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Do 'light' cigarettes deliver less nicotine to the brain than regular cigarettes?

For decades now, cigarette makers have marketed so-called light cigarettes — which contain less nicotine than regular smokes — with the implication that they are less harmful to smokers' health. A new UCLA study shows, however, that they deliver nearly as much nicotine to the brain.

Reporting in the current online edition of the *International Journal of Neuropsychopharmacology,* UCLA psychiatry professor Dr. Arthur L. Brody and colleagues found that low-nicotine cigarettes act similarly to regular cigarettes, occupying a significant percentage of the brain's nicotine receptors.

Light cigarettes have nicotine levels of 0.6 to 1 milligrams, while regular cigarettes contain between 1.2 and 1.4 milligrams.

The researchers also looked at de-nicotinized cigarettes, which contain only a trace amount of nicotine (0.05 milligrams) and are currently being tested as an adjunct to standard smoking-cessation treatments. They found that even that low a nicotine level is enough to occupy a sizeable percentage of receptors.

"The two take-home messages are that very little nicotine is needed to occupy a substantial portion of brain nicotine receptors," Brody said, "and cigarettes with less nicotine than regular cigarettes, such as 'light' cigarettes, still occupy most brain nicotine receptors. Thus, low-nicotine cigarettes function almost the same as regular cigarettes in terms of brain nicotine-receptor occupancy.

"It also showed us that de-nicotinized cigarettes still deliver a considerable amount of nicotine to the brain. Researchers, clinicians and smokers themselves should consider that fact when trying to quit."

In the brain, nicotine binds to specific molecules on nerve cells called nicotinic acetylcholine receptors, or nAChRs. When nerve cells communicate, nerve impulses jump chemically across gaps between cells called synapses by means of neurotransmitters. The neurotransmitters then bind to the receptor sites on nerve cells — in the case acetylcholine resulting in the release of a pleasure-inducing chemical called dopamine. Nicotine mimics acetylcholine, but it lasts longer, releasing more dopamine.

"It can cause specific neurons to communicate and thus increases dopamine for an extended period of time," Brody said. "Most scientists believe that's one key reason why nicotine is so addictive."

In an earlier study, researchers determined that smoking a regular, non-light cigarette resulted in the occupancy of 88 percent of these nicotine receptors. However, that study did not determine whether inhaling nicotine or any of the thousands of other chemical found in cigarette smoke resulted in this receptor occupancy. The central goal of the present study was to determine if factors associated with smoking — other than nicotine — resulted in nAChR occupancy.

The authors reasoned that if nicotine is solely responsible for receptor occupancy, then smoking a de-nicotinized cigarette or a low-nicotine cigarette would result in the occupancy of roughly 23 percent and 78 percent of nicotine receptors, respectively, based on the cigarettes' nicotine content.

"That would still be substantial," Brody said.

Fifteen smokers participated in the study. Each was given positron emission tomography (PET) scans, a brain-imaging technique that uses minute amounts of radiation-emitting substances to tag specific molecules. In this case, the tracer was designed to bind to the nicotine receptors in the brain.

The researchers could then measure what percentage of the tracer was displaced by nicotine when the research subjects smoked. In total, 24 PET scans were taken of participants' brains before and after three different conditions: not smoking, smoking a de-nicotinized cigarette and smoking a low-nicotine cigarette.

The PET data showed that smoking a de-nicotinized cigarette and a low-nicotine cigarette occupied 26 percent and 79 percent of the receptors, respectively, which was very close to what the researchers had originally estimated.

"Given the consistency of findings between our previous study with regular cigarettes and the present study — that showed us that inhaling nicotine during smoking is solely responsible for occupancy of brain nicotine receptors," Brody said.

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In addition to Brody, other authors of the study were Mark A. Mandelkern, Matthew R. Costello, Anna L. Abrams, David Scheibal, Judah Farahi, Edythe D. London, Richard E. Olmstead, Jed E. Rose and Alexey G. Mukhin. The researchers report no conflicts of interest. Rose, from the Duke University School of Medicine, has received research support for a study unrelated to the present paper from Vector Tobacco Inc., the manufacturer of Quest cigarettes.

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