**TABLE OF CONTENTS**

**WHAT WE'RE READING**

421  A Sampling of Highlights from the Literature

**CANCER IMMUNOLOGY AT THE CROSSROADS**

422  Immune Escape during Breast Tumor Progression
Carlos R. Gil Del Alcazar, Maša Alecković, and Kornelia Polyak

**PRIORITY BRIEF**

428  Inhibition of the SRC Kinase HCK Impairs STAT3-Dependent Gastric Tumor Growth in Mice

**RESEARCH ARTICLES**

436  Cancer-Associated Fibroblasts Promote Immunosuppression by Inducing ROS-Generating Monocytic MDSCs in Lung Squamous Cell Carcinoma

451  Interferon-Induced IDO1 Mediates Radiation Resistance and Is a Therapeutic Target in Colorectal Cancer
Baosheng Chen, David M. Alvarado, Micah Iticovici, Nathan S. Kau, Haeseong Park, Parag J. Parikh, Dinesh Thotala, and Matthew A. Ciorba

465  CD73 Blockade Promotes Dendritic Cell Infiltration of Irradiated Tumors and Tumor Rejection
Erik Wennerberg, Sheila Spada, Nils-Petter Rudqvist, Claire Lhuillier, Sylvia Gruber, Qiuying Chen, Fengli Zhang, Xi K. Zhou, Steven S. Gross, Silvia C. Formenti, and Sandra Demaria

479  Fatty Acid Oxidation Controls CD8+ Tissue-Resident Memory T-cell Survival in Gastric Adenocarcinoma
Run Lin, Hui Zhang, Yujie Yuan, Qiong He, Jianwen Zhou, Shuhua Li, Yu Sun, Daniel Y. Li, Hai-Bo Qiu, Wei Wang, Zhehong Zhuang, Bin Chen, Yonghui Huang, Chuwei Liu, Yingzhang, Shirong Cai, Zufu Ke, and Weiling He

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.
Metabolome of Pancreatic Juice Delineates Distinct Clinical Profiles of Pancreatic Cancer and Reveals a Link between Glucose Metabolism and PD-1 Cells
Nina Cortese, Giovanni Capretti, Marialuisa Barbagallo, Alessandra Rigamonti, Panteleimon G. Takis, Giovanni F. Castino, Debora Vignali, Gennaro Nappo, Greta Donisi, Marco Erreni, Roberta Avigni, Paolo Monti, Alessandro Zerbi, Paola Allavena, Alberto Mantovani, and Federica Marchesi

Metabolomics performed on pancreatic juice from pancreatic ductal adenocarcinoma patients identifies metabolic variables correlating with outcome and immune infiltration of tumors. Obtaining a metabolic profile could aid in the stratification of patients for more tailored immunotherapies.

Inhibition of SHP-1 Expands the Repertoire of Antitumor T Cells Available to Respond to Immune Checkpoint Blockade
Jeremy P. Snook, Ashleigh J. Soedel, H. Atakan Ekiz, Ryan M. O'Connell, and Matthew A. Williams

Checkpoint blockade enhances high-affinity T-cell responses to melanoma, but some patients do not benefit. Targeting SHP-1 expands the repertoire of T cells available to respond to treatment and induces antitumor activity from low-affinity T cells.

Improved Antitumor Efficacy of Chimeric Antigen Receptor T Cells that Secrete Single-Domain Antibody Fragments
Yushu Joy Xie, Michael Dougan, Jessica R. Ingram, Novalia Pishesha, Tao Fang, Noor Momin, and Hidde L. Ploegh

CAR T cells that target the tumor microenvironment are designed to secrete immune-modulating single-domain antibodies that can engage the innate immune system, cause epitope spreading, and evade checkpoint immunosuppression by the tumor.

γδ T-cell Receptors Derived from Breast Cancer-Infiltrating T Lymphocytes Mediate Antitumor Reactivity
Anke Janssen, Jose Villacorta Hidalgo, Dennis X. Beringer, Sanne van Dooremalen, Debora Vignali, Marialuisa Barbagallo, Alessandra Rigamonti, Panteleimon G. Takis, Giovanni F. Castino, Debora Vignali, Gennaro Nappo, Greta Donisi, Marco Erreni, Roberta Avigni, Paolo Monti, Alessandro Zerbi, Paola Allavena, Alberto Mantovani, and Federica Marchesi

Proinflammatory γδ T cells infiltrate triple-negative breast cancers and are positioned in close proximity to tumor cells. T cells engineered to express paired TCRγ/TCRδ chains are tumor reactive against an array of tumor types.

AC icon indicates AuthorChoice
For more information please visit www.aacrjournals.org
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.
### Updated version
Access the most recent version of this article at:
http://cancerimmunolres.aacrjournals.org/content/8/4

<table>
<thead>
<tr>
<th>E-mail alerts</th>
<th>Sign up to receive free email-alerts related to this article or journal.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reprints and Subscriptions</td>
<td>To order reprints of this article or to subscribe to the journal, contact the AACR Publications Department at <a href="mailto:pubs@aacr.org">pubs@aacr.org</a>.</td>
</tr>
<tr>
<td>Permissions</td>
<td>To request permission to re-use all or part of this article, use this link <a href="http://cancerimmunolres.aacrjournals.org/content/8/4">http://cancerimmunolres.aacrjournals.org/content/8/4</a>. Click on &quot;Request Permissions&quot; which will take you to the Copyright Clearance Center's (CCC) Rightslink site.</td>
</tr>
</tbody>
</table>