

IGFBP-2 Stimulates the Metastatic Phenotype of Prostate Cancer Cells in vitro

Adam Aguiar, Dave DeGraff, Christine Maguire and Robert A. Sikes. University of Delaware

Background and Objective: The progression of prostate cancer (PCa) requires alterations in the expression/activity of multiple signaling molecules. Over-expression of IGFBP-2 is correlated with Pca advancement to androgen independence. Herein we investigate mechanisms by which IGFBP-2 functions in the development and progression of PCa.

Methods: The IGFBP-2 expression levels were first measured in the human LNCaP PCa model by western blotting and densitometry. IGFBP-2 function was examined by migration and invasion assays. Effects of IGFBP-2 on the actin cytoskeleton were displayed by rhodamine phalloidin staining.

Results: IGFBP-2 levels increase in the more-metastatic, AI C42-B4 cell line when compared to the poorly-metastatic, AS LNCaP cell line. The androgen-induced reduction of IGFBP-2 is attenuated in the metastatic, AI C4-2 line. Recombinant IGFBP-2 addition, at 2µg/mL, stimulates wound-healing of LNCaP cell line, as well as the invasion through growth factor-reduced matrigel. The phalloidin staining indicates that IGFBP-2 induces the formation of filapodia at 2hrs, and continuing 24hrs post treatment.

Conclusions and Discussion These data suggest that IGFBP-2 promotes a more metastatic phenotype of LNCaP cells *in vitro* and implies its further importance in the progression of PCa *in vivo*. Therefore this study provides insight into the potential clinical relevance of IGFBP-2 as a molecular target for future therapeutic drugs, and/or as a biomarker for the screening of advanced disease.

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