

Blocking antioxidants in cancer cells reduces tumor growth in mice

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Many cancers have adapted to cope with high levels of immune systemproduced free radicals, also referred to as reactive oxygen species, by overproducing antioxidant proteins. One of these proteins, superoxide dismutase 1 (SOD1), is overproduced in lung adenocarcinomas and has been implicated as a target for chemotherapy.

In this issue of the *Journal of Clinical Investigation*, Navdeep Chandel and colleagues from Northwestern University report the effects of a SOD1 pharmacological inhibitor on non-small-cell lung cancer (NSCLC) cells. The inhibitor, called ATN-224, stunted the growth of human NSCLC cells in culture and induced their death. The researchers also found that ATN-224 inhibited other antioxidant proteins, which caused high levels of <u>hydrogen peroxide</u> inside the cells. The ability of cancer cells to produce hydrogen peroxide was required for ATN-224-dependent effects, because hydrogen peroxide activated <u>cell</u> <u>death</u> pathways.

Furthermore, ATN-224 induced cancer cell death and reduced tumor sizes in a mouse model of lung adenocarcinoma. ATN-224 dependent effects in animals were improved when the inhibitor was used in combination with another drug that activates programmed cell death.

This study suggests inhibition of antioxidants may be a viable chemotherapeutic option.

More information: Targeting SOD1 reduces experimental non-small-



cell lung cancer, J Clin Invest. DOI: 10.1172/JCI71714

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