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The Fer kinase promotes prostate cancer progression by integrating cross-talks between AR and STAT3 in the IL-6 signaling pathway

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Prostate cancer (PCa) ranks first among malignancies in incidence in North American males and second as a cause of mortality by cancer. Beside age, etiologic and progression factors are still not fully identified. A turning point in disease progression is when patients who failed surgery or radiation therapies develop castration-resistance (CRCP) while receiving androgen-deprivation therapy (ADT). Underlying mechanisms are not elucidated. Accumulating evidence, including from our team, suggests that tumor cell heterogeneity contributes to PCa progression. In fact, the androgen-dependency/sensitivity of tumor cells expressing the androgen receptor (AR) appears to gradually shift into insensitivity even if AR remains a functionally active transcription factor. Concomitant to this adaptive process, subsets of AR negative(-) and androgen-independent tumor cells of the neuroendocrine (NE) and stem-like phenotypes progressively emerge. We and others have obtained evidence that irrespectively of AR, PCa cells grow and movein response to diverse growth factors (GFs, such as EGF and IGF-1), cytokines (IL-6), and NE-products present in their microenvironment. A novelfeature of the IL-6 drivensignaling pathway in PCa cells is the intervention of the non-receptor Fer tyrosine kinase (TK) which we showed essential for STAT3 activation, nuclear translocation and transcriptional activity of IL6-dependent genes.Of significance, Fer also intervenes in the AR axis and allows IL-6 to control the transcriptional activity of genes that are normally driven by androgens. Moreover, Fer contributes but to a lesser extent to growth elicited by EGF, IGF-1, and androgens. In the latter case, Fer forms similar complexes with AR but STAT3 is not involved. Altogether Fer appears to promote PCa progression by integrating cross-talks involving STAT3 and AR as its substrates and binding partners up to the PCa cell nuclei.

Biography

Simone Chevalier completed her PhD in Biochemistry at "l' Université de Montréal", Montréal, Canada, and did her Post-doctorate training in Biochemistry and Endocrinology at the University of British Columbia (Vancouver, B.C.) and Maisonneuve-Rosemont Research Centre (Montréal), respectively. She is Associate Professor in Surgery (Urology) at McGill University and holds affiliations in Medicine and Oncology. She is the McGill Urology Director of Research and Director of the PROCURE Québec Prostate Cancer Biobank. Her career has been devoted to prostatic proliferative diseases. She published more than 90 papers in reputed journals and 350 presentations at national and international meetings.

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