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Cancer Cell
Volume 34, Issue 6, 10 December 2018, Pages 922-938.e7
CellPress

Article

Resistance to Epigenetic-Targeted Therapy Engenders Tumor Cell Vulnerabilities Associated with Enhancer Remodeling

Amanda Balboni Iniguez [1,2], Gabriela Alexe [1,2,3], Emily Jue Wang [1,2], Giovanni Roti [1,2,4], Sarvagna Pa[illegible] [1,3], Liying Chen [1,2], Samuel Kitara [1,2], Amy Conway [1], Amanda L. Robichaud [1], Björn Stolte [1,2,5], Pratiti Bandopadhayay [1,2], Amy Goodale [2], Sasha Pantel [2], Yenarae Lee [2], Dorian M. Cheff [7], Matthew D. Ha[illegible], Rajarshi Guha [7], Mindy I. Davis [7] ... Kimberly Stegmaier [1,2,12]
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Summary

Drug resistance represents a major challenge to achieving durable responses to cancer therapeutics. Resistance mechanisms to epigenetically targeted drugs remain largely unexplored. We used bromodomain and extra-terminal domain (BET) inhibition in neuroblastoma as a prototype to model resistance to chromatin modulatory therapeutics. Genome-scale, pooled lentiviral open reading frame (ORF) and CRISPR knockout rescue screens nominated the phosphatidylinositol 3-kinase (PI3K) pathway as promoting resistance to BET inhibition. Transcriptomic and chromatin profiling of resistant cells revealed that global enhancer remodeling is associated with upregulation of receptor tyrosine kinases (RTKs), activation of PI3K signaling, and vulnerability to RTK/PI3K inhibition. Large-scale combinatorial screening with BET inhibitors identified PI3K inhibitors among the most

Phosphoinositide 3-Kinase

Phosphatidyl inositol 3-kinase (PI3K) is a family of p85/p110 heterodimeric lipid kinases that generate second messenger signals downstream of tyrosine kinases activated by antigen receptors.

From: Progress in Molecular Biology and Translational Science, 2012

Related terms:

Mitogen-Activated Protein Kinase, Mammalian Target of Rapamycin, Protein, Insulin, Signal Transduction, Phosphotransferase, Phosphorylation, Protein Tyrosine Kinase

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Lipid Metabolism in Signaling Systems

Stephen P. Soltoff, ... Lewis C. Cantley, in Methods in Neurosciences, 1993

Introduction

Phosphatidylinositol 3-kinase (PtdIns 3-kinase) is a lipid kinase that is activated by a number of growth factors and takes part in the cascade of signal transduction events involved in cell growth and transformation. It associates with activated protein tyrosine kinases, including growth factor receptors that are autophosphorylated on tyrosine residues in response to the binding of the specific ligands at the plasma membrane. In addition to being activated by growth factors [including colony stimulatory factor-1, insulin, insulin-like growth factor-1 (IGF-1), epidermal growth factor (EGF), hepatocyte growth factor (HGF), and platelet-derived growth factor (PDGF],

The GPIb-IX-V Complex

Robert K. Andrews, Michael C. Berndt, in Platelets (Third Edition), 2013

3 PI 3-Kinase

PI 3-kinase is associated with GPIb-IX-V and can be co-immunoprecipitated with the receptor from resting platelet lysates.[144] The interaction with GPIb-IX-V involves the PI 3-kinase regulatory p85 subunit, which is associated with the catalytic p110 subunit. Together with 14-3-3ζ and calmodulin, PI 3-kinase translocates to the Triton-insoluble actin cytoskeleton in activated platelets, but the association of these proteins is not dependent on the GPIb-IX-V–cytoskeletal interaction because the interactions are not significantly affected by dissociation of filamin from GPIbα (using N-ethylmaleimide) or depolymerization of actin filaments (using

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