Molecular Cancer Research **Highlights**

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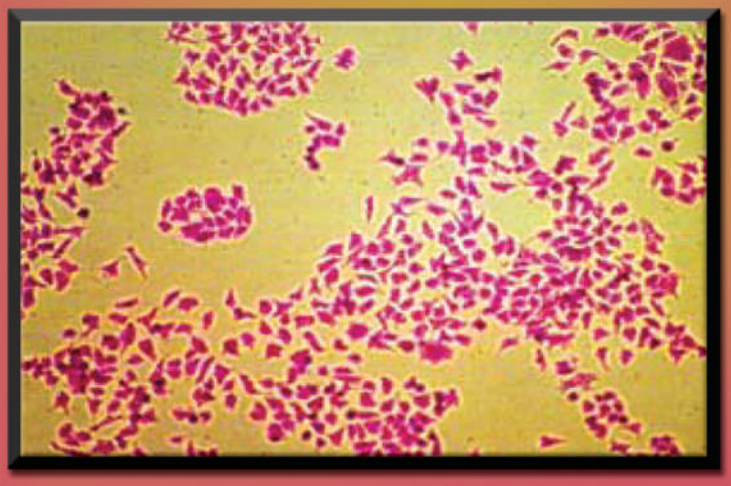
Selected Articles from This Issue



HO-3867 Inhibits Cancer Cell Migration and Invasion

Selvendiran *et al.* _____Page 1188

Fatty acid synthase (FAS) and focal adhesion kinase (FAK) are highly expressed in a variety of cancers, including ovarian cancer, and play a significant role in metastasis. Inhibition of FAS/FAK signaling is a potential target for anticancer drug development. Selvendiran and coworkers show that HO-3867, a novel analog of curcumin, attenuates the migration and invasion of ovarian cancer cells through inhibition of FAS and FAK signaling. HO-3867 has previously been shown to promote cancer cell death, while protecting normal cells. Unlike anticancer chemotherapeutics that have been shown to promote metastatic potential, HO-3867 exhibits selective anticancer potential with targeted inhibition of metastasis.

Mir-200c Expression in NSCLC

Ceppi *et al.* _____Page 1207

Disease stage and metastasis formation critically influence survival of non–small cell lung cancer (NSCLC) patients. Ceppi and colleagues investigated the functional role of a micro-RNA, miR-200c, previously shown to control epithelial-to-mesenchymal transition, a process by which cancer cells acquire dedifferentiated/invasive characteristics. The results highlight a pivotal role of miR200c in NSCLC progression and metastasis, miR-200c suppression initiating an invasive, metastatic, and a chemoresistant phenotype. Moreover, hypermethylation of the promoter region was found responsible for the loss of miR-200c expression in invasive cells. The determination of miR-200c expression could be used for the stratification of NSCLC patients into different prognostic/therapeutic groups.

RAD001 Induces Autophagy in Papillary Thyroid Cancer

Lin *et al.*_____Page 1217

Autophagy inhibition promotes papillary thyroid cancer resistance to chemotherapy and external beam radiation. To explore its potential therapeutic role, Lin and colleagues determined if the mTOR inhibitor RAD001 improved the efficacy of either cancer therapy. RAD001 activated autophagy and improved doxorubicin chemosensitivity and radiosensitivity in a synergistic fashion, suggesting that combination therapy could improve therapeutic response at less toxic concentrations. Kinome profiling analysis showed that autophagic activation dephosphorylates Met and that the chemosensitizing effects of RAD001 are mediated largely through Met attenuation in this context. Thus, RAD001 is a promising adjuvant therapy for patients with advanced thyroid cancer.

HDL Induced Proliferation of Prostate Cancer Cells

Sekine *et al.* _____Page 1284

Androgen deprivation therapy for prostate cancer leads to a significant increase of HDL, but the effect of HDL on prostate cancer is unknown. Sekine and coworkers investigated the effect of HDL on prostate cancer cells. HDL induced cell proliferation and migration of the androgen-independent PC-3 and DU145 cells by a mechanism involving ERK1/2 and Akt, but had no effect on the androgen-dependent LNCaP cell, which did not express ABCA1. Knockdown of ABCA1 by siRNA inhibited HDL-induced signal activations. These results suggest that HDL by an ABCA1-dependent mechanism can mediate signal transduction, leading to increased proliferation and migration of prostate cancer cells.