Molecular Cancer Research

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AACR American Association for Cancer Research

February 2016 • Volume 14 • Number 2

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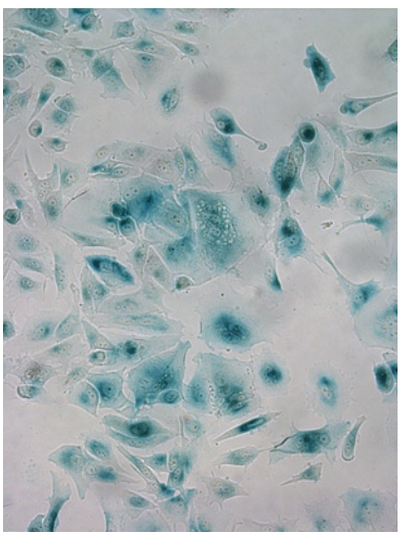
ABOUT THE COVER

In this issue, a study by Efimova and colleagues (see page 173) re-examines two nearly universal properties of cancer cells, metabolic reprogramming and cellular immortality, and describes how cancer cells may alter their metabolism to resist senescence. While the Warburg effect may provide cancer cells with biosynthetic intermediates to support rapid growth, the recent discovery of oncometabolites and their epigenetic effects suggests a more direct role in carcinogenesis. Efimova and colleagues extend the paradigm by showing that targeting metabolism overcomes the resistance of cancer cells to DNA damage, resulting in terminal senescent arrest. The cover image shows MCF-7 cells treated with the glucose uptake inhibitor 2-deoxyglucose prior to an otherwise tolerated dose of radiation, but which here induces senescence, as shown by the large, flat cells stained blue with X-Gal for senescence-associated beta-galactosidase. Treating cells with oncometabolites promotes DNA damage repair. Their work implicates metabolic reprogramming in maintaining immortality and provides a rationale for combining metabolic targeting with genotoxic therapy.



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Mol Cancer Res 2016;14:125-238.