

## Abstract

Persistence of a tumor is dependent on evasion of the host anti-tumor immune response. One effective means of immunoevasion is cancer cell driven expansion of a heterogeneous myeloid population known as myeloid derived suppressor cells (MDSC). MDSC are characterized by co-expression of the Gr-1 and CD11b antigens and the ability to inhibit the activation and proliferation of tumor specific CD8<sup>+</sup> cytotoxic T lymphocytes. When oncogenic ras expression was induced in the normal mouse epidermis using a conditional transgenic model we observed a significant increase in circulating and skin infiltrating neutrophils. Surprisingly, depletion of circulating mature neutrophils with the RB6-8C5 antibody caused derangement of myeloid production in the peripheral blood and acceleration of epidermal hyperplasia, proliferation and subsequent tumor formation. This project will test the hypothesis that accelerated tumor development following neutrophil depletion is due to increased numbers and skin infiltration of MDSC, that are normally regulated by neutrophils, by functionally and molecularly characterizing the presumptive non-neutrophil myeloid suppressor cell population.