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Article

Selective killing of oncogenically transformed cells through a ROS-mediated mechanism by β -phenylethyl isothiocyanate

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Reactive oxygen species in cancer cells: Live by the sword, die by the sword, *Cancer Cell*, Volume 10, Issue 3,Referred to by: *September 2006, Pages 175-176*Paul T. Schumacker¹, , [Abstract](#) | [Full Text + Links](#) | [PDF \(189 K\)](#)

Summary

Reactive oxygen species (ROS) stimulate cell proliferation and induce genetic instability, and their increase in cancer cells is often viewed as an adverse event. Here, we show that such abnormal increases in ROS can be exploited to selectively kill cancer cells using β -phenylethyl isothiocyanate (PEITC). Oncogenic transformation of ovarian epithelial cells with *H-Ras*^{V12} or expression of *Bcr-Abl* in hematopoietic cells causes elevated ROS generation and renders the malignant cells highly sensitive to PEITC, which effectively disables the glutathione antioxidant system and causes severe ROS accumulation preferentially in the transformed cells due to their active ROS output. Excessive ROS causes oxidative mitochondrial damage, inactivation of redox-sensitive molecules, and massive cell death. In vivo, PEITC exhibits therapeutic activity and prolongs animal survival.

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