMT plays a major role in PC cell spreading, stemness, and drug resistance, there is still debate about the role of steroids and sex steroid receptors (SRs) in this process. We present here the most recent findings on the molecular events underlying the EMT controlled by the Estrogen/Estrogen Receptors (ERs) axis in various prostate tissue and PC models. The implications of these findings for clinical management of PC patients will be discussed as well.