J Addict Res Ther 2015, 6:3
http://dx.doi.org/10.4172/2155-6105.S1.022

OMICS International
conferenceseries.com

4th International Conference and Exhibition on
Addiction Research & Therapy
August 03-05, 2015 Florida, USA

Roles of *CYP2A6* gene polymorphism in treatment of Nicotine dependence

Sandra Rasmussen
Walden University, USA

Existing nicotine dependence therapies have decreased smoking prevalence in the United States, but the decline in the number of adult smokers is stalling, due, in part, to the limited efficacy of current therapies that lack treatment personalization. Cytochrome P450 2A6 (*CYP2A6*) gene variants are known to metabolize nicotine and possibly influence nicotine dependence treatment. These gene's inconsistent information, interindividual variability, interactions with other genes, and environmental factors have made it difficult to use their information to improve nicotine dependence therapy. This cross-sectional study based on behavioral genetic theory stating that environmental and genetic factors cause behavioral disorders, assessed the impact of slow nicotine metabolizers (*CYP2A6*1H, CYP2A6*4A, CYP2A6*9*, and *CYP2A6*12A*) and normal (fast) nicotine metabolizers (*CYP2A6*1A*) gene variants and their interactions with *CYP2B*6* associated with nicotine therapy type and nicotine dependence and withdrawal syndromes on nicotine dependence outcome. Results were that *CYP2A6*4A* (*OR*=1.60, CI [1.13-1.95]; p<0.001) and *CYP2A6*9A* (*OR*=1.47, CI[1.18-1.88]; p<0.001) were the most linked to the highest odds of successful treatment outcome, indicating that carriers of slow nicotine metabolizers were more likely to maintain abstinence 6 months post period treatment than normal(fast) metabolizer *CYP2A6*1A* (*OR*=1.35, 95% CI[1.11-1.70]; p<0.003) carriers. Study findings may be useful in gene counseling and nicotine gene therapy to tailor individualized nicotine clinical treatments, to increase smoking quit rates, and to induce positive social change by improving the lives of smokers and their families.

sandra.rasmussen@waldenu.edu

Neurobiology of Nicotine addiction vulnerability in Schizophrenia: Clinical implications

R Andrew Chambers
Indiana University, USA

Understanding why nicotine use is so common in mental illness, is an important public health objective because it produces serious financial and health consequences in these patients. Although high smoking rates in schizophrenia have traditionally been assumed to represent 'self-medication', newly emerging data suggests that this comorbidity is not best explained as a therapeutic interaction between a drug and a disease process, but instead as a synergy of 2 diseases. This talk will outline a series of basic science investigations that combine a leading neurodevelopmental rat model of schizophrenia with addiction paradigms involving nicotine and other drugs. This work employs prospective experimental designs, and invasive procedures in animal models that are not ethical in human subjects but are nevertheless crucial to understanding causal-biological relationships between mental illness and addiction. The neuro developmental syndrome of schizophrenia causes increased vulnerability to nicotine and other drug addictions, through a host of biological changes involving circuits that are co-implicated in addiction and schizophrenia. These events do not depend on, and do not occur with, illness-specific effect of nicotine to act as a cognitive enhancing medication for the schizophrenia syndrome. This work suggests the inaccuracy of self-medication hypotheses as primary explanations for high nicotine use rates in mental illness while illustrating how the developmental psychopathology of schizophrenia involuntarily enhances addiction risk and severity. This line of neuroscience calls for a paradigm shift in research and treatment on nicotine use in mental illness where this comorbidity is understood as a particularly severe form of addiction, rather than it being viewed as a beneficial form of medication.

robchamb@iupui.edu