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Neurogenetics and Nutrigenomics of Neuro-Nutrient Therapy for Reward Deficiency Syndrome (RDS): Clinical Ramifications as a Function of Molecular Neurobiological Mechanisms

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In accord with the new definition of addiction published by American Society of Addiction Medicine (ASAM) it is well-known that individuals who present to a treatment center involved in chemical dependency or other documented reward dependence behaviors have impaired brain reward circuitry. They have hypodopaminergic function due to genetic and/or environmental negative pressures upon the reward neuro-circuitry. This impairment leads to aberrant craving behavior and other behaviors such as Substance Use Disorder (SUD). Neurogenetic research in both animal and humans revealed that there is a well-defined cascade in the reward site of the brain that leads to normal dopamine release. This cascade has been termed the “Brain Reward Cascade” (BRC). Any impairment environmental influences on this cascade will result in a reduced amount of dopamine release in the brain reward site. Manipulation of the BRC has been successfully achieved with neuro-nutrient therapy utilizing nutrigenomic principles. After over four decades of development, neuro-nutrient therapy has provided important clinical benefits when appropriately utilized. This is a review, with some illustrative case histories from a number of addiction professionals, of certain molecular neurobiological mechanisms which if ignored may lead to clinical complications.

It is well established that DA and a number of other linked neurotransmitters are responsible for feelings of well being. However,attempts to attenuate irregularities in the brain reward circuits using pharmaceutical agents have been met with disastrous results [4,5] and in some cases suicidal ideation [6]. The result of using powerful neurotransmitter agonists is down regulation instead of the much needed up-regulation of the specific receptors being targeted [7]. Bromocriptine a powerful DA D2 agonist has been shown to down regulate DA D2 receptors following chronic administration [8].

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