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# THE SENESCENT CELLS OF TUMOR MICROENVIRONMENT MAY INHIBIT TUMOR GROWTH IN A LEWIS LUNG CARCINOMA MOUSE MODEL

**Background/Aim.** Microenvironment plays an important role in tumor development. Thus, senescent stromal cells can function as both immune-evasion mediators and targetable immunological substrates. The aim of the present work was to demonstrate ability of senescent fibroblasts to inhibit the tumor growth in experimental animals. **Methods.** Lysates of the in vitro-induced senescent fibroblasts were used to influence the tumor growth in the Lewis lung carcinoma (LLC) mouse model. Adoptive transfer of lymphocytes from treated mice into naïve mice was performed in a prophylactic tumor challenge model. **Results.** The evidence supports a model in which senescent cells of tumor microenvironment promote immune resistance through SASP-driven inflammatory signaling, inhibition of T and NK cell activity, dendritic cell dysfunction, suppressive myeloid recruitment, and stromal shielding of malignant cells. Immune targeting of these cells may convert the tumor microenvironment from an immune-restrictive to an immune-permissive state. The preclinical data generated by pulsing dendritic cells with lysate from senescent fibroblasts provide proof of concept. Introduction of such cells in experimental animals induced recognition of senescent, but not control, stromal cells. Moreover, T-cell proliferation was stimulated and IFN- $\gamma$  and Granzyme B responses were increased. In the Lewis lung carcinoma model, prophylactic and therapeutic vaccination reduced the volume of primary tumor and a number of lung metastasis. Adoptive transfer experiments implicated CD8<sup>+</sup> T cells as key mediators of antitumor protection. Combination with anti-PD-L1 or anti-CTLA-4 antibodies enhanced tumor inhibition, supporting complementarity with checkpoint blockade. **Conclusions.** Tumor-associated senescence can be conceptualized as an actionable immune-evasion program rather than only a marker of tissue aging or treatment damage. Polyvalent senolytic dendritic cell immunotherapy offers a rational strategy to remodel the senescent tumor microenvironment and potentiate antitumor immunity. **Grants/Funding.** No specific funding was received. The author previously collaborated with Immorta Bio, Inc.; no confidential or unpublished company information is disclosed.

**Keywords:** cellular senescence, tumor microenvironment, senolytic vaccination, dendritic cells, checkpoint blockade.

## REFERENCES

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