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Cellular and Molecular Biology

“Stromal cells in prostate cancer pathobiology: friends or foes?”

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The genomic, epigenetic and metabolic determinants of prostate cancer pathobiology have been extensively studied in epithelial cancer cells. However, malignant cells constantly interact with the surrounding environment—the so-called tumour microenvironment (TME)—which may influence tumour cells to proliferate and invade or to starve and die. In that regard, stromal cells—including fibroblasts, smooth muscle cells and vasculature-associated cells—constitute an essential fraction of the prostate cancer TME. However, they have been largely overlooked compared to other cell types (i.e. immune cells). Indeed, their importance in prostate physiology starts at organogenesis, as the soon-to-be prostate stroma determines embryonal epithelial cells to commit toward prostatic differentiation. Later in life, the appearance of a reactive stroma is linked to the malignant transformation of epithelial cells and cancer progression. In this Review, we discuss the main mesenchymal cell populations of the prostate stroma, highlighting their dynamic role in the transition of the healthy prostate epithelium to cancer. A thorough understanding of those populations, their phenotypes and their transcriptional programs may improve our understanding of prostate cancer pathobiology and may help to exploit prostate stroma as a biomarker of patient stratification and as a therapeutic target.

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INTRODUCTION

Prostate adenocarcinoma (PCa) arises from the malignant transformation of the epithelial cells constituting the prostatic acini [1, 2]. Besides all the genomic, epigenomic and metabolic alterations occurring in neoplastic epithelial cells, a crucial role in PCa pathobiology is played by its ecosystem—known as the tumour microenvironment (TME). Prostate stroma is composed of a multitude of different cell populations—including fibroblasts, smooth muscle cells (SMCs), endothelium, immune cells and nerves—that actively contribute to its homeostasis in physiologic and pathologic conditions, including PCa [2–4]. Indeed, stromal populations of prostate TME are known to be partners in crime with tumour cells, and they can represent a novel actionable target in the fight against cancer [5, 6].

It is not surprising that the stroma is central in PCa pathobiology, as it has played a pivotal role since its embryological development [7, 8]. Indeed, the prostate derives from the urogenital sinus (UGS), which is composed of an epithelial layer derived from the endoderm, surrounded by the embryonal prostatic mesenchyme derived from the mesoderm. Classical experiments using tissue recombination and grafting showed that the expression of the androgen receptor (AR) and paracrine factors (e.g. fibroblast growth factor (FGF) 7 and 10) in the mesenchymal UGS is necessary for the differentiation of embryonal epithelial cells into prostatic cells and branching ducts [9–11].

Through mechanisms similar to the ones in action during organogenesis, the activation of the mesenchymal stroma represents one of the first steps in prostate carcinogenesis [12, 13]. In this Review, we summarise the current knowledge about the stromal populations in the PCa microenvironment, describing the molecular pathways beyond stromal activation in PCa tumorigenesis and highlighting potential therapeutic strategies targeting the stroma.

FIBROBLASTS

Fibroblasts represent the most studied and characterised cell population of prostate mesenchyme. In physiologic conditions, fibroblasts supervise the homeostasis of extracellular matrix (ECM) and connective tissue, and they are also involved in tissue repair processes [14]. Embryologically, most prostate fibroblasts derive from the mesodermal mesenchyme, with a smaller proportion deriving from the neural crest [3]. Fibroblasts are generally recognised by their morphology and localisation within the tissue, besides the lack of epithelial, endothelial and hematopoietic markers. Indeed, some markers may be used for their identification, including fibroblast activation protein (FAP), platelet-derived growth factor receptor- α (PDGFR α), α -smooth muscle actin (α SMA) and vimentin [15, 16]. However, such markers are not exclusively expressed by bona fide fibroblasts. At the same time, some of

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