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The $a_v b_6$ integrin regulates androgen receptor activity and prostate cancer progression

The intracellular signals governing prostate cancer (PrCa) progression in humans are not entirely understood. Androgen receptor (AR) signaling fuels PrCa, and is a major therapeutic target in this disease. However, therapeutic androgen ablation in the clinic is often unsuccessful given cancer progression to a castrate-resistant phenotype. Here, we show that the avb6 integrin, a receptor for the extracellular matrix, is significantly increased in preneoplastic lesions and prostatic adenocarcinoma, but not normal prostatic epithelium, and is required for tumor growth *in vivo* of non-castrated and castrated mice. We uncovered a novel pathway that couples signaling at the cell surface transduced by the avb6 integrin to activation of JNK1, AR-dependent gene expression and upregulation of survivin which, in turn, promotes anchorage-independent growth. This signaling network occurs preferentially in the transformed prostatic epithelium of non-castrated as well as castrated mice, triggers accelerated cell proliferation and is required for PrCa growth *in vivo*. Overall our data show that avb6 integrin pathway cross-talks with AR signaling and contributes to a castrate-resistant and aggressive PrCa phenotype.

Biography

Huimin Lu has obtained his Ph.D degree in pharmacology at Sun Yat-sen University, Guangdong, China. He is now a senior postdoctoral research fellow in Dr. Languino's laboratory at Thomas Jefferson University, Philadelphia, Pennsylvania, USA.

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