### CANCER IMMUNOLOGY RESEARCH

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Lung cancer–derived fibroblasts produce CCL2, which recruits suppressive CCR2\(^+\) myeloid cells to the tumor microenvironment, where they suppress CD8\(^+\) T-cell function. Use of various inhibitors shows that this suppression can be reversed, highlighting possible druggable targets.
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ABOUT THE COVER

Cancer-associated fibroblasts (CAFs) are a major component of the tumor stroma and can promote tumorigenesis and treatment resistance. However, the mechanisms behind how CAFs do this are not fully understood in lung squamous cell carcinoma (LSCC). By evaluating primary human LSCCs, Xiang et al. demonstrate interactions between CAFs and myeloid cells in the tumor microenvironment (TME). Lung CAFs produce CCL2, which recruits CCR2+ myeloid cells to the TME and promotes their polarization into a myeloid-derived suppressor cell (MDSC) phenotype. This results in suppression of CD8+ T-cell responses. Reduction of reactive oxygen species in CAF-induced MDSCs reverses the suppression of CD8+ T-cell proliferation and function, highlighting possible druggable targets to boost antitumor responses in LSCC. Read more in this issue on page 436. Original image from Fig. 1D. Artwork by Lewis Long.