CANCER DISCOVERY

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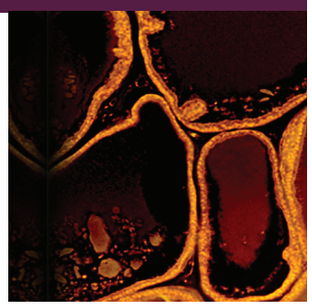

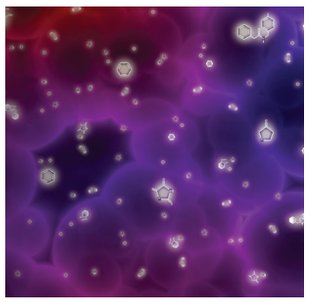

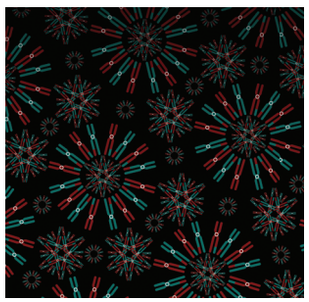

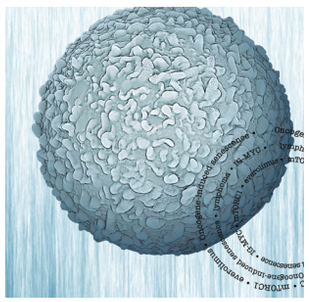

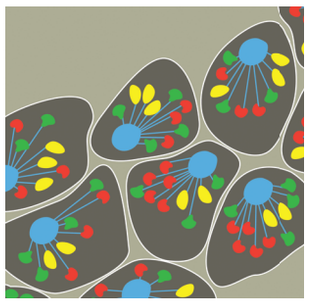

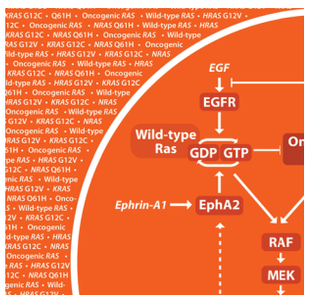

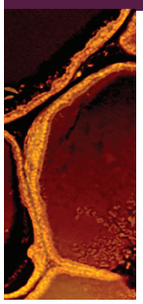

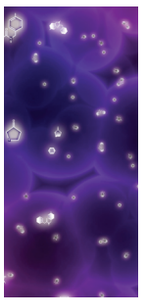

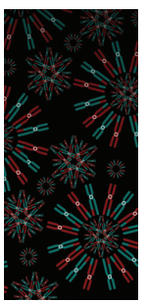

Correction

For more News and Research Watch, visit *Cancer Discovery* online at http://CDnews.aacrjournals.org. Online-only News stories include the following:

- New ADC Effective against Prostate Cancer
- Proposals Aim to Make Trials More Efficient
- Triple Jeopardy for Triple-Negative Breast Cancers
- Sandy Underlines Need for Disaster Preparation
- Inhibiting JAK2 for Inflammatory Breast Cancer
- Bevacizumab Fails to Up Breast Cancer Survival

ON THE COVER Young and colleagues show that oncogenic and wild-type RAS isoforms have nonredundant, independent roles in cancer cells. Oncogenic RAS isoforms desensitize cells to receptor tyrosine kinase (RTK) stimulation and promote basal mitogen-activated protein kinase (MAPK) signaling, whereas wild-type RAS isoforms are required for RTK-dependent activation of MAPK signaling and optimal growth of cancer cells expressing oncogenic RAS. Depletion of oncogenic RAS sensitizes cells to wild-type isoform-mediated growth factor signaling, uncovering a potential resistance mechanism employed by *RAS*-mutant cells. Combined inhibition of RAS and RTK signaling effectively blocks growth of cells expressing oncogenic RAS and may therefore be a potential approach to circumvent resistance. For details, please see the article by Young and colleagues on page 112.