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### **Genetic Addiction Risk Scores (GARS) coupled with the Comprehensive Analysis of Reported Drugs (CARD) provide diagnostic and outcome data supporting the need for dopamine D2 agonist therapy: Proposing a holistic -therapeutic model for Reward Deficiency Syndrome (RDS)**

Numerous studies have revealed an association between dopaminergic gene polymorphisms and several reward dependent thoughts and behaviors including addictive, obsessive, compulsive and impulsive tendencies. These interrelated behaviors involving dopaminergic genes and have been classified as Reward Deficiency Syndrome (RDS). Most recently, we associated polymorphisms of both the D2 receptor gene (DRD2), and the dopamine transporter gene (DAT1) in RDS subjects derived from two families over five generations of genotyping ( $P < 0.001$ ). By demonstrating this association, not only do we confirm the role of dopaminergic polymorphisms in RDS behaviors but demonstrate the importance of a nonspecific RDS phenotype. Subsequent studies published and underway reveal the important utility of a novel panel of candidate genes termed “GARS” enabling the stratification of genetically based severity of addiction liability. One study performed in both the United States and China utilizing GARS, revealed that 74% of abstinent psychostimulant and heroin dependent patients had a moderate to severe genetic liability. To evaluate treatment outcome for RDS, our associates have utilized the CARD to evaluate in six eastern states and over 24,000 specimens two important clinical issues: 1) compliance with prescribed treatment medications during in-patient or out-patient recovery programs; 2) Abstinence from all non-prescribed licit or illicit psychoactive drugs. By utilizing CARD we found significant evidence for both non-compliance ( $P < 0.0001$ ) and non-abstinence ( $P < 0.0001$ ) during treatment in all states involved. This important outcome data strongly suggests the need for better therapy. Over the last four decades our laboratory has developed the first natural dopamine D2 agonist (KB220Z) to significantly enhance brain dopamine “sensitivity” in both the PFC (prefrontal Cortex) and the Cingulate Gyrus (site of relapse) [ $P, 0.03$ ] and Nucleus Accumbens (site of reward and craving) utilizing qEEG and fMRI imaging respectively. These latter studies if confirmed will provide the rationale to include KB220Z as a frontline agent to attenuate the negative effect of unwanted hypodopaminergic function or “dopamine resistance”. Thus, we are proposing for the first time ever a holistic-therapeutic model for RDS which includes GARS (diagnostic); CARD (outcome measure) and KB220 (prolonged D2 agonist therapy) along with 12 step fellowship and other holistic modalities (e.g. low glycemic index diet; yoga, meditation etc) known to naturally release neuronal dopamine.

#### **Biography**

Kenneth Blum, PhD, research Professor of the Department of Psychiatry at the University College of Medicine, Gainesville, Florida and is currently Chairman of the Board and Chief Scientific Officer of LifeGen, Inc., San Diego, California, and a managing partner of Reward Deficiency Solutions, LLC, San Diego, California. He serves as a consultant and senior scientific advisor for many companies and a foundations. He serves as Co-Editor –in-Chief for BMC - Integrative Omics and Molecular Biology. He is also the editor in Chief for Journal of Genetic Syndromes and Gene Therapy. He has published over 500 articles and 13 books. He is considered by many to be the father of psychiatric genetics and the Father of “Neuro-nutrient Therapy” for RDS.

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