Myoblasts inhibit prostate cancer growth by paracrine secretion of tumor necrosis factor-α

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PURPOSE: Myoblasts can form muscle fibers after transplantation. Therefore, they are envisioned as a treatment for urinary incontinence after radical prostatectomy. However, to our knowledge the safety of this treatment and the interaction of myoblasts with any remaining neighboring cancer are unknown. We investigated the interactions between myoblasts and prostate carcinoma cells in vitro and in vivo. MATERIALS AND METHODS: Myoblasts isolated from the rectus abdominis were used in a series of co-culture experiments with prostate cancer cells and subcutaneously co-injected in vivo. Cell proliferation, cell cycle arrest and apoptosis of cancer in co-culture with myoblasts were assessed. Tumor volume and metastasis formation were evaluated in a mouse model. Tissue specific markers were assessed by immunohistochemistry, fluorescence activated cell sorting analysis, Western blot and real-time quantitative polymerase chain reaction. RESULTS: Myoblasts in proximity to tumor provided paracrine tumor necrosis factor-α to their microenvironment, decreasing the tumor growth of all prostate cancer cell lines examined. Co-culture experiments revealed induction of cell cycle arrest, tumor death by apoptosis and increased myoblast differentiation. This effect was largely blocked by tumor necrosis factor-α inhibition. The same outcome was noted in a mouse model, in which co-injected human myoblasts also inhibited the tumor growth and metastasis formation of all prostate cancer cell lines evaluated. CONCLUSIONS: Myoblasts restrict prostate cancer growth and limit metastasis formation by paracrine tumor necrosis factor-α secretion in vitro and in vivo.
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