# Contents

Molecular Cancer Research
Defining the Molecular Basis of Malignancy and Progression
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## CORRECTION



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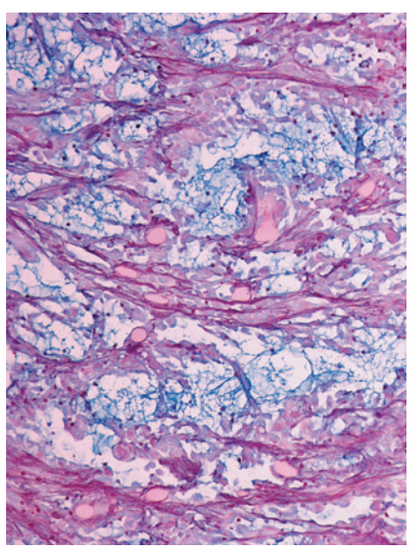
## ABOUT THE COVER



Nonmalignant immortalized human bronchial epithelial cells (HBECs, containing hTERT and CDK4 providing a bypass of p16 – two common oncogenic changes found in lung cancer) can be progressed to fully malignant cells capable of *in vivo* tumor formation following the introduction of defined oncogenic alterations (such as high levels of oncogenic KRAS, p53 knockdown, with or without exogenous c-myc expression) mimicking genetic alterations commonly found in non-small cell lung cancer. Recent genomic data shows large number of sequence altering mutations in human lung cancers requiring sorting out of "passenger" and "driver" oncogenic changes. The HBEC preclinical *in vitro* model provides a way to test these candidates and identify the minimal, most crucial set of genetic alterations required for full malignant transformation of a bronchial epithelial cell. NOD/SCID xenograft tumors of cells with similar oncogenic changes amazingly led to multiple different lung cancer histologies (adenocarcinoma, squamous carcinoma, adeno-squamous and large cell carcinoma) indicating that factors other than oncogenic changes dictate histology. Shown here is one of the $HBEC^{p53,KRAS}$ tumors that differentiated into typical lung adenocarcinoma as demonstrated by Alcian-Blue Periodic Acid Schiff (PAS) stain where mucins are stained with Alcian blue (original magnification at 10X). For examples of the other histologies and details, see article by Sato, Larsen, and colleagues on page 638.

Molecular Cancer Research

11 (6)

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