Nicotine does not promote lung cancer growth in mouse models

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Nicotine at doses similar to those found in most nicotine replacements therapies did not increase lung cancer tumor incidence, frequency or size, according to results of a mouse study presented at the AACR 102nd Annual Meeting 2011, held here April 2-6.

"If you take our data and combine it with epidemiological data from Europe, even in people who quit smoking and maintain the use of nicotine replacement therapy for months or years, there does not appear to be increased lung cancer incidence," said Phillip A. Dennis, M.D., Ph.D., senior investigator at the medical oncology branch of the National Cancer Institute. "This suggests that nicotine replacement therapy is probably safe and is certainly safer than smoking."

According to Dennis, about 20 percent of all smokers are truly addicted to tobacco. In these people, the use of nicotine replacement therapy has markedly helped them to quit smoking. The current Food and Drug Administration indication for most nicotine replacement therapies, such as a nicotine patch, is limited to 10 to12 weeks.

There is a subset of smokers who will need to stay on nicotine replacement therapy longer in order to appease addiction, according to Dennis. However, expanding the use of this therapy is controversial. Research to date has linked nicotine to cancer in various ways, including laboratory studies that indicate nicotine promotes the growth or spread of tumor cells or that it helps transform normal lung airway cells into cancerous cells, according to Dennis.

Therefore, the researchers conducted a study in mice to determine if nicotine had any tumor-promoting effects. Three groups of mice were administered nicotine in drinking water for up to 12 weeks.

In the first group, the mice were administered three weekly injections of NNK, a known tobacco carcinogen, prior to receiving nicotine. The second group of mice was genetically engineered to have activation of the KRAS oncogene, which is frequently mutated in lung cancers derived from smokers. The third group was made up of mice that were given cell lines derived from mouse lung cancers.

The researchers found that all the mice had normal water consumption. Cotinine, a metabolite of nicotine, was found to be at a level that is comparable to levels found in nicotine replacement users.

"We observed that there was no effect of nicotine on the mice in all three groups," said Dennis. "Nicotine did not increase tumor incidence, multiplicity or size."

At the levels measured in mice, nicotine did not activate signaling pathways associated with lung cancer that had been shown to be activated by high concentrations of nicotine.

"Based on our study and human epidemiological studies to date, nicotine replacement therapy is probably a safe option," he suggested.

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