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Julie H. Ko, Kyle E. Lambert, Debadrita Bhattacharya, Myung Chang Lee, Caterina I. Colón, Haley Hauser, and Julien Sage

Small cell lung cancer cells overcome deficiency of the prometastatic oncogene NFIB to gain metastatic potential through various molecular mechanisms, which may represent targets to block progression of this fatal cancer type.

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Spatial analysis identifies IFN-induced MHC-I^{hi}Gal9⁺ CAFs that form a trap for CD8⁺ T cells, providing insights into the complex networks in the tumor microenvironment that regulate T-cell infiltration and function.

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Curteisha Jacobs, Sakhi Shah, Wen-Cheng Lu, Haimanti Ray, John Wang, Natasha Hockaden, George Sandusky, Kenneth P. Nephew, Xin Lu, Sha Cao, and Richard L. Carpenter

The stress-responsive transcription factor HSF1 reduces CD8⁺ T-cell infiltration in breast tumors to prevent immune-mediated killing, indicating that cellular stress responses affect tumor-immune interactions and that targeting HSF1 could improve immunotherapies.

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Upregulation of bone-remodeling factors S100A4 and LGALS3 mediated by a circMMP2(6,7)/ β -catenin/PRMT5 complex generates a niche that supports breast cancer bone metastasis, identifying PRMT5 as a promising target for treating metastasis.

ABOUT THE COVER

The status of intratumoral CD8⁺ T cells is one of the determinants of immunotherapy efficiency. Cancer-associated fibroblasts interact with CD8⁺ T cells to impact T-cell distribution and function. The cover image depicts a subset of cancer-associated fibroblasts expressing high levels of CXCLs and MHC class I molecules that trap CD8⁺ T cells and induce dysfunction of pre-effector CD8⁺ T cells via galectin-9. For details, see article by Li and colleagues on p. 258.

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