# Contents

Molecular Cancer Research
A Journal of the Molecular and Cellular Biology of Cancer
June 2010 • Volume 8 • Number 6

## CORRECTION



## ABOUT THE COVER

This study is the first demonstration that acquired resistance mechanisms to EGFR-tyrosine kinase inhibitors (TKI) extend beyond the cancer cells and involve reprogramming of the tumor microenvironment. Using an *in vivo* GFP-tagging approach, we show that EGFR-TKI–resistant cancer cells escape therapeutic response by undergoing epithelial to mesenchymal transition (EMT) and camouflaging themselves within the host cancer-associated fibroblast (CAF) population. Shown is an image of a GFP-tagged, EGFR-TKI–resistant cancer cell within the host CAF population coexpressing the fibroblast-specific marker, N-cadherin (red), indicating the phenomenon of EMT (shown in yellow). The CAF population within the EGFR-TKI–resistant tumors further plays a role in promoting therapeutic resistance in neighboring cancer cells. Our findings highlight the need for extending investigations into potential therapeutic targets within the surrounding tumor stroma to counter EGFR-TKI–acquired resistance. For further details, please see Mink and coworkers on page 809 in this issue.