A team of researchers, led by Yi-Ching Wang, at National Cheng Kung University, Taiwan, Republic of China, has uncovered a potential mechanism by which the tobacco-specific carcinogen NNK promotes lung tumor formation and development. Specifically, they suggest that NNK induces the accumulation of a protein known as DNMT1 in the nucleus and that this protein silences genes that suppress tumor formation.

The authors generate several lines of evidence to support their suggested mechanism, one of which is the observation that DNMT1 accumulates in both lung adenomas from NNK-treated mice and tumors from lung cancer patients that were smokers. Of clinical relevance, DNMT1 overexpression in lung cancer patients who smoked continuously correlated with poor prognosis.

These data identify a potential important link between tobacco smoking and lung cancer.

**More information:** The tobacco-specific carcinogen NNK induces DNA methyltransferase 1 accumulation and tumor suppressor gene hypermethylation in mice and lung cancer patients. View this article at: [www.jci.org/articles/view/4070...be406da6c2aac829be98](http://www.jci.org/articles/view/4070...be406da6c2aac829be98)