Contents

Clinical Cancer Research
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CANCER THERAPY: PRECLINICAL

IMAGING, DIAGNOSIS, PROGNOSIS

CANCER THERAPY: CLINICAL

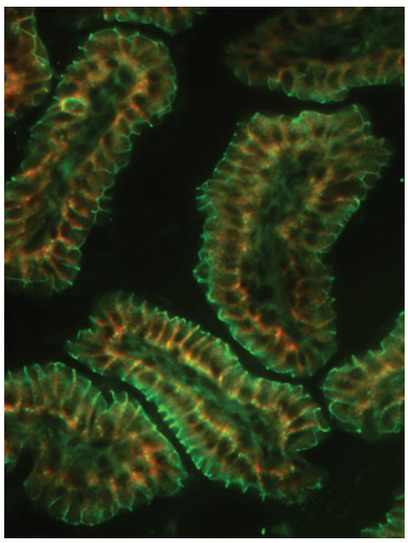
PREDICTIVE BIOMARKERS AND PERSONALIZED MEDICINE

LETTERS TO THE EDITOR

CORRECTIONS

ABOUT THE COVER

Epithelial junctions between tumor cells inhibit the penetration of anticancer drugs into tumors and represent one of the mechanisms that cancers use to protect themselves from attacks by the host-immune system as well as elimination by cancer therapeutics. Beyer and colleagues have developed a recombinant protein (JO-1), which triggers transient opening of intercellular junctions in epithelial tumors through binding to desmoglein 2 (DSG2) and thus increases the safety and therapeutic efficacy of monoclonal antibodies and chemotherapy drugs. Whereas DSG2 is readily accessible in tumors, in intestinal epithelial cells it is trapped in intercellular junctions as shown in the figure by overlapping signals for the DSG2 (green) and the junction marker claudin 7 (red). Therefore, JO-1 preferentially acts on epithelial junctions in tumors, thereby creating a "sink" for therapeutics. For details, see the article by Beyer and colleagues on page 3340 of this issue.



Clinical Cancer Research

18 (12)

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