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**Correction:** An LXR Agonist Promotes Glioblastoma Cell Death through Inhibition of an EGFR/AKT/SREBP-1/LDLR-Dependent Pathway .......... 190

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

- NCATS Is Out of the Bag
- Putting Tumors to the Blood Test
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- Web Applications Aid Clinical Trial Recruitment

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**ON THE COVER**
McKie and colleagues show that OPCML expression is silenced in multiple tumor types, including the vast majority of high-grade serous ovarian tumors, and correlates with poor prognosis. They further establish an extracellular mechanism of OPCML-mediated tumor suppression through negative regulation of a specific group of receptor tyrosine kinases (RTK). Through binding to RTK extracellular domains, OPCML induces RTK membrane redistribution, internalization, and degradation. Recombinant OPCML down-regulated the same RTKs in vivo and inhibited ovarian cancer cell growth, suggesting that extracellular protein therapy may be useful in the treatment of OPCML-deficient tumors. For details, please see the article by McKie and colleagues on page 156.
CANCER DISCOVERY

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