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CANCER PREVENTION RESEARCH

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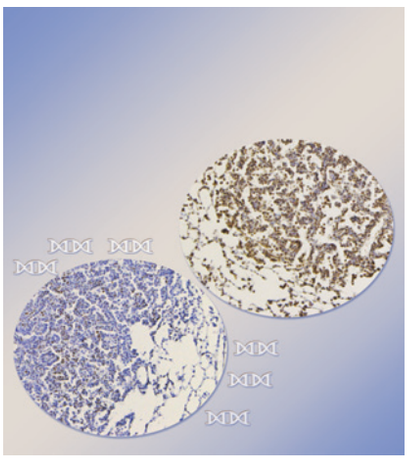
ABOUT THE COVER

Former smokers continue to be at elevated lung cancer risk, making up a large proportion of newly diagnosed lung cancer. Former smokers with airway dysplasia received some benefit in randomized trials of chemoprevention, but preventive effects to date have been modest. There is a great need for more effective chemoprevention agents for former smokers at high risk for lung cancer. Njatcha and colleagues (in a study beginning on page 735) determined the efficacy of a novel STAT3 inhibitor, CS3D, for chemoprevention using a tobacco carcinogen-induced mouse model of lung cancer that mimics an ex-smoker. CS3D is a cyclic double-stranded decoy oligonucleotide corresponding to the DNA response element for STAT3. CS3D interacts with p-STAT3 dimers to compete for their binding to the promoters of STAT3 target genes, and also triggers p-STAT3 degradation. CS3D given as a short-term intermittent therapy blocked formation and progression of airway preneoplasia and adenomas in tobacco carcinogen-exposed mice, and reduced both incidence and size of lung tumors that arose over time. The cover depicts immunohistochemical detection of p-STAT3 in lung adenomas from these mice. After treatment with CS3D (lower left image) p-STAT3 staining is reduced compared to that seen in an adenoma from a mouse treated with the inactive mutant version of the cyclic oligonucleotide, CS3M (upper right image). Detection of p-STAT3 over time showed that phosphorylated STAT3 remained suppressed eight weeks after the end of the treatment course. Results show that a short course of CS3D therapy has persistent effects to reduce STAT3 signaling in the airways and prevent lung cancer development.



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