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TNFRSF1B-DRIVEN MYELOID PLASTICITY CONSTITUTES A CLINICALLY TRACTABLE IMMUNOSUPPRESSIVE AXIS ACROSS HUMAN CANCERS

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Abstract

The immunosuppressive tumor environment (TME) is a major barrier to effective immunotherapy in ovarian cancer (OC), yet the regulatory drivers remain incompletely defined. Here, we applied a multi-omens workflow integrating single-cell RNA sequencing (scRNA-seq), bulk transcriptomics, immune decovolution, Mendelian randomization (MR), expression quantitative trait loci (eQTL) analysis, and functional

validation to dissect the heterogeneity and regulatory circuits of tumor-associated macrophages (TAMs) in OC. We identified a selective expansion of intermediate monocytes (IMo) within the OC TME, which promoted immunosuppressive niche formation. MR analysis identified TNFRSF1B (tumor necrosis factor receptor superfamily member 1B, encoding TNFR2) as a causal risk gene, enriched in immunosuppressive myeloid cells across single-cell, spatial, and pan-cancer datasets. High TNFRSF1B expression was associated with M2-like polarization, elevated immune checkpoint signatures, and poor prognosis. Functional knockdown of TNFRSF1B in vitro reduced M2 polarization and cytokine secretion, while in vivo antibody blockade suppressed tumor growth and reversed CD8+ T cell exhaustion. Mechanistically, TNFRSF1B+ TAMs secreted SPP1, which engaged CD44 on CD8+ T cells and induced TIM3-mediated exhaustion. These findings nominate TNFRSF1B as both a prognostic biomarker and a promising immunotherapeutic target in OC and potentially other solid tumors.

Keywords:

Ovarian Cancer, Tumor Microenvironment, TNFRSF1B, scRNA-seq, Mendelian Randomization, TAMs