Targeting Estrogen Signaling in Tumor Associated Myeloid Cells to Enhance Immunotherapy Responses.

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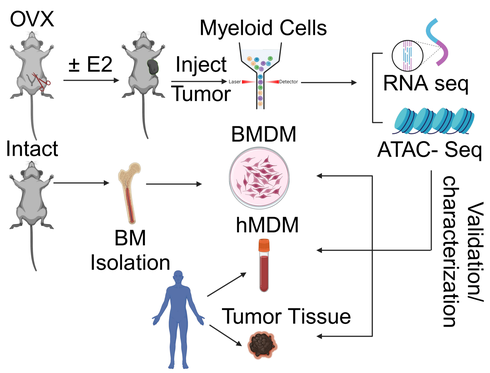
Building Interdisciplinary Research Careers in Women's Health

BACKGROUND

- Immune checkpoint inhibitors have significantly increased survival in patients with multiple cancer types.
- Gender based differences are evident in patients' response to ICB therapy
- Females respond less to ICB than do men.
- Using publicly available data we have demonstrated that tumor associated macrophages promote resistance to ICB therapy.
- Of relevance is our observation that female steroid hormone 17β- Estradiol (E2) promotes macrophage polarization towards an immune-suppressive state within tumor microenvironment.

METHODS



RESULTS

Figure 1: E2 suppress induction of type I IFN signaling in tumor associated myeloid cells via enhancing efferocytosis

