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ANGIOGENESIS, METASTASIS, AND THE CELLULAR MICROENVIRONMENT

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- 821 | **Cyclooxygenase-Deficient Pancreatic Cancer Cells Use Exogenous Sources of Prostaglandins**
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Atypical Protein Kinase C ζ Exhibits a Proapoptotic Function in Ovarian Cancer

Irina Nazarenko, Marcel Jenny, Jana Keil, Cornelia Gieseler, Karen Weisshaupt, Jalid Sehouli, Stefan Legewie, Lena Herbst, Wilko Weichert, Silvia Darb-Esfahani, Manfred Dietel, Reinhold Schäfer, Florian Ueberall, and Christine Sers

CORRECTION**Correction: A Short Hairpin DNA Analogous to miR-125b Inhibits C-Raf Expression, Proliferation, and Survival of Breast Cancer Cells****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.

