Contents

Cancer Research

January 15, 2012 • Volume 72 • Number 2

BREAKING ADVANCES

REVIEWS

PRIORITY REPORTS

CLINICAL STUDIES

MICROENVIRONMENT AND IMMUNOLOGY

MOLECULAR AND CELLULAR PATHOBIOLOGY

PREVENTION AND EPIDEMIOLOGY

THERAPEUTICS, TARGETS, AND CHEMICAL BIOLOGY

TUMOR AND STEM CELL BIOLOGY

CORRECTION

ABOUT THE COVER

Epithelial–mesenchymal transition (EMT) is a key developmental process and has also been implicated in cancer metastasis and therapeutic resistance. The factors contributing to EMT in human cancers remain unclear. Sun and colleagues show that androgen deprivation can promote EMT in normal mouse prostate as well as in human prostate cancer, revealing a potentially important consequence of a standard of care treatment for prostate malignancies. This image corresponds to an immunofluorescence staining of E-cadherin (epithelial marker) and vimentin (mesenchymal marker) in postcastration (androgen-deprived) mouse prostate tissue. Cells that express both markers are likely to be undergoing EMT. Red, Vimentin; green, E-cadherin; blue, DAPI. For details, see the article by Sun and colleagues on page 527 of this issue.