adoptive T-cell transfer, but an inflammation-induced melanoma cell dedifferentiation resulted in loss of the MART1 tumor antigen and tumor relapse.

Antitumor Activity Associated with Prolonged Persistence of Adoptively Transferred NY-ESO-1c259 T Cells in Synovial Sarcoma


Précis: In a prospective clinical study of 12 patients with synovial sarcoma, adoptive transfer of autologous NY-ESO-1c259 cells resulted in a 50% objective response rate and long-term persistence of a clonally diverse, fully functional NY-ESO-1c259 cells.

See commentary, p. 914

Clinical and Biological Correlates of Neurotoxicity Associated with CAR T-cell Therapy in Patients with B-cell Acute Lymphoblastic Leukemia


Précis: Elevation of proinflammatory cytokines and disruption of blood–CSF barrier are associated with CAR T cell–driven neurotoxicity in patients with B-ALL who were treated with CD19 CAR T cells.

Car T-cell Integration of Multiple Input Signals Allows for Precise Targeting of Cancer

D. Achkova and M. Pule

See article, p. 972

MACROD2, an Original Cause of CIN?

N. Jin and M.E. Burkard

See article, p. 988

CARs versus BiTEs: A Comparison between T Cell–Redirection Strategies for Cancer Treatment

C.Y. Slaney, P. Wang, P.K. Darcy, and M.H. Kershaw

See article, p. 924

Immunotherapy Resistance by Inflammation-Induced Dedifferentiation


Précis: A patient with metastatic melanoma initially responded after
MACROD2 Haploinsufficiency Impairs Catalytic Activity of PARP1 and Promotes Chromosome Instability and Growth of Intestinal Tumors


Précis: Deletions of the mono-ADP-ribosylhydrolase gene MACROD2 enhance intestinal tumorigenesis in a haploinsufficient manner by suppressing PARP1 activity, leading to increased sensitivity to DNA damage and driving chromosome instability.

See commentary, p. 921

Adipocyte-Derived Lipids Mediate Melanoma Progression via FATP Proteins


Précis: Melanoma tumorigenesis is accelerated by the uptake of long-chain fatty acids from adipocytes via the FATP1 lipid transporter, and this adipocyte–melanoma cell cross-talk may be disrupted by FATP inhibitors.

YAP Is Essential for Treg-Mediated Suppression of Antitumor Immunity


Précis: YAP expression in Treg cells upregulates the activin receptor to enhance SMAD/TGFβ signaling, promote Treg differentiation, and suppress antitumor immunity, suggesting potential immunotherapeutic targets.

Corrections

Correction: Identifying and Targeting Sporadic Oncogenic Genetic Aberrations in Mouse Models of Triple-Negative Breast Cancer

Correction: Linking Tumor Mutations to Drug Responses via a Quantitative Chemical–Genetic Interaction Map

ON THE COVER

Sakthianandeswaren, Parsons, Mouradov, and colleagues found that the MACROD2 locus, which encodes a mono-ADP-ribosylhydrolase implicated in the regulation of PARP1, undergoes recurrent focal deletions in a subset of human colorectal cancers. Consistent with a tumor-suppressive role, loss of MACROD2 increased the growth of intestinal tumors in mice in a haploinsufficient manner. This effect was mediated at least in part via repression of PARP1 activity in response to MACROD2 deletion, leading to impaired DNA repair and enhanced sensitivity to genotoxic stress. MACROD2 loss recapitulated the phenotype of PARP1 inhibitor treatment, including mitotic chromosome missegregation and increased structural and numerical chromosome abnormalities, suggesting that MACROD2 loss promotes chromosome instability and aneuploidy. These results define MACROD2 as a haploinsufficient tumor suppressor essential for maintaining genome integrity in human colorectal cancer. For details, please see the article by Sakthianandeswaren, Parsons, Mouradov, and colleagues on page 988.
# CANCER DISCOVERY

## 8 (8)

*Cancer Discov* 2018;8:OF2-1045.

<table>
<thead>
<tr>
<th>Updated version</th>
<th>Access the most recent version of this article at: <a href="http://cancerdiscovery.aacrjournals.org/content/8/8">http://cancerdiscovery.aacrjournals.org/content/8/8</a></th>
</tr>
</thead>
<tbody>
<tr>
<td>E-mail alerts</td>
<td>Sign up to receive free email-alerts related to this article or journal.</td>
</tr>
<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://cancerdiscovery.aacrjournals.org/content/8/8">http://cancerdiscovery.aacrjournals.org/content/8/8</a>. Click on &quot;Request Permissions&quot; which will take you to the Copyright Clearance Center's (CCC) Rightslink site.</td>
</tr>
</tbody>
</table>