Membrane Microvesicles as Actors in the Establishment of a Favorable Prostatic Tumoral Niche: A Role for Activated Fibroblasts and CX3CL1-CX3CR1 Axis

Titre	Membrane Microvesicles as Actors in the Establishment of a Favorable Prostatic Tumoral Niche: A Role for Activated Fibroblasts and CX3CL1-CX3CR1 Axis
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Auteur	Castellana, Donatello [1], Zobairi, Fatiha [2], Martinez, Maria Carmen [3], Panaro, Maria Antonietta [4], Mitolo, Vincenzo [5], Freyssinet, Jean-Marie [6], Kunzelmann, Corinne [7]
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Mots-clés	CX3CR1 [8], fibroblasts [9], microenvironment [10], microvesicles [11], tumoral [12]
Résumé en anglais	Tumor microenvironment is enriched in plasma membrane microvesicles (MV) shed from all cell types that constitute the tumor mass, reflecting the antigenic profile of the cells they originate from. Fibroblasts and tumor cells mutually communicate within tumor microenvironment. Recent evidences suggest that tumor-derived MVs (TMV) exert a broad array of biological functions in cell-to-cell communication. To elucidate their role in cancer-to-fibroblast cell communication, TMV obtained from two prostate carcinoma cell lines with high and weak metastatic potential (PC3 and LnCaP, respectively) have been characterized. TMV exhibit matrix metalloproteinases (MMP) and extracellular MMP inducer at their surface, suggesting a role in extracellular matrix degradation. Moreover, TMV not only induce the activation of fibroblasts assessed through extracellular signal-regulated kinase 1/2 phosphorylation and MMP-9 up-regulation, increase motility and resistance to apoptosis but also promote MV shedding from activated fibroblasts able in turn to increase migration and invasion of highly metastatic PC3 cells but not LnCaP cells. PC3 cell chemotaxis seems, at least partially, dependent on membrane-bound CX3CL1/fractalkine ligand for chemokine receptor CX3CR1. The present results highlight a mechanism of mutual communication attributable not only to soluble factors but also to determinants harbored by MV, possibly contributing to the constitution of a favorable niche for cancer development. [Cancer Res 2009;69(3):785–93]

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