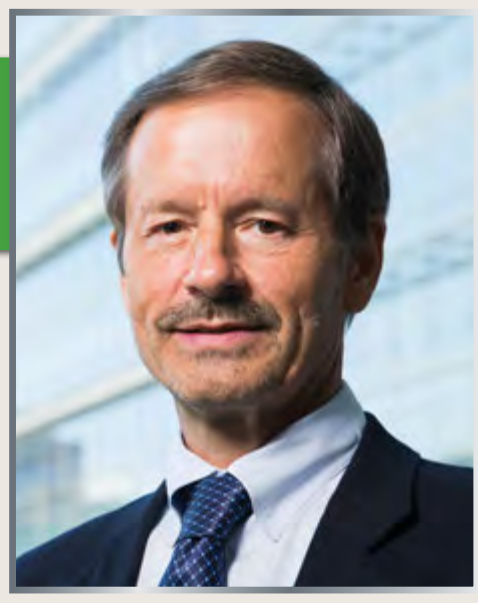
# 2011 AACR Launches High-Impact Journal: *Cancer Discovery*

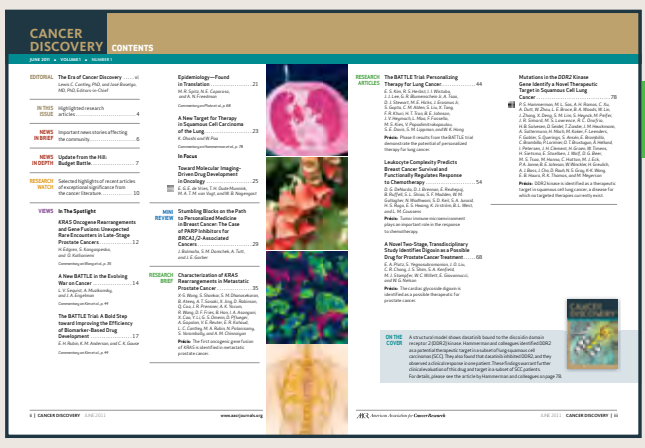


## LEWIS C. CANTLEY, PHD, FOUNDING EDITOR-IN-CHIEF, 2011-PRESENT

A leading cancer researcher whose transformative studies of the mechanisms that drive cancer development have encouraged new approaches to therapy, Dr. Cantley discovered a family of enzymes that are fundamental to cell growth. His most significant contribution has been his 1988 discovery of the phosphoinositide 3-kinase (PI3K) signaling pathway, work that revealed how biochemical signaling pathways control normal cell growth and trigger the development of cancer when they are defective. He is director of the Sandra and Edward Meyer Cancer Center at Weill Cornell Medical College and head of the Ronald P. Stanton Clinical Cancer Program at NewYork-Presbyterian Hospital.

## JOSÉ BASELGA, MD, PHD, FOUNDING EDITOR-IN-CHIEF, 2011-PRESENT

An internationally renowned physician and scientist, Dr. Baselga focuses on the clinical development of novel molecularly targeted agents for treating cancer, particularly breast cancer. He conducted the initial clinical trial which showed that patients with advanced HER2-positive breast cancer benefited from treatment with the anti-HER2 monoclonal antibody trastuzumab. His most recent research is focused on identifying mechanisms of resistance to anti-HER2 agents and on the clinical development of novel agents—including PI3 kinase inhibitors and antiestrogen therapies. Dr. Baselga is the physician-in-chief and chief medical officer at Memorial Sloan Kettering Cancer Center in New York. He is currently serving as AACR President.

