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Treating Reward Deficiency Syndrome (RDS) with a nutrigenomic dopaminergic brain activator by targeting reward gene polymorphisms utilizing genetic algorithms

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Background: Algorithms based on the identification of reward genes polymorphic targets were successfully used to customize Neuro Adaptagen Amino acid Therapy (NAAT) [KB220]1 for the treatment of obesity a subset of Reward Deficiency Syndrome (RDS). We now propose that nutrigenomic targeting of RDS risk alleles by altering NAAT ingredients may also be an applicable nutrigenomic solution for addiction (e.g. opioid) and pain. This hypothesis is based on the recent development of the Genetic Addiction Risk Score (GARS) and published rsfMRI work showing that KB220Z significantly induced enhanced dopaminergic functionality across the brain reward circuitry3.

Methods: A small subset of 1,000 obese subjects identified in the Netherlands was administered various KB220 formulae customized according to respective DNA polymorphisms. Patients attending seven diverse treatment centers n=273 who completed the ASI-Media Version were tested for ten reward genes selected for polymorphisms that had been associated in numerous studies with a hypodopaminergic trait. A crossover study placebo vs. a variant of NAAT–KB220Z, observed resting state functional connectivity in abstinent heroin addicts n=10.

Results: There was a significant decrease in both Body Mass Index (BMI) and weight in pounds. When we selected 10 genes with appropriate 11 variants, a statistically significant association between the ASI-Media Version-alcohol and drug severity scores and GARS was respectively found ($P<0.004$; $P<0.05$). KB220Z in abstinent heroin addicts, remarkably increased resting state functional connectivity was observed in a putative network that included the dorsal anterior cingulate, medial frontal gyrus, nucleus accumbens, posterior cingulate, occipital cortical areas, and cerebellum4. In other published rat work we show that KB220Z significantly activates, above placebo, seed regions of interest including the left nucleus accumbens, cingulate gyrus, anterior thalamic nuclei, hippocampus, pre-limbic and infra-limbic loci3.

Discussion: In 2005, our laboratory received the first USA patent on nutrigenomics and RDS treatment. This was awarded on the basis of our earlier work showing anti-addiction activity of a nutraceutical consisting of amino-acid precursors and enkephalinase inhibition. The results seen in rs-fMRI induced by KB220Z demonstrate significant functional connectivity, increased brain volume recruitment and enhanced dopaminergic functionality across the brain reward circuitry. This robust yet selective response implies clinical relevance.

Conclusions: We are now ready to propose a reward deficiency system solution that promotes early identification and stratification of risk alleles by utilizing GARS allowing for customized nutrigenomic targeting of these risk alleles by altering NAAT ingredients as an algorithmic function of carrying these polymorphic DNA–SNPS potentially yielding the first ever nutrigenomic solution for addiction and pain.

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