



Date: September 16, 2014
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Smoking and Schizophrenia Linked by Alterations in Brain Nicotine Signals

Reports new study in Biological Psychiatry

Philadelphia, PA, September 16, 2014 – Schizophrenia is associated with increased rates and intensity of tobacco smoking. A growing body of research suggests that the relationship between schizophrenia and smoking stems, in part, from an effort by patients to use nicotine to self-medicate symptoms and cognitive impairment associated with the disease.

A new study, published in the current issue of *Biological Psychiatry*, sheds light on this hypothesis. The authors found that the level of nicotine receptors in the brain was lower in schizophrenia patients than in a matched healthy group. Further, smoking, which is known to increase the levels of receptors for nicotine in the brain, had this effect in both groups, although was blunted in schizophrenia.

However, in the schizophrenia group, the smoking-related increase in the level of nicotine receptors was associated with lower levels of social withdrawal, blunted emotional and motivational responses, as well as better cognitive function.

Nicotine mimics the actions of a natural chemical messenger, acetylcholine, which stimulates the receptors for nicotine in the brain. So, to conduct this work, Yale University School of Medicine researchers used single photon emission computed tomography to quantify the availability of nicotinic acetylcholine receptors (β_2^* -nAChRs) in smoking and nonsmoking individuals with schizophrenia and healthy subjects.

First author and Assistant Professor Dr. Irina Esterlis details their findings, “We found a blunted effect of tobacco smoking on the β_2^* -nAChR system in individuals with schizophrenia. Furthermore, we found that lower receptor availability of β_2^* -nAChRs in smokers with schizophrenia is associated with worse negative symptoms and worse performance on tests of executive function.”

These findings may be relevant to the high rates of smoking in schizophrenia.

“The data seem to suggest that smoking might produce some clinical benefits for some patients by increasing the availability of receptor targets for nicotine in the brain,” commented Dr. John Krystal, Editor of *Biological Psychiatry*. “This finding adds to evidence that brain nicotine-related signaling might play a role for new medications developed to treat schizophrenia.”

Esterlis agreed and added, “These findings suggest that β_2^* -nAChRs may be a target for developing treatments for negative symptoms and cognitive deficits associated with schizophrenia, for which no effective treatments exist.”

The article is “In Vivo Evidence for β_2 Nicotinic Acetylcholine Receptor Subunit Upregulation in Smokers as Compared With Nonsmokers With Schizophrenia” by Irina Esterlis, Mohini Ranganathan, Frederic Bois, Brian Pittman, Marina R. Picciotto, Lara Shearer, Alan Anticevic, Jon Carlson, Mark J. Niciu, Kelly P. Cosgrove, and D. Cyril D’Souza (doi: 10.1016/j.biopsych.2013.11.001). The article appears in *Biological Psychiatry*, Volume 76, Issue 6 (September 15, 2014), published by Elsevier.

Notes for editors

Full text of the article is available to credentialed journalists upon request; contact Rhiannon Bugno at +1 214 648 0880 or Biol.Psych@utsouthwestern.edu. Journalists wishing to interview the authors may contact Dr. Irina Esterlis at irina.esterlis@yale.edu.

The authors’ affiliations, and disclosures of financial and conflicts of interests are available in the article.

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The journal publishes novel results of original research which represent an important new lead or significant impact on the field, particularly those addressing genetic and environmental risk factors, neural circuitry and neurochemistry, and important new therapeutic approaches. Reviews and commentaries that focus on topics of current research and interest are also encouraged.

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