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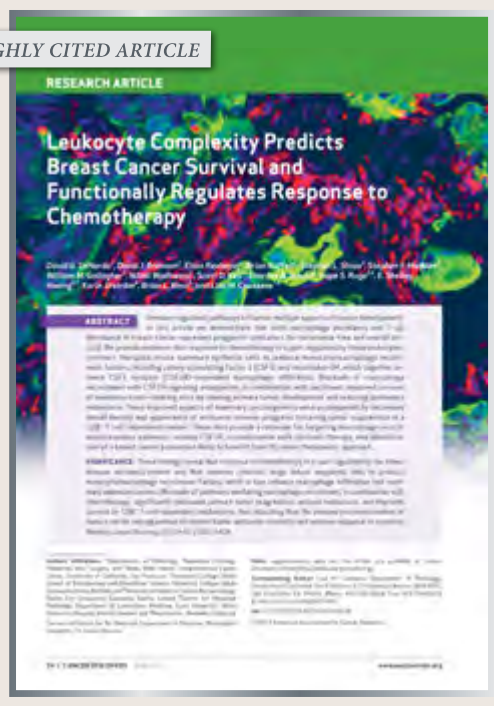
Inaugural Editorial


# Macrophage and T-cell Infiltration Determines Breast Cancer Chemosensitivity and Predicts Outcome



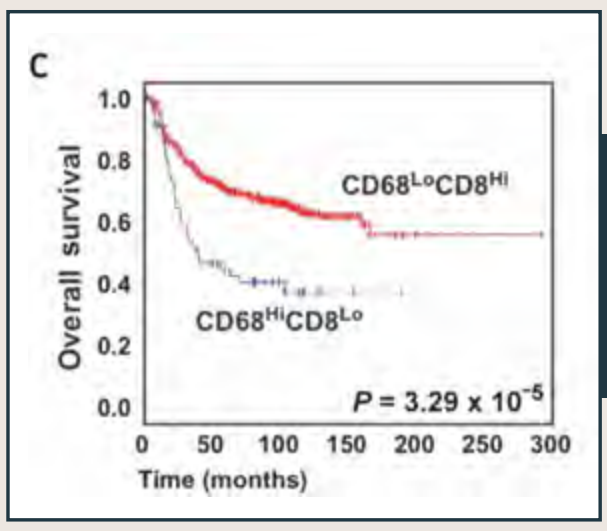




**FIGURE 7C.** Ratio of CD68 to CD8 predicts patient survival and response to neoadjuvant chemotherapy. Kaplan-Meier estimate of survival, comparing CD68high/CD8low and CD68low/CD8high immune profiles as assessed by mRNA expression from 3872 patient samples for tumors stratified into basal and HER2+ breast cancer. The log-rank (Mantel-Cox) *P* value is shown for difference in survival.

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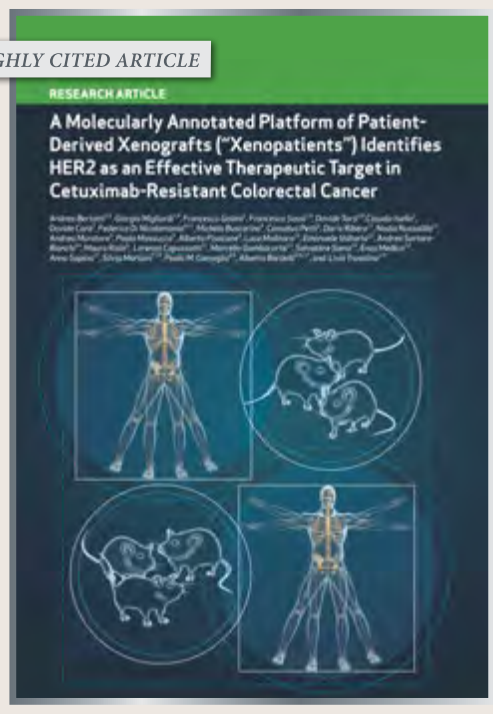
# 2011