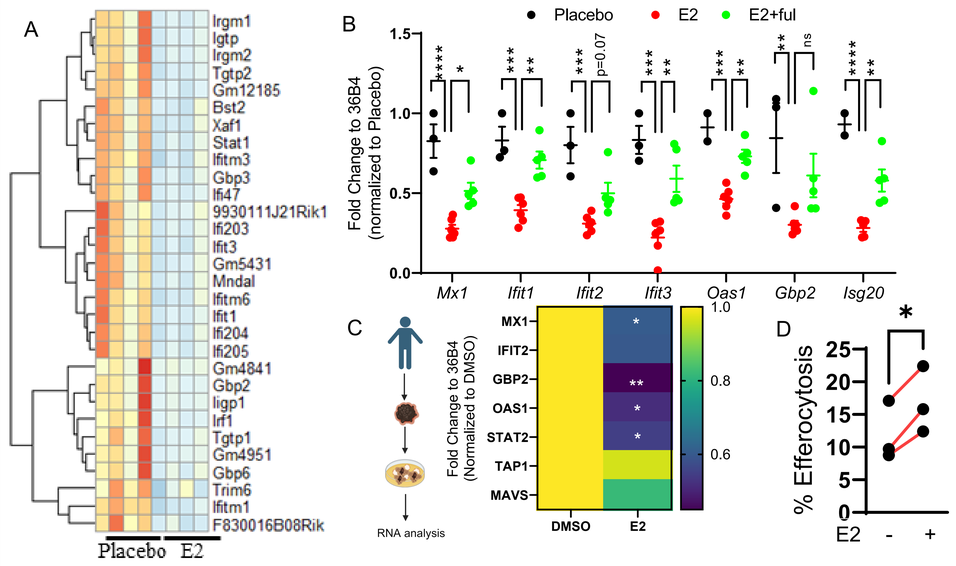
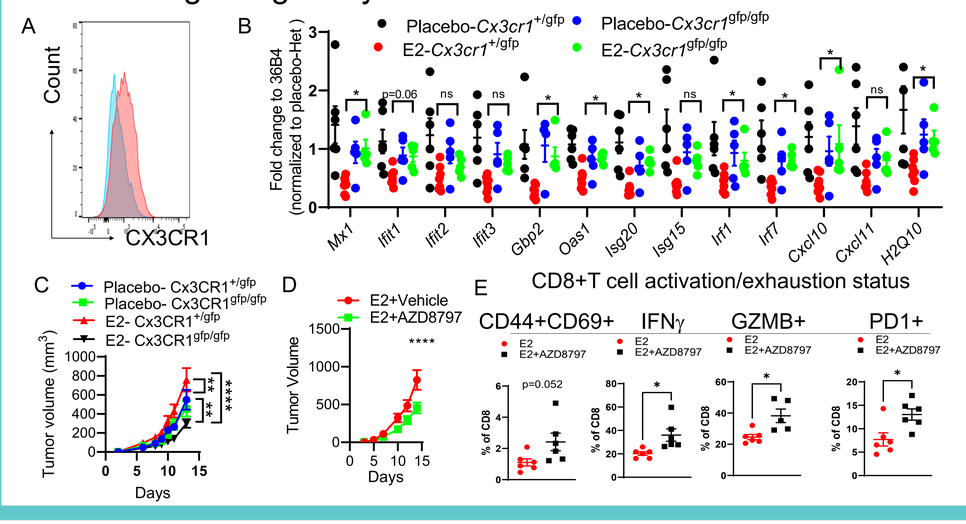
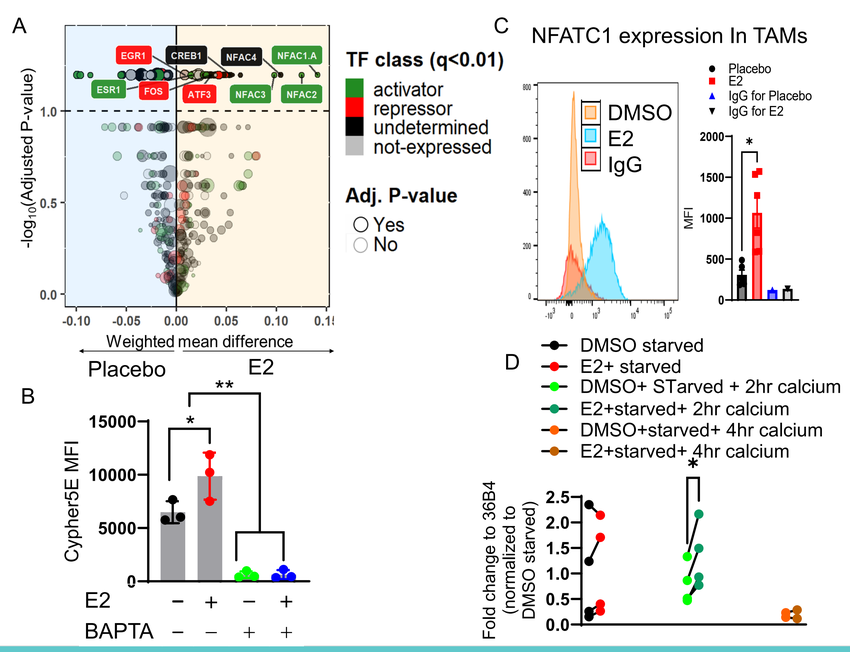


Figure 2: CX3CR1 promotes E2-mediated suppression of Type I interferon signaling in myeloid cells

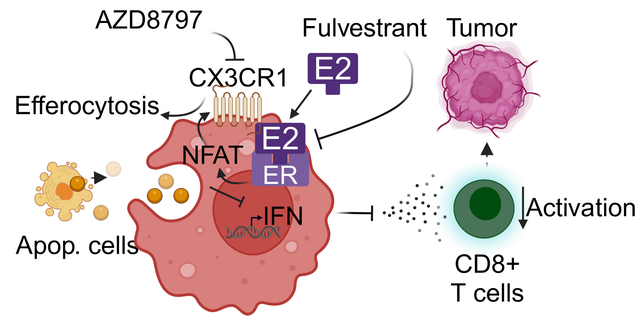


RESULTS

Figure 3. E2-mediated NFAT activation regulates CX3CR1 signaling in tumor associated myeloid cells



CONCLUSION



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