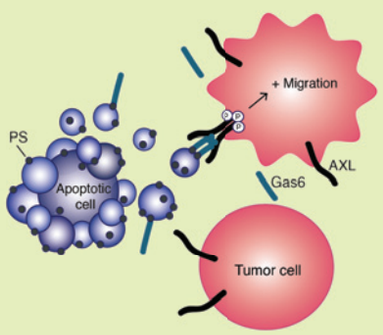
Molecular Cancer Research

Highlights

December 2017 • Volume 15 • Number 12

Selected Articles from This Issue



Apoptotic Bodies and Gas6/AXL-Induced Tumor Cell Migration

Zweemer *et al.*_____Page 1656

The receptor tyrosine kinase AXL is aberrantly expressed in many tumors, is a potent driver of invasive cell motility and has become increasingly recognized as a broadly relevant mechanism in cancer drug resistance. Here, it is reported that exposure of cancer cells to phosphatidylserine (PS)-expressing apoptotic bodies enhances migration of tumor cells via a PS-AXL signaling axis. These findings suggest that anti-cancer therapies that induce fractional cell killing contribute to the motility of surviving cells in AXL-expressing tumors, hence generating potentially problematic invasive and metastatic behavior.

NAMPT as a Treatment Target for Chondrosarcoma

Peterse *et al.*_____Page 1714

Nicotinamide phosphoribosyltransferase (NAMPT) and nicotinic acid phosphoribosyltransferase (NAPRT) are rate-limiting enzymes in the nicotinamide adenine dinucleotide (NAD+) synthesis pathway. In this study, *in vitro* experiments reveal that chondrosarcoma cells exhibit a dose-dependent decrease in cell viability, 3D collagen invasion and colony formation upon treatment with NAMPT inhibitors. Strikingly, higher methylation of the NAPRT promoter was observed in high-grade versus low-grade chondrosarcomas. Therefore, chondrosarcoma patients, especially those of high histological grade with lower expression and hypermethylation of NAPRT, may benefit clinically from inhibition of the NAD synthesis pathway.

Genomic Subtypes of Nasopharyngeal Carcinoma

Zhang *et al.*_____Page 1722

Few actionable oncogenic driver mutations have been identified in nasopharyngeal carcinoma (NPC). However, a marked lymphocytic infiltrate and association with viral infection make immunotherapy an intriguing treatment option. Zhang and colleagues perform the first integrated analysis of gene expression, mutation, copy number, and clinical data in NPC, identifying gene expression subtypes characterized by distinct immune and Epstein-Barr virus (EBV) associated gene expression patterns. Prognostic associations with stromal tumor-infiltrating lymphocytes and a proliferation signature were also identified. This study is an important step in understanding how NPC tumor cells and EBV influence the tumor microenvironment (TME), and may help guide immunotherapy strategies in this disease.

Harmine is a TWIST1 Inhibitor in NSCLC

Yochum *et al.*_____Page 1764

TWIST1 promotes tumorigenesis through induction of EMT, invasion and metastasis, as well as suppression of oncogene-induced senescence and apoptosis. Additionally, TWIST1 expression is required for oncogene-driven lung cancer, including tumors driven by mutant KRAS. Using a chemical-bioinformatic approach, Yochum and colleagues identified harmine as a first-in-class TWIST1 inhibitor with marked anti-tumor activity in oncogene-driven NSCLC including EGFR mutant, KRAS mutant and MET altered NSCLC. Targeting TWIST1 with a small-molecule therapeutic such as harmine and its potential analogues/derivatives, represents a novel treatment strategy in oncogene-driven lung cancer and other solid malignancies in which TWIST1 promotes tumorigenesis.

Downloaded from mcr.aacrjournals.org on October 22, 2021. © 2017 American Association for Cancer Research.

AACR American Association for Cancer Research

Molecular Cancer Research

Highlights of This Issue

Mol Cancer Res 2017;15:1635.

Updated version Access the most recent version of this article at:
http://mcr.aacrjournals.org/content/15/12/1635

E-mail alerts Sign up to receive free email-alerts related to this article or journal.

Reprints and Subscriptions To order reprints of this article or to subscribe to the journal, contact the AACR Publications Department at pubs@aacr.org.

Permissions To request permission to re-use all or part of this article, use this link
http://mcr.aacrjournals.org/content/15/12/1635.
Click on "Request Permissions" which will take you to the Copyright Clearance Center's (CCC) Rightslink site.

Downloaded from mcr.aacrjournals.org on October 22, 2021. © 2017 American Association for Cancer Research.