There is a well-established connection between smoking and depression, with depressed individuals over-represented among smokers and ex-smokers often experiencing increased depressive symptoms immediately after quitting. Nicotine in tobacco binds to, activates and desensitizes nicotinic acetylcholine receptors (nAChRs), but it is not known whether activation or desensitization is more important for nicotine’s effects on depressive symptoms. Our hypothesis is that blockade rather than activation of neuronal nAChRs may be important for the effects of nicotinic agents on depressive symptoms based on clinical and pre-clinical studies of nicotinic drugs. The endogenous neurotransmitter for nAChRs is acetylcholine, and the effects of nicotine on depression-like behaviors support the idea that dysregulation of the cholinergic system may contribute to the etiology of major depressive disorder. Thus, pharmacological agents that limit acetylcholine signaling through neuronal nAChRs might be promising for the development of novel antidepressant medications.

Thursday, 4: 00 PM, October 6th, 2011

Hughes Auditorium
R. H. Lurie Medical Research Center, Chicago Campus
303 East Superior St., Chicago IL 60611

Video Conference to Evanston Campus
Technological Institute, Room L251
2145 Sheridan Road, Evanston, IL 60208

A reception will follow the lecture in Chicago

Host: Dr. Anis Contractor. For more information please contact: s-stade@northwestern.edu