Alternative inclusion of fibroblast growth factor receptor 2 exon IIIc in Dunning prostate tumors reveals unexpected epithelial mesenchymal plasticity. [1]

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Abstract	In epithelial cells, alternative splicing of fibroblast growth factor receptor 2 (FGFR2) transcripts leads to the expression of the FGFR2(IIIb) isoform, whereas in mesenchymal cells, the same process results in the synthesis of FGFR2(IIIc). Expression of the FGFR2(IIIc) isoform during prostate tumor progression suggests a disruption of the epithelial character of these tumors. To visualize the use of FGFR2 exon IIIc in prostate AT3 tumors in syngeneic rats, we constructed minigene constructs that report on alternative splicing. Imaging these alternative splicing decisions revealed unexpected mesenchymal-epithelial transitions in these primary tumors. These transitions were observed more frequently where tumor cells were in contact with stroma. Indeed, these transitions were frequently observed among lung micrometastases in the organ parenchyma and immediately adjacent to blood vessels. Our data suggest an unforeseen relationship between epithelial mesenchymal plasticity and malignant fitness.
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