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  See related article by Murai and colleagues, Cancer Res 2012;72:5588–99

## CANCER RESEARCH HIGHLIGHTS
- **5608** mTOR-Dependent ARID1A Degradation: A New Twist in the Genetic-Epigenetic Interplay Driving Hepatocellular Carcinoma  
  David R. Pease and Martin E. Fernandez-Zapico  
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- **5611** Collagen Linearization within Tumors  
  Craig E. Barcus and Gregory D. Longmore  
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## GENOME AND EPIGENOME
- **5613** Therapeutic Potential of Chemically Modified, Synthetic, Triplex Peptide Nucleic Acid-Based Oncomir Inhibitors for Cancer Therapy  
  Karishma Dhuri, Ravinder Reddy Gaddam, Ajit Vikram, Frank J. Slack, and Raman Bhal  
  This study demonstrates the utility of novel oncomir inhibitors as cancer therapeutics, providing a new approach for targeting miRNAs and other noncoding RNAs.
- **5625** Diverse Oncogenic Fusions and Distinct Gene Expression Patterns Define the Genomic Landscape of Pediatric Papillary Thyroid Carcinoma  
  Ana Stosic, Fabio Fuligni, Nathaniel D. Anderson, Scott Davidson, Richard de Borja, Meryl Acker, Vito Forte, Paolo Campisi, Evan J. Propst, Nikolaus E. Wolter, Rose Chami, Ozgur Mete, David Malkin, Adam Shlien, and Jonathan D. Wasserman  
  This study highlights important distinctions between the genomes and transcriptomes of pediatric and adult papillary thyroid carcinoma, with implications for understanding the biology, diagnosis, and treatment of advanced disease in children.

## MOLECULAR CELL BIOLOGY
- **5638** CstF64-Induced Shortening of the BID 3'UTR Promotes Esophageal Squamous Cell Carcinoma Progression by Disrupting ceRNA Cross-talk with ZFP36L2  
  Ai Lin, Ping Ji, Xiangjie Niu, Xuan Zhao, Yamei Chen, Weiling Liu, Yachen Liu, Wenyi Fan, Yanxia Sun, Chuanwang Miao, Shaosen Zhang, Wen Tan, Dongxin Lin, Eric J. Wagner, and Chen Wu  
  High-throughput analysis of alternative polyadenylation in esophageal squamous cell carcinoma identifies recurrent shortening of the BID 3'UTR as a driver of disease progression.
- **5652** mTORC1 Promotes ARID1A Degradation and Oncogenic Chromatin Remodeling in Hepatocellular Carcinoma  
  Shanshan Zhang, Yu-Feng Zhou, Jian Cao, Stephen K. Burley, Hui-Yun Wang, and X.F. Steven Zheng  
  mTOR promotes oncogenic chromatin remodeling by controlling ARID1A degradation, which is important for liver tumorigenesis and response to mTOR- and YAP-targeted therapies in hepatocellular carcinoma.  
  See related commentary, p. 5608
Matricellular Protein WISP2 Is an Endogenous Inhibitor of Collagen Linearization and Cancer Metastasis

Jagadeesh Janjanam, Glendin Pano, Ruishan Wang, Benjamin A. Minden-Birkenmaier, Hannah Breeze-Jones, Eleanor Baker, Cecile Garcin, Georgia Clayton, Abbas Shirinifard, Ana Maria Zaske, David Finkelstein, and Myriam Labelle

Two secreted factors, WISP1 and WISP2, antagonistically regulate collagen linearization, and therapeutically increasing the WISP2:WISP1 ratio in tumors limits collagen linearization and inhibits metastasis.

See related commentary, p. 5611

LncRNA HIF1A-AS1 Promotes Gemcitabine Resistance of Pancreatic Cancer by Enhancing Glycolysis through Modulating the AKT/YB1/HIF1a Pathway

Fengyu Xu, Mengqi Huang, Qingyong Chen, Yi Niu, Yuhang Hu, Ping Hu, Ding Chen, Chi He, Kang Huang, Zhu Zeng, Jiang Tang, Fan Wang, Yong Zhao, Chunyou Wang, and Gang Zhao

These findings show that a reciprocal feedback of HIF1A-AS1 and HIF1α promotes gemcitabine resistance of pancreatic cancer, which provides an applicable therapeutic target.
Computational Analysis of Cholangiocarcinoma Phosphoproteomes Identifies Patient-Specific Drug Targets
Shirin Elizabeth Khorsandi, Arran D. Dokal, Vinothini Rajeeve, David J. Britton, Megan S. Illingworth, Nigel Heaton, and Pedro R. Cutillas

Phosphoproteomic and computational analyses identify patient-specific drug targets in cholangiocarcinoma, supporting the potential of a machine learning method to predict personalized therapies.

Correction
Correction: CXCL12 Promotes Metastatic Castration-Resistant Prostate Cancer by Inducing Cancer Stem Cell and Neuroendocrine Phenotypes

ABOUT THE COVER
Various types of cancer overexpress oncogenic miRNAs, making them a potential therapeutic target. Next-generation chemically modified triplex peptide nucleic acid–based miR-155 inhibitors possess superior therapeutic efficacy compared with conventional full-length anti-miR-155. The cover depicts intratumoral treatment with the next-generation anti-miRNA-155 inhibitor. For details, see article by Dhuri and colleagues on page 5613.

doi: 10.1158/0008-5472.CAN-81-22-CVR