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Summary

CANCER-ASSOCIATED FIBROBLASTS REGULATE THE FORMATION OF MACROPHAGE EXTRACELLULAR TRAPS VIA CCL11/CCR5 AXIS TO RESIST IMMUNOTHERAPY IN PANCREATIC DUCTAL ADENOCARCINOMA

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Cancer-associated fibroblasts promote macrophage extracellular trap formation via CCL11/CCR5 signaling in pancreatic cancer, and the CCR5 inhibitor maraviroc suppresses METs formation and enhances immunotherapy efficacy in preclinical models.

KEY POINTS

- High levels of macrophage extracellular traps (METs) in pancreatic ductal adenocarcinoma tissue were significantly associated with poor patient survival, advanced tumor grade and stage, fewer infiltrating CD8+ T-cells, increased CD86+ macrophages, and increased α-SMA+ cancer-associated fibroblasts in 46 surgical samples.
- CCL11 secreted by cancer-associated fibroblasts bound to CCR5 on macrophages and promoted PAD2 expression, facilitating METs formation.
- Maraviroc, a CCR5 inhibitor, effectively suppressed METs formation and increased PD-L1 expression and CD8+ T cell infiltration in pancreatic cancer tissue.
- In mouse orthotopic transplantation models using KPC-derived organoids, maraviroc effectively enhanced the anti-tumor effects of immunotherapy.
- The findings suggest that antagonizing CCR5 to inhibit METs formation reshaped the immunosuppressive microenvironment of pancreatic cancer and enhanced sensitivity to immunotherapy.

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