## A Patient-Derived Xenograft Preclinical Trial Reveals Targeted Therapy Resistance Mechanisms



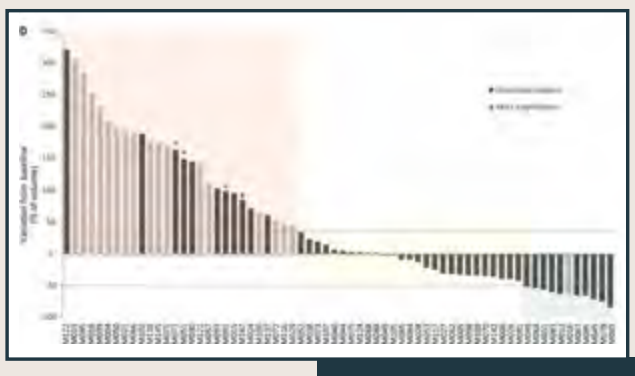



**FIGURE 4D.** Correlation between *HER2* amplification and therapeutic resistance to cetuximab in xenopatients. Genotype-response correlations in the *KRAS* wild-type subpopulation as previously shown in Figure 3A. Light gray histograms indicate cases with rare mutations of *KRAS* or mutations of *NRAS*, *BRAF*, and *PIK3CA*.

# 2012

## Targeted Deep Sequencing to Detect Genomic Alterations is Feasible in Archival Tumor Samples

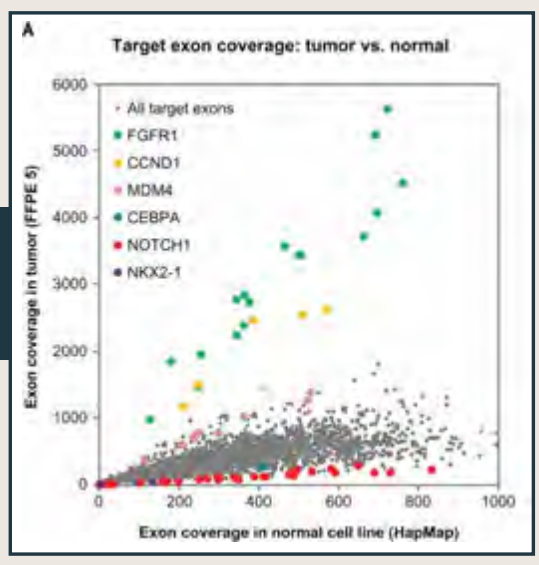



**FIGURE 2A.** Copy-number alterations in an archival breast cancer sample. Sequence coverage is shown for each target in the tumor sample compared with a normal diploid sample. Exon targets from several genes with copy-number gains and losses are highlighted.


## EGFR Underlies the Relative Insensitivity of Colorectal Cancer to BRAF Inhibition Compared with Melanoma

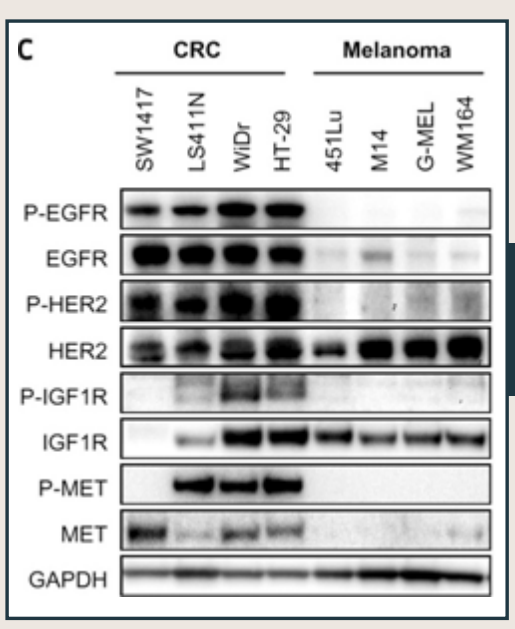




**FIGURE 2C.** Increased RTK activation in *BRAF*-mutant colorectal cancer (CRC). Lysates from *BRAF*-mutant colorectal cancer and melanoma cell lines were evaluated by the use of Western blot to determine total and phosphorylated protein levels of the RTKs identified in (B).

## First Impact Factor: 10.143

# 2014

## AACR Record High Impact Factor: 19.453

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