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Frequent Alterations and Epigenetic Silencing of Differentiation Pathway Genes in Structurally Rearranged Liposarcomas


Précis: Dedifferentiated liposarcomas harbor recurring HDAC1 mutations and exhibit aberrant methylomes, suggesting that epigenetic therapies may be effective in these tumors.

Combination Epigenetic Therapy Has Efficacy in Patients with Refractory Advanced Non–Small Cell Lung Cancer

Authors: R.A. Juergens, J. Wrangle, F.P. Vendetti, S.C. Murphy, M. Zhao, B. Coleman, R. Sebree, K. Rodgers, C.M. Hooker, N. Franco, B. Lee, S. Tsai, I.E. Delgado, M. A. Rudek, S.A. Belinsky, J.G. Herman, S.B. Baylin, M.V. Brock, and C.M. Rudin

Précis: Objective, long-lasting responses are observed in patients with NSCLC treated with azacitidine and entinostat.

Amplification of CRKL Induces Transformation and Epidermal Growth Factor Receptor Inhibitor Resistance in Human Non–Small Cell Lung Cancers


Précis: Overexpression of the CRKL adaptor protein activates oncogenic signaling pathways and promotes drug resistance in NSCLC.

Corrections

Correction: Ovarian Cancer Spheroids Use Myosin-Generated Force to Clear the Mesothelium

Correction: Forty Years of Translational Cancer Research

Correction: Drugs, Diagnostic Tests Approved Quickly

Acknowledgment to Reviewers

For more News and Research Watch, visit Cancer Discovery online at www.AACR.org/CDnews. Online-only News stories include the following:

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- Phenotypic Profiling Identifies Novel Anticancer Drugs
- Automated Pathology Gives Accurate Predictions
- Triple-Acting Drug Boosts Prostate Cancer Survival
- Analyzing Intact Proteins with Mass Spectrometry
- FDA Pulls Approval for Avastin in Breast Cancer

ON THE COVER

Juergens and colleagues present results from a phase I/II trial showing that combined epigenetic therapy with azacitidine and entinostat can elicit objective responses, including one complete and one partial response, in refractory metastatic non–small cell lung cancer (NSCLC). A decreased methylation signature in response to treatment was associated with longer overall and progression-free survival, indicative of on-target epigenetic effects. Furthermore, several patients had objective responses to subsequent anticancer therapies. This combination epigenetic therapy may therefore be effective in reversing the epigenetic mechanisms driving the progression and resistance of NSCLC. For details, please see the article by Juergens and colleagues on page 598.
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