Molecular Cancer Therapeutics

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COMPANION DIAGNOSTICS AND CANCER BIOMARKERS

MODELS AND TECHNOLOGIES

CORRECTION

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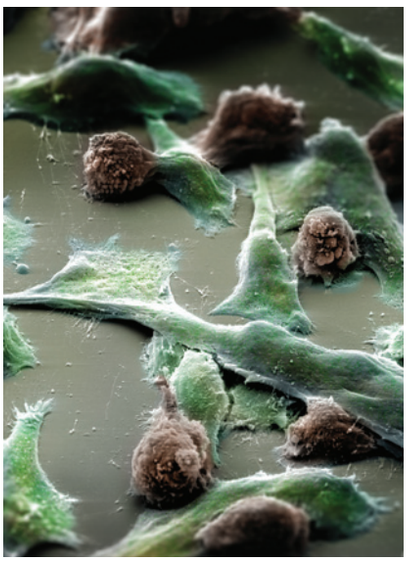
ABOUT THE COVER



The FDA-approved BRAF inhibitor vemurafenib achieves outstanding clinical response rates in melanoma patients, but early resistance is common. Understanding of the pathomechanisms of drug resistance and identification of effective therapeutic alternatives are key challenges for melanoma research. Despite intensive efforts, the breakthrough to understand and prevent resistance to RAF inhibition has not been achieved. This might be due to the plasticity and heterogeneity of melanoma, which allow the tumor cells to adapt to biological processes. Proteomics facilitate a broad insight into the active, complex pathomechanisms. The authors' proteome data demonstrate in a melanoma cell model with acquired resistance that vemurafenib resistance can be characterized by enhanced expression of the lysosomal compartment, enhanced cell adhesion, and by epithelial-mesenchymal transition (EMT) associated with an invasive, more aggressive phenotype. The typical morphology of EMT was demonstrated using a scanning electron microscopy. As visualized in the cover, the vemurafenib-sensitive cells had a ball-like structure and were smaller, whereas sprouted cells were almost exclusively found in the resistant cell lines. For details, see the article by Paulitschke and colleagues on page 757.

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